



Technology Transfer Network  
Clean Air Technology Center - RACT/BACT/LAER Clearinghouse

## Process Information - Details

For information about the pollutants related to this process, click on the specific pollutant in the list below.

[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**DRAFT****RBLC ID:** MI-0445**Corporate/Company:** INDECK NILES, LLC**Facility Name:** INDECK NILES, LLC**Process:** FGCTGHRSG

### Pollutant Information - List of Pollutants

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**Primary Fuel:** Natural gas  
**Throughput:** 3421.00 MMBTU/H  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	1911481.0000 T/YR	BACT-PSD	NO
<u>Carbon Monoxide</u>	4.0000 PPM	BACT-PSD	UNKNOWN
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPM	BACT-PSD	NO
<u>Particulate matter, filterable (FPM)</u>	9.9000 LB/H	BACT-PSD	UNKNOWN
<u>Particulate matter, total &lt; 10 μ (TPM<sub>10</sub>)</u>	19.8000 LB/H	BACT-PSD	UNKNOWN
<u>Particulate matter, total &lt; 2.5 μ (TPM<sub>2.5</sub>)</u>	19.8000 LB/H	BACT-PSD	UNKNOWN
<u>Sulfur Dioxide (SO<sub>2</sub>)</u>	11.7000 LB/H	BACT-PSD	UNKNOWN
<u>Sulfuric Acid (mist, vapors, etc)</u>	4.6000 LB/H	BACT-PSD	NO
<u>Volatile Organic Compounds (VOC)</u>	4.0000 PPM	BACT-PSD	UNKNOWN

**Process Notes:** 3421 MMBTU/H for each turbine 740 MMBTU/H for each duct burner for a combined throughput of 4161 MMBTU/H or 8322 MMBTU/H for both trains. Two combined-cycle natural gas-fired combustion turbine generators (CTGs) with Heat Recovery Steam Generators (HRSG) (EUCTGHRSG1 & EUCTGHRSG2 in FGCTGHRSG). The total hours for startup and shutdown for each train shall not exceed 500 hours per 12-month rolling time period.



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[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**DRAFT****RBLC ID:** VA-0332**Corporate/Company:** CHICKAHOMINY POWER LLC**Facility Name:** CHICKAHOMINY POWER LLC**Process:** Three (3) Mitsubishi Hitachi Power Systems combustion turbine generators

### Pollutant Information - List of Pollutants

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**Primary Fuel:** natural gas  
**Throughput:** 35000.00 MMCF/YR  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	812.0000 LB/CO <sub>2</sub> E /MW-HR	BACT-PSD	NO
<u>Carbon Monoxide</u>	1.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPMVD 15% O <sub>2</sub>	BACT-PSD	NO
<u>Particulate matter, filterable (FPM)</u>	0.0052 LB/MMBTU	BACT-PSD	UNKNOWN
<u>Particulate matter, total &lt; 10 μ (TPM<sub>10</sub>)</u>	0.0052 LB/MMBTU	BACT-PSD	NO
<u>Particulate matter, total &lt; 2.5 μ (TPM<sub>2.5</sub>)</u>	0.0052 LB/MMBTU	BACT-PSD	NO
<u>Sulfur Dioxide (SO<sub>2</sub>)</u>	0.0011 LB/MMBTU	BACT-PSD	NO
<u>Sulfuric Acid (mist, vapors, etc)</u>	0.0012 LB/MMBTU	BACT-PSD	NO
<u>Volatile Organic Compounds (VOC)</u>	0.7000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO

**Process Notes:** One on one configuration: 4,066 MMBtu/hr combustion turbine. Emission limits reflect the operation of each of the three turbines.





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[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**FINAL**

**RBLC ID:** TN-0162

**Corporate/Company:** TENNESSEE VALLEY AUTHORITY

**Facility Name:** JOHNSONVILLE COGENERATION

**Process:** Natural Gas-Fired Combustion Turbine with HRSG

### Pollutant Information - List of Pollutants

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**Primary Fuel:** Natural Gas  
**Throughput:** 1339.00 MMBtu/hr  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	1800.0000 LB/MWH	BACT-PSD	UNKNOWN
<u>Carbon Monoxide</u>	2.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	UNKNOWN
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	UNKNOWN
<u>Particulate matter, total (TPM)</u>	0.0050 LB/MMBTU	BACT-PSD	UNKNOWN

**Process Notes:** Turbine throughput is 1019.7 MMBtu/hr when burning natural gas and 1083.7 MMBtu/hr when burning No. 2 oil. Duct burner throughput is 319.3 MMBtu/hr. Duct burner firing will occur during natural gas combustion only.



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[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**FINAL****RBLC ID:** VA-0328**Corporate/Company:** NOVI ENERGY**Facility Name:** C4GT, LLC**Process:** GE Combustion Turbine - Option 1 - Normal Operation

### Pollutant Information - List of Pollutants

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**Primary Fuel:** natural gas  
**Throughput:** 34000.00 MMCF/YR  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	883.0000 LB CO <sub>2</sub> E/MW-H	BACT-PSD	NO
<u>Carbon Monoxide</u>	1.0000 PPMVD@ 15% O <sub>2</sub>	BACT-PSD	NO
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO
<u>Particulate matter, total &lt; 10 μ (TPM<sub>10</sub>)</u>	0.0069 LB/MMBTU WITHOUT DUC	BACT-PSD	NO
<u>Particulate matter, total &lt; 2.5 μ (TPM<sub>2.5</sub>)</u>	0.0069 LB/MMBTU WITHOUT DUC	BACT-PSD	NO
<u>Sulfur Dioxide (SO<sub>2</sub>)</u>	0.0011 LB/MMBTU	OTHER CASE-BY-CASE	NO
<u>Sulfuric Acid (mist, vapors, etc)</u>	2.5000 LB/H	BACT-PSD	NO
<u>Volatile Organic Compounds (VOC)</u>	0.7000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO

**Process Notes:** Option 1: Two on one configuration: 3,482 MMBtu/hr combustion turbine with 475 MMBtu/hr duct-fired HRSG. Emission limits reflect the operation of one turbine with or without duct firing.



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[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**FINAL****RBLC ID:** VA-0328**Corporate/Company:** NOVI ENERGY**Facility Name:** C4GT, LLC**Process:** Siemens Combustion Turbine - Option 2 - Normal Operation

### Pollutant Information - List of Pollutants

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**Primary Fuel:** Natural Gas  
**Throughput:** 35000.00 MMBTU/YR  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	883.0000 LB CO <sub>2</sub> E/MW H	BACT-PSD	NO
<u>Carbon Monoxide</u>	1.8000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO
<u>Particulate matter, total &lt; 10 μ (TPM<sub>10</sub>)</u>	0.0065 LB/MMBTU	BACT-PSD	NO
<u>Particulate matter, total &lt; 2.5 μ (TPM<sub>2.5</sub>)</u>	0.0065 LB/MMBTU	BACT-PSD	NO
<u>Sulfur Dioxide (SO<sub>2</sub>)</u>	0.0011 LB/MMBTU	OTHER CASE-BY-CASE	NO
<u>Sulfuric Acid (mist, vapors, etc)</u>	2.2000 LB/H	BACT-PSD	NO
<u>Volatile Organic Compounds (VOC)</u>	1.0000 PPMVD @ 15% O <sub>2</sub>	BACT-PSD	NO

**Process Notes:** Option 2: Two on one configuration: 3,116 MMBtu/hr combustion turbine with 991 MMBtu/hr duct-fired HRSG. Emission limits reflect the operation of one turbine with or without duct firing.



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[RBLC Home](#)[New Search](#)[Search Results](#)[Facility Information](#)[Process List](#)[Process Information](#)[Help](#)**FINAL****RBLC ID:** VA-0325**Corporate/Company:** VIRGINIA ELECTRIC AND POWER COMPANY**Facility Name:** GREENSVILLE POWER STATION**Process:** COMBUSTION TURBINE GENERATOR WITH DUCT-FIRED HEAT RECOVERY STEAM GENERATORS (3)

### Pollutant Information - List of Pollutants

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**Primary Fuel:** natural gas  
**Throughput:** 3227.00 MMBTU/HR  
**Process Code:** 15.210

Pollutant	Primary Emission Limit	Basis	Verified
<u>Carbon Dioxide Equivalent (CO<sub>2</sub>e)</u>	890.0000 LB/MWH	OTHER CASE-BY-CASE	UNKNOWN
<u>Carbon Monoxide</u>	1.6000 PPMVD	N/A	UNKNOWN
<u>Nitrogen Oxides (NO<sub>x</sub>)</u>	2.0000 PPMVD	N/A	UNKNOWN
<u>Particulate matter, filterable &lt; 2.5 μ (FPM2.5)</u>	0.0039 LB/MMBTU	N/A	UNKNOWN
<u>Particulate matter, total &lt; 10 μ (TPM10)</u>	0.0039 LB/MMBTU	N/A	UNKNOWN
<u>Sulfur Dioxide (SO<sub>2</sub>)</u>	0.0011 LB/MMBTU	N/A	UNKNOWN
<u>Sulfuric Acid (mist, vapors, etc)</u>	0.0006 LB/MMBTU	N/A	UNKNOWN
<u>Volatile Organic Compounds (VOC)</u>	1.4000 PPMVD	N/A	UNKNOWN

**Process Notes:** 3227 MMBTU/HR CT with 500 MMBTU/HR Duct Burner, 3 on 1 configuration.

174 FERC ¶ 61,126  
UNITED STATES OF AMERICA  
FEDERAL ENERGY REGULATORY COMMISSION

Before Commissioners: Richard Glick, Chairman;  
Neil Chatterjee, James P. Danly,  
Allison Clements, and Mark C. Christie.

Algonquin Gas Transmission, LLC  
Maritimes & Northeast Pipeline, LLC

Docket No. CP16-9-012

ORDER ESTABLISHING BRIEFING

(Issued February 18, 2021)

1. On September 24, 2020, Commission staff issued an order authorizing Algonquin Gas Transmission, LLC (Algonquin) and Maritimes & Northeast Pipeline, LLC (Maritimes) (together, Applicants) to place facilities associated with the Atlantic Bridge Project into service (Authorization Order).<sup>1</sup> On October 23, 2020, the Fore River Residents Against the Compressor Station (Fore River Residents), the City of Quincy, Massachusetts, Weymouth Councilor Rebecca Haugh, Michael Hayden, and Food and Water Watch (collectively Petitioners) filed a timely joint request for rehearing of the Authorization Order. Since issuance of the Authorization Order the Commission has also received numerous other pleadings expressing safety concerns regarding the operation of the project.

2. We believe that the concerns raised regarding the operation of the project warrant further consideration by the Commission and set the matter for paper briefing to address the questions listed below. Initial briefs will be due 45 days from the date of this order. Reply briefs will be due 30 days thereafter. The facilities placed in service pursuant to the Authorization Order may remain in service while the Commission considers the issues raised here. The Commission asks for briefing on the following matters:

- In light of the concerns expressed regarding public safety, is it consistent with the Commission's responsibilities under the Natural Gas Act (NGA) to allow the Weymouth Compressor Station to enter and remain in service?

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<sup>1</sup> *Algonquin Gas Transmission, LLC*, Docket No. CP16-9-000, at 1 (Sept. 24, 2020) (delegated order) (Authorization Order).

- Should the Commission reconsider the current operation of the Weymouth Compressor Station in light of any changed circumstances since the project was authorized? For example, are there changes in the Weymouth Compressor Station's projected air emissions impacts or public safety impacts the Commission should consider? We encourage parties to address how any such changes affect the surrounding communities, including environmental justice communities.
- Are there any additional mitigation measures the Commission should impose in response to air emissions or public safety concerns?
- What would the consequences be if the Commission were to stay or reverse the Authorization Order?

The Commission orders:

Briefing procedures are hereby established, as discussed in the body of this order. Initial briefs are due 45 days from the date of this order and reply briefs are due 30 days thereafter.

By the Commission. Commissioner Danly is dissenting with a separate statement attached.  
Commissioner Christie is dissenting with a separate statement attached.

( S E A L )

Kimberly D. Bose,  
Secretary.

UNITED STATES OF AMERICA  
FEDERAL ENERGY REGULATORY COMMISSION

Algonquin Gas Transmission, LLC  
Maritimes & Northeast Pipeline, LLC

Docket No. CP16-9-012

(Issued February 18, 2021)

DANLY, Commissioner, *dissenting*:

1. I dissent in full from the majority's "Order Establishing Briefing" in *Algonquin Gas Transmission, LLC* Docket No. CP16-9-012. This order is both contrary to law and bad policy. Before I explain my reasoning, a complete recitation of the background facts is necessary.

**I. Background**

2. Over four years ago, on January 25, 2017, the Commission issued Algonquin Gas Transmission, LLC (Algonquin) a certificate authorizing the construction and operation of the Weymouth Compressor Station as part of the Atlantic Bridge Project.<sup>1</sup> The Commission found the project to be in the public convenience and necessity after considering the project need and the environmental effects of the project, including the effects that constructing and operating the Weymouth Compressor Station would have on safety, air quality, and environmental justice communities.<sup>2</sup> The Certificate Order found that the Weymouth Compressor Station would not result in a significant increase in risk to the nearby public "[b]ased on Algonquin's commitment to comply with [Pipeline and Hazardous Materials Safety Administration (PHMSA)] requirements."<sup>3</sup> In addition, the Commission's Environmental Assessment (EA) estimated the fugitive emissions (including blowdowns) at the Weymouth Compressor Station, compared the emissions to

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<sup>1</sup> *Algonquin Gas Transmission, LLC*, 158 FERC ¶ 61,061 (2017) (Certificate Order). Chairman Bay, Commissioner LaFleur, and Commissioner Honorable unanimously approved the certificate.

<sup>2</sup> See *id.* PP 225-238 (safety), *id.* PP 194-216 (air quality), *id.* PP 185-189 (environmental justice). The Certificate Order also addressed specific air quality and health effects from blowdowns. See *id.* PP 198, 223

<sup>3</sup> *Id.* P 226.

a past health risk assessment performed on a similar facility, and found that the health risks from operating the compressor station would not be significant.<sup>4</sup>

3. In December 2018, the U.S. Court of Appeals for the D.C. Circuit upheld the Certificate Order, including the Commission's assessment of impacts on public safety and environmental justice.<sup>5</sup>

4. On November 27, 2019, Commission staff authorized Algonquin to commence construction of the Weymouth Compressor Station after confirming that Algonquin had received all federal authorizations relevant to the approved activities. Those federal authorizations included its Air Quality Plan approved by the Massachusetts Department of Environmental Protection (Massachusetts DEP).

5. Late summer last year, Algonquin began testing its compressor station facilities as required by PHMSA.<sup>6</sup> Section 192.503 of PHMSA's regulations prohibits any person from operating a new segment of pipeline until "(1) [i]t has been tested in accordance with this subpart and § 192.619 to substantiate the maximum allowable operating pressure; and (2) [e]ach potentially hazardous leak has been located and eliminated."<sup>7</sup> Further, section 192.503 requires the test medium to be "liquid, air, natural gas, or inert gas."<sup>8</sup>

6. On September 11, 2020, during Algonquin's testing of equipment, a gasket failed, triggering the manual activation of its emergency shutdown system. Section 192.167 of the PHMSA's regulations requires compressor stations to have emergency shutdown systems that blow down the station piping.<sup>9</sup> Consequently, Algonquin's emergency shutdown system blew down natural gas, releasing 169,000 standard cubic feet (scf) of natural gas and 35 pounds (lbs) (or 0.0175 tons<sup>10</sup>) of Volatile Organic Compounds (VOCs), which is approximately 0.19 percent of the estimated 9.0 tons of annual fugitive

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<sup>4</sup> EA at 2-95, 2-98.

<sup>5</sup> *Town of Weymouth v. FERC*, No. 17-1135, 2018 WL 6921213 (D.C. Cir. Dec. 27, 2018) (unpublished opinion).

<sup>6</sup> Algonquin September 29, 2020 Weekly Status Report for No. 176 for Reporting Period Ending September 4, 2020 at 2.

<sup>7</sup> 49 C.F.R. § 192.503(a) (2020).

<sup>8</sup> *Id.* § 192.503(b).

<sup>9</sup> *Id.* § 192.167(a)(1).

<sup>10</sup> One ton equals 2,000 lbs.



VOCs evaluated by the EA.<sup>11</sup> Thereafter, Algonquin continued testing and calibrating activities.<sup>12</sup> The record does not show Massachusetts DEP initiating a compliance action.

7. On September 16, 2020, Algonquin requested authorization to place the Weymouth Compressor Station into service pursuant to Environmental Condition 10 of the Certificate Order. Environmental Condition 10 requires Algonquin to “receive written authorization from the Director of OEP before commencing service on each discrete facility of the Project” and provided that “[s]uch authorization will only be granted following a determination that rehabilitation and restoration of the right-of-way and other areas affected by the Project are proceeding satisfactorily.”<sup>13</sup>

8. On September 24, 2020, Commission staff authorized Algonquin to place its Weymouth Compressor Station into service, finding that “Algonquin and Maritimes [had] adequately stabilized areas disturbed by construction and that restoration is proceeding satisfactorily.”<sup>14</sup>

9. On September 30, 2020, the Weymouth Compressor Station experienced an unplanned emergency shutdown, releasing approximately 195,000 scf of natural gas, including 27 lbs (or 0.0135 tons) of VOCs, which is approximately 0.15 percent of the estimated 9.0 tons of annual fugitive VOCs evaluated by the EA. The cause of the unplanned shutdown was unknown. That same day, Algonquin voluntarily shut in its system.<sup>15</sup> The record does not show Massachusetts DEP initiating a compliance action.

10. On October 1, 2020, as amended on October 30, 2020, PHMSA issued a Corrective Action Order directing Algonquin to not operate the compressor station until authorized to do so, develop a Restart Plan for approval, and complete a root cause failure analysis.<sup>16</sup>

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<sup>11</sup> EA at 2-95, tbl. 2.7.4-3.

<sup>12</sup> Algonquin October 7, 2020 Weekly Status Report No. 177 for the Reporting Period Ending September 11, 2020 at 3.

<sup>13</sup> Certificate Order, 158 FERC ¶ 61,061 at Appendix B, Environmental Condition 10.

<sup>14</sup> Commission Staff September 24, 2020 Letter Order Authorizing Commencement of Service at 1 (Authorization Order).

<sup>15</sup> Algonquin October 7, 2020 Weekly Status Report No. 180 for the Reporting Period Ending October 2, 2020 at 2.

<sup>16</sup> PHMSA, Corrective Action Order (Oct. 1, 2020),

11. On October 23, 2020, Petitioners<sup>17</sup> filed a timely request for rehearing of Commission staff's September 24, 2020 Letter. First, they argued that the Commission "failed to complete a situational assessment and strategic responses for public safety and environmental impacts associated with incidents involving natural gas infrastructure."<sup>18</sup> Second, they argued "[t]he unplanned emergency shutdowns and COVID-19 pandemic . . . rise to the level of a change in core circumstances" requiring the Commission to reopen the record under Rule 716 of the Commission's Rules of Practice and Procedure.<sup>19</sup> Petitioners did not challenge Commission staff's finding that restoration and rehabilitation was proceeding satisfactorily.

12. On November 23, 2020, the Commission issued a notice denying Petitioners' rehearing request by operation of law.

13. On November 25, 2020, PHMSA approved Algonquin's Restart Plan and authorized Algonquin to return the compressor station facilities to a pressure not exceeding 80 percent of full operating pressure.<sup>20</sup>

14. On January 22, 2021, PHMSA approved the temporary operation of the Weymouth Compressor Station at full pressure, stating "PHMSA has reviewed the [root cause failure analysis] and the data submitted on [Algonquin's] preventative and mitigative measures performed and based on our technical review, it is our determination to allow the temporary removal of the pressure restriction."<sup>21</sup>

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[https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2020-10/12020014CAO\\_Corrective%20Action%20Order\\_10012020-Algonquin%20Gas%20Transmission.pdf](https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2020-10/12020014CAO_Corrective%20Action%20Order_10012020-Algonquin%20Gas%20Transmission.pdf).

<sup>17</sup> Petitioners include the Fore River Residents Against Compressor Station; City of Quincy, Massachusetts; Weymouth Councilor Rebecca Haugh; Michael Hayden; and Food & Water Watch.

<sup>18</sup> Petitioners Oct. 23, 2020 Rehearing at 2.

<sup>19</sup> *Id.* at 3. Although not explicitly stated, it is apparent that the Petitioners sought to reopen the Certificate Order. *Id.* at 5 ("The issuance of the Certificate Order on January 25, 2017 could not possibly have foreseen the impact of COVID-19, nor could the Certificate Order have anticipated the disparate impact the pandemic would have upon environmental justice communities in the Commonwealth of Massachusetts.")

<sup>20</sup> PHMSA, Letter Approving Restart Plan (Nov. 25, 2020), [https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2020-11/12020014CAO\\_PHMSA%20Approval%20of%20Weymouth%20Restart%20Plan\\_11252020.pdf](https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2020-11/12020014CAO_PHMSA%20Approval%20of%20Weymouth%20Restart%20Plan_11252020.pdf).

<sup>21</sup> PHMSA, Letter Approving Enbridge Allowing Temporary Removal of Pressure

15. On January 25, 2021, Algonquin placed the Weymouth Compressor Station into service.<sup>22</sup>

16. Now on February 18, 2021—over four years after the Commission issued the Certificate Order authorizing the operation of the Weymouth Compressor Station, nearly four months after Petitioners’ timely rehearing request, after PHMSA has authorized Algonquin to resume operating the Weymouth Compressor Station at full pressure, and without any indication that Algonquin is out of compliance with its air permit—the Commission is issuing this “Order Establishing Briefing.”

## **II. The Order is an Attempt to Revisit the Certificate Proceeding and is Contrary to Law**

### **A. This Order is an Attempt to Revisit the Certificate Order**

17. It is somewhat difficult to make sense of this order. On its face, it bears the benign-sounding title “Order Establishing Briefing.” Those sorts of orders are issued now and again; they are procedural and, one would think, warrant little scrutiny. But briefing for what? The Certificate Order and the Authorization Order are both final—the Certificate Order was issued more than four years ago, and as for the Authorization Order, rehearing was denied by operation of law and the opportunity to appeal lapsed without a petition for review. Both of those proceedings appear to be irretrievably final. And, in fact, this order is *neither* of those proceedings. The Commission has assigned a new sub-docket number, -012, to distinguish it from the rehearing proceeding.<sup>23</sup> Confusion is justified as to what exactly is at issue since the Order Establishing Briefing cites to pleadings filed in the rehearing sub-docket.

18. Procedural oddities aside, this order does not look like other orders, by which I mean that those few people who spend a large amount of their time reading Commission orders will enjoy the familiarity of the caption and paragraph format but will be left with vague unease as they notice that the order is missing some fairly standard contents. It has no background section. It offers no basis in law for the Commission’s action. It provides no explanation as to what it is trying to achieve other than a vague promise of the “further

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Restriction at Weymouth Compressor Station at 1 (Jan. 22, 2021), [https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2021-01/12020014CAO\\_Region%20Response%20to%20Corrective%20Action%20Item%205\\_01222021.pdf](https://www.phmsa.dot.gov/sites/phmsa.dot.gov/files/2021-01/12020014CAO_Region%20Response%20to%20Corrective%20Action%20Item%205_01222021.pdf).

<sup>22</sup> Algonquin January 25, 2021 Notice of Commencement of Service.

<sup>23</sup> With a new docket number may come a new intervention period. Every pipeline company, shipper, and pipeline investor should consider intervening in this “new” proceeding.

consideration” of something.<sup>24</sup> In fact, in the last 10 years, the Commission has never issued an order captioned “Order Establishing Briefing” and to the extent that free-standing briefing orders have issued during that time, they have issued following remand from appellate courts, or to address issues not resolved in settlement, motions for interlocutory appeal, and investigations into the justness and reasonableness of rates.<sup>25</sup>

19. So what exactly does this order purport to do? It states that staff authorized Algonquin to place the Weymouth Compressor Station into service and it mentions that a timely rehearing request and other pleadings were filed. Then it states that the Commission “believe[s] that the concerns raised regarding the operation of the project warrant further consideration by the Commission and set[s] the matter for paper briefing to address” a series of appended questions.<sup>26</sup> By its plain language, the order requests

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<sup>24</sup> This formulation, “further consideration,” is particularly unfortunate and perhaps even provocative in an order issued in a closed docket following the D.C. Circuit’s issuance of *Allegheny Defense Project v. FERC*, 964 F.3d 1 (D.C. Cir. 2020) (en banc).

<sup>25</sup> See, e.g., *Am. Elec. Power Serv. Corp. v. Midcontinent Indep. Sys. Operator, Inc.*, 168 FERC ¶ 61,178 (2019) (order establishing briefing procedures to investigate potentially unjust and unreasonable rates); *Duke Energy Corp.*, 163 FERC ¶ 61,102 (2018) (order establishing briefing schedule following remand); *Black Oak Energy, L.L.C.*, 146 FERC ¶ 61,099 (2014) (same); *Duquesne Light Co.*, 135 FERC ¶ 61,237 (2011) (order establishing briefing procedures to develop a record to enable the Commission to respond to a district court’s questions); *Midwest Indep. Transmission Sys. Operator, Inc.*, 108 FERC ¶ 61,248 (2004) (order establishing briefing schedule to consider rehearing requested 13 days before order issuance); *Nat. Gas Pipeline Co. of Am.*, 82 FERC ¶ 61,061 (1998) (order establishing briefing schedule to consider pipeline’s request to flow through refunds); *El Paso Nat. Gas Co.*, 82 FERC ¶ 61,060 (1998) (same); *Union Pac. Fuels, Inc.*, 75 FERC ¶ 61,071 (1996) (order establishing briefing schedule on complaint regarding violation of NGA); *Williston Basin Interstate Pipeline Co.*, 66 FERC ¶ 61,169 (1994) (order establishing briefing schedule to address issues not resolved in settlement); *Tenn. Gas Pipeline Corp.*, 63 FERC ¶ 61,204 (1993) (same); *Northwest Pipeline Corp.*, 63 FERC ¶ 61,028 (1993) (same); *Trunkline Gas Co.*, 57 FERC ¶ 61,314 (1991) (same); *Panhandle Eastern Pipe Line Co.*, 57 FERC ¶ 61,313 (1991) (same); *Transcontinental Gas Pipe Line Corp.*, 38 FERC ¶ 61,142 (1987) (order establishing briefing on interlocutory appeal from rulings of the presiding judge); *Am. Elec. Power Serv. Corp.*, 30 FERC ¶ 61,011 (1985) (order establishing briefing schedule following remand); *City of Cleveland v. Cleveland Elec. Illuminating Co.*, 56 F.P.C. 2673 (same).

<sup>26</sup> *Algonquin Gas Transmission, LLC*, 174 FERC ¶ 61,126, at P 2 (2021) (Order

information on a set of discrete topics for the Commission’s “further consideration.” To what end? Among other things, the questions ask, rather ominously, (1) whether the Commission “should allow the Weymouth Compressor Station to enter and remain in service”; (2) whether the Commission should “reconsider” the current operation of the compression station; (3) whether the Commission “should consider” changes in air emissions or public safety impacts; (4) whether there are any “additional mitigation measures” the Commission should “impose” (presumably by means of revising Environmental Condition 10 of the Certificate Order); and (5) what would happen if the Commission were to “stay or reverse” the Authorization Order.

20. It would appear that the Commission is collecting comments in order to determine whether it should re-litigate the Certificate Order absent a breach or violation of the certificate terms and conditions. Though the majority may be laboring under the impression that this Order Establishing Briefing is no more than a late attempt to grant a (now denied and final, non-appealable) rehearing request sought following the Authorization Order, the Order asks questions that go directly the Certificate Order only. Only by re-litigating the Certificate Order and modifying Environmental Condition 10 of the Certificate Order can the Commission “reconsider the current operation of the Weymouth Compressor Station,” consider “changes in . . . projected air emissions or public safety impacts,” “impose” “additional mitigation measures,” or “stay or reverse the Authorization Order.” Moreover, none of the questions address the basis for the Authorization Order—whether the rehabilitation and restoration of lands affected by project construction were proceeding satisfactorily.

### **B. The Order Establishing Briefing is Contrary to Law**

21. This Order is legally infirm because the action is simply beyond the Commission’s authority. Even if it were not *ultra vires*, the Commission has fallen short of its Administrative Procedure Act (APA) obligations by failing to explain why it departs from the Commission’s rules and policies.

22. There is a good reason for why the Commission fails to cite legal authority for today’s order—“[t]he Commission has already approved the [c]ertificate, and there is nothing in the law that allows us to revisit that decision.”<sup>27</sup> Just so. The current Commission may believe that the Commission, voting unanimously, acted improvidently in early 2017. They may believe that circumstances have changed.<sup>28</sup> They may believe

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Establishing Briefing).

<sup>27</sup> Commissioner (now Chairman) Glick, Comments at Open Meeting at 29 (Jan. 19, 2021).

<sup>28</sup> Circumstances, however, have not changed and additional briefing on this matter is not needed to make this finding. The Certificate Order found that there were no

that the parties seeking rehearing were completely correct and that rehearing should have been granted. They may be right.<sup>29</sup> Regardless, there is no basis in law to re-examine final orders.

23. The Commission, as a mere creature of statute, can only act pursuant to law by which Congress had delegated its authority.<sup>30</sup> Although courts afford agencies great discretion to establish the procedures by which they conduct their business, that business, however fashioned, must be conducted within the bounds of that delegation.<sup>31</sup>

24. Nowhere does NGA section 7 authorize the Commission to unilaterally revisit final certificate orders or establish briefing schedules to inform such actions. Quite the contrary. NGA section 7(e) states: “a certificate shall be issued . . . if it is found that the applicant is able and willing properly to do the acts . . .”<sup>32</sup> and “[t]he Commission shall have the power to attach to the issuance of the certificate and to the exercise of the rights granted thereunder such reasonable terms and conditions . . .”<sup>33</sup> So conditioned, the

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significant impacts on safety because Algonquin would comply with PHMSA regulations. Certificate Order, 158 FERC ¶ 61,061 at P 226. Algonquin has done so. *See supra* PP 5-6, 9-10, 13-14. Further, the Commission considered blowdown events, such as those that occurred in September, and found they would not have significant effects on air quality and health. EA at 2-98. And moreover, the amount of VOCs released by the events amounted to only 0.34 percent of the estimated blowdown emissions from the Weymouth Compressor Station in the EA. *See supra* PP 6, 9.

<sup>29</sup> This is unlikely. Every subject raised in the rehearing requests was fully litigated at various stages of the underlying proceedings. *See* Appendix.

<sup>30</sup> *Bowen v. Georgetown Univ. Hosp.*, 488 U.S. 204, 208 (1988) (“It is axiomatic that an administrative agency’s power to promulgate legislative regulations is limited to the authority delegated by Congress.”); *accord, e.g., Atl. City Elec Co. v. FERC*, 295 F.3d 1 (D.C. Cir. 2002) (“As a federal agency, FERC is a ‘creature of statute,’ having ‘no constitutional or common law existence or authority, but *only* those authorities conferred upon it by Congress.’”) (quoting *Michigan v. EPA*, 268 F.3d 1075, 1081 (D.C. Cir. 2001)) (emphasis in original).

<sup>31</sup> For example, the Commission established a tolling procedure for rehearing requests in which the D.C. Circuit found was contrary to the Natural Gas Act (NGA). *See Allegheny Def. Project v. FERC*, 964 F.3d 1.

<sup>32</sup> 15 U.S.C. § 717f(e).

<sup>33</sup> *Id.* *See also Trunkline LNG Co.*, 22 FERC ¶ 63,028, at 65,135-39 (1983) (Chief Administrative Law Judge Recommended Decision).

Commission's regulations require the pipeline to then accept the certificate order.<sup>34</sup> In sum, the Commission's power is to grant, with conditions, a certificate of public convenience and necessity and to enforce the certificate. Absent a violation of those conditions, once the certificate issues and becomes final, the Commission has never revisited a certificate order and has in fact always doubted its ability to do so.<sup>35</sup>

25. Many are quick to turn to NGA section 16 when all else has failed, but it is often freighted with more weight than it can bear. Section 16 does not represent an independent grant of authority: "[t]he Commission shall have power to perform any and all acts, and to prescribe, issue, make, amend, and rescind such orders, rules, and regulations as it may find necessary or appropriate to carry out the provisions of this chapter."<sup>36</sup> This does not create new powers under the NGA or obviate NGA section 7(e), which limits the Commission's authority over a certificate to the certificate's conditions.<sup>37</sup> Moreover, like its counterpart in FPA section 309, the use of NGA

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<sup>34</sup> 18 C.F.R. § 157.20(a) (2020) ("The certificate shall be void and without force or effect unless accepted in writing by applicant within 30 days from the issue date of the order issuing such certificate."). *Cf.* 16 U.S.C. § 799 ("Each such license shall be conditioned upon acceptance by the licensee of all of the terms and conditions of this chapter and such further conditions, if any, as the Commission shall prescribe in conformity with this chapter, which said terms and conditions and the acceptance thereof shall be expressed in said license."); *Clifton Power Corp. v. FERC*, 88 F.3d 1258, 1261 (D.C. Cir. 1996) (concluding that the Commission erred in finding the license required the licensee to operate the project in a run-of-river mode because the license order did not contain an explicit condition requiring the licensee to operate run-of-river).

<sup>35</sup> *Trunkline LNG Co.*, 22 FERC ¶ 61,245, at 61,442 (1983). In *Trunkline*, the Commission declined to address whether it had the authority to revisit a certificate. However, to the extent the Commission has the authority, the Commission stated that action, "would be an extraordinary step and would, in our judgment, require a compelling showing of a fundamental shift of a long-term nature in the basic premises on which the certificate was issued." *Id.* at 61,442. The Commission also stated "because the project had previously been approved by the Commission and funds committed based on that approval, the Commission would be obligated to revoke or modify the certificate in a manner that would leave investors in the project in substantially the same position they would have been had the Commission not revoked or modified the certificate." *Id.* at 61,442 n.5. The record shows no fundamental shift, and the Order Establishing Briefing asks no questions on how to leave investors in substantially the same position they would have been.

<sup>36</sup> 15 U.S.C. § 717o.

<sup>37</sup> *Fla. Gas Transmission Co. v. FERC*, 604 F.3d 636, 647 (D.C. Cir. 2010)

section 16 must be “consistent with the authority delegated to it by Congress.”<sup>38</sup> But the order here does not do so because it flies in the face of the statutory process for rendering final orders subject to judicial review.

26. No other law, regulation, or policy can be relied upon to revisit a certificate. Rule 716, which allows the Commission to reopen the record in certain proceedings,<sup>39</sup> explicitly applies only to initial or revised initial decisions and, moreover, *does not apply* to final, unappealable orders.<sup>40</sup> And even if there were another source of authority, the Commission has failed to explain how the exercise of that authority in this proceeding can be squared with the Commission’s longstanding practice of leaving final,

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(“[W]hile section 16 gives the Commission ancillary jurisdiction to carry out the statute’s other provisions, it does not confer additional jurisdiction . . . otherwise outside the Commission’s jurisdiction.”) (citing *Pub. Serv. Comm’n of N.Y. v. FERC*, 866 F.2d 487, 491-92 (D.C. Cir. 1989)).

<sup>38</sup> *Verso Corp. v. FERC*, 898 F.3d 1, 7 (D.C. Cir. 2018) (citing *Xcel Energy Servs. Inc. v. FERC*, 815 F.3d 947, 952 (D.C. Cir. 2016)); *accord id.* at 10 (“Section 309 accordingly permits FERC to advance remedies not expressly provided by the FPA, *as long as they are consistent with the Act.*”) (emphasis added) (citing *TNA Merch. Projects, Inc. v. FERC*, 857 F.3d 354, 359 (D.C. Cir. 2017) (citing *Niagara Mohawk Power Corp. v. Fed. Power Comm’n*, 379 F.2d 153, 158 (D.C. Cir. 1967))).

<sup>39</sup> 18 C.F.R. § 385.716 (2020). “Initial decision” is “any decision rendered by a presiding officer in accordance with Rule 208”—meaning a decision rendered by the Administrative Law Judges, not the Commission. *Id.* § 385.702. The Commission has previously applied Rule 716 to Commission orders despite the Commission’s regulations to the contrary. *See Panhandle E. Pipe Line Co. v. FERC*, 613 F.2d 1120, 1135 (D.C. Cir. 1979) (“[W]e do not believe the Commission should have authority to play fast and loose with its own regulations. It has become axiomatic that an agency is bound by its own regulations.”). To my knowledge, the Commission has never reopened a record of a final order that was affirmed on appeal. Nor can the majority square reopening the record of the Authorization Order with its long-standing policy to reopen only where there is “a change in the core circumstance that goes to the very of the case,” *CSM Midland, Inc.*, 56 FERC ¶ 61,177, at 61,624 (1991), as the safety and air emissions are entirely unrelated to the issuance of the Authorization Order. Similarly, the majority has not explained its departure from its long-standing policy.

<sup>40</sup> *See N. Nat. Gas Co.*, 113 FERC ¶ 61,060, at 61,170 (2005); *Old Dominion Elec. Coop.*, 105 FERC ¶ 61,094, at 61,485 (2003).



unappealable orders undisturbed. Failure to set forth that explanation, in the face of so long a practice, is necessarily a violation of the APA.<sup>41</sup>

### III. The Order is Bad Policy

27. On top of being unlawful, the Order is bad policy. Issuing an order that appears to revisit final, unappealable certificate orders impairs regulatory certainty and arrogates to the Commission authority it does not have.

28. Regulatory certainty, of which finality is a large part, is absolutely critical to achieving the goals of the NGA. “[W]ithout the sanctity of certificates granted under Sections 3 and 7 of the Natural Gas Act, there would be no private financing, and without private financing, there would be no projects.”<sup>42</sup> Further, “the revocation or adverse modification of a certificate or authorization . . . when the certificate or authorization forms the basis of project financing would be a clear violation of the basic constitutional principles of due process.”<sup>43</sup>

29. Worse still, the Order Establishing Briefing impairs the finality normally enjoyed by certificate holders, based on issues well outside our jurisdiction. The Order asks: whether the Commission should revisit the Certificate Order on the basis of pipeline operational safety and air emissions. Reading this, one would presume that Algonquin is not in compliance with pipeline safety and air emission requirements and the Commission has the authority and expertise to address the non-compliance. Neither of those presumptions, however, is correct.

30. First, as I note above, PHMSA and Massachusetts DEP appear satisfied that Algonquin is complying with their regulations and requirements. Nearly one month ago, PHMSA authorized Algonquin to resume operating the Weymouth Compressor Station at

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<sup>41</sup> See *FCC v. Fox Television Stations, Inc.*, 556 U.S. 502, 515 (2009) (“[T]he requirement that an agency provide reasoned explanation for its action would ordinarily demand that it display awareness that it *is* changing position.”) (emphasis in original); *id.* (“[A]n agency may not . . . depart from a prior policy *sub silentio* or simply disregard rules that are still on the books.”); *New England Power Generators Ass’n, Inc. v. FERC*, 881 F.3d 202, 211 (D.C. Cir. 2018) (finding “that FERC did not engage in the reasoned decisionmaking required by the Administrative Procedure Act” because it “failed to respond to the substantial arguments put forward by Petitioners and *failed to square its decision with its past precedent*”) (emphasis added).

<sup>42</sup> *Trunkline LNG Co.*, 22 FERC ¶ 63,028 at 65,139.

<sup>43</sup> *Id.*

full pressure.<sup>44</sup> Massachusetts DEP approved the Air Quality Plan for the Weymouth Compressor Station, finding it is in compliance with the Air Pollution Control regulations and current air pollution control engineering practice.<sup>45</sup> The record does not show Massachusetts DEP initiating a compliance action.

31. Second, Congress expressly delegated to the Secretary of the Department of Transportation the authority to regulate pipeline safety<sup>46</sup> and to the U.S. Environmental Protection Agency (EPA) the authority to regulate air emissions.<sup>47</sup> The Commission's long-standing practice is to rely on PHMSA to regulate pipeline safety and the EPA, or its state delegated agency, to regulate air emissions.<sup>48</sup> It is baffling on what factual basis the Commission could modify the Certificate Order and what additional measures the Commission could impose that PHMSA and Massachusetts DEP have not considered and would not interfere with their approvals.

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<sup>44</sup> See *supra* P 14.

<sup>45</sup> Massachusetts DEP, Air Quality Plan Approval at 2 (Aug. 26, 2019), <https://www.mass.gov/doc/air-quality-plan-approval-august-2019/download>. Massachusetts affirmed the plan on September 29, 2020. Massachusetts DEP, Final BACT Determination for Weymouth Compressor Station at 1, <https://www.mass.gov/doc/final-bact-determination-september-29-2020/download>.

<sup>46</sup> See 49 U.S.C. § 60102(a)(2) (2018) (“The Secretary shall prescribe minimum safety standards for pipeline transportation and for pipeline facilities.”); see also FERC, Natural Gas Safety and Inspections, <https://www.ferc.gov/industries-data/natural-gas/safety-and-inspections> (“[o]nce Natural Gas pipeline projects become operational, safety is regulated, monitored, and enforced by the Department of Transportation”); FERC, Strategic Plan FY2018-2021 at vii, <https://www.ferc.gov/sites/default/files/2020-04/FY-2018-FY-2022-strat-plan.pdf> (lists “[r]esponsibility for pipeline safety” under the heading “What FERC does not do”).

<sup>47</sup> See *Wyoming v. U.S. Dep’t of Interior*, 2020 WL 7641067 \*9 (Oct. 8, 2020) (“The rub here, however, is whether the Rule, or at least certain provisions of the Rule, was promulgated *for the prevention of waste* or instead for the *protection of air quality*, which is expressly within the ‘substantive field’ of the EPA and States pursuant to the Clean Air Act.”) (emphasis in original).

<sup>48</sup> See *Town of Weymouth*, No. 17-1135, 2018 WL 6921213 at \*1 (“although the challengers argue that FERC impermissibly relied on the pipeline companies’ assertions that they would comply with certain federal safety regulations, FERC was entitled, ‘[a]bsent evidence to the contrary,’ to ‘assume . . . that [the companies] will exercise good faith.’ *Murray Energy Corp. v. FERC*, 629 F.3d 231, 240 (D.C. Cir. 2011).”).

32. Intended or not, the message from this order is clear: even if a pipeline has its certificate, a court upholds that certificate, and that pipeline is in compliance, the Commission can now find a way to modify, or even possibly revoke, the certificate. This order requires Algonquin to relitigate the Certificate Order affirmed over three years ago. Algonquin has now been aggrieved.<sup>49</sup> This order threatens the certainty of the certificate upon which the pipeline's business is founded, disregards the principles of final judgement upon which all litigants rely, and violates the specific statutory procedures devised by Congress to render and challenge final orders. The order manufactures what is essentially an end-run around the statutory process for rehearing and judicial review that is far more dangerous and disruptive than the Commission's past abuse of tolling orders,<sup>50</sup> because tolling orders only delayed the final resolution of cases, but did not constitute surprise attacks on long-final orders. Algonquin should appeal immediately.

For these reasons, I respectfully dissent.

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James P. Danly  
Commissioner

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<sup>49</sup> *Cf. Papago Tribal Util. Auth. v. FERC*, 628 F.2d 235, 245 (D.C. Cir. 1980) (explaining that Mobile-Sierra claims are immediately reviewable in the courts).

<sup>50</sup> *See Allegheny Def. Project v. FERC*, 964 F.3d 1.

## Appendix

### FERC Process

- On January 25, 2017, the Commission issued a Certificate Order to Algonquin, considering the safety risk of the compressor station, the air quality and health impacts of blowdowns, and impacts on environmental justice communities near the compressor station. *See Algonquin Gas Transmission, LLC*, 158 FERC ¶ 61,061 (2017).
- On December 13, 2017, the Commission denied rehearing after considering the safety risks of the Weymouth Compressor Station (PP 27-28, 32, 134-139), the effects of blowdowns (P 132), and environmental justice (PP 91-99). *See Algonquin Gas Transmission, LLC*, 161 FERC ¶ 61,255 (2017).
- On December 27, 2018, the U.S. Court of Appeals for the D.C. Circuit affirmed the Commission's Certificate Order, including its consideration of impacts on safety and environmental justice. *Town of Weymouth, Massachusetts*, No. 17-1135, 2018 WL 6921213 (D.C. Cir. Dec. 27, 2018) (unpublished opinion).

### Massachusetts DEP Air Quality Plan Approval

- In March 2017, Massachusetts DEP issued a proposed Air Quality Plan Approval, determining that the Massachusetts Environmental Justice Policy does not apply because the anticipated emissions would not exceed emission thresholds. *See* Massachusetts DEP, Air Quality Proposed Plan Approval (Mar. 30, 2017), <https://www.mass.gov/doc/proposed-air-quality-plan-approval-march-2017/download>.
- In the spring of 2017, Massachusetts DEP held a public comment period on the proposed Air Quality Plan Approval. *See* Massachusetts DEP Algonquin Natural Gas Compressor Station, Weymouth, <https://www.mass.gov/service-details/algonquin-natural-gas-compressor-station-weymouth>.
- In July 2017, Governor Baker directed Massachusetts DEP and the Massachusetts Department of Public Health to perform a comprehensive health impact assessment. *See id.*
- In January 2019, Massachusetts DEP and the Massachusetts Department of Health issued the *Health Impact Assessment of a Proposed Natural Gas Compressor Station in Weymouth*. The assessment considered health and environmental justice

impacts of the Weymouth Compressor Station. *See* Massachusetts Department of Health et al., *Health Impact Assessment of a Proposed Natural Gas Compressor Station in Weymouth*, MA (January 2019), [http://foreriverhia.wpengine.com/wp-content/uploads/2019/01/Final-Report\\_20190104.pdf](http://foreriverhia.wpengine.com/wp-content/uploads/2019/01/Final-Report_20190104.pdf).

- On January 11, 2019, Massachusetts DEP issued a Non-Major Comprehensive Air Quality Plan Approval to Algonquin for its construction and operation of the Weymouth Compressor Station. *See* Massachusetts DEP, Air Quality Plan Approval (January 11, 2019).
- In May and June 2019, an adjudicatory hearing was held on six appeals of Massachusetts DEP's approval. *See* Massachusetts DEP Algonquin Natural Gas Compressor Station, Weymouth, <https://www.mass.gov/service-details/algonquin-natural-gas-compressor-station-weymouth>.
- On August 26, 2019, Massachusetts DEP issued a Non-Major Comprehensive Air Quality Plan Approval, which incorporated conditions required by the final decisions resulting from the adjudicatory hearing and found that Massachusetts Environmental Justice Policy does not apply because the anticipated emissions would not exceed emission thresholds. Massachusetts DEP, Air Quality Plan Approval (Aug. 26, 2019), <https://www.mass.gov/doc/air-quality-plan-approval-august-2019/download>.
- On June 3, 2020, the U.S. Court of Appeals for the First Circuit affirmed in part Massachusetts DEP's Air Quality Plan Approval, including its assessment of environmental justice. *See Town of Weymouth, Massachusetts v. Mass. Dep't of Environmental Protection*, 961 F.3d 34, 54-55 (1st Cir. 2020), *amended*, 973 F.3d 143 (1st Cir. 2020).

UNITED STATES OF AMERICA  
FEDERAL ENERGY REGULATORY COMMISSION

Algonquin Gas Transmission, LLC  
Maritimes & Northeast Pipeline, LLC

Docket No. CP16-9-012

(Issued February 18, 2021)

CHRISTIE, Commissioner, *dissenting*:

1. What the majority does in this order is inconsistent with the purpose and principle behind a future-looking review of certification applications. Today, the Commission makes a foray into retroactively changing the rules long after the fact: long after construction was begun and long after investors committed significant funds, as described below, to a project. Today's capricious action violates the most basic standards of regulatory due process and regulatory finality, both of which are absolutely necessary to balance appropriate regulatory protections for people who live in geographic proximity to infrastructure projects with regulatory certainty for those who are building and financing needed infrastructure to provide vital services to consumers and create jobs for Americans.

2. On January 25, 2017 – more than four years ago – this Commission authorized Applicants to construct and operate certain pipeline and compression facilities in New York, Connecticut, and Massachusetts (Atlantic Bridge Project), and, in so doing, found that the “*public convenience and necessity require approval and certification of the Atlantic Bridge Project under section 7 of the NGA,*” subject to certain conditions.<sup>1</sup> In reliance on the issuance of that certificate of public convenience and necessity (CPCN), investors committed hundreds of millions of dollars to construct the project.<sup>2</sup> Construction took place and on September 24, 2020, Commission staff issued a delegated letter order authorizing the remaining facilities associated with the Atlantic Bridge

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<sup>1</sup> *Algonquin Gas Transmission, LLC*, 158 FERC ¶ 61,061 at P 31 (Certificate Order) (emphasis added), *order on reh'g*, 161 FERC ¶ 61,255 (2017) (Certificate Rehearing Order), *aff'd sub nom., Town of Weymouth v. FERC*, No. 17-1135, 2018 WL 6921213, at \*1 (D.C. Cir. Dec. 27, 2018) (unpublished opinion).

<sup>2</sup> The Certificate Order states that, at the time of the applications, Applicants estimated the cost of the Atlantic Bridge Project to be \$451,791,440. Certificate Order at P 10 (footnote omitted).

Project be placed into service, including the Weymouth Compressor Station in Norfolk, Massachusetts, and the Maritimes Westbrook Metering and Regulator Station in Cumberland, Maine, and finding that “Algonquin and Maritimes have adequately stabilized areas disturbed by construction and that restoration is proceeding satisfactorily.”<sup>3</sup>

3. Now, four years after finding *public convenience and necessity require approval and certification of the Atlantic Bridge Project* and inviting investors to commit substantial funds to build it, and without recognizing the request for rehearing was denied by operation of law, the majority literally invites opponents of the project to re-litigate the core question of whether the project should even have been built. The majority’s order unquestionably raises the specter of shutting down this completed and functioning project even permanently, although it offers no discussion as to how it would do so under the law.

4. The majority’s decision is apparently – it is unclear – based on an alleged safety issue with a compressor station that is no longer under this Commission’s jurisdiction, but is rather under that of another federal agency.<sup>4</sup> The Certificate Rehearing Order states that the U.S. Department of Transportation’s Pipeline and Hazardous Materials Safety Administration (PHMSA) is the agency charged with developing safety regulations for the design and operation of natural gas pipeline facilities and enforces compliance with these regulations. To compound the Kafkaesque quality of the Commission’s action, PHMSA has already investigated and given the compressor facility a temporary green light to operate.<sup>5</sup>

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<sup>3</sup> *Algonquin Gas Transmission, LLC*, Docket No. CP16-9-000, at 1 (Sep. 24, 2020) (delegated order) (Authorization Order). Subsequent to the Authorization Order, on October 23, 2020, the Fore River Residents Against the Compressor Station (Fore River Residents), the City of Quincy, Massachusetts, Weymouth Councilor Rebecca Haugh, Michael Hayden, and Food and Water Watch filed what was styled as request for rehearing of the Authorization Order.

<sup>4</sup> The Commission’s action *may* also be based on an argument in the request for rehearing that has already been denied by operation of law, that the Weymouth Compressor Station poses a threat to neighboring communities during the COVID-19 pandemic and represents a change in core circumstances that requires the Commission to re-open the record in this proceeding. Such an argument appears to be another attempt to re-open and re-litigate the original certificate proceeding with a goal of overturning that decision and shutting the project down permanently.

<sup>5</sup> See *In the Matter of Algonquin Gas Transmission, LLC*, Corrective Action Order, CPF No. 1-2020-014-CAO, Dep’t of Transp. (Oct. 2020) (prohibiting Algonquin from operating the Weymouth Compressor station following two unplanned emergency

5. Fairness and due process in the regulatory consideration of project certification applications means litigating all relevant issues during the original proceeding, providing for robust public participation, and then issuing a decision well-grounded in law and fact. Then out of fairness to all concerned, the regulatory body should stand behind its decision. Today's decision violates this basic standard.

6. Instead, today's order creates more questions than it answers and leaves uncertainty only in its wake. Nothing in today's order suggests that the Commission has not left open the possibility that it will shut down this project. As a result, today's order may, regrettably, impact investment in *all* infrastructure projects making them less appealing to engage in by those who normally seek to build the projects and harder to finance or, at the very least, more expensive to finance due to the increased risk created by this specter of uncertainty.

7. Mark Twain said the art of prophecy is very difficult, especially with respect to the future; however, I suspect that the use of the legal weapons of unending litigation and collateral attacks against infrastructure projects long after they have been approved, as is enabled by today's order, will not be limited to natural gas projects, even though they are today's primary target. Campaigns of unending legal warfare may well be used one day against other types of infrastructure projects, including those the majority may well want to promote.

For these reasons, I respectfully dissent.

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Mark C. Christie  
Commissioner

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shutdowns on September 11 and 30, 2020); *see also In the Matter of Algonquin Gas Transmission, LLC*, Region Approves Restart Plan, CPF No. 1-2020-014-CAO, Dep't of Transp. (Nov. 2020) (approving Algonquin's restart plan for the Weymouth Compressor Station at 80 percent capacity), January 22, 2021 Letter from PHMSA to Enbridge, CPF 1-2020-014-CAO (permits the temporary removal of the pressure restriction and approves the temporary operation of the compressor units in the station). As a result of the January 22, 2021 PHMSA Letter, Applicants filed a Notice of Commencement of Service with the Commission in this docket on January 25, 2021. Even if additional measures are ordered by PHMSA, that would be under PHMSA's authority not the Commission's.



# **Health Effects Associated with Stack Chemical Emissions from NYS Natural Gas Compressor Stations: 2008-2014**

A Technical Report Prepared for the Southwest Pennsylvania Environmental Health Project underwritten by the Park Foundation

12 October 2017

P.N. Russo & D.O. Carpenter

## **Institute for Health and the Environment**

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## From Raina Rippel, Environmental Health Project Director

*Health Effects Associated with Chemical Emissions from NYS Natural Gas Compressor stations: 2008-2014* is a report on the chemical and particulate emissions of eighteen compressor stations in New York State (NYS), based on what companies are required to report to NY Department of Environmental Conservation (DEC) and National Emissions Inventory (NEI) of the U.S. Environmental Protection Agency (EPA). In addition, it presents the potential health effects of the 70 chemicals catalogued. The Report is aimed primarily at New York state and local governmental officials and administrators in order to raise their awareness of the size and scope of the air emissions generated by unconventional natural gas compressor stations. Many public officials are in positions to make decisions about siting compressor stations so that people who live nearby are relatively safe. EHP and IHE believe that the data provided in this Report should play a central role in that decision-making.

Secondarily, the Report is intended to inform communities, NGOs, and health care professionals about emissions from the eighteen compressor stations in NYS. It is not, however, designed to help quantify the risk of any particular community. Here's the reason why:

The presence of a chemical with disease-causing properties does not necessarily result in disease in any one individual. For instance, some chemicals are only harmful beyond a certain level of exposure (often referred to as a "dose"). Some are more likely to be harmful if exposure is repeated before the body has had the opportunity to clear the preceding exposure. Others are more likely to cause disease or symptoms in vulnerable populations. That said, some of the chemicals reported by the companies will likely produce health effects in individuals living, working or going to school near the compressor stations. (David Brown, ScD, EHP Toxicologist and Public Health Scientist)

There are important reasons, however, for communities, NGOs and health care professionals to make use of this report. These 18 compressor stations are the seventh largest "Point source" of air pollution in New York State, and emit a large array of chemicals, in conjunction with fine and ultrafine particles. Some communities will experience intense exposures and these exposures will be to multiple contaminants simultaneously. Researchers do not know the combined effects of the possible mix of chemicals, but it is an important feature of the UNGD process (Unconventional Natural Gas Development). Health care professionals can use the Report, and specifically Chapter 3, to identify the actual health conditions produced by the reported chemicals.

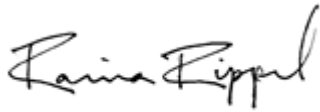
By volume, the largest emissions are NO<sub>2</sub>, CO, VOCs, Formaldehyde, and Particulate Matter. Exposure to these chemicals can cause respiratory and cardiovascular diseases, neurological and developmental diseases and cancer. The New York State Bureau of Vital Statistics reports that, as of 2012, the leading causes of death were heart disease and cancer, followed by chronic lower respiratory disease. What we know from our work and that of researchers across the country is that symptoms associated with UNGD exposure and reported by residents include respiratory, cardiovascular and neurological health effects. Thus, exposure to emissions from these compressor stations may contribute to these prevalent diseases.

**What does this data mean for impacted residents and communities?**

A question often asked of us by residents living near UNGD sites is “how will these emissions affect my health or my community?” This report shows that every compressor station routinely releases large volumes of chemicals associated a variety of diseases and disorders. The level of risk to any individual or community from a compressor station can be estimated by applying specific statistical analyses. The analysis should include modeling the reported chemical emissions from the compressor station based on local weather patterns. The exposure levels at varying distances from the site, and the duration of extreme exposures can then be estimated. The largest emissions by volume are likely to produce the greatest exposures and consequent health impacts.

EHP expects that this compilation of readily available information will be helpful in assuring the health of residents near compressor stations. We welcome feedback, questions and comments on the use of this report.

In good health,

A handwritten signature in black ink, reading "Raina Rippel". The signature is written in a cursive, flowing style.

Raina Rippel, Director

Southwest Pennsylvania Environmental Health Project

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# Purpose of the Report

## Is it Safe?

People living in communities where natural gas compressor stations are sited or are proposed, have repeatedly asked: “Is it safe?” This study represents an attempt to answer that crucial question.

### Industry’s answer

Each of the compressor stations operating in New York State (NYS) have been approved by the state’s Department of Environmental Conservation (DEC) based on the conclusion that they comply with all federal and state air quality requirements.

When members of the public or local officials question the potential health effects of compressor station pollution, invariably the response from industry, EPA, DEC and DOH is that “all legal requirements have been met” -- the clear **implication** being that if these “legal requirements” have been met, there is no reason to be concerned about adverse health effects.

For example, in a public statement issued by Dominion Transmission concerning its New Market Project, it states: “The FERC approved New Market on April 28, 2016 after 23 months of evaluating all environmental, health and safety concerns associated with the project.” Dominion poses the question, “What will be the environmental and public health concerns?” And answers:

Any emissions from the compressor station will comply with all air quality requirements, which are established to protect the public health, safety and welfare. We would not operate the compressor station if we could not operate it according to stringent air quality regulations.

Ensuring compliance with environmental requirements falls either to the Environmental Protection Agency (EPA) or state environmental agencies (states by delegation), depending on the specific permit and rule. (Dominion 2016)

At best, such fact free statements are ill-informed.

In this connection, the three most essential points are these:

First, federal and state environmental laws and regulations are at best designed to protect the general health of regional populations and often fail to protect any single group of locally exposed persons. The “stringent air quality regulations” that Dominion refers to are those established by EPA for the purpose of controlling regional levels of pollution.

Second, “controlling regional levels of air pollution” is not equivalent to ensuring that the air in a region is free of pollutants that cause illness and early death. The air quality in any given region of the state may fully meet all federal and state air standards even though it is saturated with hundreds of dangerous chemicals and tens of thousands of pounds more are added each and every day.

Third, industry’s assurances are hollow. They provide neither the empirical information nor the theoretical framework necessary for the public and local and state officials need to begin to understand the potential adverse health effects of existing and proposed compressor stations.

## Our answer

Our answer to the question, “Is it safe?” is fundamentally different from that of industry and federal and state government.

***In our opinion, the routine emissions surrounding the operation of natural gas compressor stations in New York State (NYS) increase the risk for most major categories of human disease in the state but especially in the communities where they are sited.***

This conclusion is based on (1) an empirical assessment of the volume and content of contaminants released by 18 compressor stations operating under Title V of Clean Air Act in NYS (2) a comprehensive review of peer-reviewed scholarship associating the chemical constituents of those emissions with known and suspected human diseases.

## What this study shows

The air in NY contains chemicals that are linked to 19 of 20 major categories of human disease.

There are 56 operational natural gas compressor stations in New York. Based on data collected by the natural gas industry and reported by EPA, we show that in **a 7-year period 18 of these sites released an estimated 40.2 million toxic pollutants made up of 70 different chemicals**. These 70 chemicals are also linked to 19 of 20 major categories of human disease.

Adding 40.2 million pounds of 70 contaminants to air already contaminated makes the air we breathe **more contaminated** and, by extension, increases the potential for human disease. It is, we think, as simple as that.

A few specific examples:

**Cancer and air pollution:** The International Agency for Research on Cancer, a division of the World Health Organization, is the most authoritative source of information on the effects of chemical and radiologic contamination on human health. In 2016, the Agency released what is perhaps its most important finding: “Outdoor air pollution is carcinogenic to humans.” After reviewing the extensive peer-reviewed literature IARC concluded that (1) in industrialized countries simply breathing the air increases the risk of cancer compared to breathing the uncontaminated air or relatively uncontaminated air and (2) the responsible pollutants are largely the result of human activity, that occur in both rural and urban areas from many different sources.

Although there are hundreds of sources of outdoor air pollution, the source categories that are the largest contributors to most air pollutants in many locations are: vehicle emissions; stationary power generation; other industrial and agricultural emissions; residential heating and cooking; re-emission from terrestrial and aquatic surfaces; the manufacturing, distribution, and use of chemicals; and natural processes. (IARC 2016)

Cancer is the second leading cause of death in the United States and the second leading cause of death in NYS, and in the near future it is very likely to surpass cardiovascular disease, currently the leading cause of death. The 40.2 million pounds of chemicals released by the state’s compressor stations from 2008 to 2014 includes 9.5 million pounds associated with cancer. Of this amount, 7.9 million pounds (83%) is made up of 20 chemicals classified as “known human carcinogens” by one or more authoritative governmental authorities. These cancers are known to cause cancers of the digestive tract (biliary tract, hepatocellular and liver), respiratory tract (lung, nasal cavity and paranasal sinuses), male genital organs (prostate), urinary tract (bladder and kidney), and hemolymphatic organs (acute myeloid leukemia/acute non-lymphocytic leukemia). Adding 5.7 million pounds of carcinogens to the state’s air each year can only increase the risk of cancer.

**Birth defects:** Fifty-seven of the 70 chemicals releases are associated with congenital malformation and deformations, including nervous system, deformations: eye, ear, face and neck, and circulatory system malformations and deformations.

**Reproductive disorders:** Thirty-seven chemicals are associated with diseases of the pelvis, genitals and breasts that affect reproduction. For males, this includes: epididymis, low hormone levels, male impotence, reduced fertility, semen (chemical contamination of semen, low amount of semen and low number of swimming semen), seminal vesicle injury, sperm (abnormalities, irregular shape and low number), and sterility. In women these chemicals are associated with diseases of female pelvic organs as well as noninflammatory disorders of female genital tract--both primary infertility (infertility without any previous pregnancy) and secondary infertility (fertility problems occurring in a couple that has conceived on their own and had a child in the past), as well as cervical erosion, effects on the ovaries (damage, weight changes and unspecified effects), menstrual problems including dysmenorrhea, endometrial stromal polyps, and vagina effects.

**Circulatory system disease:** Cardiovascular disease is the leading cause of death in the United States. In a 7-year period New York's compressor stations released 16 million pounds of cardiovascular toxicants. Compressor station pollutants are linked to hypertensive disease, chronic rheumatic heart diseases, cardiac arrhythmia, heart weight change, increased cardiovascular mortality, acute pulmonary edema, diseases of arteries, arterioles and capillaries (blood vessel changes and regional, general arteriolar or venous dilation).

This information has not previously been reported.

Without hesitation, we can say not only that the volume and known health effects of these pollutants increase the **risk of disease**, but that they will result **in actual illness**. However, given the limited scope of this study, we cannot quantify the nature or extent of potential increased risk.

### The public's right-to-know

In our opinion, the public has a right-to-know the basic facts surrounding the operation of a compressor station, including the number and volume of pollutants and their known or suspected health effects. But beyond this basic information, the public also has the right to expect the opportunity to review a scientifically sound study of the potential health impacts of a compressor station **before** it is built.

### In the State of New York

Neither industry nor government has provided the public with basic data about the extent of compressor station pollution or its likely health effects. In communities where new compressor stations have been planned, the public has asked the industry and state agencies to provide them with "health impact statements," "risk assessments" or "cost-benefit analysis." To public these terms are essentially synonymous, but they represent very different types of studies to the public health community.

In this connection, we would make three points. First and foremost, "health impact statements," "risk assessments" or "cost-benefit analysis" as conducted by federal and state agencies or industry and its paid consultants (a) rarely predict the likely qualitative impact of pollution, as any number of investigators have noted, (b) seldom present relevant information to the public in coherent fashion, or (c) never present a morally persuasive argument why some populations should be subjected against their will to greater levels of pollution with its attendant risk than other (usually more affluent) populations. Generally, the definition of "acceptable risk" adopted by industry and government is one death per 1 million people who are exposed, though various industries have

sought (in some cases successfully) to lower the accepted standard to one death per 100,000 exposed—a tenfold increase.

Second, notwithstanding our skepticism of the value or “health impact statements” and “risk assessments” broadly defined, it is worth noting that we could not find a single existing or proposed compressor station in NYS that has been the subject of such reviews by industry, NYS’s DEC or Department of Health (DOH). Such analyses by the natural gas industry are not those of disinterested investigators. Much the same could be said of FERC, which is widely viewed as a “captive agency,” i.e., an agency effectively controlled by the industry it is responsible for regulating. It is not the responsibility of DEC to perform health-based analyses, nor does EPA routinely require health impact statements. This only leaves the DOH which, unfortunately, is missing in action.

And finally, the lack of information about the potential adverse health effects of compressor stations on local communities has played no role in preventing their construction or expansion.

### **This study’s rationale**

All industrial development involves tradeoffs between short- and long-term economic benefits (real and perceived) and potential harm to human health and the environment.

To date, the criticisms of the expansion of the natural gas industry in the U.S. have focused primarily on four concerns.

**Natural gas versus coal:** First: exaggerated claims for the advantages of using natural gas compared to coal in terms of global warming. As a rule, burning natural gas to produce electricity produces half as much carbon monoxide as coal. While natural gas combustion produces fewer greenhouse gases than coal at the point of combustion, when the chemicals released in the production, transportation and distribution of natural gas are taken into account, the comparative advantage of natural gas are far less clear cut (Dove 2016, Grossman 2015, Moskowitz 2015, UCS, Zielinski S. 2014). Fugitive emissions of methane, roughly 30 times more potent as a heat-trapping gas than carbon dioxide, are of particular concern.

**Climate change:** Second: the more profound and most important argument that whatever natural gas’s relative advantage compared to coal, the planet cannot sustain continued reliance on fossil fuels. In this connection, perhaps most startling is a recent study showing that climate sensitivity is nonlinear. Based on past, current and probable future greenhouse gas emissions, the Earth could heat up as much as 6°C (almost 11°F) in a single lifetime. (Friedrich 2016). Scientists estimate the range of sea rise from 1 to 6 meters (1.3 to 20 feet). The Australian Earth and paleoclimate scientist, Andrew Glikson, describes the likely future: “The consequences of open ended rise in atmospheric CO<sub>2</sub> are manifest in the geological record. . . At 460 ppm CO<sub>2</sub>-equivalent, the climate is tracking close to the upper stability limit of the Antarctic ice sheet, defined at approximately 500 ppm. Once transcended, mitigation measures would hardly be able to re-form the cryosphere. According to Joachim Schellnhuber, Director of the Potsdam Climate Impacts Institute and advisor to the German government: ‘We’re simply talking about the very life support system of this planet.’. . . Humans cannot argue with the physics and chemistry of the atmosphere. What is needed are urgent measures including: Deep cuts in carbon emissions; Parallel Fast track transformation to non-polluting energy utilities – solar, solar-thermal, wind, tide, geothermal, hot rocks; Global reforestation and re-vegetation campaigns, including application of biochar. The alternative does not bear contemplation.” (Glikson 2010) Twenty-two scientists writing in *Nature* depict the situation with equal starkness: “[T]he next few decades offer a brief window of opportunity to minimize [but not prevent] large-scale and potentially catastrophic climate change that will extend longer than the entire history of human civilization thus far. “ (Clark et al. 2016).

In a recent paper reviewing 40 years of climate data and conclusions drawn from that data, researchers found that scientists have underestimated the likelihood of dangerous to catastrophic climate changes.

The historic Paris Agreement calls for limiting global temperature rise to “well below 2 °C.” Because of uncertainties in emission scenarios, climate, and carbon cycle feedback, we interpret the Paris Agreement in terms of three climate risk categories and bring in considerations of low-probability (5%) high-impact (LPHI) warming in addition to the central (~50% probability) value. The current risk category of dangerous warming is extended to more categories, which are defined by us here as follows: >1.5 °C as dangerous; >3 °C as catastrophic; and >5 °C as unknown, implying beyond catastrophic, including existential threats. With unchecked emissions, the central warming can reach the dangerous level within three decades, with the LPHI warming becoming catastrophic by 2050. (Xu and Ramanathan 2017)

**Damage to local environments:** Third: the damage fracking does to local environments in the form of air, water and soil contamination. In February 2014, there were an estimated 1.1 million active oil and gas wells in the U.S. In August 2015, the number of active and dry holes was estimated to be 1.7 million (Kelso 2015). To frack a single well requires up to 5 million gallons of water, and wells can be fracked multiple times—18 times or more. If we assume that each of the 1.1 million active wells were fracked once and used 5 million gallons of water, it means ~12 trillion gallons of water has been contaminated with hundreds of toxic chemicals. This waste water is either collected in surface ponds (many of which are unlined) where it inevitably contaminates surface and groundwater and the air when it vaporizes, or else it is injected underground where it contaminates groundwater and the environment for decades if not centuries.

**Health impacts of fracking:** Fourth: the health impacts of fracking on local communities. A study by the Wall Street Journal examining fracking operations in 11 of the biggest energy producing states found that, “At least 15.3 million Americans live within a mile of a well that has been drilled since 2000. That is more people than live in Michigan or New York City.” (WSJ)

**Health impacts of transportation of natural gas:** To these concerns, we would add a fifth: the health and environmental damage caused by the transportation of natural gas. To our knowledge, this subject has not been previously addressed.

Much of the environmental damage caused by the natural gas industry is largely unseen. The physical damage to local environments where fracking occurs is in part obvious to anyone who cares to look. Roads are cut through forests and hillsides, large bodies of wastewater are collected in ponds and lagoons, dust from diesel trucks and construction equipment is constant as is the noise they make in what were once relatively quiet rural places. But the chemical pollution associated with the extraction, refinement, transportation, storage and combustion of natural gas for energy and heat is largely invisible. Industry advertisements tout natural gas as the “clean alternative” to coal--and in some ways, it is, though it's comparative advantages are wildly exaggerated. But part of what makes natural gas “clean” is that the public can't see the pollution it causes. One reason most people are unaware and unconcerned by pollution associated with unconventional gas development (UGD) is because most don't live in areas where fracking takes place or compressor stations are sited. But more fundamentally we're unconcerned by the UGD pollution because for the most part it's invisible as are its impacts on the public's health--realities which are denied both by the industry and its supporters in and out of government.

The task we set for ourselves here, is to show what the eye can't see: the volume of pollution associated with the transportation of natural gas in New York and its potential to harm human health. The potential health impacts of the large volumes of pollutants generated by natural gas compressor

stations have not been addressed, let alone answered, by those arguing for their construction and expansion.

This report has been prepared to provide the data necessary to understand and evaluate the potential immediate and long-term health outcomes connected with the pollution generated by the routine operations of natural gas compressor stations in New York State (NYS) by examining the actual volume of airborne releases generated by 18 plants and the diseases associated with the chemical pollutants they contain. It is directed at 4 primary audiences:

- Communities with existing compressor stations (to help them recognize the potential adverse health outcomes associated with their continued operation).
- Communities where compressor stations are proposed (to help them understand the potential health threats their construction and operation will introduce).
- Physicians and health practitioners in affected communities.
- Public officials responsible for protecting the safety and health of the public.

There are, by our preliminary estimates, more than 2,000 compressor stations operating under Title V permits in the U.S. (and an equal or greater number of non-Title V stations). Given the volume and toxicity of chemicals released by the 18 Title V facilities we studied, natural gas compressor stations represent a significant national public health problem.

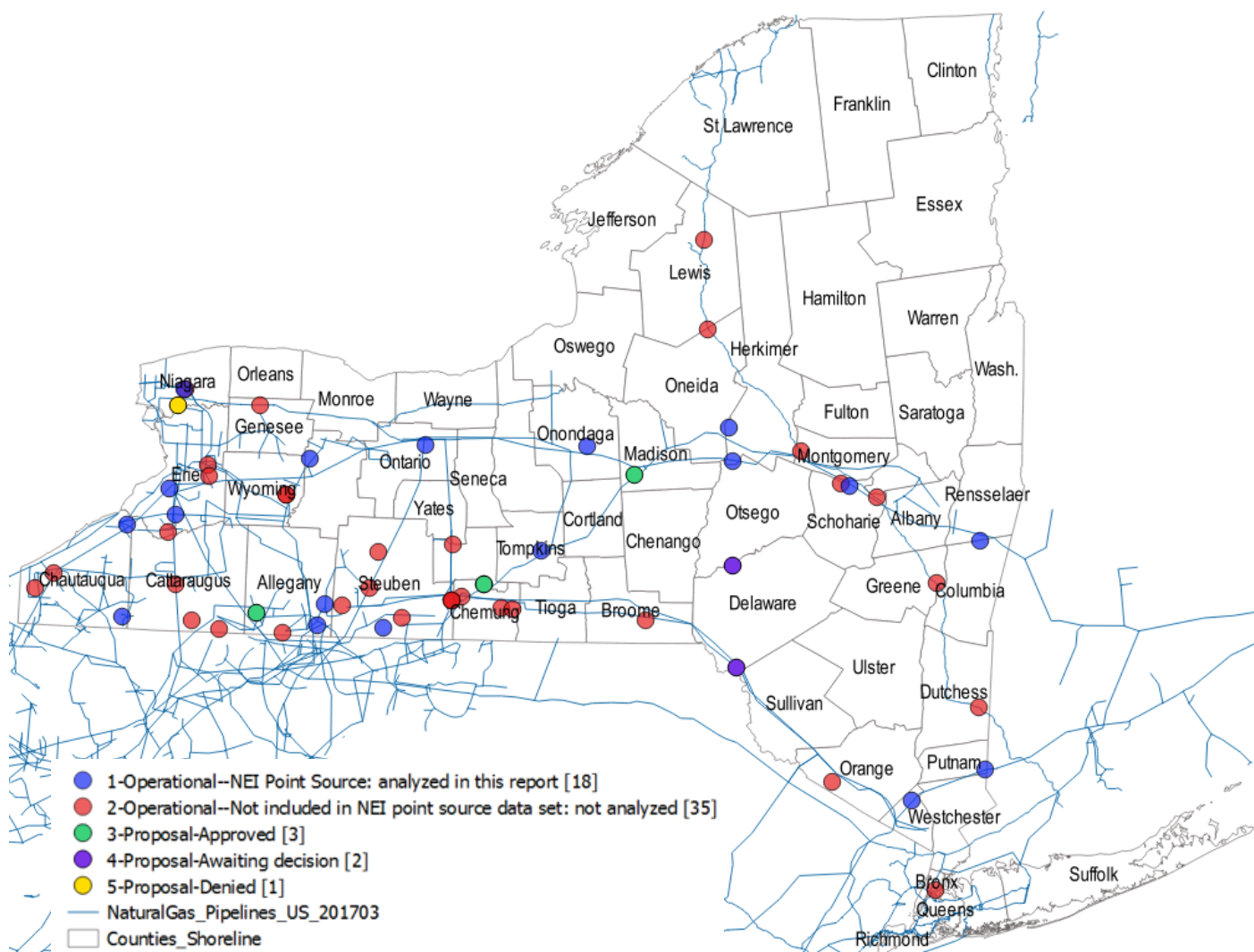


# Executive Summary

## 1. Natural Gas Compressor Stations in NYS

This report analyzes the emissions data for 18 as reported to the National Emissions Inventory (NEI) of the U.S. Environmental Protection Agency (EPA) as point sources of air pollution for the period 2008 to 2014. For the period 2008 to 2014, a total of 58 compressor stations were operational or seeking state and federal approval: operational (54), approved (3), awaiting approval (2) permit denied (1). National Emissions Inventory data is available for 18 of the state's 54-operational natural gas compressor stations. Four operational stations are seeking significant modifications requiring DEC approval. Eighteen of the state's 54 operational compressor stations are classified as "major polluters" and operate under Title V of the Clean Air Act (CAA). Pollution data for these sites is part of NEI's point source data set. These 18 sites are the subject of this analysis. The remaining 37 operational stations are permitted as a NYS "Air State Facility."

GHS emissions data is only available for 8 of the 18 compressor stations for which NEI data is available.



## 2. Total Releases: 40.2 million pounds

For the period 2008 to 2014, an estimated 1.5 billion pounds of point sources of air pollution were reported to NEI by facilities in NYS.

**Releases from the state's national gas compressor stations accounted for approximately 40.2 million pounds or 2.7% of total on-site pollution reported to NEI.**

**This amounts to an annual average of 5.7 million pounds or 478,485 pounds per month, 15,731 pounds per day, 655 pounds per hour.**

**If each of the state's 19.8 million residents were given their fair share, each would receive a little more than 2 pounds over 7 years.**

**Analyzing emissions by each site's 5-digit NAICS code, based on NEI data national gas compressor stations were the 6<sup>th</sup> largest point (stationary) source of air-pollution in NYS.** (If we were to include other sources of air pollution associated with natural gas not included in NAICS 48621, the volume and percentage would be significantly higher. By far the point source of air pollution in NYS is electric power generation (NAICS code 22111) which accounts for approximately 42.3% of the state total. A significant part of this amount is generated by burning natural gas.)

### 3. Total Releases by Chemical: 70

NYS's compressor stations reported releasing 70 individual chemicals or chemicals categories in the period 2008 to 2011 totaling approximately 40 million pounds. The volume of releases varies tremendously. Twelve chemicals have reported releases of less than one pound.

**The largest pollutant, nitrogen oxides, had releases totaling 18.1 million pounds or 45.2% of the aggregate.** Carbon monoxide ranked second (12.4 million pounds or 31%), followed by volatile organic compounds as a group (4.9 million pounds or 12.3%), formaldehyde (1,309,336 pounds or 3.27%), and PM10 Primary (Filt + Cond) (1,259,744 pounds or 3.15%). These five chemicals accounted for 95% of the total.

### 4. Total Releases by Compressor Stations: 18

All 18 compressor stations reporting to NEI reported toxic emissions which totaled 40,192,733 pounds.

The volume of total pollution by station varied widely. The lowest amount reported was one-quarter of a million pounds--a still considerable sum.

**The largest release was from Tennessee Gas Pipeline Company's (TGPC) Compressor Station 245 in Herkimer County: 10.5 million pounds or slightly more than one-quarter (26.1%) of the state total.** TGPC's Compressor Station 229 & TEG Dehydration Facility in Erie County ranked second (5.1 million pounds or 12.8%), followed by TGPC's Compressor Station 249 in Schoharie County (4.3 million pounds or 10.8%).

These three facilities accounted for 19.9 million pounds or slightly less than one-half (49.54%) of all releases.

The top 5 stations accounted for 25.3 million pounds or slightly less (63.1%) than two-thirds of the state total.

#### Total Compressor Station Estimated Releases by Station: 2008-2014

Rank	Facility	County	Pounds	①	②	③	④	⑤	⑥	⑦	⑧	⑨	⑩	⑪
1	TGPC CS 245	Herkimer	10,465,389	①	②	③	④	⑤	⑥	⑦	⑧	⑨	⑩	⑪
2	TGPC 229 & TEG DF	Erie	5,124,427	①	②	③	④	⑤						
3	TGPC CS 249	Schoharie	4,323,285	①	②	③	④							
4	TGPC CS 241	Onondaga	3,039,661	①	②	③								
5	TGPC CS 254	Columbia	2,393,661	①	②	④								
6	TGPC CS 237	Ontario	2,298,394	①	②	③								
7	AGT Stony Point CS	Rockland	2,013,478	①	②									
8	NFGSC Concord CS	Erie	1,733,171	①	⑦									
9	AGT Southeast CS	Putnam	1,688,815	①	⑦									
10	NFGSC Beech Hill CS	Allegany	1,387,592	①	④									
11	NFGSC Independ. CS	Allegany	1,353,931	①	③									
12	TGPC CS 224	Chautauqua	1,146,797	①	①									
13	DTI Woodhull Station	Steuben	829,223	⑧										
14	DTI Borger CS	Tompkins	780,159	⑧										
15	NFGSC Nashville CS	Chautauqua	622,791	⑥										
16	TGPC CS 230-C	Niagara	485,610	⑤										
17	DTI Utica Station	Herkimer	281,369	③										
18	TGPC CS 233	Livingston	224,978	②										

40,192,733

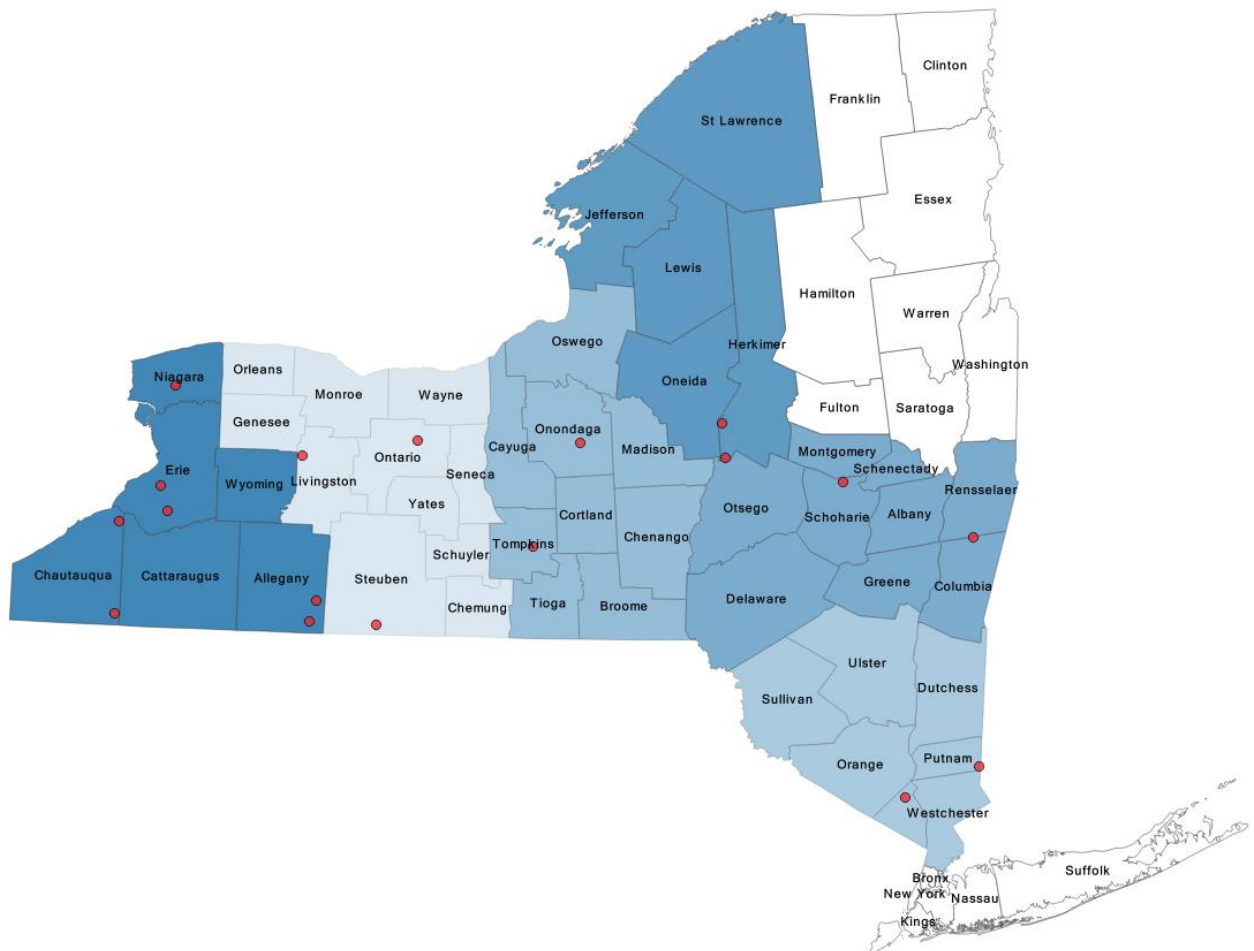
## 5. Total Releases by DEC Regions: 6

NYS DEC divides the state's 62 counties into 9 regions. The 18 operational compressor stations reporting to NEI are in 6 of NYS's 9 DEC regions encompassing 46 counties: 3-Lower Hudson Valley: 2, 4-Capital Region/Northern Catskills: 2, 6-Western Adirondacks/Eastern, Lake Ontario: 2, 7-Central New York: 2, 8-Western Finger Lakes: 3, 9-Western New York: 7.

**Region 9, Western New York, ranked first with an estimated 11.7 million pounds (29.5%), closely followed by Region 2 (10.8 million pounds or 26.7%).** Region 4 reported 6.7 million pounds (16.7%). These three regions accounted for nearly three-fourths (73%) of the state total.

### Total Compressor Station Releases by DEC Region: 2008-2014

Rank	DEC Region	Pounds											
1	9: Western New York	11,646,722	①	②	③	④	⑤	⑥	⑦	⑧	⑨	⑩	⑪
2	6: W. Adirondacks / E. Lake Ontario	10,746,758	①	②	③	④	⑤	⑥	⑦	⑧	⑨	⑩	⑪
3	4: Capital Region / N. Catskills	6,716,946	①	②	③	④	⑤	⑥	⑦				
4	7: Central New York	3,819,820	①	②	③								
5	3: Lower Hudson Valley	3,702,293	①	②	③								
6	8: Western Finger Lakes	3,352,596	①	②	③								

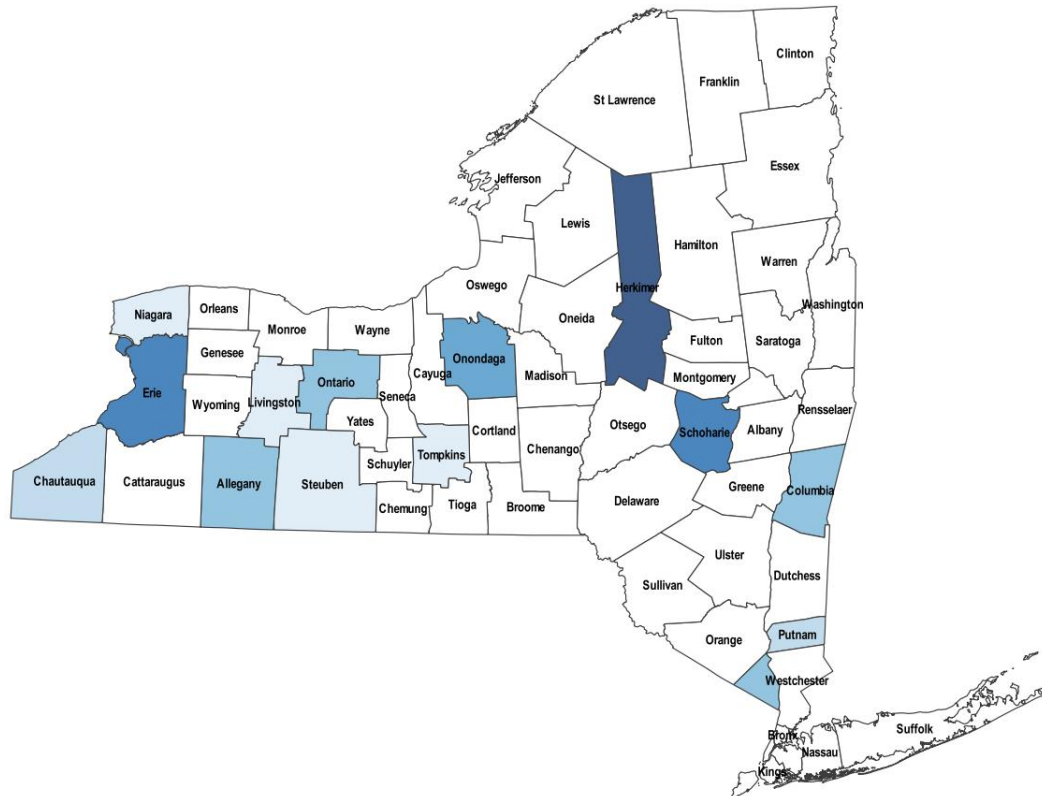


## 6. Total Releases by County: 14

The 18 natural gas compressor stations reporting to NEI are in 14 of NY's 62 counties: Allegany, Chautauqua, Columbia, Erie, Herkimer, Livingston, Niagara, Onondaga, Ontario, Putnam, Rockland, Schoharie, Steuben, and Tompkins. **Herkimer County ranked first with 10.7 million pounds or slightly more than one-fifth of the total (26.7%),** followed by Erie County with 6.9 million pounds (17.1%), and Schoharie with 4.3 million pounds (10.8%). These three counties accounted for slightly more than one-half (54.6%) of all releases: 22 million pounds. The top five counties were responsible for 27.7 million pounds or slightly more than two-thirds (69%) of the aggregate.

### Total Compressor Station Releases by County: 2008-2014

Rank	County	DEC Region	Pounds	1	2	3	4	5	6	7	8	9	10	7
1	Herkimer	6: W Adirondacks/E. L Ontario	10,746,757	1	2	3	4	5	6	7	8	9	10	7
2	Erie	9: Western New York	6,857,598	1	2	3	4	5	6	9				
3	Schoharie	4: Capital Region/N. Catskills	4,323,285	1	2	3	4	3						
4	Onondaga	7: Central New York	3,039,661	1	2	3								
5	Allegany	9: Western New York	2,741,523	1	2	7								
6	Columbia	4: Capital Region/N. Catskills	2,393,660	1	2	4								
7	Ontario	8: Western Finger Lakes	2,298,394	1	2	3								
8	Rockland	3: Lower Hudson Valley	2,013,478	1	2									
9	Putnam	3: Lower Hudson Valley	1,688,814	1	7									
10	Chautauqua	9: Western New York	1,561,991	1	6									
11	Steuben	8: Western Finger Lakes	829,223	8										
12	Tompkins	7: Central New York	780,159	8										
13	Niagara	9: Western New York	485,609	5										
14	Livingston	8: Western Finger Lakes	224,978	2										



## 7. Total Releases by Zip Codes: 18

The 18 operational compressor stations reporting to NEI are in 18 zip codes. **Compressor stations were responsible for 92% of all recorded industrial emissions in their respective zip codes.** In 14 of these zip codes, emissions from natural gas compressor stations were the **only** point source of air pollution reported by NEI.

## 8. Total Releases per Square Mile

The distance and direction pollution travels from each natural gas compressor station on any given day (or any hour) is dependent on many factors, including: the height of the stack, chemical composition of the fuel, chemical composition of emissions, meteorological conditions (wind speed and direction, atmospheric stability and cloud cover), as well as local and regional geographical features.

Absent an independent analysis, most epidemiological studies assume that if stacks are short (which is the case for NYS compressor stations), on a typical day most air pollution that is inhaled has traveled a relatively short distance from a plant—something on the order of less than 10 miles—recognizing that on certain days pollution from a single plant can travel hundreds or even thousands of miles before it reaches the ground and is inhaled.

If we assume that the 10.5 million pounds of toxic releases generated by the largest polluter, TGPC's Compressor Station 245, fell within a 1-mile radius of the plant (a 2-mile diameter circle of 3.14 square miles), it amounts to 3.3 million pounds per square mile or approximately 0.12 pounds per square foot.

If, instead, we assume it fell within 1.5-mile radius of the plant (a 3-mile diameter circle of 7.07 square miles), it amounts to 1.5 million pounds per square mile.



## 9. Total Releases: Circular Area Population Profiles

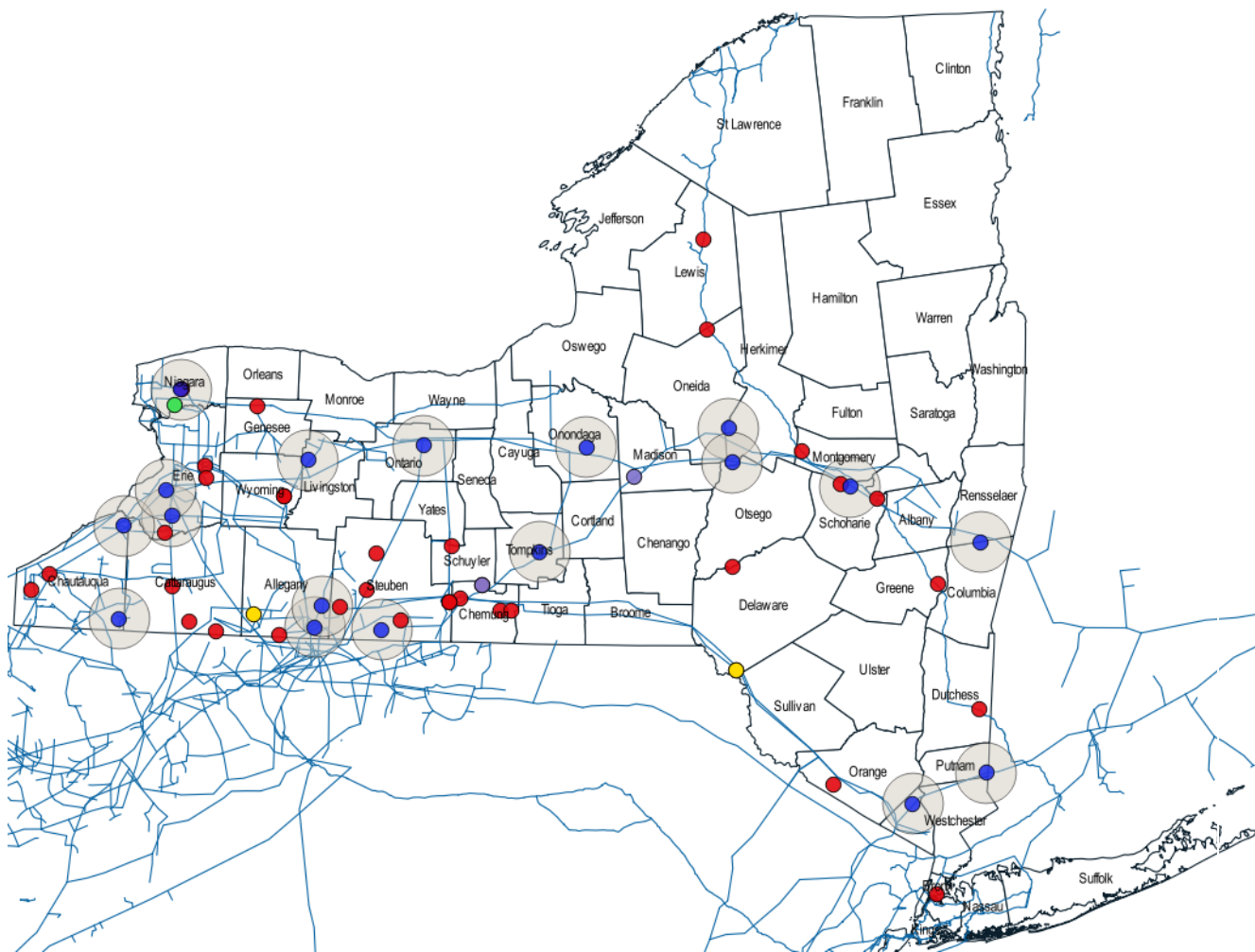
The number of people in New York State exposed to air pollution from natural gas compressor stations is significantly larger than generally recognized.

### 10-Mile Radius

Air pollution from a compressor station can easily travel 10 miles or more before returning to ground level.

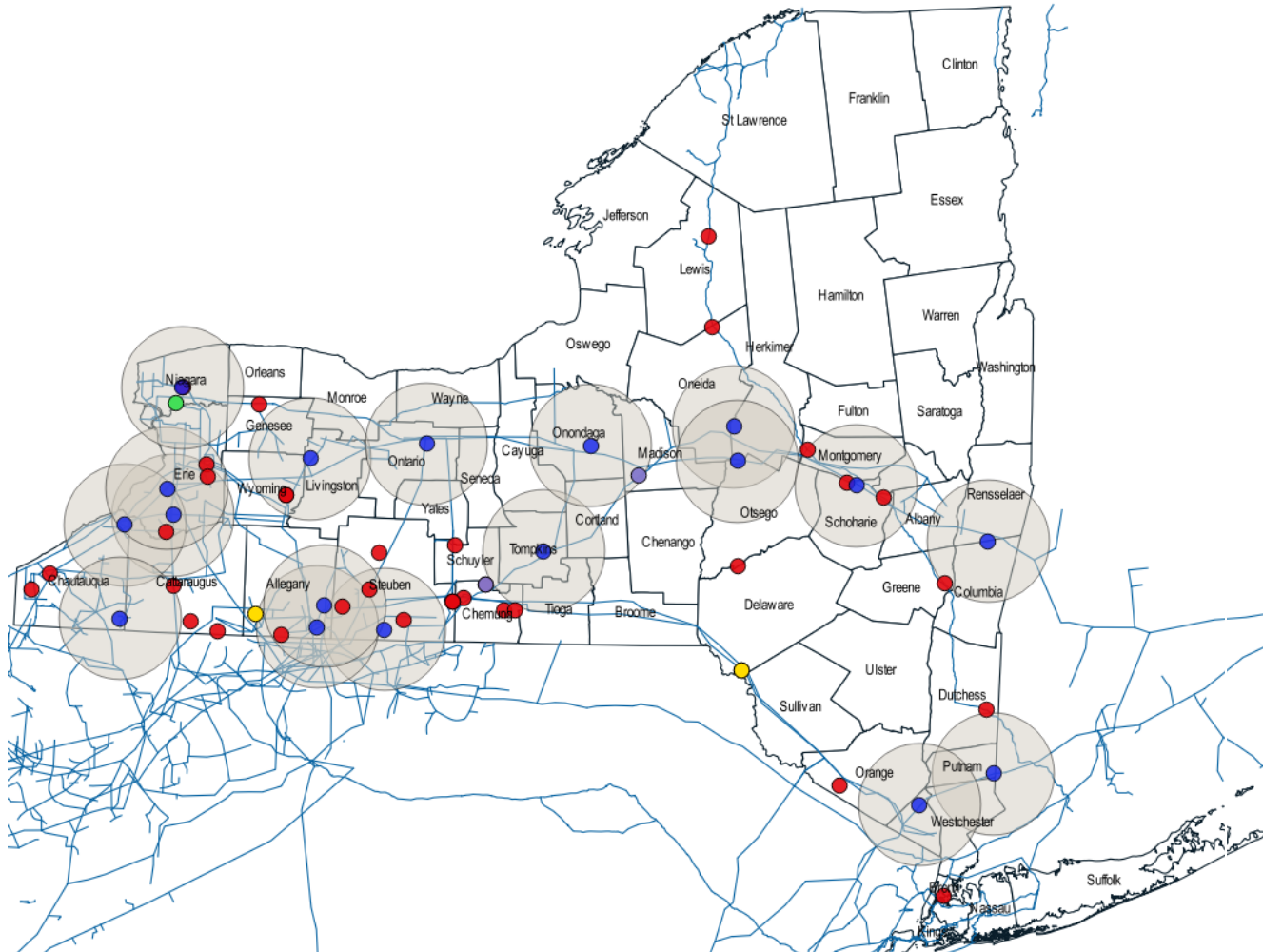
***Approximately 1.6 million people live within 10 miles of the 18 natural gas compressor stations analyzed in this report—more than 1 out of 8 New Yorkers or 12.5% of the population, which works out to about 25 pounds per person over 7 years.***

At this 10-mile radius, people in ~31 counties are potentially breathing air contaminated by compressor station pollutants: NY (27), CT (1), and PA (3).



## 20-Mile Radius

Expanded to 20 miles the number potentially affected is 5.7 million (more than 1 out of every 3 people) in 52 counties: NY (39), CT (3), MA (1), NJ (3), and PA (6).



## 2-Mile Radius

Approximately 33,516 people live within a 2-mile radius. If we assume all the pollution was limited to this radius, it works out to 1,201 pounds per person over 7 years.

(See section 2.5c.1. for more details)



## 10. Total Releases by Health Effects

The 70 chemicals released by NYS's natural gas compressor stations are linked to all 17 of the major categories of human disease as classified by the International Statistical Classification of Diseases and Related Health Problems, 10<sup>th</sup> edition (ICD-10). These are summarized in the table below.

Most chemicals are known to cause multiple categories of diseases. Formaldehyde is a good example. NEI shows releases totaling approximately 1.3 million pounds of this chemical.

Formaldehyde is a known human carcinogen, so it is included as a chemical associated with neoplasms (ICD-10, Chapter 2). But it is also associated with virtually every other major category of human disease, so it would be included as contributing to the totals in the table below for chapters 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 14, 15, 16, 17, and 18.

**There were, for example 9.5 million pounds of 59 chemicals related to neoplastic diseases (malignant and benign neoplasms) released by 18 facilities in 14 counties, averaging 1.4 million pounds annually (ICD-10, Ch. 2).** Or, to put it differently, 23.9% of all 40.2 million pounds of toxic chemicals released are carcinogens.

There were 16.2 million pounds of pollutants associated with circulatory diseases such as heart attacks and strokes (ICD-10, Ch. 9). The table below lists health effects by their ICD Chapter.

### Toxic Releases by ICD-10 Chapter

New York State Natural Gas Compressor Stations: 2008 to 2014

ICD Ch.	Disease \ Disorders	Chemicals	Facilities	Counties	DEC Reg.	Pounds Annual Average	Pounds Total	% of Total Lbs.
1	Certain infectious and parasitic diseases	1	18	14	9	2,583,224	18,082,570	45.0
2	Neoplasms (malignant and benign)	59	18	14	9	1,362,610	9,538,272	23.9
3	Blood and blood-forming organs and certain disorders involving the immune mechanism	41	18	14	9	2,678,763	18,751,319	47.0
4	Endocrine, nutritional and metabolic	51	18	14	9	1,016,765	7,117,352	17.8
5	Mental and behavioral	34	18	14	9	2,678,042	18,746,295	47.0
6	Nervous system	42	18	14	9	2,713,070	18,991,490	47.6
7	Eye and adnexa	40	18	14	9	3,547,275	24,830,922	61.8
8	Ear and mastoid process	15	18	14	9	2,494,582	17,462,077	43.5
9	Circulatory system	31	18	14	9	2,321,403	16,249,821	40.4
10	Respiratory system	51	18	14	9	5,663,824	39,646,765	98.6
11	Digestive system	45	18	14	9	5,496,041	38,472,286	95.7
12	Skin and subcutaneous tissue	48	18	14	9	3,963,161	27,742,125	69.0
13	Musculoskeletal system and connective tissue	17	18	14	9	176,168	1,233,174	3.1
14	Genitourinary system	43	18	14	9	5,706,861	39,948,030	99.4
	1. Urinary system	33	18	14	9	915,867	6,411,070	16.0
	2. Reproductive system: pelvis, genitals and breasts	37	18	14	9	5,706,424	39,944,967	99.4
15	Pregnancy, childbirth and the puerperium	18	18	14	9	2,803,817	19,626,720	48.8
16	Certain conditions originating in the perinatal period	20	18	14	9	3,215,181	22,506,319	56.0
17	Congenital malformations, deformations, chromosomal abnormalities	59	18	14	9	5,663,578	39,645,048	98.7
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	43	18	14	9	5,663,743	39,646,203	98.7
	<b>All Releases</b>	<b>70</b>	<b>18</b>	<b>14</b>	<b>9</b>	<b>5,741,819</b>	<b>40,192,733</b>	<b>100%</b>

## NYS Compressor Station Toxicants: 2008-2014

<b>Total Releases by ICD-10 Disease Category (millions of pounds)</b>	<b>~40.2</b>
1. Certain infectious and parasitic diseases*	18.1
2. Neoplasms (malignant and benign)	9.5
3. Blood and blood form, certain immune disorders	18.8
4. Endocrine and metabolic	18.8
5. Mental and behavioral	18.7
6. Nervous system	19.0
7. Eye and adnexa	24.8
8. Ear and mastoid process	17.5
9. Circulatory system	16.3
10. Respiratory system	39.6
11. Digestive system	38.5
12. Skin and subcutaneous tissue	27.7
13. Musculoskeletal system\connective tissue	1.2
14. Genitourinary system	39.9
Urinary system	6.4
Reproductive system: Pelvis, genitals and breasts	40.0
15. Pregnancy, childbirth, puerperium	19.6
16. Certain conditions originating in the perinatal period	22.5
17. Birth defects, chromosomal abnormalities	39.6
18. Symptoms, signs, findings nec	39.6

\* Systemic effects resulting from the release of greenhouse gases.

## 11. Visualizing the Data

### Scenario 1

It's difficult to visualize what 40.2 million pounds of pollution looks like.

The following might help.

Everyone's familiar with a 5-pound bag of flour. There's one in every kitchen.

Assume that the 40.2 million pounds of toxic pollution generated by the state's 18 compressor stations has the same density as flour, i.e., that 5-pounds of pollution would fit in a bag equivalent in size to a 5-pound bag of flour.

To put the 40.2 million pounds of compressor station pollution in 5-pound bags would require 8,038,545 bags.

### Scenario 2

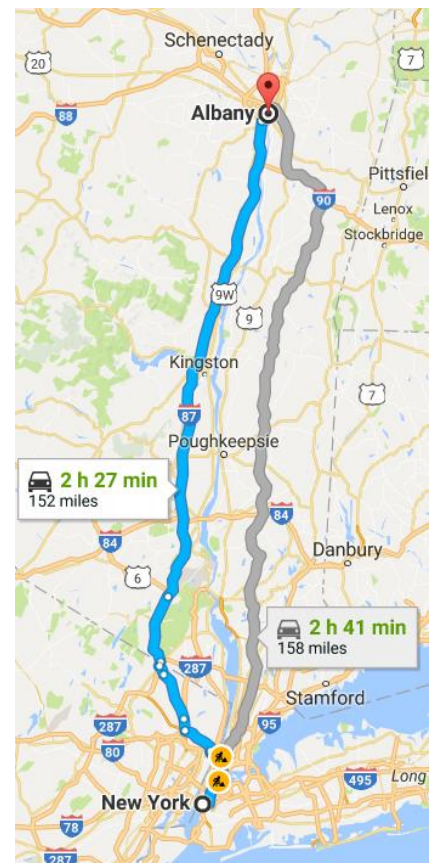
Let's go a step further. Let's say we wanted to take our 8 million bags of toxic pollution for a ride.

Assume we loaded all 7 years of compressor station pollution onto  $\frac{1}{2}$ -ton pickup trucks.

Each pickup could safely carry 1,000 pounds or 200 5-pound bags.

So, we'd have to load up 40,193 1-ton pickups (40.2 million pounds / 1,000)

Let's say each pickup is 20' long and we were backed up on the highway literally bumper to bumper: Our line of 40,193 1-ton pickup trucks would stretch 152 miles-- exactly the distance from New York City to Albany.



### Scenario 3

Another scenario.

Let's assume we aren't stuck in traffic and instead our 40,193 trucks filled with compressor station pollution are traveling 65 mph on the nation's highways with 576 feet between each truck (the distance the average driver needs to react in 6 seconds).

In this case our pollution convey would stretch about 4,537 miles (40,193 trucks x 596 feet / 5,280 feet) -- almost long enough to stretch from the easternmost location in the U.S., Houlton Maine, down to Miami, back up to Tampa, along the Gulf Coast to Houston, across Texas, New Mexico, Arizona and California to Los Angeles.



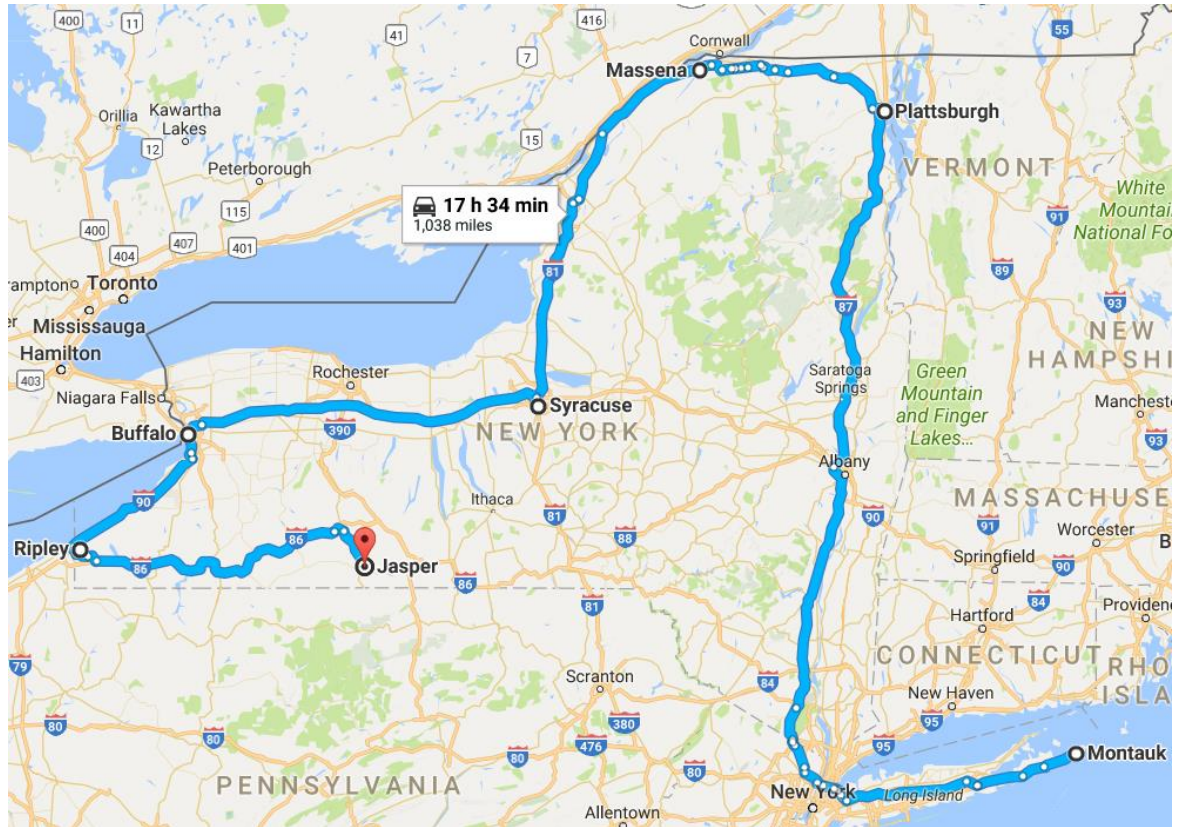


## Scenario 4

A flour bags is 8" tall.

If we laid each of our 8,038,545 bags on their side they would stretch approximately 1,034 miles.

This is enough bags to line the interstates from Montauk Long Island, up to Plattsburgh, west to Massena, down to Syracuse, went to Buffalo, down to Ripley and east to Jasper.



## Scenario 5

Everyone knows what a football field looks like.

New Era Field, home to the Buffalo Bills, is the only professional football field in NYS.

A football field measures 160' x 360' (57,600 sq. ft.).

Our flour bags measure 8" x 6" x 5".

If we lay a flour bag on its widest sides, it measures 8" x 6" or 48 sq. inches.

It takes 14,400 bags to cover a football field with a single layer of 5-lb. bags to a height of 5".

If we placed all 8 million bags one atop the other, we could cover New Era Field to a height of about 97' -- roughly the height of a 10-story office building.

A second scenario: If we laid the bags next to each other along the 5" dimension (the shortest), since a football field is 100 yards or 300 feet,  $0.417 \text{ ft per bag} \times 8.04 \text{ million bags} \times 1 \text{ mile per } 5280 \text{ ft}$  yields 634 miles. Since a football field is 0.057 mi long, we would need  $634/0.057$  or about 11,000 football fields all lined up one after the other to "hold" this line of bags.



# Introduction

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## Contents of the Report

This report is divided into four chapters.

- Chapter 1, Background: provides a brief review of the issues which motivated this report.
- Chapter 2, Compressor Station Releases: identifies the locations of natural gas compressor stations in New York State (NYS) and the total volume of air pollution they generate based on the three most recent years of data collected by the U.S. Environmental Protection Agency's (EPA) National Emissions Inventory. Detailed analysis of total air pollution is analyzed by chemical, chemical category, NYS Department of Environmental Conservation (DEC) Regions, counties, and zip codes.
- Chapter 3, Health Effects: provides a detailed analysis of compressor station air pollutants for each of 17 major disease categories as defined by the International Classification of Diseases, 10th edition (ICD-10). For each disease category, gas compressor station air pollutants are analyzed by chemicals, by ICD categorization, facilities, DEC regions, and by counties.
- Chapter 4, Facilities: provides a profile of each of the compressor station studied in this report along with a summary of the health effects associated with the chemicals each generates.

## Materials and Methods

### Health effects

To facilitate the identification of toxic exposures and their potential health and environmental impacts, the author has created several proprietary SQL-compliant databases used in environmental and epidemiological studies:

1. Chemical Database: Contains essential data on slightly more than 21,000 unique chemicals or chemical categories, including names, synonyms, identification numbers, chemical and physical characteristics, and inclusion in federal, state and international reporting programs.
2. Occupational Database: Contains essential data on approximately 500 occupations or occupational grouping for which there are epidemiological assessments. Occupations are categorized according to the U.S. Bureau of Labor Statistics Standard Occupational Classification (2010).
3. Health Effects Database: Indexes approximately 120,000 peer-reviewed studies examining the impacts of toxic chemicals on human health and the environment. Each article is indexed by the relevant ICD-10 code. Fields include: chemical name or identifier, author, full reference, PMID, DOI, subject (human/animal), acute/chronic exposure, route of exposure (inhalation, skin, drinking water, diet, etc.). This database can be used to identify all health effects associated with a specific chemical or chemical category.

## U.S. National Emissions Inventory

The rationale for the creation of NEI and some of its limitations are described in the draft of NEI's Technical Support Document, published in June 2014 (USEPA 2014):

The NEI is created to provide EPA, federal and state decision makers, the U.S. public, and other countries the U.S.'s best and most complete estimates of CAP and HAP emissions. While EPA is not directly obligated to create the NEI under the Clean Air Act, the Act authorizes the EPA Administrator to implement data collection efforts needed to properly administer the NAAQS program. Therefore, the Office of Air Quality Planning and Standards (OAQPS) maintains the NEI program in support of the NAAQS. Furthermore, the Clean Air Act requires states to submit emissions to EPA as part of their State Implementation Plans (SIPs) that describe how they will attain the NAAQS. The NEI is used as a starting point for many SIP inventory development efforts and for states to obtain emissions from other states needed for their modeled attainment demonstrations.

While the NAAQS program is the basis on which EPA collects CAP emissions from the state, local, and tribal (S/L/T) air agencies, it does not require collection of HAP emissions. For this reason, the **HAP reporting requirements are voluntary**. [authors' emphasis] Nevertheless, the HAP emissions are an essential part of the NEI program. These emissions estimates allow EPA to assess progress in meeting HAP reduction goals described in the Clean Air Act 4 amendments of 1990. These reductions seek to reduce the negative impacts to people of HAP emissions in the environment, and the NEI allows EPA to assess how much emissions have been reduced since 1990.

If "HAP reporting requirements are voluntary" by extension it seems reasonable to conclude that EPA isn't legally obligated to analyze the results of the inventory to identify all potential health impacts, to prioritize chemicals in terms of their greatest harm to health, or communicate this information to the general public or state regulators effectively.

The National Emissions Inventory is available to the public on EPA's website.

Data is published every 3 years. This report uses the last 3 years of published data: 2008, 2011, and 2014.

To estimate total releases over the 7-year period from 2008 through 2014, the average for 3 years was determined and multiplied by 7. Given the characteristics of the data, performing these calculations at different levels (e.g., facility versus country) sometimes produces slightly different totals, though the difference is small and not statistically meaningful.

## U.S. EPA Greenhouse Gas Inventory

The major source of emissions of greenhouse gases is EPA's Greenhouse Gas Inventory:

[T]he U.S. Greenhouse Gas Inventories developed by the U.S. government to meet U.S. commitments under the United Nations Framework Convention on Climate Change (UNFCCC). Article 4.1a of the UNFCCC requires that all countries periodically publish and make available to the Conference of the Parties (COP) inventories of anthropogenic emissions and removals by sinks of all greenhouse gases not controlled by the Montreal Protocol.

Subsequent decisions by the COP require the United States to submit these reports on an annual basis and include emissions of carbon dioxide (CO<sub>2</sub>), methane (CH<sub>4</sub>), nitrous oxide (N<sub>2</sub>O), hydrofluorocarbons (HFCs), perfluorocarbons (PFCs), and sulfur hexafluoride (SF<sub>6</sub>) and removal of these gases by sinks. (EPA GGI).

## Available EPA Data: Chemicals, Emissions Types, Years

Data on pollution from natural gas compressor stations in New York State is drawn from 2 federal sources: U.S. EPA National Emissions Inventory (point sources) and U.S. EPA Greenhouse Gas Inventory.

### Stationary Sources

NEI's point source data set provides data on releases from stationary sources (aka point or stack) and provides information on 70 specific chemicals.

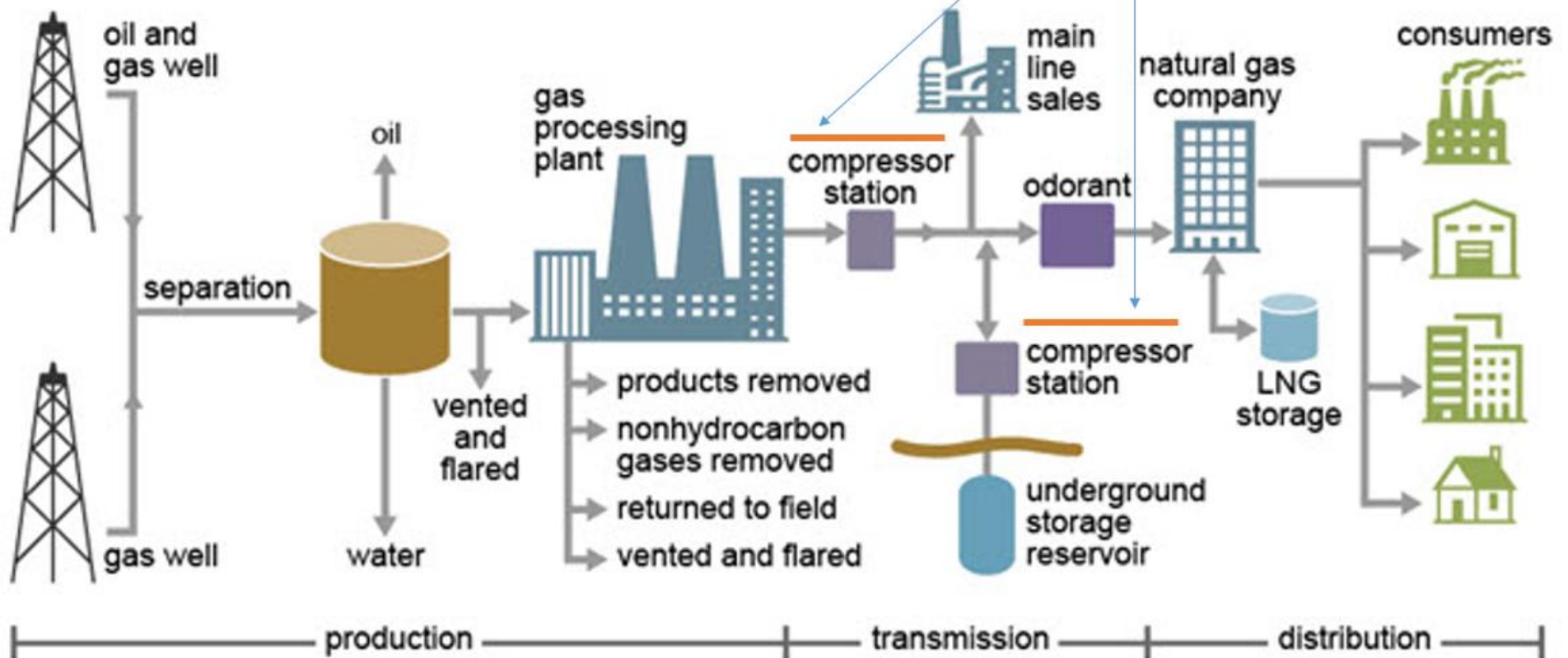
EPA's GHGI provides data on 2 chemicals not included in NEI's point data set for compressor stations: carbon dioxide and methane.

### Fugitive Releases

EPA's GHGI provides data on 3 chemicals from fugitive sources at the compressor station site: carbon dioxide, methane and NO<sub>x</sub>. However, data is only available for 8 of NYS's 18 Title V compressor stations and this only begins in 2010.

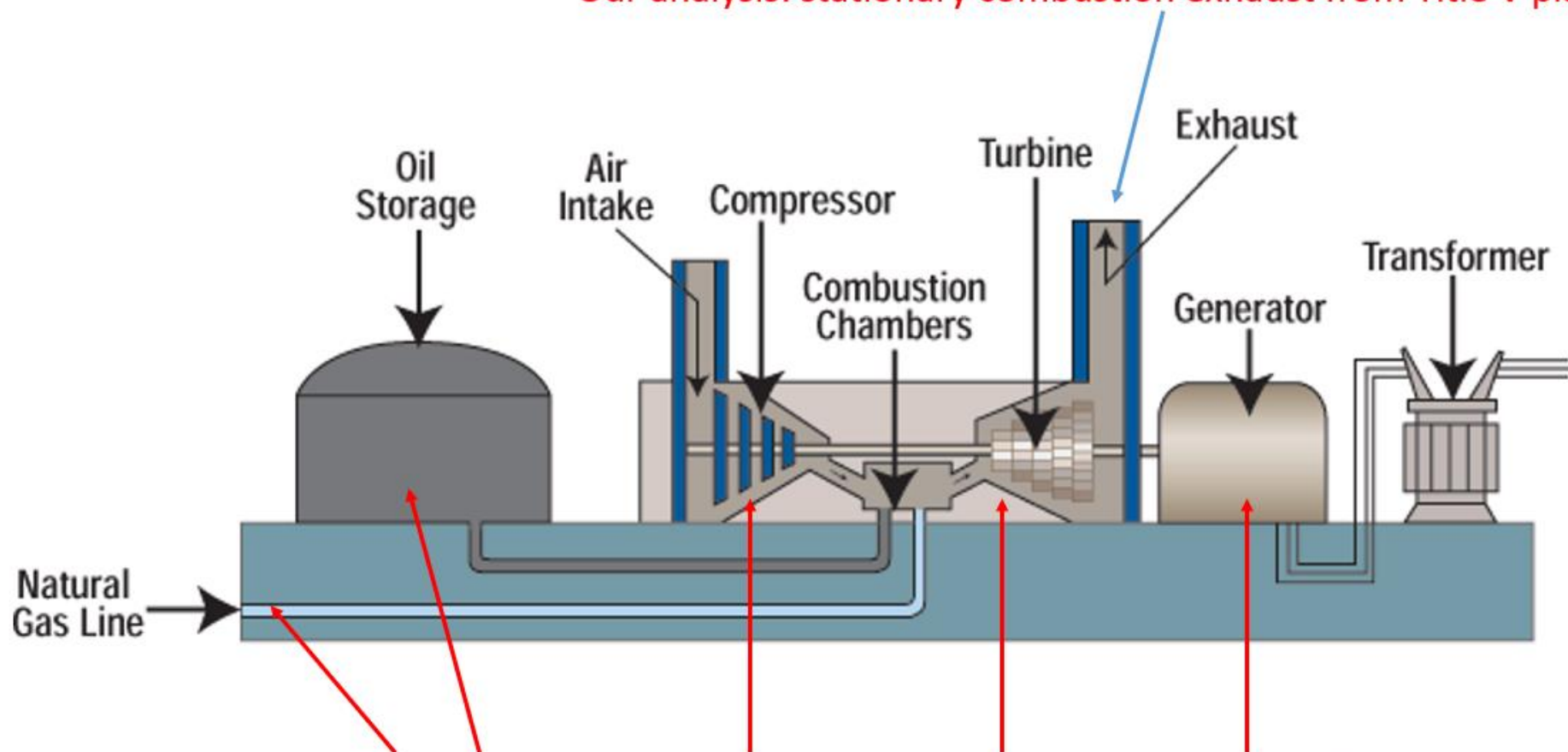
# Natural gas production and delivery

**This Study**



Source: EIA

Our analysis: stationary combustion exhaust from Title V plant



In this report it was **not** possible to analyze compressor station fugitive releases



## National Fuel's planned compressor station in Hinsdale (Cattaraugus County)



Source: National Fuel Gas Supply Corp.

## Years of available data

EPA NEI reports data every third year—data is available for 2008, 2011 and 2014.

The data reported for NYS is fairly complete: 17 stations report data for all 3 years, while one reports for 2008 and 2011 but not for 2014.

The national data seems fairly incomplete: (a) 409 compressor stations that reported releases in 2008 had not data for 2011, and (b) 196 that reported data in 2008 showed no data for 2014, and (c) 426 that reported data in 2011 showed no data for 2014. In a few cases we suspect this is due to plant modifications but it is unlikely that this explains the extent of missing data.



# Available EPA Natural Gas Compressor Station Data: Chemicals, Emissions Types, Years, Sources

✓ Stationary (Stack) Combustion	70 chemicals <i>not</i> including Carbon dioxide or Methane	U.S. EPA National Emissions Inventory (Point Sources)
✓ Stationary (Stack) Combustion	Carbon dioxide	U.S. EPA Greenhouse Gas Inventory (GHGI)
✓ Stationary (Stack) Combustion	Methane	Not reported in either NEI or GHGI
✓ Fugitive Emissions	Carbon dioxide	U.S. EPA Greenhouse Gas Inventory
✓ Fugitive Emissions	Methane	U.S. EPA Greenhouse Gas Inventory
✓ Fugitive Emissions	???	U.S. EPA NEI (Non-Point Sources): To be determined

#	Compressor Station	Town	County	2008	2009	2010	2011	2012	2013	2014	2015
Title V Permit				18 Operational Compressor Stations							
1	AG SE CS	Southeast	Putnam	✓		✓	✓	✓	✓	✓	✓
2	AG Stony Point CS	Stony Point	Rockland	✓		✓	✓	✓	✓	✓	✓
3	DTI Borger CS	Ithaca	Tompkins	✓			✓			✓	
4	DTI Utica CS	Frankfort	Herkimer	✓			✓			✓	
5	DTI Woodhull CS	Woodhull	Steuben	✓			✓			✓	
6	NFGSC Beech Hill CS	Willing	Allegany	✓			✓			✓	
7	NFGSC Concord CS	Concord	Erie	✓			✓		✓	✓	✓
8	NFGSC Independence CS	Andover	Allegany	✓			✓		✓	✓	✓
9	NFGSC Nashville CS	Hanover	Chautauqua	✓			✓				
10	TGPC CS 224	Clymer	Chautauqua	✓			✓	✓	✓	✓	✓
11	TGPC CS 229	Eden	Erie	✓		✓	✓	✓	✓	✓	✓
12	TGPC CS 230-C	Lockport	Niagara	✓			✓			✓	✓
13	TGPC CS 233	York	Livingston	✓			✓			✓	
14	TGPC CS 237	Manchester	Ontario	✓			✓			✓	
15	TGPC CS 241	LaFayette	Onondaga	✓		✓	✓	✓	✓	✓	✓
16	TGPC CS 245	Winfield	Herkimer	✓		✓	✓	✓	✓	✓	✓
17	TGPC CS 249	Carlisle	Schoharie	✓		✓	✓	✓	✓	✓	✓
18	TGPC CS 254	Chatham	Columbia	✓			✓	✓	✓	✓	✓

#	Compressor Station	Town	County	2008			2009			2010			2011			2012			2013			2014			2015																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																	
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## Identification of NYS Natural Gas Compressor Stations

The method used to identify natural gas compressor stations operating in NYS is described in Chapter 2.

## Abbreviations

AG	Algonquin Gas Transmission LLC
DTI	Dominion Transmission Inc.
NFGSC	National Fuel Gas Supply Corp.
CFR	Code of Federal Regulations
ch	Chemical or chemicals
cs	Compressor Station
DEC	New York State Department of Environmental Conservation
deh	Dehydration
DOH	New York State Department of Health
EPA	U.S. Environmental Protection Agency
est'd	estimated
Fac	Facility or facilities
TGPC	Tennessee Gas Pipeline Company
FDA	Food and Drug Administration
FERC	Federal Energy Regulatory Commission
GHG	Greenhouse gas
IARC	International Agency for Research on Cancer
ICD-10	International Classification of Disease, 10 <sup>th</sup> edition
Lbs.	pounds
ng	natural gas
nec	not elsewhere classified
ngfsct	natural gas fired stationary compressor turbine
NSPSs	New Source Performance Standards
NYS	New York State
REL	Recommended exposure limit
src	source
TBD	To be determined

# Chapter 1. Background

# 1. Introduction

## 1.1. Pollution as a Cause of Human Disease

The causes of human disease are various and complex. The siting of industrial facilities is inherently problematic and political. Communities facing the prospect of a new natural gas compressor station or the expansion of an existing station, must grapple with both sets of concerns and, more difficult still, the specific question of how compressor station emissions may potentially harm human health.

It was only in relatively recent years that a large part of the scientific community accepted the view that environmental and occupational exposures to man-made chemicals and radiation are a significant threat to health. The creation of the U.S. Environmental Protection Agency (EPA) and the U.S. Occupational Agency (OSHA) in 1970 and the passage of the National Cancer Act the next year were each **partly** motivated by growing evidence of pollution as a cause of human diseases.

In the near half-century since these agencies and programs were created, significant progress has been made in reducing pollution in the U.S. But when the actual history is studied, it is clear that these advances only came because of sustained political efforts by people outside of government attempting to pressure those in government to do the right thing over the opposition of vested economic interests. Legislative measures are generally only taken many years after scientific warnings are first raised. And more often than not, these long overdue legislative measures are half-steps that are inadequately funded and ineffectively enforced.

While the positive steps that have been made should be recognized, the difference between what society needed to do to confront the problem of pollution and what has actually been done, is stark.

The two leading causes of death in the U.S. are cardiovascular disease and cancer. Environmental and occupational exposure to chemicals are a significant risk factor for both diseases. Based on data reported by the natural gas industry, this report shows that 18 compressor stations operating in New York State released a total of more than 40 million pounds of toxic air pollution in the period from 2008 to 2014, including 16.3 million pounds association with cardiovascular disease and 9.5 million pounds of carcinogens.

We are approaching the 50<sup>th</sup> anniversary of the creation of EPA and OSHA and President Nixon's declaration of "The War on Cancer". In 50 years the nation has made little progress in protecting the public from environmental exposures to known and suspected human carcinogens. Perhaps most importantly, this includes the failure of the existing approach to identifying and communicating environmental and occupational chemical risk.

## 1.2. President's Cancer Panel (2010)

The long-recognized failure of federal agencies to address the environmental causes of cancer led to the creation of the President's Cancer Panel (PCP) which between September 2008 and January 2009 convened four national meetings "to assess the state of environmental cancer research, policy and programs addressing known and potential effects of environmental exposure on cancer." More specifically, the Panel's assigned task was to appraise the National Cancer Program as established in accordance with the National Cancer Act of 1971 (P.L. 92-218), the Health Research Extension Act of 1987 (P.L. 99-158), the National Institutes of Health Revitalization Act of 1993 (P.L. 103-43), and Title V, Part A, Public Health Service Act (42 U.S.C. 281 et seq.). The Panel's overarching conclusion:

Research on environmental causes of cancer has been limited by low priority and inadequate funding. . . There is a lack of emphasis on environmental research as a route to primary cancer prevention. . . Cancer prevention efforts have focused narrowly on smoking, other lifestyle behaviors and chemo-preventive interventions. Scientific evidence on individual and multiple environmental exposure effects on disease initiation and outcomes, and consequent health system and societal costs, are not being adequately integrated into national policy decisions and strategies for disease prevention, health care access and health system reform. (U.S. DHHS 2010)

With regard to this paper's primary concern, identifying the potential health risk associated with routine operations of natural gas compressor stations, four of the Panels critical conclusions are particularly relevant:

### **1. The Present Approach to Risk Assessment is Inadequate:**

[Exposure assessment] is needed more broadly to evaluate cancer risk associated with workplace or environmental exposures in the aggregate. In the U.S., most available exposure assessments are badly outdated. A comprehensive assessment of the extent of all environmental and workplace exposures, for example, has not been conducted since the flawed Doll and Peto estimates published in 1981 (Doll and Peto 1981). Although OSHA's mission is to ensure that workplace environments are safe, it does not conduct a comprehensive national review of carcinogens in the workplace. (U.S. DHHS 2010, p. 15)

Cancer risk assessment also is hampered by lack of access to existing exposure data, especially for occupational/industrial exposures, and regarding levels of radon, asbestos, and other contaminants in schools and day care centers. (U.S. DHHS 2010, p. viii)

Research Methodology and Data Collection Issues: In addition to measurement and standard-setting issues, environmental and occupational cancer research and assessment have suffered from methodologic and data collection weaknesses. (U.S. DHHS 2010, p. 10)

Testimony, Paul Schulte, NIOSH: Right now, the numbers for how many workers are exposed to most of the known carcinogens are 20 to 30 years old so we don't really know what the contemporary workforce is experiencing in terms of exposure.

Testimony, Sandra Steingraber, Ithaca College: Estimates of "attributable fractions" of the cancer burden due to occupation (approximately 4 percent), pollution (2 percent), industrial products (<1 percent), and medicines and medical procedures

(1 percent) are now believed to underestimate significantly the true toll of cancer related to these exposures.” (U.S. DHHS 2010, p. 2)

Recommendation: A thorough new assessment of workplace chemical and other exposures is needed to quantify current health risks. Previous estimates of occupational cancer risk are outdated and should no longer be used by government or industry. (xii)

Recommendation: Measurement tool development and exposure assessment research, including the development of new research models and endpoints, should be accelerated to enable better quantification of exposures at individual, occupational, and population levels. (U.S. DHHS 2010, p. xiv)

Recommendation: Epidemiologic and hazard assessment research must be continued and strengthened in areas in which the evidence is unclear, especially research on workplace exposures, the impact of in utero and childhood exposures, and exposures that appear to have multigenerational effects. Current funding for federally supported occupational and environmental epidemiologic cancer research is inadequate. (U.S. DHHS 2010, p. 105)

## **2. Workers, Other Populations with Known Exposures, and the General Public Require Full Disclosure of knowledge about Environmental Cancer Risks**

Individuals and communities are not being provided all available information about environmental exposures they have experienced, the cumulative effects of such exposures, and how to minimize harmful exposures. (U.S. DHHS 2010, p. ix)

Continued Epidemiologic and Other Environmental Cancer Research Is Needed: Cancer risk assessment . . . is hampered by lack of access to existing exposure data, especially for occupational/industrial exposures, and regarding levels of radon, asbestos, and other contaminants in schools and day care centers. (U.S. DHHS 2010, p. 98)

## **3. Medical Professionals Need to Consider Occupational and Environmental Factors When Diagnosing Patient Illness**

Physicians and other medical professionals rarely ask patients about their workplace and home environments when taking a medical history. Such information can be invaluable in discovering underlying causes of disease. Moreover, gathering this information would contribute substantially to the body of knowledge on environmental cancer risk. (U.S. DHHS 2010, p. ix)

## **4. Inadequate Funding**

Testimony: Elizabeth Fontham, Louisiana State University: Unfortunately, while budgets have waxed and waned on the federal level, a consistent finding, I would say, is that occupational and environmental exposures have been under addressed. (U.S. DHHS 2010, p. 5)

NIOSH Work Group: In 1996, NIOSH convened a group of experts from academia, business, labor, and government to identify the gaps in occupational cancer research methods.<sup>60</sup> The group’s recommendations for strengthening research methods, which became part of NIOSH’s National Occupational Research Agenda, focused on four broad areas: identification of occupational carcinogens, design of epidemiologic studies, risk assessment, and primary and secondary prevention (U.S. DHHS 2010, p. 10)



For those who believe that environmental factors are a much-neglected risk factor for cancer (as well as for non-neoplastic diseases) PCP's conclusion is an important step in the right direction. (Everything the Panel has stated about the lack of attention to environmental and occupational causes of cancer could, in our opinion, also be said of non-neoplastic diseases.)

It is, we think, remarkable that those advancing the view that environmental exposures play only the smallest role in human cancer do so without ever discussing let alone conducting a detailed assessment of exposure, i.e., the extent to which Americans are exposed to chemical and radiologic carcinogens.

The starting place of scientific inquiry is identifying all possible factors which might in some measure affect the phenomenon under investigation. How is it possible to conclude that environmental causes are bit players without first having examined fundamental questions related to the extent and significance of exposures to man-made carcinogens? Such questions include: the total pounds of chemicals manufactured and imported, their number and characteristics, the number of chemicals approved for commercial use, the number and volume of chemicals produced by combustion (not intentionally manufactured), where and how exposures occur, their persistence in the environment, chemical synergism, issues related to exposure assessment, and the number of carcinogens found in human urine, blood and adipose tissue as well as evidence of neo-natal contamination?

Any summary account attempting to answer the question (however tentatively) "What causes cancer?" must include an analysis of these critical issues as well as a number of pertinent methodological concerns. Absent this framework any analysis which purports to claim that X% of Y cancers are caused by Z (or some combination of factors) is logically unsound—all of the possible relevant explanations have not been considered.

In this paper, we show that 18 of the state's ~40 natural gas compressor stations released an estimated 40.2 million pounds of toxic into the air over a 7-year period—an annual average of about 5.7 million pounds. The 7-year total included an estimated 9.5 million pounds of carcinogens (80% of which are classified as "known human carcinogens")—approximately 1.4 million pounds a year. The significance of this finding we believe, speaks for itself. Would a reasonable person who is presented with fact persist in the assertion that environmental factors are only a small causal factor? We don't think so.

Our establishing the extent of carcinogenic exposures in a single facility does not prove that they cause a specific percentage of a given cancer or all cancers at this work site, but it does demonstrate that an analysis consistent with the principles, methods and logic of scientific inquiry must seriously take environmental considerations into account.

In *Discourse on Method* Descartes advises that "when it is not in our power to determine what is true, we ought to follow what is most probable." It is this precept that has been the hallmark of modern science. It is, however, one which cannot be said to guide the nation's approach to preventing cancer. The tired assertion endlessly repeated that most cancers have little or no connection to environmental pollution is made without context or reference to physical realities--the extent and characteristics of chemical contamination and its documented effects. The environmental hypothesis is in no meaningful way refuted. It is simply dismissed. ***The physical reality of widespread, unavoidable chemical contamination is the large picture that must frame any meaningful discussion of cancer's etiology as well as risk assessment.***

### 1.3. Outdoor Air and Particulate Air Pollution: Known Human Carcinogens

What is that larger picture?

***In 2013, the International Association for Research on Cancer, the specialized cancer agency of the World Health Organization, classified outdoor air as a known human carcinogen and a leading cause of cancer deaths.***

IARC's study was "based on the independent review of more than 1,000 scientific papers from studies on five continents. The reviewed studies analyze the carcinogenicity of various pollutants present in outdoor air pollution, especially particulate matter and transportation-related pollution. The evaluation is driven by findings from large epidemiologic studies that included millions of people living in Europe, North and South America, and Asia."

After reviewing the extensive scientific literature IARC concluded that there is sufficient evidence to conclude that exposure to outdoor air pollution is a cause of lung cancer (Group 1) and that there is a positive association with an increased risk of bladder cancer.

From 2008 to 2014, NYS's natural gas compressor stations released an estimated 2.9 million pounds of particulate pollution, a major component of outdoor air pollution. Particulate airborne pollution was evaluated separately by IARC and was also classified as carcinogenic to humans (Group 1).

The IARC evaluation showed an increasing risk of lung cancer with increasing levels of exposure to particulate matter and air pollution. Although the composition of air pollution and levels of exposure can vary dramatically between locations, the conclusions of the Working Group apply to all regions of the world.

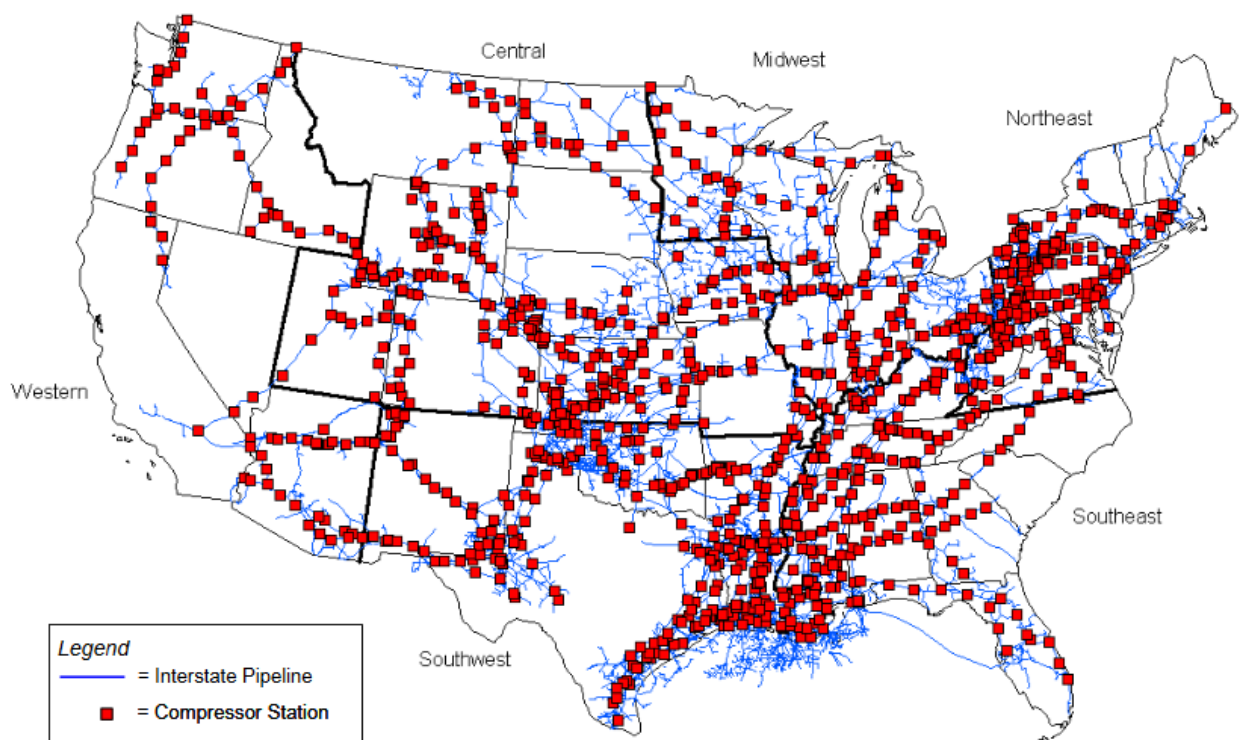
A recent Chinese study observed that cancer is rising in China in significant part to air pollution but that policies to systematically use cancer registry statistics and air pollution data to understand this problem are inadequate (which is also true in this country).

Analyses on the cancer registry data show that cancer burden related to air pollution is on the rise in China and will likely increase further, but there is a lack of data to accurately predict the cancer burden. Past experience from other countries has sounded alarm of the link between air pollution and cancer. The quantitative association requires dedicated research as well as establishment of needed monitoring infrastructures and cancer registries. The air pollution-cancer link is a serious public health issue that needs urgent investigation. (Huang et al. 2014)

#### 1.4. Expansion of Fracking Operations and Natural Gas Compressor Stations in the U.S.

In 2010, the U.S. Department of Energy reported there were 510,000 operational natural gas wells in the U.S., almost twice as many as there were in 2000, and that on average 13,000 new wells drilled each year during this 10-year period.

To keep pace with the unprecedented expansion of fracking operations, over the last two years the number of natural gas pipeline compressor stations has grown significantly. In 1996, there were approximately 1,047 compressor stations attached to the mainline grid with an installed horsepower of 13.4 million, capable of a daily combined throughput of 743 billion cubic feet. Ten years later there were 1,201 comparable stations (an increase of 17%) with 16.9 million installed horsepower capable of 881 billion cubic feet or a 19% increase in output. (EIA 2007-11)



Note: EIA has determined that publication of this figure does not raise security concerns, based on the application of Federal Geographic Data Committee's Guidelines for Providing Appropriate Access to Geospatial Data in Response to Security Concerns.  
Source: Energy Information Administration, Natural Gas Division, Natural Gas Transportation Information System, Compressor Station Database.

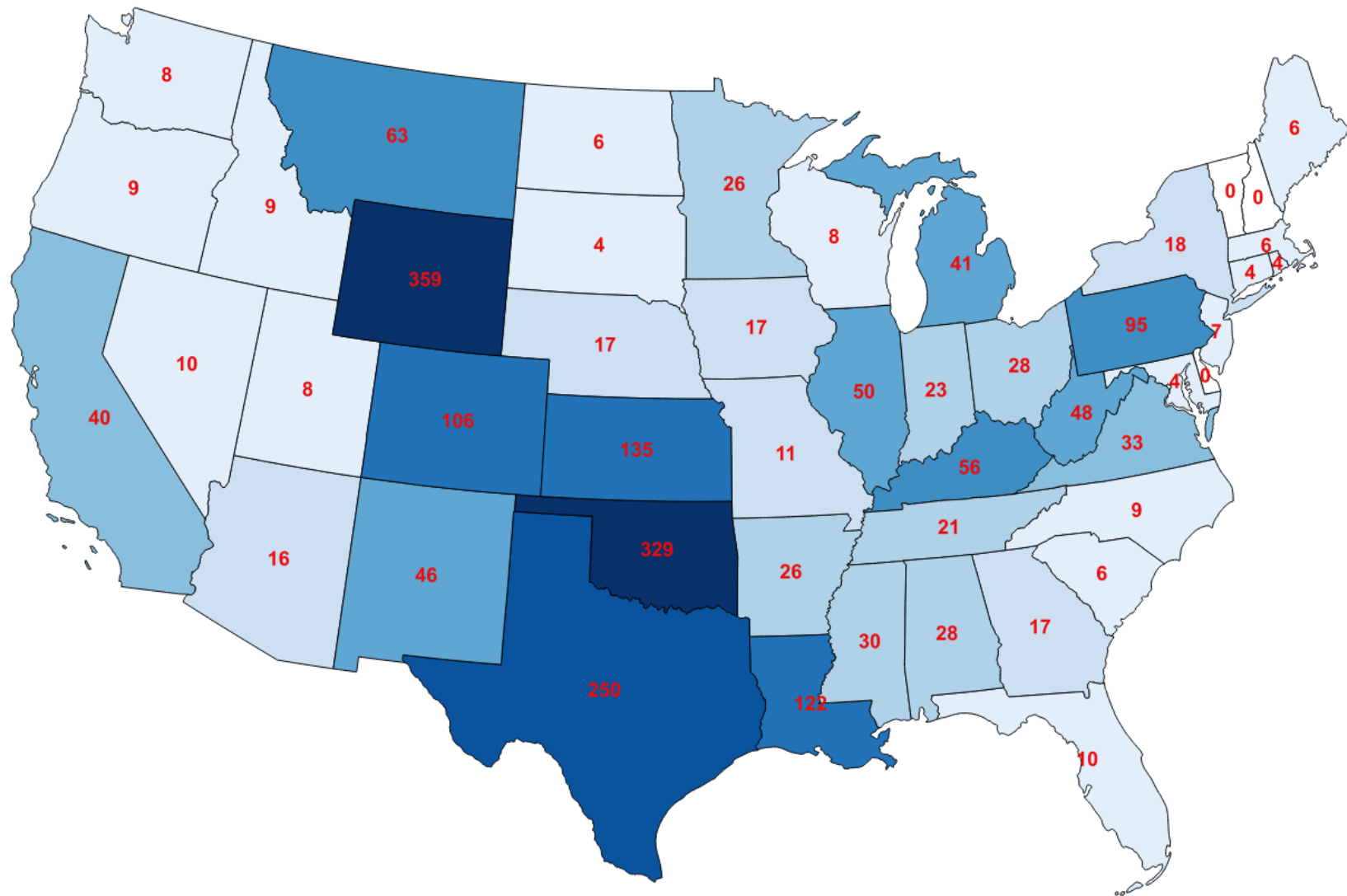
Our own analysis of NEI's data for the period 2008 to 2014 shows 2,177 compressor stations as identified by NAICS 48261.

In NYS Title V compressor stations are classified as 48621. It is unclear to the authors whether all 2,177 stations with NAICS 48621 operate under a Title V permit.

***Our preliminary estimate is that these 2,177 stations are responsible for more than 2.1 trillion pounds of stationary air pollutants.*** This may actually be an underestimation: (a) a significant number of stations reported releases in a given year but failed to report in one or more subsequent years, (b) some stations only reported releases as small as 2 pounds, and (c) it may not include all compressor stations (e.g., non-Title V stations).

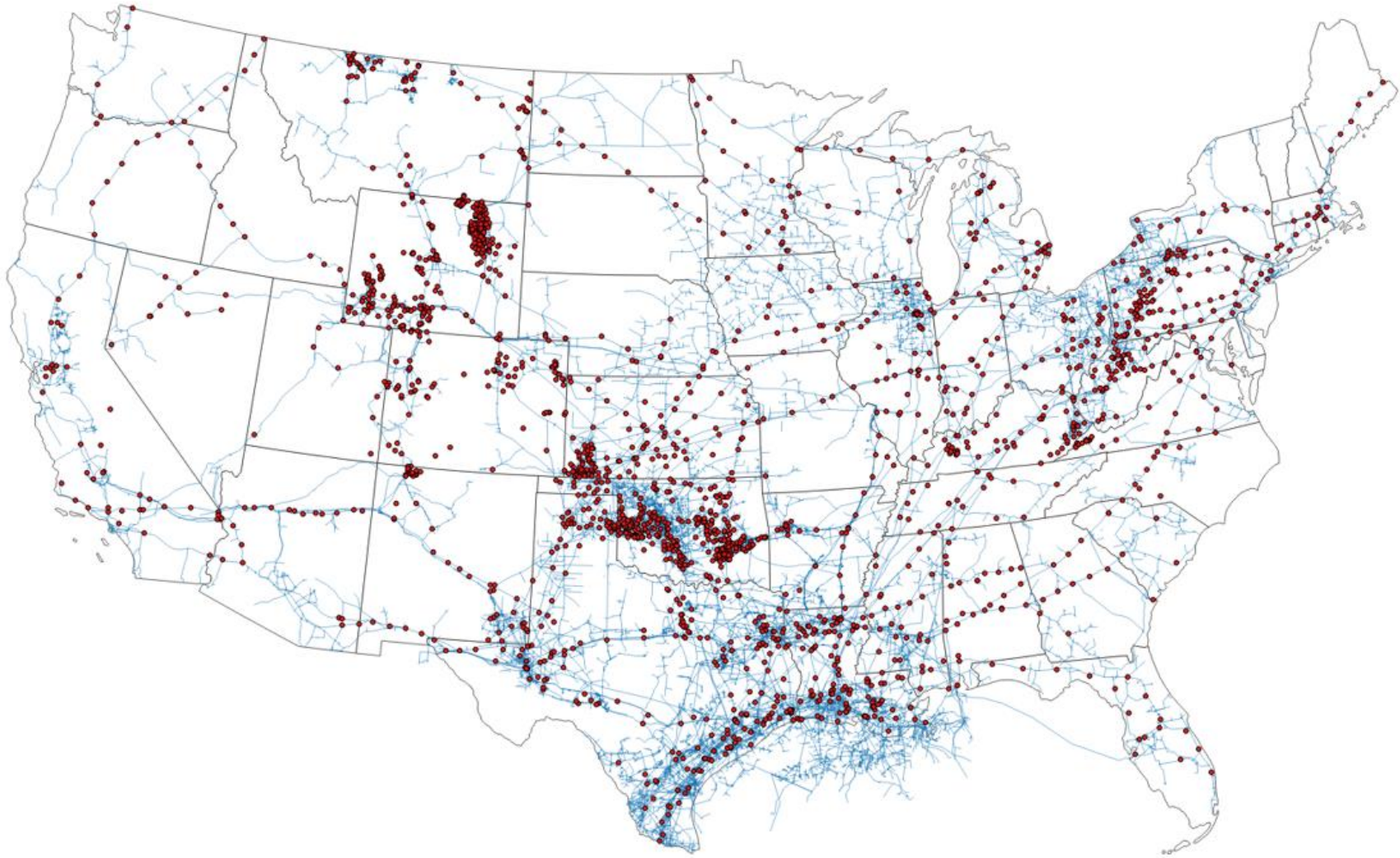
**Table 1.4.**

**U.S. National Gas Compressor Stations (NAICS Code 486210): 2,177**



## Table 1.4.

### U.S. National Gas Compressor Stations (NAICS Code 486210): 2,177



## 1.5. The Legal Framework for Accessing the Health and Environmental Risks of Natural Gas Compressor Stations

### 1.5.1. The Halliburton Loophole

The expansion of unconventional gas operations in the U.S. has largely occurred without benefit of the nominal health and environmental safeguards governing the siting and operation of oil and gas operations in force prior to 2004.

Under the terms of the Energy Policy Act of 2005, oil and gas operations were exempted from almost all existing federal air and water regulations.

The provision in the Act that exempts the oil and gas operations from federal rules covering all other industrial operations is the result of then Vice President Dick Cheney's Energy Task Force and is universally known as the "Halliburton loophole".

Halliburton Corporation is one of the three largest manufacturers of fracking fluids, making it one of the largest beneficiaries of the Act's disregard for even minimal health and safety concerns. Before becoming Vice President in 2001, Cheney was Halliburton's CEO.

In 1997 EPA was ordered to regulate hydraulic fracturing under the Safe Drinking Water Act (SDWA) by the U.S. Court of Appeals for the 11th Circuit (Atlanta), following the contamination of residential drinking water wells in Alabama. The Halliburton Loophole prohibits EPA from regulating the injection of fracturing fluids under the SDWA. In consequence, fracking wastewater is injected directly into or adjacent to underground drinking water without governmental oversight.

In response EPA undertook a 3-year study of the issue. Oil and gas industry representatives made up 5 of the 7 members of EPA's peer review panel, including a representative from Halliburton. The panel's findings, published in 2004, concluded that fracturing "poses little or no threat" to drinking water and that no further study of the question was necessary.

In its investigation, the transparently self-serving panel had ignored or concealed well documented evidence that fracking presents a significant threat to drinking water. Courageously, Weston Wilson, a 30-year veteran of the EPA in Denver, blew the whistle on the panel's violation of scientific principles and available evidence and the Agency's refusal to regulate what is clearly a hazard to public health. The weight of evidence in Wilson's charge that the panel's findings were "scientifically unsound" and the public outcry that followed, forced EPA inspector General Nikki Tinsley to conclude in March 2005 that there was sufficient evidence to justify a review of the panel's work.

It is at this point in the story that Cheney and his former employer Halliburton jumped into the breach--the passage of the Energy Policy Act effectively removed EPA from a meaningful regulation of fracking and related operations, not only of drinking water but also the terms of the Clean Water Act (CWA), the Clean Air Act, the Superfund Act, the Emergency Planning and Community Right-to-Know Act, and the National Environmental Policy Act.

### 1.5.2. Public Concerns about the Health Risks of Natural Gas Compressor Stations

The explosion in unregulated fracking operations made possible by the Halliburton Loophole has in turn resulted, as previously noted, in the expansion of natural gas compressor station operations and the constructions of thousands of miles of new pipelines.

As many knowledgeable observers have noted, existing federal, state and local policies that govern industrial site approval and the actual approval process fail adequately to protect the immediate and long-term health of the public or safeguarding the environment.

Many reasons account for this failure.

First and foremost is the view that development (“job creation”) is the paramount concern of government.

Policies certainly exist on paper stating that assessments of potential harm to public health and the environment are an integral part of the approval process.

In actual practice, these “safeguards” are largely a legal formality at great variance from governmental assurances that the primary concern is the public’s health and safety.

Only a small percentage of these proposals are rejected on grounds that they would unduly threaten public health and the environment; in most of these cases it is only because public interest groups could mount and sustain long legal and political struggles.

In the case of struggles attempting to prevent the construction of new or expansion of existing natural gas compression stations, they are waged without any quantitative assessment of potential immediate and long-term health risks associated with air emissions from these facilities.

When local officials or the public raise concerns about the potential health risks associated with natural gas compressor stations, the answer is always the same: “all required studies have been completed”.

What does this mean?

When an average person hears an “expert’s” confidently assertion that “all required studies have been completed”, they conclude this should be taken to mean it won’t cause immediate- or long-term damage to human health or the environment.

That, of course, is precisely the intention.

And to be truthful, this is what many people want to hear; they welcome such an assurance because it means the proposed plant is something they don’t have to worry about or spend time and effort organizing against.

The purpose of the assertion “all required studies have been completed” is to quell public doubt and to silence scientific criticism.

***The most important thing the public and local officials need to understand is that “all required studies have been completed” is not the same thing as saying (or the reality) that “there is no potential for increased risk of death or disease.”***

This is to say, the “required studies” do not, in our opinion, meaningfully protect human health.

The public works under the false assumption that the primary purpose of governmental regulations such as those under consideration is to determine what’s “safe”.

At best, such safeguards prevent egregious potential for harm, but often they don’t even do that.

There are several reasons for this.

The first is that preventing the public from any increased health risk is not the overriding goal of federal and state regulations. Rather, it is “balancing” potential harm from potential “benefits”.

This raises three obvious questions:

Who determines “the potential for harm”?

Who determines what constitutes a “benefit”?

And who determines what the balance between the two should be?

To state the obvious, it is not within the authority of the public, public health experts or independent scientific agencies to answer these questions.

The rules such as they are, have largely been written to advance the interests of industry even when public health and the environment are compromised. The case of the Halliburton Loophole is one obvious example. The refusal to take measures to address climate change is another.

Each of the 18 compressor stations analyzed in this report were required to complete 6 or more federal or state studies before they were given building permits. A few of these studies directly address health concerns, and in each case, they concluded that the proposed facility would meet governmental public health standards.

Let’s throw a few balls up in the air and try to juggle them.

*First ball:* As previously indicated, IARC has concluded that breathing outdoor air increases everyone’s risk of cancer, meaning that each and every minute of our lives we’re breathing a cocktail of chemical carcinogens.

*Second ball:* For more than 30 years EPA has maintained that any exposure to a known human carcinogen increases cancer risk.

*Third ball:* Based on data collected by the natural gas industry and reported by EPA, the 40.2 million pounds of pollution released by the state’s compressor in a 7-year period included 9.5 million pounds of carcinogens (83% of which were classified as “known human carcinogens” by one or more authoritative agency).

*Fourth ball:* All 18 compressor stations in NYS met all regulatory requirements and were granted building permits.

***If the air we breathe is already filled with hundreds of known human carcinogens, any exposure to a single carcinogen increases the risk of developing cancer and***



***compressor stations add 5.7 million pounds of carcinogens to the state's air every year, how can compressor stations not increase the risk of cancer?***

And is the assertion that “all required studies” fully protects public health in any meaningful sense plausible?

Anyone looking for an insight into the growing disillusionment of ordinary citizens with the regulatory process should attempt this logical juggling act.

### **Engineers and Industry Spokesmen Public Relations Posing as Public Health Experts**

It also needs to be understood that the industry spokesman and DEC officials attempting to assure the public that compressor stations pose no threat to human health, are almost without exception people who have no training in public health, epidemiology or toxicology.

In the case of industry representatives, they are public relations specialists or company spokesman who have memorized their lines.

And in the case of the DEC, they are well-qualified scientifically trained engineers with different areas of expertise, but this is not equivalent to being a scientifically trained medical or public health expert.

### **The NYS DOH**

It must also be recognized that historically DOH is reluctant to weigh in on such matters, preferring to let DEC carry the load.

In this connection, it's worth noting that we could locate no systematic analysis of compressor station pollution authored by the DOH.

The public only hears what engineers and public relations officials have to say on the health effects of compressor stations and natural gas pipelines (“all required studies have been completed”) and little or nothing from the agency specifically tasked with protecting public health.

## 1.6. The Precautionary Principle and Legal Damage Awards

In most cases legislative action to protect the public from the danger of chemical exposures generally only occurs long after harm has been done. This highlights the weakness of the existing approach to chemical regulation—if regulations were adequate there would not be so many effected populations.

Rather than waiting for harm to occur, progressive public health advocates believe regulation should be based on the precautionary principle --concept that when there is some evidence for harm from a particular exposure, but the level of evidence falls short of being clearly established, steps should none-the-less be taken immediately to reduce exposure. Study of hazards from environmental exposures often take many years for definitive results to be obtained, and if one waits to take action until you can count the bodies you will have unnecessarily harmed people. Furthermore, the latency for many diseases is long, and therefore if you take no steps to reduce exposure once the evidence becomes totally clear there will be people who have been unnecessarily exposure who will be developing diseases for many years to come.

Finally, in legal cases it is common for juries to award damages based on proof of exposure to a substance known to cause a disease such as cancer, even if that person does not have cancer him or herself. The level of proof of exposure is usually that the body burden of a substance, for example PCBs or dioxins, is significantly higher than most Americans (usually somewhere between the 75-95th percentile from NHANES). Therefore, courts accept the concept that exposure to a chemical that causes a known disease proves elevated risk of the disease even if the disease has not yet occurred.

## 1.2. The Existing Literature

### 1.2.1. Peer-reviewed studies of natural gas compressor stations

To the authors' knowledge, there are no peer-reviewed papers examining the health effects associated with pollutants generated by natural gas compressor stations.

PubMed (pubmed.gov), a free resource developed and maintained by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine® (NLM), provided access to more than 27 million citations for biomedical and life science research. The search terms "compressor station" and "natural gas compressor" only generate 28 citations. Only 9 of the 28 have as the primary subject matter compressor stations. In the others, compressor stations are essentially a passing reference.

None provide a quantitative assessment of the volume of compressor station emissions, chemical content of these emissions or their potential health impacts.

Subject	Author
<b>Compressor Stations: major subject of the paper</b>	
Compressor stations, criteria pollutants	Goetz et al. 2015
Compressor stations, methane emissions	Jakober et al. 2014, Litto et al. 2012, Mayfield et al. 2017, Nathan et al. 2015, Subramanian et al. 2015
Compressor stations, ocular melanoma associated w\electric motor frequency	Milham and Stetzer 2017.
Compressor stations, operations, pipeline energy optimization	Liu et al. 2014
Compressor stations, operations, turbine improvement	Mohamed et al. 2016
Compressor stations, triaryl phosphate poisoning in cattle.	Beck et al. 2012
Compressor stations, vent operations	García et al. 2012
Compressor stations, wildlife impacts from noise	Bunkley, et al. 2017
Fracking operations, animal health	Bamberger and Oswald 2014
<b>Compressor Stations: passing reference</b>	
Fracking operations, chemical pollution	Brown et al. 2015, Hildenbrand et al. 2016, Pekney et al. 2014, Lan et al. 2015, Lavoie et al. 2015, Chepenko et al. 2012)
Fracking operations, radon	Chepenko et al. 2012
Gas processing, ozone formation	Olaguer 2012
Health impacts, noise	Boyle et al. 2016
Methane emissions, plume characteristics	Payne et al. 2016
Natural gas operations, beef cattle reproduction and calf mortality	Waldner et al. 2012
Natural gas operations, CAP	Roy et al. 2014
Natural gas operations, methane emissions	Allen et al. 2014
Natural gas operations, methane emissions	Yacovitch et al. 2015
Natural gas operations, methane emissions	Zimmerle et al.
Natural gas operations, VOC emissions	Zielinska et al. 2015

#### 1.2.2. NYS DEC

The author could locate no specific statement reviewing the volume of releases of emissions from compressor stations, their content or the impact of these chemicals on human health on DEC's website.

#### 1.2.3. NYS DOH

In December 2014, NYS DOH published *A Public Health Review of High Volume Hydraulic Fracturing for Shale Gas Development* (NYSDOH 2014). The report provides a useful summary of peer-reviewed studies of the health impacts of unconventional gas development including compressor stations. To be precise, it contains 5 references to "compressor stations". But nowhere in this 186-page report is there a reference to the volume of emissions from compressor stations, their content or the impact of these chemicals on human health.

#### 1.2.4. National Academy of Science's Health Impact Assessment of Shale Gas Extraction

In 2014 the National Academy of Science published *Health Impact Assessment of Shale Gas Extraction: Workshop Summary* (NYAS 2014). The report contains a great deal of important information about the pollution associated with fracking but almost nothing concerning the pollution associated with the transportation of natural gas.

The phrase "compressor station" appears 13 times. None of these references provide any quantitative assessment of compressor station emissions.

But the last reference to compressor stations which appears in the report's final paragraph does say something quite important.

Finally, the panel was asked to comment on the testing of acute, short-term exposures versus low-level chronic exposures, for example, the low-level chronic exposures of farmers who leased out their land for hydraulic fracturing or homeowners who are living 100 feet from a compressor station and live with these emissions daily. The audience member noted that there has been remarkably little air and water testing in the U.S. gas fields to date, and the available testing efforts have shown exposures at "safe" levels, which is disheartening for people experiencing a multiplicity of health symptoms at these levels. . .

### 1.2.5. U.S. Environmental Protection Agency, Inspector General

A 2013 report from the United States Environmental Protection Agency 's Inspector General states that there is inadequate information available on direct measurement emissions from oil and gas production activities.

High levels of growth in the oil and natural gas (gas) production sector, coupled with harmful pollutants emitted, have underscored the need for EPA to gain a better understanding of emissions and potential risks from the production of oil and gas. However, EPA has limited directly-measured air emissions data for air toxics and criteria pollutants for several important oil and gas production processes and sources, including well completions and evaporative ponds. Also, EPA does not have a comprehensive strategy for improving air emissions data for the oil and gas production sector; the Agency did not anticipate the tremendous growth of the sector, and previously only allocated limited resources to the issue. (USEPA 2013)

### 1.2.6. U.S. Agency for Toxic Substances and Disease Registry

A 2016 report by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) of health particulate matter exposures in the vicinity of the Williams Central natural gas compressor station in Brooklyn Township, Susquehanna County, Pennsylvania, reached 2 fundamental conclusions:

**Conclusion 1, Short term exposures:** Exposure to maximum levels of PM<sub>2.5</sub> may be harmful to unusually sensitive populations, such as those with respiratory or heart disease, but are not at levels that are a concern to the general population.

**Conclusion 2, Chronic exposures:** The estimated annual average PM<sub>2.5</sub> concentration of 15 to 16 µg/m<sup>3</sup> may be harmful to the general population and sensitive subpopulations, including the elderly, children, and those with respiratory or heart disease.

(USATSDR 2016)

### 1.3. Requests for Information

The need for quantitative information about the volume of pollution and its potential to cause adverse health impacts has been the subject of resolutions by local governments, public health advocates as well as local, regional and state environmental organizations.

#### 1.3.1. Letter to Mr. Michael Higgins, NYS DEC, Division of Environmental Permits From the Westchester County Board of Legislators (February 2015)

Board of Legislators Resolution No. 80-2014 Resolution No. 80-2014 (“Resolution”) calls for independent, transparent, continuous and comprehensive baseline air testing to establish the public’s exposure to toxins that are currently being emitted from the compressor stations prior to allowing any increase in emissions. Furthermore, the resolution calls upon all involved agencies, including NYSDEC, to evaluate cumulative short and long-term health impacts of the entire proposed AIM project, including the impact of emissions from all regional sources of emissions, prior to any expansion of these compressor stations. (Westchester 2015)

#### 1.3.2. Letters to Governor Cuomo and Health Commissioner Howard A. Zucker From Concerned Health Professionals of New York, Physicians for Social Responsibility -- U.S., Physicians for Social Responsibility -- New York (October 14, 2015, May 29, 2014, February 27, 2013, October 4, 2012, March 16, 2012, December 12, 2011, October 5, 2011, March 26, 2011, February 28, 2011)

Compressor stations and pipelines are both major sources of air pollutants, including benzene and formaldehyde, that create serious health risks for those living nearby while offering little or no offsetting economic benefits. Compressor stations – used along regular intervals of most pipelines – in particular, are semi-permanent facilities that pollute the air 24 hours a day and expose nearby residents to levels of noise pollution known to induce negative health effects. Moreover, emerging data show that their day-to-day air emissions are highly episodic and create periods of potentially extreme exposures.

We have particular concerns about the air pollution events created by compressor station “blowdown” events, which are used for maintenance and to control pressure and can last for hours. The intentional or accidental releases of gas through valves create 30- to 60-meter-high gas plumes, causing high levels of contaminant release. Anecdotal accounts associate blowdowns with short term effects such as nosebleeds, burning eyes and throat, skin irritation, and headache. Given the chemicals released, we are deeply concerned about the possible long-term effects of these exposures, including cancer, asthma, heart disease and severe neurological impairments. We note that there exists neither a national nor a state inventory of compressor station accidents. We have yet to accumulate an extensive body of peer-reviewed research on the public health impacts of compressor stations, but our new report includes very troubling documentation of extensive leakage of methane and other contaminants. (CHPNY)

### 1.3.3. Letter to Mr. Christopher Hogan, NYSDEC, Division of Environmental Permits

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From 14 NYS Physicians (September 12, 2016)

We are . . . greatly concerned that no state agency appears to be monitoring the ever increasing flow of information and scientific studies based on effects of UNGD and its associated infrastructure. On this point we would like to be mistaken; and please correct us if we are. But since the two-year Public Health Review of HVHF activities concluded in December 2014, it appears that DEC and DOH have “washed their hands” of the issue. Nevertheless, huge problems - with health impacts, quality of life and climate impacts associated with the proliferation of natural gas infrastructure in our state - are not going away. Someone needs to be paying attention to this, and “connecting the dots” - individual infrastructure projects must be considered not only separately but in their cumulative health, environmental, and climate impacts. We do not see this happening in New York State. (Medical Professionals 2016)

### 1.3.4. Public Statement: Mothers Out Front Mobilizing for a Livable Climate (Monroe County NY)

It is somewhat difficult to find scientific literature completely focused on the impacts of compressor stations. . . (MOF)

## 1.4. Summary of health effects

Based on data submitted by the natural gas industry and collected by DEC and EPA we show that 18 compressor stations in NYS were responsible for releasing more than 40 million pounds of toxic air pollutants over a 7-year period, including:

- 9.5 million pounds of human carcinogens,
- 18.8 million pounds of chemicals associated with blood and immune system disorders,
- 18.8 million pounds associated with endocrine, nutritional and metabolic disorders,
- 18.7 million pounds of chemicals with mental and behavioral effects,
- 18.7 million pounds that affect the brain and central nervous system,
- 24.7 million pounds that affect the eye and adnexa,
- 17.5 million pounds that affect the ear and mastoid process,
- 16.3 million pounds associated with circulatory system diseases including heart attacks and strokes,
- 39.6 million pounds linked to respiratory system diseases,
- 38.5 million pounds linked to digestive system diseases,
- 27.7 million pounds associated with skin and subcutaneous tissue disorders,
- 1.2 million pounds linked to musculoskeletal system and connective tissue diseases,
- 39.9 million pounds with genitourinary system diseases,
- 6.4 million pounds with urinary system diseases and disorders,
- 39.9 million pounds connected to pelvis, genital and breast diseases including reproductive disorders
- 19.6 million pounds that affect pregnancy, childbirth and the puerperium,
- 22.5 million pounds with certain conditions originating in the perinatal period,
- 39.6 million pounds with congenital malformations, deformations, chromosomal abnormalities, and
- 39.6 million pounds with symptoms, signs and abnormal clinical and laboratory findings, nec.

### **What should the public make of this?**

#### 1.4.1. A substantial amount of health relevant information is not reaching the public

A tenant of health and community governance is public information and health and safety. Community members depend on Public Officials to provide information needed to protect their health. In the case of gas compressor stations substantive amounts of health relevant information is not reaching the public. Instead of informing the public, the information is mired in bureaucratic processes.



#### 1.4.2. Governments' failure to analyze or communicate the results of its own data collection

The data analyzed in this report is collected by DEC and EPA. Neither agency has reported the total volume of pollution associated with the transportation of natural gas, let alone analyzed these releases in terms of how they potentially impact human health.

It is the responsibility of each compressor station in NYS to estimate the total volume of pollution they generate as well as its chemical constituents and to report this information to DEC--and they do.

DEC is required to review this information and forward it to EPA--and they do.

EPA assembles this data and make it available to the public, which they do, not just for compressor station but for millions of other sites nationally.

It is not the most difficult thing in the world to use the information collected and apply a little math. But, if either the EPA or DEC have ever done so, it is not something to our knowledge that has been presented to the public. Nor have we found this information on their respective web sites.

The information that is collected is presented in formats that are not readily understood by local residents. To be useful, the most important conclusions and insights of technical data must be comprehensible to the average person.

#### 1.4.3. DOH's failure to analyze the potential health impacts of compressor station pollution

For its part, even if EPA and DEC are not interested in analyzing the compressor station emission data they collect, there is nothing to prevent DOH from doing so.

DEC\EPAs' compressor station data is publicly available. DOH could download it and do the math.

Again, as far as the authors are aware, DOH has not done so.

#### 1.4.4. Industry and governmental assurances that gas compressor stations “comply with all air quality requirements” and that they therefore pose no unreasonable threat to public health

Each of the 57 compressor stations that have been permitted by DEC have been approved based on the conclusion that they comply with all federal and state air quality requirements.

When members of the public or local officials question the potential health effects of compressor station pollution, the response from industry, EPA, DEC and DOH is always that “all legal requirements have been met” -- the clear implication being that if these “legal requirements” have been met, there is no reason to be concerned about adverse health effects.

For example, in a public statement issue by Dominion Transmission concerning its New Market Project, it states: “The FERC approved New Market on April 28, 2016 after 23 months of evaluating all environmental, health and safety concerns associated with the project.”

Dominion poses the question, “What will be the environmental and public health concerns?”

Its answer:

Any emissions from the compressor station will comply with all air quality requirements, which are established to protect the public health, safety and welfare. We would not operate the compressor station if we could not operate it according to stringent air quality regulations.

Ensuring compliance with environmental requirements fall either to the Environmental Protection Agency (EPA) or state environmental agencies (states by delegation), depending on the specific permit and rule. (Dominion 2016)

Dominion is disingenuous. Their health and safety officials recognize, or should recognize, that Federal and State Environmental laws and regulations are designed to protect the general health of regional populations and not to protect any single group of locally exposed persons. The “stringent air quality regulations” that Dominion refers to are those established by EPA for the purpose of controlling regional levels of pollution and even in this limited context these regulations fail to protect the public from all manner of demonstrably harmful exposures.

Therefore, these regulations make three critical, scientifically questionable assumptions when applied to local pollution.

The “stringent air quality regulations” that Dominion refers to are those established by EPA.

It is beyond the scope of this project to provide a full analysis of this claim, but several brief observations are in order.

First, the regulation used to determine the potential health impacts of chemical exposure make three critical, scientifically questionable assumptions.

##### 1. Individual Chemicals versus Chemical Mixtures

First, it effectively assumes that an air standard can be set for a single chemical to protect against unnecessary risk. The problem here is that setting standards for individual chemicals makes very little sense when people are continuously exposed to multiple complex chemical mixtures.

The mixture of any two chemicals can be additive ( $1 + 1 = 2$ ), less than additive ( $1 + 1 = 1.5$ ), or synergistic ( $1 + 1 = 2+$ ).

NYS's national gas compressor stations reported releasing more than 70 chemicals.

This number includes 31 chemicals associated with circulatory diseases like heart attacks and strokes. Knowing how much risk is associated with a single circulatory toxicant tells us nothing about the real-world risk of being exposed to 31.

The state's compressor station reported releasing a total of 59 carcinogens, including 23 chemicals classified as "known human carcinogens" by an authoritative international, federal or state agency. Of the 9.6 million pounds of carcinogens released from 2008 to 2014, 83% were known to cause cancer in humans. The effect of being exposed to multiple carcinogens is not considered in EPA or DEC regulations.

## **2. Average versus Acute Exposures**

EPA and DEC guidelines assume that the exposures from a facility occur evenly over time. In fact, for any given facility air pollution releases fluctuate widely. What this means is that at times of peak exposure individuals may breathe chemical concentrations greater than what is assumed to be safe. Studies have shown that levels of the carcinogens formaldehyde, benzene and 1,3-butadiene periodically exceed what federal and state guidelines consider safe at natural gas compressor stations in four states. (Macey et al. 2014).

## **3. Healthy versus health-compromised or particularly vulnerable populations**

With a few exceptions, EPA and DEC guidelines assume that everyone is at equal risk from the harmful effects of air pollution. This is simply not the case. There are many populations who are at greater risk, including, infants, children, pregnant women, the elderly, those with compromised immune systems, and those already suffering from specific diseases or disorders.

Studies have shown that the cancer rates are proportional to levels of regional air pollution. These three points aside, it is plausible that the release of 9.6 million pounds of carcinogens in a 7-year period, and continued releases over the active use of the facility does increase the incidence of certain types of cancer.

When national economic concerns are given priority higher than community health it is necessary to provide simple and clear guidance to the person whose risk is increased. In those situations, it is not appropriate to compare risks to other sources or situations. Individuals who know that they are of enhanced risks, avoid polluted areas.

The environmental and health risks can be handled much more fairly. To do so requires recognition of commonly understood concepts and readily accessible information.

## **4. Other general concerns**

Problems associated with risk assessments broadly understood and air pollution standards have been identified by a number of researchers (Goodman et al. 2013, Maynard et al. 1995, McClellan 2012, Mauderly et al. 2010.)

#### 1.4.5. The absence of concrete information about potential health impacts in industry proposals

In the proposals we have reviewed, there is an absence of concrete information about exposures and their potential health impacts.

Millennium's proposal for its Highland Compressor Station is a case in point.

In July 2016, the company submitted its application proposal, *Millennium Pipeline Company, LLC Highland Compressor Station, Eastern System Upgrade Project, Air State Facility Permit Application* to regulators.

The 83-page report contains a great amount of important information but almost nothing substantive about potential adverse human health impacts.

A search of the document shows that the word "health" only appears four times

First, as a footnote to table **Table 2-1: Proposed Facility Emissions (tons/year)**.

(3) Trivial per 201-3.3(94) for emissions of "...oxygen, carbon dioxide, nitrogen, simple asphyxiants including methane and propane, trace constituents included in raw materials or byproducts, where the constituents are less than 1 percent by weight for any regulated air pollutant, or 0.1 percent by weight for any carcinogen listed by the United States Department of **Health** and Human Services' Seventh Annual Report on Carcinogens (1994). The definition of "regulated air pollutant" under 200.1(bu) does not include methane or ethane.

Second, in section **3.4.1 Exempt and Trivial Sources**, the same sentence is repeated.

Blowdowns are considered a trivial activity per 6 NYCRR 201-3.3(94) which covers "Emissions of the following pollutants: water vapor, oxygen, carbon dioxide, nitrogen, inert gases such as argon, helium, neon, krypton and xenon, hydrogen, simple asphyxiants including methane and propane, trace constituents included in raw materials or byproducts, where the constituents are less than 1 percent by weight for any regulated air pollutant, or 0.1 percent by weight for any carcinogen listed by the United States Department of **Health** and Human Services' Seventh Annual Report on Carcinogens (1994)." The natural gas composition at the Highland Station meets the definition in 6 NYCRR 201-3.3 as shown in Appendix B.

Third, in section **3.6 New York State Department of Environmental Conservation Regulations:**

If the agency considers that any project triggering minor NSR permitting could threaten attainment with the National Ambient Air Quality Standards (NAAQSs) or human **health** from toxic air pollutant (TAP) concentrations, NYSDEC can require air dispersion modeling for the Project. A site wide modeling analysis for criteria pollutants has been performed in accordance with their impact analysis modeling guidance, Policy DAR-10. In addition, a modeling analysis that addresses TAPs is performed per Policy DAR-1. This section details the NAAQS and TAPs modeling assessment for the proposed Highland Station.

And fourth, as an unchecked box in Rule Citation 201-3.2(c), item 20, "Municipal/Public **Health** Related."

A subsequent report by the company, Millennium Pipeline Company, LLC, Eastern System Upgrade Project, Hancock and Highland Compressor Stations, Human Health Risk Assessment (February 2017), addresses health concerns more explicitly. But, again, there is (a) no attempt to place compressor station pollutants within the context of the overall burden of pollution in NYS or (b) explicit discussion of the what the peer-reviewed science has to say about the potential health effects of releases.

## 1.6. Other sources of exposure to the 70 chemicals released by natural gas compressor stations

In the course of a single day each of us is exposed to hundreds of toxic chemicals. Over a lifetime, the number is in the thousands or, more probably, tens of thousands.

It is often assumed, mistakenly in our view, that continuous exposure to high levels of toxic chemicals are required to produce illness. In fact, illness may result from a small exposure, especially if exposure occurs continuously over time.

Compressor stations represent a significant source of airborne pollution in NYS, increasing the likelihood of disease, particularly for local communities. The potential health effects of compressor stations are clearly a principal concern for communities opposing their construction or expansion. As we try to show in these pages, the potential for harm is real and their concern justified. At the same time, it's necessary to not lose sight of the forest when we're looking at the trees. Pollution from compressor stations is a significant threat to human health, but it is one of many. All need to be understood and addressed.

A few essential facts:

- Each year the U.S. manufactures or imports trillions of pounds of chemicals. Of the 70 chemicals released as combustion pollutants from natural gas compressor stations, 27 are produced at a volume of more than 1 million pounds annually in the U.S. Of this number, 13 are produced at more than 1 billion pounds and 3 at more than 10 billion pounds. (Given the limitations of EPA's reporting on chemical manufacturing and importation, these numbers are in all probably underestimations.)
- Companies don't manufacture or import chemicals with the intention of creating pollution but to use them in products. In the course of **production**, a relatively small percentage of chemicals are released into the environment. In absolute numbers, of course, the volume of air and water pollution released and hazardous waste generated is extraordinary, but the most significant source of pollution occurs **after production**, when chemicals incorporated into products and are used and in many cases, discarded. The single most important source of airborne pollution in the U.S. is the combustion of gasoline and diesel fuel in on-road vehicles. At present, the U.S. has 139 operating petroleum refineries, located in 39 states. Eighty-nine are located just four states: Texas, California, Oklahoma, and Louisiana, and these account for more 10 million of the 16.7 billion barrels produced daily. Studies consistently find extremely high levels of air and water pollution and significantly higher rates of human disease around refineries. But far more pollution is created and more people are exposed to its harmful effects when the 7 billion barrels of petroleum produced each year in the U.S. are used, most notably, when they are burned in cars, trucks, buses and planes or used as fuel for heat or electricity generation.
- Prior to EPA's creation in 1970, there was literally no national regulation of chemical production or chemical waste disposal and only the flimsiest air and water pollution regulations. In 1970, an estimated 65,000 chemicals were in use in the U.S. EPA "grandfathered" these chemicals, meaning that companies could continue to use them until the Agency had time to determine if their use should be continued. New chemicals would have to be approved for use by EPA prior to introduction, but they didn't have to be tested. Nearly a half century later, more 85,000 chemicals have now been approved for use by EPA under Toxic Substances Control Act (TSCA), but fewer than 1,000 have been systematically

evaluated for their potential to harm human health and the environment by a federal agency (EPA and NTP), and only a small number have been withdrawn from use.

- Even when a chemical is clearly shown to present significant harm to human health or the environment, it is extremely difficult to have its use terminated. The fact that a chemical has been shown to be a known human carcinogen does not, for example, mean its use is automatically prohibited or restricted. This only happens in the rarest of circumstances and only longer after the problem has been documented. The current controversy concerning chlorpyrifos, a pesticide known to affect childhood brain development, is a case in point.
- Of the estimated 85,000 chemicals approved for use in the U.S. by EPA, fewer than 1,500 are systematically tracked as environmental pollutants or as food and water contaminants. Of this number, fewer than 900 are tracked as air pollutants.
- Of the 319 chemicals in EPA National Emissions Inventory, 198 were reported as air pollutants in NYS in the period from 2008 to 2014, meaning the state's residents were exposed to 128 additional chemicals not related as compressor station releases.
- The 70 chemicals released as compressor station stack air pollutants can be found in many other point sources or air pollution reported by NEI. These same 70 chemicals are reported as non-point sources by NEI, and 40 as on-road and non-road sources. Thirty-five of these 70 chemicals are residential air pollutants.
- Forty-one of the 70 chemicals released as compressor station stack can be found in clothing and textiles, jewelry, personal care products, cosmetics, perfumes, skin, hair care products, hair dyes, shoes and leather products, tobacco products/smoking.
- Forty-four of the 70 chemicals released as compressor station stack pollutants can be found on food items.
- In recent years CDC's NHANES has studied the number of chemical contaminants found in our bodies. These studies and those of the Environmental Working Group (EWG) have shown that chemical contaminants found in our bodies are varied as are their potential health impacts., and that even those working in relatively "clean" occupations also suffer significant contamination. Perhaps the most startling finding is that chemical contamination occurs before births. Dozens of toxic chemicals can be found in umbilical cord blood or placenta, including many chemicals known or suspected of causing human cancer.

Of the 70 chemicals released as compressor station stack pollutants, 48 are documented body burden contaminants, including: blood (29), breast milk (17), umbilical cord (20), placenta, sweat (3), urine (11), and unspecified (1).

- Air Pollution \ Cancer
  - Outdoor air is a known human carcinogen. (IARC)
  - In 2005, nearly all U.S. children (99.9%) lived in census tracts in which hazardous air pollutant (HAP) concentrations combined to exceed the 1-in-100,000 cancer risk benchmark. (US EPA)
  - 7% of children lived in census tracts in which HAPs combined to exceed the 1-in-10,000 cancer risk benchmark. (US EPA)
- Air Pollution \ Non-neoplastic diseases (health effects other than cancer)
  - 56% of children lived in census tracts in which at least one HAP exceeded the benchmark for health effects other than cancer. (U.S. EPA)

- In 2015, 59% of U.S. children lived in counties with measured pollutant concentrations above the levels of one or more national ambient air quality standards. (U.S. EPA)



- Drinking Water \ Health Standards
  - In 2015, approximately 7% of children served by community drinking water systems that did not meet all applicable health-based standards. (U.S. EPA)
  - Between 1993 and 2015, the estimated percentage of children served by community water systems that had at least one monitoring and reporting violation fluctuated between about 10% and 21%, and was 12% in 2015. (U.S. EPA)
- Drinking Water \ Detectable organophosphate pesticide residues (U.S. EPA)
  - In 2009, 35% of sampled apples
  - In 2007, 5% of sampled carrots
  - In 2008, 9% of sampled tomatoes
  - In 2009, 8% of sampled grapes
- Hazardous Waste
  - As of 2009, approximately 6% of all children in the United States lived within one mile of a Corrective Action or Superfund site that may not have had all human health protective measures in place, disproportionately affecting more Black children. (U.S. EPA)

Table 1.6a

Other sources of exposure to the 70 chemicals released by New York's natural gas compressor stations

<b>High production chemicals: &gt;= 1 million pounds annually</b>		<b>27</b>
<b>High production chemicals: &gt;= 1 billion pounds annually</b>		<b>13</b>
<b>High production chemicals: &gt;= 10 billion pounds annually</b>		<b>3</b>
<b>Ambient air</b>		<b>70</b>
01	Ambient, point	70
02	Ambient, non-point	40
03	Ambient, mobile, on-road	40
04	Ambient, mobile, non-road	40
<b>Residential exposures</b>		<b>35</b>
<b>01</b>	<b>Residential, indoor</b>	<b>21</b>
01.01	Residential, indoor: buildings materials, furniture	14
01.02	Residential, indoor: air fresheners, candles, incense	23
01.03	Residential, indoor: home maintenance	2
01.04	Residential, indoor: home office	2
01.04	Residential, indoor: pet care	12
<b>02</b>	<b>Residential, outdoor</b>	<b>10</b>
02.01	Residential, outdoor, landscape and yard	7
02.02	Residential, outdoor, pesticides	7
<b>Our Bodies</b>		<b>41</b>
01	Clothing and textiles	3
02	Jewelry	19
03	Personal care products	3
04	Cosmetics, perfumes, skin	0
05	Hair care products	1
06	Hair dyes	7
07	Shoes and leather products	28
10	Tobacco products / smoking	28
<b>Food</b>		<b>44</b>
<b>01</b>	<b>Food items</b>	<b>10</b>
01.01	Dairy products	12
01.02	Fats, oils, fat emulsions	6
01.03	Edible ices	17
01.04	Fruits, vegetables, nuts, seeds	10
01.05	Confectionery	10
01.06	Cereals and cereal products	12
01.07	Baked products	14
01.08	Meat, poultry, game	25
01.09	Fish and shellfish products	6
01.10	Eggs and egg products	0
01.11	Sweeteners, including honey	5
01.12	Salts, spices, soups, sauces, salads, protein products	8
01.13	Baby food	7
01.14	Beverages, excluding dairy products	14
01.15	Ready-to-eat savorys	9
01.16	Prepared foods	7
01.17	Fast food	5
01.18	Additives, colorings, flavorings	<b>36</b>

Sources: EPA Chemical Data Reporting System (CDRS), FDA Total Dietary Study (TDS), NLM Hazardous Substances Data Bank (HSDB).

Table 1.6b

## Body Burden of Compressor Station Chemicals

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

		Blood	Breast milk	Umbilical cord	Placenta	Sweat	Urine	Unspecified
#	Chemical	29	17	20	2	3	11	1
1	Acenaphthene	Y		Y				
2	Acenaphthylene	Y		Y				
3	Acetaldehyde		Y					
4	Anthracene	Y		Y				
5	Arsenic					Y	Y	
6	Benzene	Y	Y	Y				
7	Benzo(j,k)fluorene	Y		Y				
8	Benzo[a]pyrene							
9	Benzo[b]fluoranthene	Y						
10	Benzo[g,h,i]perylene	Y		Y	Y			
11	Benzo[k]fluoranthene	Y						
12	Beryllium						Y	
13	Butadiene, 1,3-						Y	
14	Cadmium	Y		Y		Y	Y	
15	Carbon monoxide	Y						
16	Carbon tetrachloride	Y	Y	Y				
17	Chlorobenzene	Y	Y					
18	Chloroform	Y		Y				
19	Cobalt						Y	
20	Ethyl benzene	Y	Y					
21	Ethyl chloride (Chloroethane)		Y					
22	Ethylene dichloride	Y	Y					
23	Ethylidene dichloride	Y						
24	Fluorene	Y		Y				
25	Hexane, n-							
26	Indeno[1,2,3-cd]pyrene	Y	Y	Y	Y			
27	Lead	Y				Y	Y	
28	Manganese	Y					Y	
29	Mercury	Y		Y			Y	
30	Methane dichloride	Y	Y	Y				
31	Methanol		Y					
32	Methylnaphthalene, 2-		Y					
33	Naphthalene	Y	Y	Y			Y	
34	Nickel							Y
35	Perchloroethylene	Y	Y					
36	Perylene	Y		Y				
37	Phenanthrene	Y		Y			Y	
38	Polycyclic aromatic hydrocarbons, total (PAHs Total)			Y				
39	Propylene dichloride	Y						
40	Pyrene	Y		Y				
41	Selenium	Y		Y				
42	Styrene		Y	Y				
43	Sulfur dioxide		Y					
44	Tetrachloroethane, 1,1,2,2-	Y						
45	Toluene	Y	Y					
46	Trichloroethane, 1,1,2-	Y						
47	Vinyl chloride						Y	
48	Xylene (mixed isomers)	Y	Y					

## 1.7. President's Obama's Cancer Panel

These specific points aside, it's useful to consider the claim that environmental pollution from natural gas compressor stations poses no threat to human health in the context of the findings of the President's Cancer Panel (2010).

Between September 2008 and January 2009, the President's Cancer Panel (PCP) convened four national meetings "to assess the state of environmental cancer research, policy and programs addressing known and potential effects of environmental exposure on cancer."

The Panel's report, released in 2010, came to this essential conclusion:

"Research on environmental causes of cancer has been limited by low priority and inadequate funding. . . There is a lack of emphasis on environmental research as a route to primary cancer prevention. . . Cancer prevention efforts have focused narrowly on smoking, other lifestyle behaviors and chemo-preventive interventions. Scientific evidence on individual and multiple environmental exposure effects on disease initiation and outcomes, and consequent health system and societal costs, are not being adequately integrated into national policy decisions and strategies for disease prevention, health care access and health system reform." (U.S. DHHS 2010)

In the light of Panel's conclusion, the results of our study raise three significant questions:

First, what percentage of cancers is likely a consequence of exposure to chemical carcinogens?

Secondly, what is the impact of occupation as compared to non-occupation exposures?

Finally, how adequate are policies that both inform people of hazards and act to reduce exposure to chemical carcinogens?

There is considerable debate on the question of what percentages of cancers are due to exposure to chemical carcinogens, both in the workplace and elsewhere. Doll and Peto (1981) "provisionally estimated" that 4% of cancer was due to occupational exposures, but attributed most of this to lung cancer. Mokdad et al. (2004) ascribed only 2.3% of causes of death in the US to "toxic agents", but then attributed 18.1% to tobacco and 16.6% to poor diet and physical inactivity, not distinguishing the degree to which either was due to chemicals in tobacco or food. Schottenfeld et al. (2013) list tobacco, alcohol, ionizing radiation, solar radiation, infectious agents and obesity as risk factors for cancer, but totally ignore other chemical carcinogens other than occupational exposures. Prüss-Ustün and colleagues from the World Health Organization (2016) attribute 19% of all cancer to environmental factors, which includes 2-8% due to exposure in occupational exposures. Their report does not consider smoking, alcohol, diet or genetic factors. In discussing specific cancers, they attributed colon and rectal cancer to low physical activity, radiation and asbestos, but do not mention other chemical carcinogens in food. Chemical exposure is identified as a risk factor for breast, lymphoma, multiple myeloma, leukemia, larynx, bladder and melanoma cancers. Clapp et al. (2008) note that while overall cancer rates are declining (especially lung among men and colorectal in both sexes), some are rising (esophagus, liver, thyroid, melanoma, non-Hodgkin's, multiple myeloma, testicular, bladder, brain, and lung in women). Childhood cancers (leukemia and brain) are also rising. They and Belpomme et al. (2007) provide strong evidence that exposure to carcinogenic chemicals plays a major role in risk of these cancers. Christiani (2011) has suggested that 85-95% of cancer arise because of exposure to specific carcinogenic agents.

In addition to exposure to chemical carcinogens, cancer can be caused by genetics, infection and inflammation. Lichtenstein et al. (2000) reported an analysis of mono- and di-zygotic twins in Scandinavia in an effort to distinguish genetic from environmental factors in causation of cancer. They concluded that most cancers were due to environmental factors. Genetic factors were relatively unimportant in most cancers, although were significant in prostate (42%), colorectal (35%) and breast cancer (27%). Wu et al. (2016) examined intrinsic and extrinsic risk factors for cancer, and concluded that intrinsic factors contribute only modestly (less than 10-30%) to the risk. This is not to imply that individual genetic differences are unimportant, because polymorphisms of drug metabolizing enzymes serve as modulators of cancer susceptibility (Taningher et al., 1999).

These reports indicate that we do not have good understanding of the relative role of exposure to chemical carcinogens in overall cancer incidence beyond general knowledge that many chemicals to which humans are exposed cause cancer. Clearly carcinogenic chemicals are found in both the occupational and non-occupational environment. While the chemical exposure in an occupational setting differs from that of the general population, there are many carcinogens found in food, tobacco, personal care products, and indoor and outdoor air. Many use terms such as “life-style” to encompass such behaviors, without considering the carcinogenic chemicals that result from these behaviors. Workers have all of these non-occupational exposures as well as those specific to the workplace.

EPA’s official policy is that exposure to any level of carcinogen increases the risk of cancer. At the same time, EPA has a methodology to determine the extent of risk that rarely finds excessive risk. It is hard to fathom how the release of 9.6 million pounds of carcinogens in a 7-year period does not increase the incidence of cancer.

## Chapter 2. Compressor Station Releases

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2.1. Number, Categorization and Operational Status of Facilities

This report analyzes the emissions data for 18 natural gas compressor stations in New York State (NYS) as reported to the National Emissions Inventory (NEI) of the U.S. Environmental Protection Agency (EPA) as point sources of air pollution for the period 2008 to 2014.

The author could not locate a single list of facilities involved in the storage and transportation of natural NYS from either state or federal sources.

One was created by reviewing all DEC air permits and identifying those that are compressor stations and comparing it to those listed in EPA’s Envirofacts System.

This is a little more complicated than it might seem because there are a large number of permits and permit modifications, all compressor stations are not necessarily labeled as such, there are apparent inconsistency in NAICS and SIC classifications, and some sites have multiple functions.

We identified a total of 58 compressor stations, including operational (55), approved (2), denied (1), and pending (3).

This list provides what the author believes is an accurate characterization of major natural gas facilities in NYS but it should not be considered comprehensive or final. Additional research would undoubtedly identify additional sites, especially minor ones, and clarify the function of several sites.

Table 2.1

Facility Type	Total
Compressor stations	58
Operational	55
Proposed: Approved	2
Proposed: Denied approval	1
Dehydration facility	1
Gas turbine facility	4
Gate	3
Holding point tap	2
Metering & regulation station	19
Pig launching or receiving	1
Storage & filling	1
Unknown	2



## 2.2. NYSDEC Air Pollution Control Permits and Registrations

Under the Clean Air Act and under New York State law and regulation, most notably 6 NYCRR Part 201, NYSDEC is required to issue permits for polluters. The two most common permits for large on-site polluters are: “Air Title V Facility permits” and “State facility permits”.

NYSDEC describes these as follows:

### 2.2.1. State Facility Permits

State facility permits are issued to facilities that are not considered to be major (as defined in the department's regulations), but that meet the criteria of Subpart 201-5. ([link leaves DEC](#)) These are generally large facilities with the following characteristics:

- Their actual emissions exceed 50 percent of the level that would make them major, but their potential to emit as defined in 6NYCRR Part 200 does not place them in the major category
- They require the use of permit conditions to limit emissions below thresholds that would make them subject to certain state or federal requirements
- They have been granted variances under the department's air regulations, or
- They are new facilities that are subject to New Source Performance Standards (NSPS) or that emit hazardous air pollutants. Instead, all DEC site.

### 2.2.2. Title V Permits

Title V facility permits, the second type of permit, are issued to facilities subject to Subpart 201-6. These include facilities that are judged to be major under the department's regulations, or that are subject to New Source Performance Standards (NSPSs), to a standard or other requirements regulating hazardous air pollutants or to federal acid rain program requirements.

Title V permits reduce violations of air pollution laws and improve enforcement of those laws by:

- Recording in one document all of the air pollution control requirements that apply to the source. This gives members of the public, regulators, and the source a clear picture of what the facility is required to do to keep its air pollution under the legal limits.
- Requiring the source to make regular reports on how it is tracking its emissions of pollution and the controls it is using to limit its emissions. These reports are public information, and you can get them from the permitting authority.
- Adding monitoring, testing, or record keeping requirements, where needed to assure that the source complies with its emission limits or other pollution control requirements.
- Requiring the source to certify each year whether or not it has met the air pollution requirements in its title V permit. These certifications are public information.
- Making the terms of the title V permit federally enforceable. This means that EPA and the public can enforce the terms of the permit, along with the State.

(Source: NYSDEC)

The legal intention of a Title V operating air permit is described by DEC as:

The Title V operating air permit is intended to be a document containing only enforceable terms and conditions as well as any additional information, such as the identification of emission units, emission points, emission sources and processes, that makes the terms meaningful. 40 CFR Part 70.7(a)(5) requires that each Title V permit have an accompanying "...statement that sets forth the legal and factual basis for the draft permit conditions". The purpose for this permit review report is to satisfy the above requirement by providing pertinent details regarding the permit/application data and permit conditions in a more easily understandable format. This report will also include background narrative and explanations of regulatory decisions made by the reviewer. It should be emphasized that this permit review report, while based on information contained in the permit, is a separate document and is not itself an enforceable term and condition of the permit.

### 2.2.3. Changing permit status over time

A facility's permit status can change over time.

A station initially permitted with a permit type "Air State Facility" may subsequently seek site expansion or modifications which DEC may determine requires a "Title V" permit, or the reverse might be true.

To determine the present status of a particular station, see NYS DEC air permits:

**Issued Title V Permits**

[http://www.dec.ny.gov/dardata/boss/afs/issued\\_atv.html](http://www.dec.ny.gov/dardata/boss/afs/issued_atv.html)

**Draft Title V Permits**

[http://www.dec.ny.gov/dardata/boss/afs/draft\\_atv.html](http://www.dec.ny.gov/dardata/boss/afs/draft_atv.html)

**Issued State Facility Permits**

[http://www.dec.ny.gov/dardata/boss/afs/issued\\_asf.html](http://www.dec.ny.gov/dardata/boss/afs/issued_asf.html)

**Draft State Facility Permits**

[http://www.dec.ny.gov/dardata/boss/afs/draft\\_asf.html](http://www.dec.ny.gov/dardata/boss/afs/draft_asf.html)

## 2.3. Reporting Requirements for Compressor Stations with Title V Permits

Each permitted point-source of pollution must meet one or more state or federal reporting requirements.

The two tables which follow summarize the principal air pollution regulatory programs applicable for each of the 18 NYS compressor stations under review.

Table 2.3.1.

### Applicable State and Federal Air Pollution Regulatory Programs by Facility

#### NYS Natural Gas Compressor Stations

			State and Federal Air Pollution Regulatory Programs									
Facility	County	Town	PSD	NSR	NESHAP	MACT	NSPS	Title IV	Title V	Title VI	RACCT	SIP
Count ("Yes"):			4	0	0	14	7	0	18	0	18	18
AGT Southeast CS	Putnam	Southeast	No	No	No	No	Yes	No	Yes	No	Yes	Yes
AGT Stony Point CS	Rockland	Stony Point	Yes	No	No	Yes	Yes	No	Yes	No	Yes	Yes
DTI Borger CS	Tompkins	Ithaca	Yes	No	No	No	Yes	No	Yes	No	Yes	Yes
DTI Utica Station	Herkimer	Frankfort	Yes	No	No	No	No	No	Yes	No	Yes	Yes
DTI Woodhull Station	Steuben	Woodhull	No	No	No	Yes	Yes	No	Yes	No	Yes	Yes
NFGSC Beech Hill CS	Allegany	Willing	No	No	No	Yes	No	No	Yes	No	Yes	Yes
NFGSC Concord CS	Erie	Concord	No	No	No	Yes	Yes	No	Yes	No	Yes	Yes
NFGSC Independ. CS	Allegany	Andover	No	No	No	Yes	No	No	Yes	No	Yes	Yes
NFGSC Nashville CS	Chautauqua	Hanover	No	No	No	No	No	No	Yes	No	Yes	Yes
TGPC 229 & TEG DF	Erie	Eden	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 224	Chautauqua	Clymer	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 230-C	Niagara	Lockport	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 233	Livingston	York	No	No	No	Yes	Yes	No	Yes	No	Yes	Yes
TGPC CS 237	Ontario	Manchester, Phelps	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 241	Onondaga	LaFayette	Yes	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 245	Herkimer	Winfield	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 249	Schoharie	Carlisle	No	No	No	Yes	No	No	Yes	No	Yes	Yes
TGPC CS 254	Columbia	Chatham	No	No	No	Yes	Yes	No	Yes	No	Yes	Yes

Source: Authors' review of NYS DEC permits.

Table 2.3.2.

## Applicable State and Federal Air Pollution Regulatory Programs: Summary

## NYS Natural Gas Compressor Stations

Regulatory Program	Description	No
<b>Prevention of Significant Deterioration</b> <b>PSD</b> <b>40 CFR 52</b>	Prevention of Significant Deterioration (40 CFR 52) - requirements which pertain to major stationary sources located in areas which are in attainment of National Ambient Air Quality Standards (NAAQS) for specified pollutants.	4
<b>New Source Review</b> <b>NSR</b> <b>6 NYCRR Part 231</b>	New Source Review (6 NYCRR Part 231) - requirements which pertain to major stationary sources located in areas which are in non-attainment of National Ambient Air Quality Standards (NAAQS) for specified pollutants.	0
<b>National Emission Standards for Hazardous Air Pollutants</b> <b>NESHAP</b> <b>40 CFR 61</b>	National Emission Standards for Hazardous Air Pollutants (40 CFR 61) - contaminant and source specific emission standards established prior to the Clean Air Act Amendments of 1990 (CAAA) which were developed for 9 air contaminants (inorganic arsenic, radon, benzene, vinyl chloride, asbestos, mercury, beryllium, radionuclides, and volatile HAP's).	0
<b>Maximum Achievable Control Technology</b> <b>MACT</b> <b>40 CFR 63</b>	Maximum Achievable Control Technology (40 CFR 63) - contaminant and source specific emission standards established by the 1990 CAAA. Under Section 112 of the CAAA, the US EPA is required to develop and promulgate emissions standards for new and existing sources. The standards are to be based on the best demonstrated control technology and practices in the regulated industry, otherwise known as MACT. The corresponding regulations apply to specific source types and contaminants.	14
<b>New Source Performance Standards</b> <b>NSPS</b> <b>40 CFR 60</b>	New Source Performance Standards (40 CFR 60) - standards of performance for specific stationary source categories developed by the US EPA under Section 111 of the CAAA. The standards apply only to those stationary sources which have been constructed or modified after the regulations have been proposed by publication in the Federal Register and only to the specific contaminant(s) listed in the regulation.	7
<b>Title IV Acid Rain Control Program</b> <b>Title IV</b> <b>40 CFR 72-78</b>	Title IV Acid Rain Control Program (40 CFR 72 thru 78) - regulations which mandate the implementation of the acid rain control program for large stationary combustion facilities.	0
<b>Title V</b>		18
<b>Title VI Stratospheric Ozone Protection</b> <b>Title VI</b> <b>40 CFR 82, Subparts A-G</b>	Title VI Stratospheric Ozone Protection (40 CFR 82, Subparts A thru G) - federal requirements that apply to sources which use a minimum quantity of CFC's (chlorofluorocarbons), HCFC's (hydrofluorocarbons) or other ozone depleting substances or regulated substitute substances in equipment such as air conditioners, refrigeration equipment or motor vehicle air conditioners or appliances.	0
<b>Reasonably Available Control Technology</b> <b>RACCT</b> <b>6 NYCRR Parts 212.10, 226, 227-2, 228, 229, 230, 232, 233, 234, 235, 236)</b>	Reasonably Available Control Technology (6 NYCRR Parts 212.10, 226, 227-2, 228, 229, 230, 232, 233, 234, 235, 236) - the lowest emission limit that a specific source is capable of meeting by application of control technology that is reasonably available, considering technological and economic feasibility. RACT is a control strategy used to limit emissions of VOC's and NOx for the purpose of attaining the air quality standard for ozone. The term as it is used in the above table refers to those state air pollution control regulations which specifically regulate VOC and NOx emissions.	18
<b>State Implementation Plan</b> <b>SIP</b> <b>40 CFR 52, Subpart HH</b>	State Implementation Plan (40 CFR 52, Subpart HH) - as per the CAAA, all states are empowered and required to devise the specific combination of controls that, when implemented, will bring about attainment of ambient air quality standards established by the federal government and the individual state. This specific combination of measures is referred to as the SIP. The term here refers to those state regulations that are approved to be included in the SIP and thus are considered federally enforceable.	18

## 2.4. U.S. EPA NEI Reporting for Compressor Stations

How a facility is permitted determines how its pollution data is tracked by NEI.

Compressor stations receiving a “Title V” permit are tracked by the system NEI uses for point air pollution sources. Compressor stations receiving a “State Facility Permit” are tracked by the system NEI uses for non-point sources of air pollution.

There is one exception: A single station with a “State Facility Permit” is being tracked as an NEI on-site polluter. It is unclear why this is the case.

### 2.4.1. Compressor Stations with a “Title V Permit”

The NEI for 2008-2014 includes data for 18 compressor stations in NYS.

There are, however, several additional sites classified as “Title V” facilities on the DEC’s website which are not found in NEI. (We notified DEC of this discrepancy and are in conversation to determine why these sites are not included as NEI point polluters. The compressor sites found in NEI are the exclusive source or data for this report.)

North American Industry Classification System (NAICS) code 48621 is used to designate facilities whose main purpose is the transportation of natural gas. Each of the 18 sites analyzed have this classification.

### 2.4.2. Compressor Stations with a “State Facility Permit”

We have identified 19 non-Title V compressor stations which, based on federal and state guidelines, should be reported as non-point NEI sources. Because these stations are not easily identified within NEI and the time limits and scope of this project, an analysis of the pollution associated with their operation is not included in this report.

Table 2.4.2.

## Facilities Categorization: Transportation of Natural Gas (NAICS 48621)

## New York State

#	Status*	Name	Address	Town	Zip	DEC Reg.	County	NEI Point Src.	GHG
1	Op-EM	AGT Southeast CS	142 Tulip Rd	Southeast	10509	3	Putnam	√	√
2	Op-EM	AGT Stony Point CS	1 Lindberg Rd	Stony Point	10980	3	Rockland	√	√
	Op	CGTC Corning CS	4401 College Ave	Corning	14830	8	Steuben		√
	Op	CGTC Dundee CS	4620 Rte. 226	Starkey	14878	8	Yates		
	Op	CGTC North Greenwood CS	Brown Hollow Rd @ Kelly Rd	Greenwood	14839	8	Steuben		
	Op	DTI Borger CS	219 Ellis Hollow Creek Rd	Ithaca	14850	7	Tompkins	√	
	Op-EM	DTI Brookman CS	201 Casler Rd	Minden	13339	4	Montgomery		
	Op	DTI Canajoharie MRS	110 Gogus Rd	Canajoharie	12010	4	Montgomery		
	Op-EM	DTI Utica Station	Higby Rd	Frankfort	13340	6	Herkimer	√	
	Op	DTI Woodhull Station	974 CO RTE 99	Woodhull	14898	8	Steuben	√	
	Op	HSC	4511 Egypt Rd	Canandaigua	14424	8	Ontario		
	Op	Hunts Point Ave CS	332 Hunts Point Avenue		10474	2	Bronx		
	Op	IGTS Athens CS	915 Schoharie Tpk Cr 28	Athens	12015	4	Greene		
	Op	IGTS Boonville CS	3338 East Rd	Boonville	13309	6	Oneida		
	Op	IGTS Croghan CS	Old State Rd	Croghan	13327	6	Lewis		
	Op	IGTS Dover CS	186 Dover Furnace Rd	Dover Plains	12522	3	Dutchess		
	Op	IGTS Wright CS	320 Westfall Road	Delanson	12053	4	Schenectady		√
	Op	Millennium Minisink CS	107 Jacobs Rd	Wawayanda	10998	3	Orange		√
	Op	NFGSC Beech Hill CS	1161 Peet Rd	Willing	14895	9	Allegany	√	
	Op	NFGSC Concord CS	5510 Genesee Rd	Concord	14141	9	Erie	√	√
	Op	NFGSC Independence CS	2210 County Road 22	Andover	14806	9	Allegany	√	√
	Op	NFGSC Nashville CS	11413 Allegany Rd	Hanover	14062	9	Chautauqua	√	
	Op-EM	NFGSC Porterville CS	350 Hemstreet Rd	Aurora	14052	9	Erie		
	Op	NFGSC Zoar CS	Wilson & Conerts Rd	Collins	14034	9	Erie		
	Op	NP Hanover Mayville CS	5644 Bently Rd	Chautauqua	14757	9	Chautauqua		
	Op	TE Catlin Hill CS	Brown and Cemetery Rds	Catlin	14812	8	Chemung		
	Op-EM	TGP CS 230-C	7586 East Eden Road	Eden	14057	9	Erie		
	Op	TGPC 229 & TEG DF	7586 East Eden Road	Eden	14057	9	Erie	√	√
	Op	TGPC CS 224	9766 Ravlin Hill Rd	Clymer	14724	9	Chautauqua	√	√
	Op	TGPC CS 230-C	5186 Lockport Junction Rd	Lockport	14094	9	Niagara	√	√
	Op	TGPC CS 233	2262 Dow Rd	York	14533	8	Livingston	√	
	Op	TGPC CS 237	2001 Archer Road	Manchester, Phelps	14432	8	Ontario	√	
	Op	TGPC CS 241	3447 Sentinel Heights Rd	LaFayette	13084	7	Onondaga	√	√
	Op	TGPC CS 245	457 Burrows Rd	Winfield	13491	6	Herkimer	√	√
	Op	TGPC CS 249	2480 US Route 20	Carlisle	12031	4	Schoharie	√	√
	Op	TGPC CS 254	ST Rte 66	Chatham	12123	4	Columbia	√	√
	Op	TNG CS 249 - B	2840 US Route 20 East	Carlisle	12031	4	Schoharie		
	Op	TNG CS 405A	Mackey Rd	Woodhull	14809	8	Steuben		
	Op	WP Dunbar CS	414 Dunbar Rd	Windsor	13865	7	Broome		√
	App	DTI Horseheads CS	End of Bush Rd	Veteran	14845	8	Chemung		
	App	ESPC Oakfield CS	3309 Lockport Rd	Oakfield	14125	8	Genesee		
	Prp	DTI CS Prp.		Nassau		4	Rensselaer		
	Prp	DTI Sheds CS Prp.	Wilcox Rd	Georgetown	13072	7	Madison		
	Prp	NFGSC Hinsdale CS	SE of Philips Rd	Hinsdale	14743	9	Cattaraugus		
	Prp	TNG CS Prp.	2060 Otego Rd	Franklin	13775	3	Sullivan		
	Prp	TNG Hancock CS	1579 Hungary Hill Rd	Hancock	13783	4	Delaware		√
	Prp	TNG Market Path CS-Prp		Not released		4	Schoharie		
	Prp	TNG Supply Path Trail CS-Prp		Not released		4	Schoharie		
	Prp-D	NFGSC Pendleton CS Prp-Denied	Killian Rd	Pendleton		9	Niagara		

\* Op-Operational, OP-EM -- Operational-Enhancements\Modifications, App -- Approved, Prp -- Proposed, Prp-D -- Proposal Denied

## 2.5. Total Releases

### 2.5a. Releases by Chemical

Table 2.5a.1.

#### Total Pounds by Chemical (ranked)

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank		Pounds							
	Description	2008	2011	2014	3-Years	3-Yr-Avg	7 Years	%	
1	Nitrogen oxides (NO2)	2,269,341	2,993,049	2,487,284	7,749,673	2,583,224	18,082,571	45.22	
2	Carbon monoxide	1,415,996	2,030,629	1,850,403	5,297,028	1,765,676	12,359,731	30.91	
3	Volatile organic compounds	374,277	831,915	902,548	2,108,741	702,914	4,920,396	12.31	
4	Formaldehyde	110,334	229,882	220,928	561,144	187,048	1,309,336	3.27	
5	PM10 Primary (Filt + Cond)	107,946	242,279	189,665	539,890	179,963	1,259,744	3.15	
6	PM 2.5 Primary (Filt + Cond)	92,595	220,983	160,507	474,085	158,028	1,106,198	2.77	
7	PM Condensable	43,227	109,501	78,815	231,543	77,181	540,267	1.35	
8	Sulfur dioxide	7,587	14,174	58,287	80,048	26,683	186,778	0.47	
9	Acetaldehyde	4,385	15,091	8,797	28,272	9,424	65,969	0.16	
10	Acrolein	3,226	11,742	7,628	22,596	7,532	52,723	0.13	
11	Benzene	2,029	3,876	3,199	9,103	3,034	21,241	0.05	
12	Methanol	1,381	4,324	2,580	8,286	2,762	19,333	0.05	
13	Toluene	1,267	3,633	3,375	8,275	2,758	19,308	0.05	
14	Hexane, n-	1,939	1,780	1,502	5,222	1,741	12,184	0.03	
15	Xylene (mixed isomers)	360	1,460	1,777	3,598	1,199	8,394	0.02	
16	Butadiene, 1,3-	273	999	751	2,022	674	4,719	0.01	
17	Trimethylpentane, 2,2,4-	238	931	735	1,905	635	4,445	0.01	
18	Ethyl benzene	155	577	466	1,198	399	2,794	0.01	
19	Ammonia	262	238	174	674	225	1,573	0.00	
20	Phenol	33	149	121	303	101	706	0.00	
21	Naphthalene	50	154	94	298	99	696	0.00	
22	Nickel	169	21	107	296	99	692	0.00	
23	Biphenyl	68	178	49	296	99	690	0.00	
24	Methane dichloride [1910.1052]	31	118	120	269	90	629	0.00	
25	Propylene oxide	7	115	142	263	88	615	0.00	
26	Manganese	104	0	47	150	50	350	0.00	
27	Ethylene dibromide	29	71	49	149	50	347	0.00	
28	Tetrachloroethane, 1,1,2,2-	26	64	42	132	44	309	0.00	
29	Carbon tetrachloride	24	59	38	121	40	282	0.00	
30	Trichloroethane, 1,1,2-	21	52	33	106	35	247	0.00	
31	Styrene	18	49	33	100	33	234	0.00	
32	Chloroform	18	45	19	83	28	193	0.00	
33	Methylnaphthalene, 2-	15	55	12	82	27	191	0.00	
34	Chlorobenzene	19	36	19	74	25	172	0.00	
35	Propylene dichloride	17	35	18	70	23	164	0.00	
36	Dichloropropene, 1,3	17	34	18	69	23	161	0.00	
37	Ethylene dichloride	16	32	17	65	22	151	0.00	
38	Ethylidene dichloride	15	31	16	62	21	144	0.00	
39	Vinyl chloride	10	24	12	46	15	107	0.00	
40	Mercury	17	7	6	30	10	70	0.00	
41	Chromium (III) compounds (as Cr)	16	0	7	24	8	56	0.00	
42	Phenanthrene	4	14	2	21	7	48	0.00	
43	Polycyclic aromatic hydrocarbons, total (PAHs Total)		0	15	15	5	35	0.00	
44	Cadmium	9	0	4	13	4	30	0.00	
45	Fluorene	2	8	1	12	4	28	0.00	
46	Benz[a]anthracene	4	2	2	8	3	19	0.00	
47	Benzo(j,k)fluorene	2	2	1	5	2	11	0.00	



Rank	Description	Pounds			3-Years	3-Yr-Avg	7 Years	%
		2008	2011	2014				
48	Anthracene	0	4	0	4	1	10	0.00
49	Perchloroethylene [PERC PCE, Tetrachloroethylene]	1	2	1	4	1	9	0.00
50	Acenaphthene	1	2	1	4	1	8	0.00
51	Pyrene	1	2	0	3	1	7	0.00
52	Ethyl chloride (Chloroethane)	1	2	0	3	1	6	0.00
53	Acenaphthylene	2		-	2	1	5	0.00
54	Chrysene	0	1	0	2	1	4	0.00
55	Chromium (VI) & inorganic Cr6+ compounds	1	0	0	1	0	2.3	0.00
56	Benzo[g,h,i]perylene	0	1	0	1	0	1.7	0.00
57	Benzo[b]fluoranthene	0	0	0	0	0	0.7	0.00
58	Lead	0	0	0	0	0	0.6	0.00
59	Benzo[e]pyrene	0			0	0	0.09	0.00
60	Arsenic	0	0	0	0	0	0.06	0.00
61	Cobalt	0	0	0	0	0	0.03	0.00
62	Indeno[1,2,3-cd]pyrene	0	0	0	0	0	0.02	0.00
63	Benzo[a]pyrene	0	0	0	0	0	0.01	0.00
64	Selenium	0	0	0	0	0	0.01	0.00
65	Perylene	0			0	0	0.00	0.00
66	Beryllium	0	0	0	0	0	0.00	0.00
67	Dimethylbenz[a]anthracene, 7,12-		0	0	0	0	0.00	0.00
68	Benzo[k]fluoranthene	0			0	0	0.00	0.00
69	Methylcholanthrene, 3-		0	-	0	0	0.00	0.00
70	Dibenz[a,h]anthracene	0			0	0	0.00	0.00

Table 2.5a.2.

## Total Pounds by Chemical Category

## NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Category	Rank	Description	2008	2011	2014	3-Years	3-Yr-Avg	7 Years	%
			<b>4,437,584</b>	<b>6,718,435</b>	<b>5,980,468</b>	<b>17,136,487</b>	<b>5,712,162</b>	<b>39,985,136</b>	<b>100%</b>
Biphenyls	23	Biphenyl	68	178	49	296	99	<b>690</b>	0.00
CAPs	1	Nitrogen oxides (NO2)	2,269,341	2,993,049	2,487,284	7,749,673	2,583,224	18,082,571	45.22
CAPs	2	Carbon monoxide	1,415,996	2,030,629	1,850,403	5,297,028	1,765,676	12,359,731	30.91
CAPs	8	Sulfur dioxide	7,587	14,174	58,287	80,048	26,683	186,778	0.47
CAPs	19	Ammonia	262	238	174	674	225	1,573	0.00
			<b>3,693,186</b>	<b>5,038,090</b>	<b>4,396,148</b>	<b>13,127,423</b>	<b>4,375,808</b>	<b>30,630,653</b>	<b>76.6</b>
Metals	66	Beryllium	0	0	0	0	0	0.00	0.00
Metals, heavy	22	Nickel	169	21	107	296	99	692	0.00
Metals, heavy	26	Manganese	104	0	47	150	50	350	0.00
Metals, heavy	40	Mercury	17	7	6	30	10	70	0.00
Metals, heavy	41	Chromium (III) compounds (as Cr)	16	0	7	24	8	56	0.00
Metals, heavy	44	Cadmium	9	0	4	13	4	30	0.00
Metals, heavy	55	Cr6+ compounds	1	0	0	1	0	2.3	0.00
Metals, heavy	58	Lead	0	0	0	0	0	0.6	0.00
Metals, heavy	60	Arsenic	0	0	0	0	0	0.06	0.00
Metals, heavy	61	Cobalt	0	0	0	0	0	0.03	0.00
Metals, heavy	64	Selenium	0	0	0	0	0	0.01	0.00
			<b>316</b>	<b>28</b>	<b>171</b>	<b>514</b>	<b>171</b>	<b>171</b>	<b>0.00</b>
PAHs	43	Polycyclic aromatic hydrocarbons		0	15	15	5	35	0.00
PAHs	50	Acenaphthene	1	2	1	4	1	8	0.00
PAHs	59	Benzo[e]pyrene	0			0	0	0.09	0.00
PAHs	65	Perylene	0			0	0	0.00	0.00
PM10	5	PM10 Primary (Filt + Cond)	107,946	242,279	189,665	539,890	179,963	1,259,744	3.15
PM25	6	PM 2.5 Primary (Filt + Cond)	92,595	220,983	160,507	474,085	158,028	1,106,198	2.77
			<b>200,542</b>	<b>463,262</b>	<b>350,172</b>	<b>1,013,994</b>	<b>337,997</b>	<b>2,365,985.09</b>	<b>5.92</b>
PM-CON	7	PM Condensable	<b>43,227</b>	<b>109,501</b>	<b>78,815</b>	<b>231,543</b>	<b>77,181</b>	<b>540,267</b>	<b>1.35</b>
Solvents	20	Phenol	<b>33</b>	<b>149</b>	<b>121</b>	<b>303</b>	<b>101</b>	<b>706</b>	<b>0.00</b>
SVOCs	21	Naphthalene	50	154	94	298	99	696	0.00
SVOCs	33	Methylnaphthalene, 2-	15	55	12	82	27	191	0.00
SVOCs	42	Phenanthrene	4	14	2	21	7	48	0.00
SVOCs	45	Fluorene	2	8	1	12	4	28	0.00
SVOCs	46	Benzo[a]anthracene	4	2	2	8	3	19	0.00
SVOCs	47	Benzo[j,k]fluorene	2	2	1	5	2	11	0.00
SVOCs	48	Anthracene	0	4	0	4	1	10	0.00
SVOCs	51	Pyrene	1	2	0	3	1	7	0.00
SVOCs	53	Acenaphthylene	2	0	0	2	1	5	0.00
SVOCs	54	Chrysene	0	1	0	2	1	4	0.00
SVOCs	56	Benzo[g,h,i]perylene	0	1	0	1	0	1.7	0.00
SVOCs	57	Benzo[b]fluoranthene	0	0	0	0	0	0.7	0.00
SVOCs	62	Indeno[1,2,3-cd]pyrene	0	0	0	0	0	0.02	0.00
SVOCs	63	Benzo[a]pyrene	0	0	0	0	0	0.01	0.00
SVOCs	67	Dimethylbenz[a]anthracene, 7, 12-	0	0	0	0	0	0.00	0.00
SVOCs	68	Benzo[k]fluoranthene	0	0	0	0	0	0.00	0.00
SVOCs	69	Methylcholanthrene, 3-	0	0	0	0	0	0.00	0.00
SVOCs	70	Dibenz[a,h]anthracene	0	0	0	0	0	0.00	0.00
			<b>80</b>	<b>243</b>	<b>112</b>	<b>438</b>	<b>146</b>	<b>1021.43</b>	<b>0.0</b>

Category	Rank	Description	2008	2011	2014	3-Years	3-Yr-Avg	7 Years	%
VOCs	3	Volatile organic compounds	374,277	831,915	902,548	2,108,741	702,914	4,920,396	12.31
VOCs	4	Formaldehyde	110,334	229,882	220,928	561,144	187,048	1,309,336	3.27
VOCs	9	Acetaldehyde	4,385	15,091	8,797	28,272	9,424	65,969	0.16
VOCs	10	Acrolein	3,226	11,742	7,628	22,596	7,532	52,723	0.13
VOCs	11	Benzene	2,029	3,876	3,199	9,103	3,034	21,241	0.05
VOCs	12	Methanol	1,381	4,324	2,580	8,286	2,762	19,333	0.05
VOCs	13	Toluene	1,267	3,633	3,375	8,275	2,758	19,308	0.05
VOCs	14	Hexane, n-	1,939	1,780	1,502	5,222	1,741	12,184	0.03
VOCs	15	Xylene (mixed isomers)	360	1,460	1,777	3,598	1,199	8,394	0.02
VOCs	16	Butadiene, 1,3-	273	999	751	2,022	674	4,719	0.01
VOCs	17	Trimethylpentane, 2,2,4-	238	931	735	1,905	635	4,445	0.01
VOCs	18	Ethyl benzene	155	577	466	1,198	399	2,794	0.01
VOCs	24	Methane dichloride [1910.1052]	31	118	120	269	90	629	0.00
VOCs	25	Propylene oxide	7	115	142	263	88	615	0.00
VOCs	27	Ethylene dibromide	29	71	49	149	50	347	0.00
VOCs	28	Tetrachloroethane, 1,1,2,2-	26	64	42	132	44	309	0.00
VOCs	29	Carbon tetrachloride	24	59	38	121	40	282	0.00
VOCs	30	Trichloroethane, 1,1,2-	21	52	33	106	35	247	0.00
VOCs	31	Styrene	18	49	33	100	33	234	0.00
VOCs	32	Chloroform	18	45	19	83	28	193	0.00
VOCs	34	Chlorobenzene	19	36	19	74	25	172	0.00
VOCs	35	Propylene dichloride	17	35	18	70	23	164	0.00
VOCs	36	Dichloropropene, 1,3-	17	34	18	69	23	161	0.00
VOCs	37	Ethylene dichloride	16	32	17	65	22	151	0.00
VOCs	38	Ethylidene dichloride	15	31	16	62	21	144	0.00
VOCs	39	Vinyl chloride	10	24	12	46	15	107	0.00
VOCs	49	Perchloroethylene [PERC PCE, Tetrachloroethylene]	1	2	1	4	1	9	0.00
VOCs	52	Ethyl chloride (Chloroethane)	1	2	0	3	1	6	0.00
			<b>500,134</b>	<b>1,106,979</b>	<b>1,154,863</b>	<b>2,761,978</b>	<b>920,659</b>	<b>6,444,612</b>	<b>16.1</b>

## 2.5c. Releases by Facility

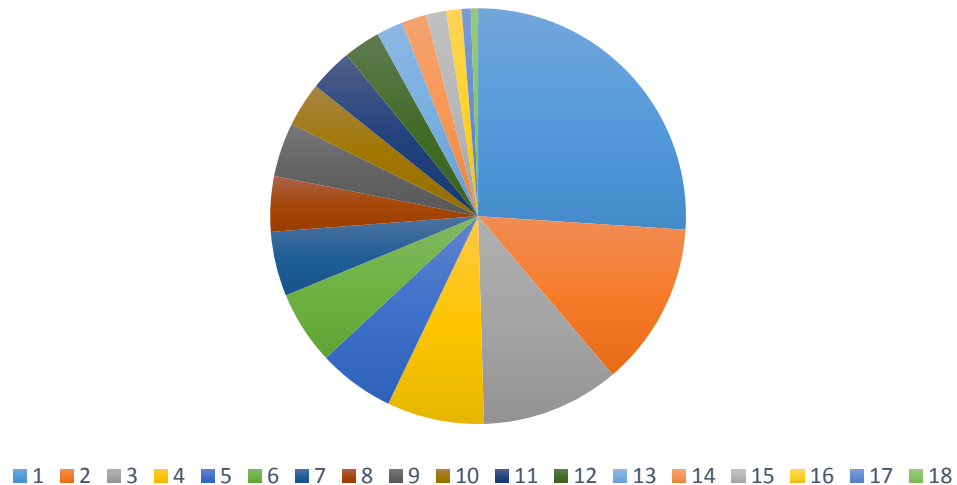
Table 2.5c.

### Total Pounds by Facility (ranked)

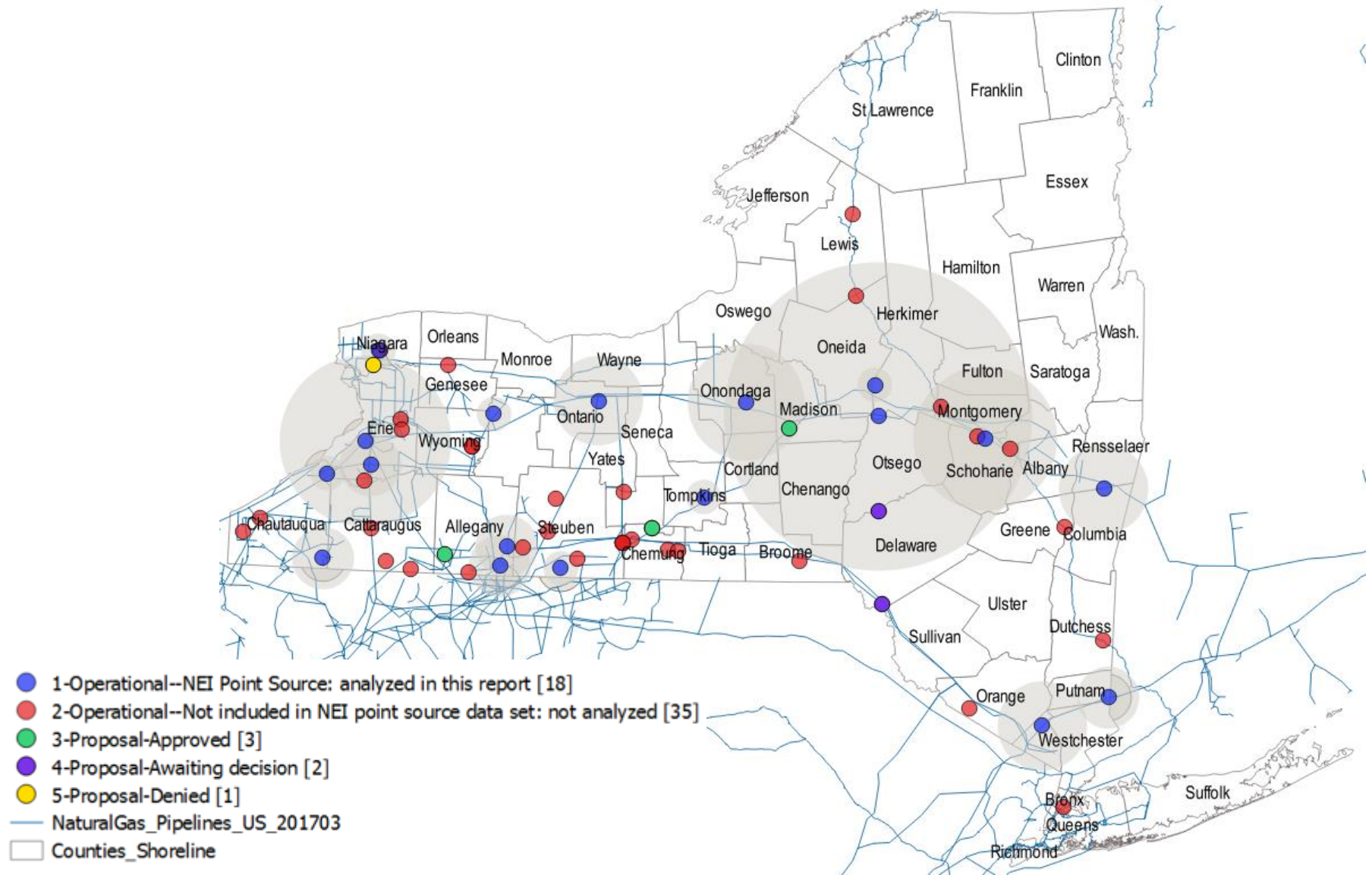
NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Chemicals			Pounds				7 Years (estimate)	
	Facility Name (Short)	Town	County	'08	'11	'14	2008	2011	2014	Average	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	49	47	25	750,288	1,877,949	1,856,930	1,495,056	10,465,389	26.04
2	TGPC 229 & TEG DF	Eden	Erie	52	47	48	499,504	1,160,934	535,745	732,061	5,124,427	12.75
3	TGPC CS 249	Carlisle	Schoharie	49	27	26	712,001	569,088	571,747	617,612	4,323,285	10.76
4	TGPC CS 241	LaFayette	Onondaga	47	37	46	297,485	574,214	431,014	434,237	3,039,661	7.56
5	TGPC CS 254	Chatham	Columbia	27	16	9	288,373	260,770	476,712	341,952	2,393,661	5.96
6	TGPC CS 237	Manchester, Phelps	Ontario	9	8	5	321,292	482,043	181,691	328,342	2,298,394	5.72
7	AGT Stony Point CS	Stony Point	Rockland	46	24	23	244,039	268,064	350,815	287,640	2,013,478	5.01
8	NFGSC Concord CS	Concord	Erie	11	10	12	364,989	993	376,805	247,596	1,733,171	4.31
9	AGT Southeast CS	Southeast	Putnam	27	18	46	161,097	255,290	307,392	241,259	1,688,815	4.20
10	NFGSC Beech Hill CS	Willing	Allegany	20	20	21	115,405	202,835	276,443	198,227	1,387,592	3.45
11	NFGSC Independ. CS	Andover	Allegany	15	10	17	119,762	210,879	249,615	193,419	1,353,931	3.37
12	TGPC CS 224	Clymer	Chautauqua	47	45	47	44,133	391,407	55,945	163,828	1,146,797	2.85
13	DTI Woodhull Station	Woodhull	Steuben	44	54	58	104,802	209,130	41,449	118,460	829,223	2.06
14	DTI Borger CS	Ithaca	Tompkins	44	45	19	129,004	83,412	121,938	111,451	780,159	1.94
15	NFGSC Nashville CS	Hanover	Chautauqua	38	36		100,466	77,474		88,970	622,791	1.55
16	TGPC CS 230-C	Lockport	Niagara	27	26	27	83,451	2,791	121,877	69,373	485,610	1.21
17	DTI Utica Station	Frankfort	Herkimer	38	43	57	45,899	59,846	14,841	40,196	281,369	0.70
18	TGPC CS 233	York	Livingston	27	16	4	55,594	31,316	9,510	32,140	224,978	0.56
				67	65	66	4,437,584	6,718,435	5,980,468	5,712,162	40,192,733	100%

### NYS Compression Station Releases by Facility: Total Pounds: 2008-2014



# New York's 59 Compressor Stations



### 2.5c.1. Releases by Facility: Circular Area Air Pollution Profiles

Because most compressor stations are located in sparsely populated areas, it is widely believed that relatively few people are directly exposed to their air releases. An examination of actual population counts by distance from each station, reveals a more complex picture. While it is true that only 2,660 people live within ½ mile of the 18 compressor stations analyzed in this report, nearly 1.7 million live within 10-mile radius—more than 1 out of every 8 New Yorkers.

Table 2.5c.1a.

#### NYS Compressor Stations, Circular Area Profile, .05 to 30 Mile Radius: 2010

##### Total Population

Compressor Station	Location			Radius in miles								
	Reg	County	Town	.05	1	2	3	5	10	15	20	30
AG SE CS	3	Putnam	Southeast	261	799	3,323	12,564	57,347	236,568	402,810	761,783	2,341,903
AG Stony Point CS	3	Rockland	Stony Point	704	2,158	10,310	24,626	62,433	330,569	700,546	1,292,599	5,268,668
DTI Borger CS	7	Tompkins	Ithaca	144	396	2,184	5,155	53,097	84,565	115,705	170,961	328,040
DTI Utica Station	6	Herkimer	Frankfort	45	254	1,406	6,243	56,734	148,087	192,498	255,438	363,367
DTI Woodhull Station	8	Steuben	Woodhull	2	57	371	950	2,130	12,947	24,941	66,963	175,182
NFGSC Beech Hill CS	9	Allegany	Willing	43	64	329	687	2,999	14,592	27,665	49,547	116,261
NFGSC Concord CS	9	Erie	Concord	-	125	579	1,346	4,168	38,139	129,370	262,634	866,137
NFGSC Independ. CS	9	Allegany	Andover	839	1,080	1,377	1,639	2,638	19,772	42,188	59,407	132,614
NFGSC Nashville CS	9	Chautauqua	Hanover	41	166	579	1,320	6,920	31,268	78,625	121,441	432,158
TGPC CS 224	9	Chautauqua	Clymer	95	103	622	1,645	4,689	51,965	84,954	111,105	203,396
TGPC CS 229	9	Erie	Eden	151	726	3,803	11,106	34,960	131,667	323,483	684,972	1,066,965
TGPC CS 230-C	9	Niagara	Lockport	12	359	2,202	5,922	39,624	145,809	485,700	836,986	1,095,236
TGPC CS 233	8	Livingston	York	15	109	841	2,140	4,538	40,531	78,013	176,242	794,615
TGPC CS 237	8	Ontario	Manchester, Phelps	27	211	796	5,815	12,654	72,831	143,122	266,572	810,144
TGPC CS 241	7	Onondaga	LaFayette	218	460	1,627	4,484	25,469	257,224	385,855	496,520	704,663
TGPC CS 245	6	Herkimer	Winfield	-	166	1,366	1,969	4,470	16,826	84,588	210,758	379,224
TGPC CS 249	4	Schoharie	Carlisle	-	71	497	1,623	4,791	22,593	48,605	113,059	437,636
TGPC CS 254	4	Columbia	Chatham	10	137	643	1,622	7,455	40,695	127,791	441,231	841,606

Table 2.5c.1b.

## NYS Title V Compressor Stations, Circular Area Profile at 10-Mile Radius, Counties and Exposed Population

## Total Population

Compressor Station	Location		Counties						Exposed Population					
	Reg	County	NY	CT	MA	NJ	PA	Total	NY	CT	MA	NJ	PA	Total
AG SE CS	3	Putnam	3	0	0	0	0	3	83,417	148,176	0	0	0	231,593
AG Stony Point CS	3	Rockland	4	0	0	0	0	4	331,090	0	0	0	0	331,090
DTI Borger CS	7	Tompkins	1	0	0	0	0	1	80,226	0	0	0	0	80,226
DTI Utica Station	6	Herkimer	2	0	0	0	0	2	150,877	0	0	0	0	150,877
DTI Woodhull Station	8	Steuben	1	0	0	0	1	2	6,800	0	0	0	4,192	10,992
NFGSC Beech Hill CS	9	Allegany	2	0	0	0	1	3	12,650	0	0	0	1,305	13,955
NFGSC Concord CS	9	Erie	1	0	0	0	0	1	36,020	0	0	0	0	36,020
NFGSC Independ. CS	9	Allegany	2	0	0	0	0	2	19,472	0	0	0	0	19,472
NFGSC Nashville CS	9	Chautauqua	3	0	0	0	0	3	28,503	0	0	0	0	28,503
TGPC CS 224	9	Chautauqua	2	0	0	0	1	3	49,999	0	0	0	2,695	52,694
TGPC CS 229	9	Erie	1	0	0	0	0	1	136,180	0	0	0	0	136,180
TGPC CS 230-C	9	Niagara	2	0	0	0	0	2	144,562	0	0	0	0	144,562
TGPC CS 233	8	Livingston	3	0	0	0	0	3	37,769	0	0	0	0	37,769
TGPC CS 237	8	Ontario	2	0	0	0	0	2	68,821	0	0	0	0	68,821
TGPC CS 241	7	Onondaga	1	0	0	0	0	1	254,062	0	0	0	0	254,062
TGPC CS 245	6	Herkimer	4	0	0	0	0	4	16,828	0	0	0	0	16,828
TGPC CS 249	4	Schoharie	3	0	0	0	0	3	24,041	0	0	0	0	24,041
TGPC CS 254	4	Columbia	2	0	0	0	0	2	39,315	0	0	0	0	39,315

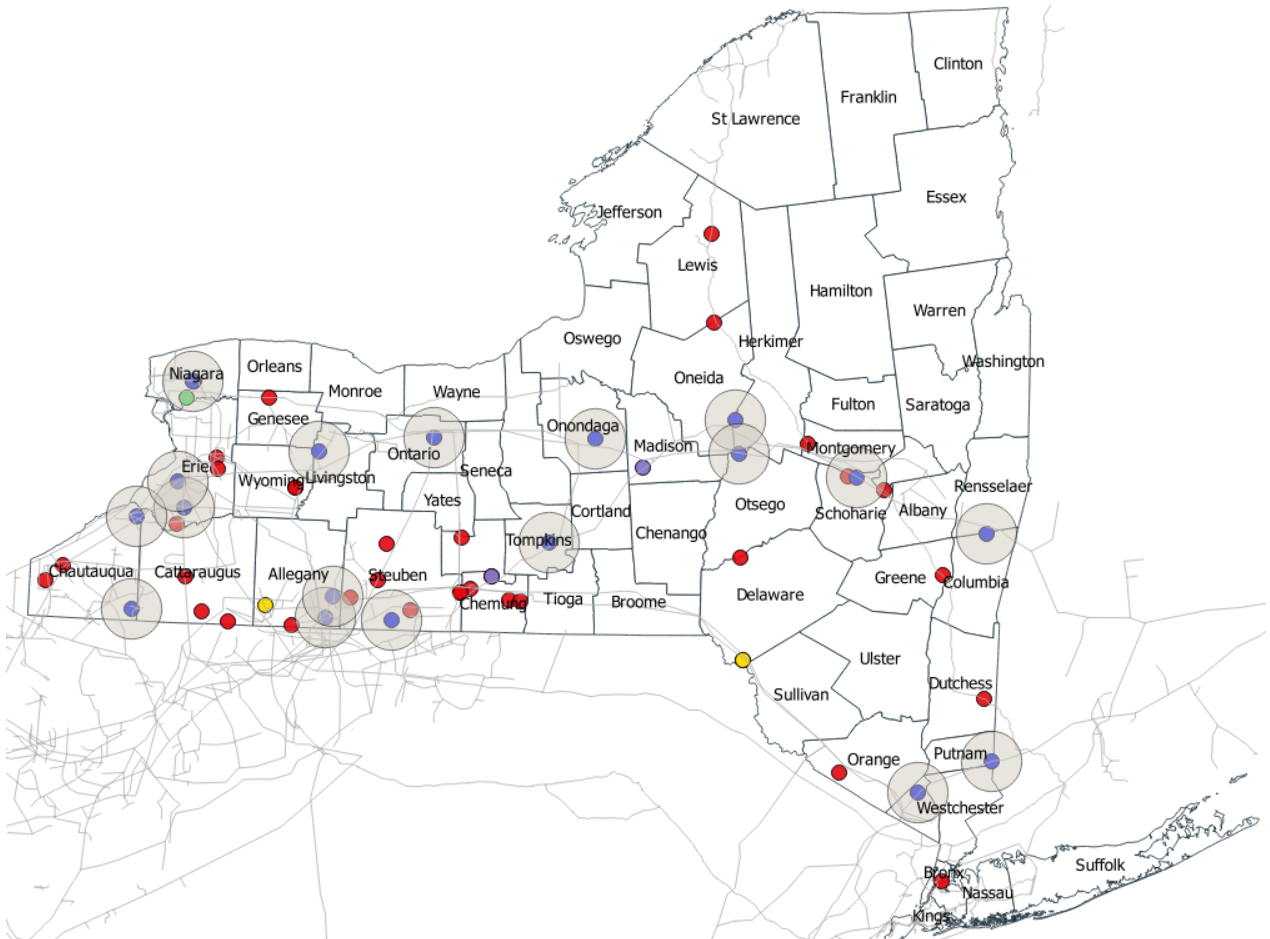


Table 2.5c.1c.

## NYS Title V Compressor Stations, Circular Area Profile at 10-Mile Radius, By State and County

Total Population

#	ST	County	1. AG SE CS	2. AG Stony Point CS	3. DTI Borger CS	4. DTI Utica Station	5. DTI Woodhull Station	6. FGSC Beech Hill CS	7. NFGSC Concord CS	8. NFGSC Independence CS	9. NFGSC Nashville CS	10. TGPC CS 224	11. TGPC CS 229 & TEG DF	12. TGPC CS 230-C	13. TGPC CS 233	14. TGPC CS 237	15. TGPC CS 241	16. TGPC CS 245	17. TGPC CS 249	18. TGPC CS 254
1	CT	Fairfield	√																	
2	NY	Allegany						√		√										
3	NY	Cattaraugus									√	√	√							
4	NY	Chautauqua									√	√								
5	NY	Columbia																		√
6	NY	Dutchess																		
7	NY	Erie							√		√			√						
8	NY	Genesee													√					
9	NY	Herkimer				√												√		
10	NY	Livingston													√					
11	NY	Madison																√		
12	NY	Montgomery																	√	
13	NY	Niagara												√						
14	NY	Oneida				√												√		
15	NY	Onondaga															√			
16	NY	Ontario														√				
17	NY	Orange		√																
18	NY	Otsego																√		
19	NY	Putnam	√	√																
20	NY	Rensselaer																		√
21	NY	Rockland		√																
22	NY	Schenectady																	√	
23	NY	Schoharie																	√	
24	NY	Steuben					√	√		√										
26	NY	Tompkins			√															
25	NY	Wayne														√				
27	NY	Westchester	√	√																
28	NY	Wyoming													√					
29	PA	Potter						√												
30	PA	Tioga					√													
31	PA	Warren										√								



Table 2.5c.1d.

## NYS Title V Compressor Stations, Circular Area Profile at 20-Mile Radius, Counties and Exposed Population

## Total Population

Compressor Station	Location		Counties						Exposed Population					
	Reg	County	NY	CT	MA	NJ	PA	Total	NY	CT	MA	NJ	PA	Total
AG SE CS	3	Putnam	3	3	0	0	0	6	320,502	440,274	0	0	0	760,776
AG Stony Point CS	3	Rockland	4	0	0	2	0	6	983,807	0	0	300,950	0	1,284,757
DTI Borger CS	7	Tompkins	8	0	0	0	0	8	168,038	0	0	0	0	168,038
DTI Utica Station	6	Herkimer	4	0	0	0	0	4	258,872	0	0	0	0	258,872
DTI Woodhull Station	8	Steuben	2	0	0	0	2	4	48,746	0	0	0	15,138	63,884
NFGSC Beech Hill CS	9	Allegany	2	0	0	0	2	4	37,820	0	0	0	11,062	48,882
NFGSC Concord CS	9	Erie	4	0	0	0	0	4	258,402	0	0	0	0	258,402
NFGSC Independ. CS	9	Allegany	2	0	0	0	1	3	55,368	0	0	0	4,846	60,214
NFGSC Nashville CS	9	Chautauqua	3	0	0	0	0	3	122,243	0	0	0	0	122,243
TGPC CS 224	9	Chautauqua	2	0	0	0	3	4	83,777	0	0	0	34,618	118,395
TGPC CS 229	9	Erie	3	0	0	0	0	3	687,974	0	0	0	0	687,974
TGPC CS 230-C	9	Niagara	4	0	0	0	0	4	834,828	0	0	0	0	834,828
TGPC CS 233	8	Livingston	5	0	0	0	0	5	172,667	0	0	0	0	172,667
TGPC CS 237	8	Ontario	5	0	0	0	0	5	271,633	0	0	0	0	271,633
TGPC CS 241	7	Onondaga	4	0	0	0	0	4	504,522	0	0	0	0	504,522
TGPC CS 245	6	Herkimer	5	0	0	0	0	5	211,083	0	0	0	0	211,083
TGPC CS 249	4	Schoharie	6	0	0	0	0	6	115,788	0	0	0	0	115,788
TGPC CS 254	4	Columbia	4	0	1	0	0	5	376,937	0	65,680	0	0	442,617

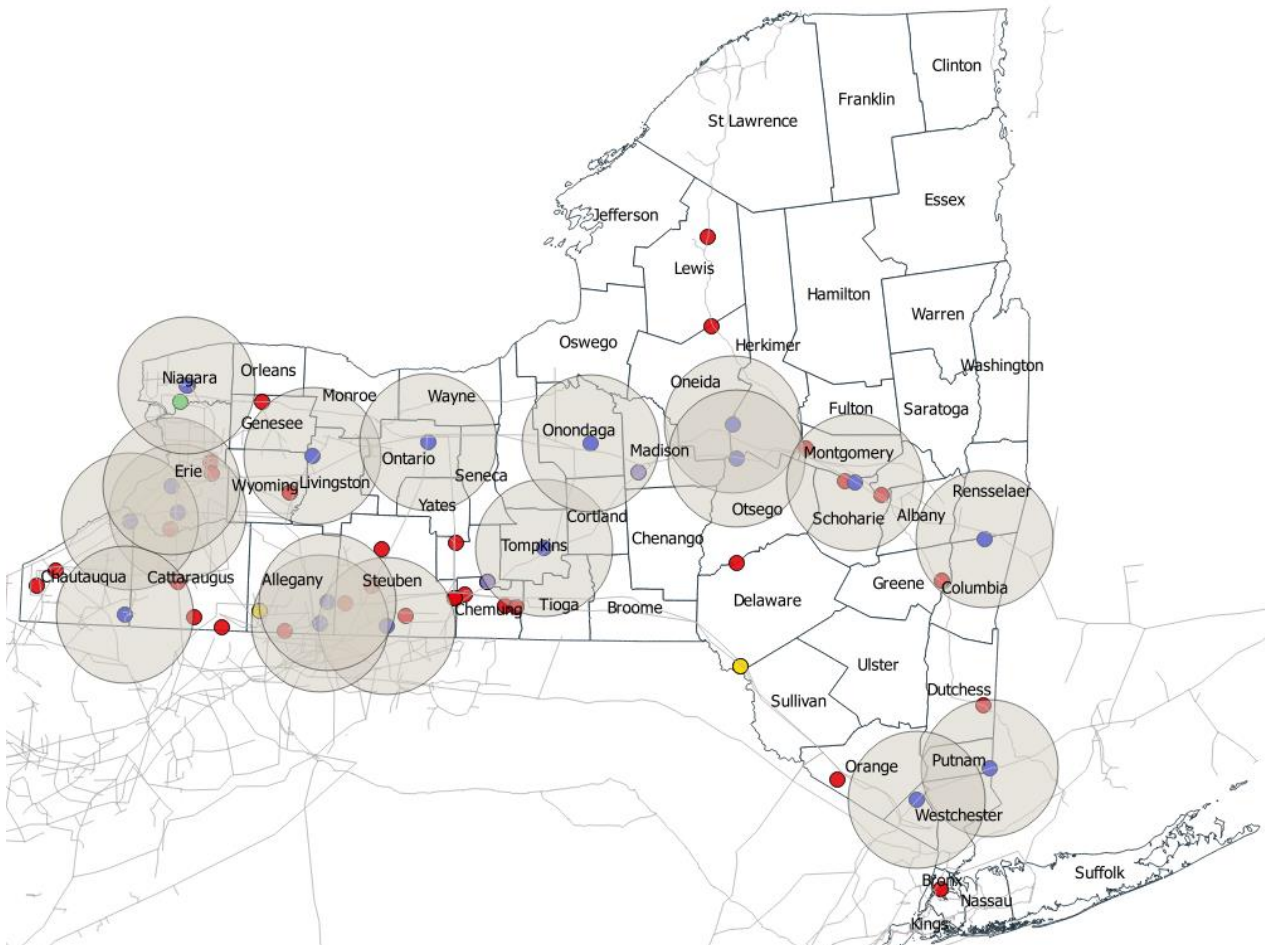


Table 2.5c.1e.

## NYS Title V Compressor Stations, Circular Area Profile at 10-Mile Radius, By State and County

## Total Population

#	ST	County	1. AG SE CS	2. AG Stony Point CS	3. DTI Borger CS	4. DTI Utica Station	5. DTI Woodhull Station	6. FGSC Beech Hill CS	7. NFGSC Concord CS	8. NFGSC Independence CS	9. NFGSC Nashville CS	10. TGPC CS 224	11. TGPC CS 229 & TEG DF	12. TGPC CS 230-C	13. TGPC CS 233	14. TGPC CS 237	15. TGPC CS 241	16. TGPC CS 245	17. TGPC CS 249	18. TGPC CS 254
1	CT	Fairfield	√	√																
2	CT	Litchfield	√																	
3	CT	New Haven	√																	
4	MA	Berkshire																		√
5	NJ	Bergen		√																
6	NJ	Passaic		√																
7	NJ	Sussex		√																
8	NY	Albany																	√	
9	NY	Allegany					√	√		√										
10	NY	Broome			√															
11	NY	Cattaraugus							√		√	√	√							
12	NY	Cayuga			√												√			
13	NY	Chautauqua							√		√	√	√							
14	NY	Chemung			√															
15	NY	Chenango																√		
16	NY	Columbia																		√
17	NY	Cortland															√			
18	NY	Dutchess		√																
19	NY	Erie							√		√		√	√						
20	NY	Fulton																	√	
21	NY	Genesee													√					
22	NY	Greene																		√
23	NY	Herkimer				√												√		
24	NY	Livingston												√	√					
25	NY	Madison				√											√	√		
26	NY	Montgomery																	√	
27	NY	Monroe												√		√				
28	NY	Niagara												√						
29	NY	Oneida				√												√		
30	NY	Onondaga															√			
31	NY	Ontario												√		√				
32	NY	Orange	√	√																
33	NY	Otsego				√												√	√	
34	NY	Putnam	√	√																
35	NY	Rensselaer																		√
36	NY	Rockland	√	√																
37	NY	Schenectady																	√	
38	NY	Schoharie																	√	
39	NY	Schuyler			√															
40	NY	Seneca																		
41	NY	Steuben					√	√		√										
42	NY	Tioga			√															
43	NY	Tompkins			√												√			
44	NY	Wayne														√				
45	NY	Westchester	√	√																
46	NY	Wyoming							√					√	√					
47	NY	Yates			√											√				
48	PA	Erie										√								
49	PA	Potter					√	√		√										
50	PA	Mc Kean										√								
51	PA	Tioga					√	√												
52	PA	Warren										√								

# **NYS Compressor Stations: 2008-2014** **Exposed Populations by Facility: Circular Area Profile – 2, 5, 10, 15, 20 Mile Radii**

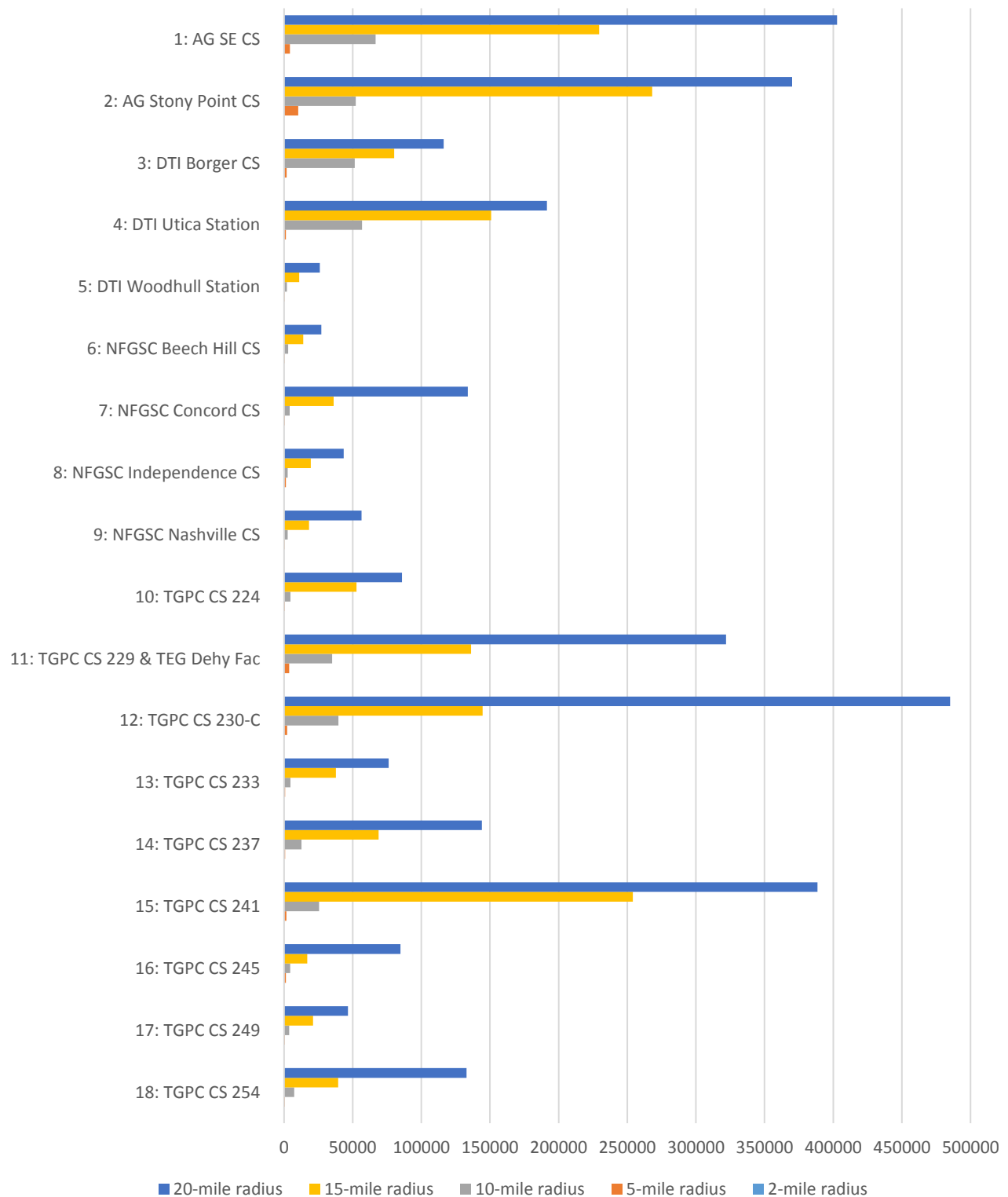


Table 2.5c.1c.

## Total Population by Facility: Circular Area Profile – .05 to 30 Mile Radii

## NYS Natural Gas Compressor Stations: 2008-2014

Facility		Location		Radius								
Address \ County		ST	County	.05	1	2	3	5	10	15	20	30
1	AG SE CS	CT	Fairfield	0	328	1,463	7,535	37,921	148,176	210,298	388,132	916,829
	Southeast NY	CT	Litchfield	0	0	0	0	0	0	11,706	33,700	81,789
	Putnam County	CT	New Haven	0	0	0	0	0	0	0	18,442	276,243
		NY	Dutchess	0	0	0	0	0	0	13,654	58,321	110,574
		NY	Orange	0	0	0	0	0	0	0	0	110,574
		NY	Putnam	0	471	1,640	4,143	16,964	58,575	83,472	96,206	99,710
		NY	Rockland	0	0	0	0	0	0	0	165,975	135,426
		NY	Westchester	0	0	220	886	2,462	24,842	84,264	760,776	493,074
				261	799	3,323	12,564	57,347	231,593	403,394	760,776	2,354,578
2	AG Stony Point CS	CT	Fairfield	0	0	0	0	0	0	0	0	259,310
	Stony Point NY	NJ	Bergen	0	0	0	0	0	0	65,233	269,249	875,306
	Rockland County	NJ	Essex	0	0	0	0	0	0	0	0	67,681
		NJ	Hudson	0	0	0	0	0	0	0	0	10,027
		NJ	Morris	0	0	0	0	0	0	0	0	75,012
		NJ	Passaic	0	0	0	0	0	0	2,463	31,701	501,226
		NJ	Sussex	0	0	0	0	0	0	0	23,392	35,974
		NY	Bronx	0	0	0	0	0	0	0	0	1,225,424
		NY	Dutchess	0	0	0	0	0	0	0	213,509	126,685
		NY	New York	0	0	0	0	0	0	0	0	363,983
		NY	Orange	0	0	0	0	0	37,831	109,807	48,959	346,935
		NY	Putnam	0	0	0	0	0	3,599	18,565	311,687	98,438
		NY	Rockland	704	2,158	10,310	24,626	55,121	213,075	304,874	386,260	311,687
		NY	Ulster	0	0	0	0	0	0	0	0	22,511
		NY	Westchester	0	0	0	0	7,312	76,585	192,761	1,284,757	949,113
				704	2,158	10,310	24,626	62,433	331,090	693,703	1,284,757	5,269,312
3	DTI Borger CS	NY	Broome	0	0	0	0	0	0	0	1,444	53,802
	Ithaca NY	NY	Cayuga	0	0	0	0	0	0	985	5,103	16,463
	Tompkins County	NY	Chemung	0	0	0	0	0	0	0	510	30,899
		NY	Chenango	0	0	0	0	0	0	0	0	801
		NY	Cortland	0	0	0	0	0	0	0	0	48,356
		NY	Onondaga	0	0	0	0	0	0	0	0	4,424
		NY	Schuyler	0	0	0	0	0	0	0	5,297	15,137
		NY	Seneca	0	0	0	0	0	0	0	2,154	6,979
		NY	Tioga	0	0	0	0	0	0	6,003	14,105	43,956
		NY	Tompkins	144	396	2,184	5,155	53,097	80,226	101,564	101,564	101,564
		NY	Yates	0	0	0	0	0	0	0	0	4,428
				0	0	0	0	0	80,226	116,305	168,038	326,809

Facility		Location		Radius							
Address \ County	ST	County	.05	1	2	3	5	10	15	20	30
4	DTI Utica Station	NY	Chenango	0	0	0	0	0	0	0	5,724
	DTI Utica CS	NY	Fulton	0	0	0	0	0	0	0	1,924
	Herkimer County	NY	Herkimer	45	254	1,083	2,181	4,163	29,631	42,888	57,351
		NY	Madison	0	0	0	0	0	0	0	4,167
		NY	Montgomery	0	0	0	0	0	0	0	0
		NY	Oneida	0	0	323	4,062	52,571	121,246	146,635	193,064
		NY	Otsego	0	0	0	0	0	1,901	4,290	19,201
				45	254	1,406	6,243	56,734	150,877	191,424	258,872
5	DTI Woodhull Station	NY	Allegany	0	0	0	0	0	0	684	20,261
	Woodhull NY	NY	Chemung	0	0	0	0	0	0	0	22,692
	Steuben County	NY	Schuyler	0	0	0	0	0	0	0	1,609
		NY	Steuben	2	57	371	950	2,031	6,800	16,285	48,062
		PA	Bradford	0	0	0	0	0	0	0	814
		PA	Potter	0	0	0	0	0	1,037	1,037	5,689
		PA	Tioga	0	0	0	0	99	4,192	8,680	14,101
				2	57	371	950	2,130	10,992	26,002	63,884
6	FGSC Beech Hill CS	NY	Allegany	43	64	329	687	2,605	12,105	19,352	28,464
	Willing NY	NY	Cattaraugus	0	0	0	0	0	0	0	11,656
	Allegany County	NY	Steuben	0	0	0	0	0	545	1,955	9,356
		PA	Mc Kean	0	0	0	0	0	0	0	5,843
		PA	Potter	0	0	0	0	394	1,305	5,867	7,868
		PA	Tioga	0	0	0	0	0	0	0	3,194
				43	64	329	687	2,999	13,995	27,174	48,882
7	NFGSC Concord CS	NY	Allegany	0	0	0	0	0	0	0	2,130
	Concord NY	NY	Cattaraugus	0	0	0	0	0	11,290	19,042	46,639
	Erie County	NY	Chautauqua	0	0	0	0	0	0	0	5,989
		NY	Erie	0	125	579	1,346	4,168	36,020	122,570	230,067
		NY	Genesee	0	0	0	0	0	0	0	1,155
		NY	Wyoming	0	0	0	0	0	0	0	3,304
				0	125	579	1,346	4,168	36,020	133,860	258,402
8	NFGSC Independ. CS	NY	Allegany	839	1,080	1,377	1,639	2,491	18,062	25,189	31,630
	Andover NY	NY	Cattaraugus	0	0	0	0	0	0	0	4,246
	Allegany County	NY	Livingston	0	0	0	0	0	0	0	7,318
		NY	Steuben	0	0	0	0	147	1,410	16,860	23,738
		PA	Mc Kean	0	0	0	0	0	0	0	641
		PA	Potter	0	0	0	0	0	1,305	4,846	12,175
		PA	Tioga	0	0	0	0	0	0	0	7,472
				839	1,080	1,377	1,639	2,638	19,472	43,354	60,214
9	NFGSC Nashville CS	NY	Cattaraugus	0	0	19	147	866	4,721	8,266	11,894
	Hanover NY	NY	Chautauqua	41	166	560	1,173	5,505	10,774	39,199	48,450
	Chautauqua County	PA	Erie	0	0	0	0	549	13,008	32,427	61,899
				41	166	579	1,320	6,920	28,503	79,892	122,243

Facility		Location		Radius								
Address \ County		ST	County	.05	1	2	3	5	10	15	20	30
10	TGPC CS 224	NY	Cattaraugus	0	0	12	43	253	2,866	5,803	13,694	33,184
	Clymer NY	NY	Chautauqua	95	103	610	1,602	4,321	47,133	59,704	70,083	104,211
	Chautauqua County	PA	Erie	0	0	0	0	0	0	0	0	886
		PA	Mc Kean	0	0	0	0	0	0	0	4,540	24,334
		PA	Warren	0	0	0	0	115	2,695	20,317	30,078	39,983
				95	103	622	1,645	4,689	52,694	85,824	118,395	206,884
11	TGPC CS 229 & TEG DF	NY	Cattaraugus	0	0	0	0	0	0	115	9,341	29,317
	Eden NY	NY	Chautauqua	0	0	0	0	0	0	0	5,152	29,114
	Erie County	NY	Erie	151	726	3,803	11,106	34,960	136,180	321,782	673,481	917,797
		NY	Genesee	0	0	0	0	0	0	0	0	4,787
		NY	Niagara	0	0	0	0	0	0	0	0	72,143
		NY	Wyoming	0	0	0	0	0	0	0	0	17,502
				151	726	3,803	11,106	34,960	136,180	321,897	687,974	1,070,660
	12	TGPC CS 230-C	NY	Erie	0	0	0	0	0	33,009	277,098	607,651
Lockport NY		NY	Genesee	0	0	0	0	0	0	0	1,494	18,499
Niagara County		NY	Niagara	12	359	2,202	5,922	39,624	111,553	208,081	216,469	216,469
		NY	Orleans	0	0	0	0	0	0	0	9,214	26,607
		NY	Wyoming	0	0	0	0	0	0	0	0	2,454
				12	359	2,202	5,922	39,624	144,562	485,179	834,828	1,086,847
13	TGPC CS 233	NY	Allegany	0	0	0	0	0	0	0	0	5,331
	York NY	NY	Erie	0	0	0	0	0	0	0	0	5,496
	Livingston County	NY	Genesee	0	0	0	22	368	6,581	14,360	42,808	60,079
		NY	Livingston	15	109	841	2,013	3,579	26,388	41,687	56,647	65,393
		NY	Monroe	0	0	0	0	0	0	5,632	44,228	571,226
		NY	Ontario	0	0	0	0	0	0	0	2,148	36,095
		NY	Orleans	0	0	0	0	0	0	0	0	11,958
		NY	Wyoming	0	0	0	105	591	4,800	14,538	26,836	38,776
				15	109	841	2,140	4,538	37,769	76,217	172,667	800,524
14	TGPC CS 237	NY	Cayuga	0	0	0	0	0	0	0	0	20,233
	Manchester, Phelps NY	NY	Livingston	0	0	0	0	0	0	0	0	22,671
	Ontario County	NY	Monroe	0	0	0		0	0	3,963	75,265	509,857
		NY	Ontario	27	211	796	5,815	12,654	53,584	93,899	100,200	107,931
		NY	Seneca	0	0	0	0	0	0	7,558	20,829	33,097
		NY	Steuben	0	0	0	0	0	0	0	0	3,370
		NY	Wayne	0	0	0	0	0	15,237	37,245	64,847	90,461
		NY	Yates	0	0	0	0	0	0	1,458	10,492	24,006
				27	211	796	5,815	12,654	68,821	144,123	271,633	811,626
15	TGPC CS 241	NY	Cayuga	0	0	0	0	0	0	0	7,841	71,871
	LaFayette NY	NY	Chenango	0	0	0	0	0	0	0	0	2,522
	Onondaga County	NY	Cortland	0	0	0	0	0	0	2,228	5,932	41,458
		NY	Madison	0	0	0	0	0	0	16,456	30,214	63,437
		NY	Oneida	0	0	0	0	0	0	0	0	8,338
		NY	Onondaga	218	460	1,627	4,484	25,469	254,062	369,779	460,535	467,026
		NY	Oswego	0	0	0	0	0	0	0	0	36,965
		NY	Tompkins	0	0	0	0	0	0	0	0	8,227
		218	460	1,627	4,484	25,469	254,062	388,463	504,522	699,844		

Facility		Location		Radius									
Address \ County		ST	County	.05	1	2	3	5	10	15	20	30	
16	TGPC CS 245	NY	Chenango	0	0	0	0	0	0	0	3,307	20,317	
	Winfield NY	NY	Fulton	0	0	0	0	0	0	0	0	1,924	
	Herkimer County	NY	Herkimer	0	166	1,263	1,528	2,261	5,179	36,884	49,510	60,443	
		NY	Madison	0	0	0	0	213	1,426	2,545	12,243	34,278	
		NY	Montgomery	0	0	0	0	0	0	0	0	9,217	
		NY	Oneida	0	0	0	108	966	5,933	35,350	128,462	195,132	
		NY	Otsego	0	0	103	333	1,030	4,290	10,013	17,561	53,366	
		NY	Schoharie	0	0	0	0	0	0	0	0	1,000	
				0	166	1,366	1,969	4,470	16,828	84,792	211,083	375,677	
17	TGPC CS 249	NY	Albany	0	0	0	0	0	0	0	8,411	96,415	
	Carlisle NY	NY	Delaware	0	0	0	0	0	0	0	0	2,701	
	Schoharie County	NY	Fulton	0	0	0	0	0	0	0	0	17,648	51,847
		NY	Greene	0	0	0	0	0	0	0	0	0	2,887
		NY	Herkimer	0	0	0	0	0	0	0	0	0	6,586
		NY	Montgomery	0	0	21	174	728	4,630	5,093	46,944	50,219	
		NY	Otsego	0	0	0	0	0	0	0	3,476	13,485	
		NY	Saratoga	0	0	0	0	0	0	0	0	0	23,460
		NY	Schenectady	0	0	0	0	0	0	1,437	9,936	154,727	
		NY	Schoharie	0	71	476	1,449	4,063	16,500	17,511	29,373	32,749	
				0	0	497	1,623	4,791	21,130	24,041	115,788	435,076	
18	TGPC CS 254	MA	Berkshire	0	0	0	0	0	0	10,311	65,680	126,293	
	Chatham NY	NY	Albany	0	0	0		0	0	24,105	194,709	298,289	
	Columbia County	NY	Columbia	10	65	298	761	2,635	18,420	27,699	43,451	56,855	
		NY	Greene	0	0	0	0	0	0	2,869	14,566	38,909	
		NY	Rensselaer	0	72	345	861	4,820	20,895	67,895	124,211	152,634	
		NY	Schenectady	0	0	0	0	0	0	0	0	102,292	
		VT	Bennington	0	0	0	0	0	0	0	0	5,096	
				10	137	643	1,622	7,455	39,315	132,879	442,617	839,481	

At a given site the concentrations of pollutants is largely directly dependent on local emissions, but there are many important exceptions to this general rule.

In this connection, the most important fact to bear in mind is that ***human illness or an adverse environmental effect is not necessarily the result of the preponderance of pollutants in a place but may be caused by a single pollutant which may have traveled great distances and that relatively small quantities can be extremely dangerous.***

As we have already indicated, the distance air pollution travels and how much reaches ground level is dependent on many factors. On any given day, pollution from a given site can travel less than a mile, a few miles, hundreds of miles, thousands of miles, or around the globe.

A few local examples.

### Chernobyl disaster

The meltdown of the Chernobyl nuclear power plant in the Ukraine on April 26, 1986, released 100 times more airborne radiation than the fallout from U.S. nuclear bombs dropped on Hiroshima and Nagasaki. More than 40% of Europe's land mass to the north and west and had measurable amounts of radiation contamination, including Austria, Belarus, Bulgaria, Finland, France, Germany, Great Britain, Greece, Iceland, Italy, Norway, Romania, Slovenia, Sweden, and Switzerland, wide territories to the south including Armenia, Georgia, northern Africa and the Emirates, and China to the west. By May 6<sup>th</sup>, contamination reached Canada and the U.S. – more than half-way around the globe. (Yablokov and Nesterenko 2009, Gould 1990). The conclusion reached by the UN Scientific Committee on the Effects of Atomic Radiation is that the Chernobyl disaster "Resulted in radioactive material becoming widely dispersed and deposited . . . throughout the northern hemisphere." And that "[r]eleases of radioactive materials were such that contamination of the ground was found to some extent in every country in the Northern Hemisphere." (UNSCEAR 2011). Measurable amounts of Iodine-131 from Chernobyl fallout were found in fresh milk (Feely et al. 1988) and New York City's air along with Cesium-137 (U.S. DOE 1986), total ground deposition of Iodine-131 and Cesium-137 in Chester, New Jersey (U.S. DOE 1986), and gross beta particles in precipitation in Montpelier, Vermont (U.S. EPA 1986).

### Fallout from U.S. Nuclear Weapons Tests: Rochester and Troy NY

At dawn on July 16, 1945 in the dessert of Alamogordo, New Mexico, America exploded the world's first atomic weapon, code named "Trinity". Over the next few weeks Eastman Kodak headquarters were flooded with complaints from customers who had purchased sensitive X-ray film that it had been rendered unusable due to "fogging". Within a few weeks the company's scientists had determined that the strawboard, used as a stiffener board between film sheets produced in mills in Vincennes, Indiana had been contaminated "a new type radioactive containment not hitherto encountered." This, in turn, had produced black exposed spots on the company's film.

Alerted to the danger of open-air testing of nuclear weapons, Kodak began routinely measuring ambient radioactivity. In late January 29, 1951, the company's Geiger counters measured elevated levels of radioactivity brought to the ground by



a winter snow storm. The radiation was the result of a 1-kiloton nuclear test that had taken place in Nevada two days earlier. On February 3, the *New York Times* ran a front-page story on the incident, highlighting the work of University of Rochester scientists who had quantified trace amounts of radioactivity in the city's snow. (Mehmott 2016)

On April 28, 1953, the Geiger counters of Rensselaer Polytechnic Institute chemistry professor Herbert Clark began crackling away at surprising high levels. A severe rainstorm had brought down radiation from a nuclear test that had occurred three days earlier in the Nevada desert. The blast from the 11,000-lb. nuclear bomb code-named Simon had risen to a height of 44,000 feet above sea level, where 115 miles an hour winds carried it to Troy, some 2,300 miles downwind in just a few days. Levels of radioactivity in drinking water measured the next day were 100 to 1000 times greater than natural background radioactivity. (Clark 1954, Lade 1953, Lade 1962, Heller 2003).

### Depleted Uranium Contamination: Albany and Colonie NY

From the late 1950s through 1980, the National Lead company and the U.S. government operated a facility on Central Avenue in Colonie, New York, that fashioned depleted uranium (DU) for use in U.S. armor-piercing shot and shell, and in the process exposed its workforce and nearby residents to significant levels of radioactive contaminants. During its years of operation there no efforts were made to systematically monitor air, soil, surface water or groundwater for excessive contamination at either the 18-acre work site or in the community where the plant was located. More than 20 years after the plant closed, researchers found measurable levels of DU among former workers and Colonie residents (Parrish et al. 2008). But DU contamination was not confined to National Lead property or even the nearby neighborhood of Roessleville. In the 1990s, air filters at Knolls Atomic Power Laboratory in Niskayuna, about 3.5 miles away, detected DU from National Lead. Even more alarming is that more than 25 miles away DU contamination was detected by the Kesselring Naval Nuclear Laboratory in Milton.

### Elevated Rates of Birth Defects 10 miles from Natural Gas Wells

The chemicals found in the air around natural gas wells are generally the same chemicals found in compressor station emissions. McKenzie et al. found elevations in rates of birth defects of the cardiovascular system, and border-line elevations in rates of neurotube birth defects among people who live within ten miles of natural gas wells. This is a striking finding, as the study was of 124,842 births between 1996 and 2009, and the fact that birth defects are relatively rare and that more than half of the birth were the controls that did not live within ten miles of gas wells. There was also a significant association with the numbers of well and the distance. The author conclude that the result suggests a positive association between density and proximity to gas wells within a ten-mile radius and birth defects of the heart and possibly neurotube defects, but not with oral clefts, preterm birth or reduced fetal growth.

### Small-Scale Spatial Variations

On the other end of the scale, small-scale spatial variations of only a few feet or yards have been shown to significantly effect personal exposure to ambient PAH concentrations. (Lovinsky-Desir et al. 2016)

Table 2.5c.1b.

#### Total Pounds by Facility: Circular Area Air Pollution Profile – .05-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	.05-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	DTI Woodhull Station	Woodhull	Steuben	829,223	2	414,611
2	TGPC CS 254	Chatham	Columbia	2,393,660	10	239,366
3	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	27	85,125
4	TGPC CS 249	Carlisle	Schoharie	4,323,285	94	45,992
5	TGPC CS 230-C	Lockport	Niagara	485,609	12	40,467
6	TGPC CS 229 & TEG DF	Eden	Erie	5,124,426	151	33,936
7	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	43	32,269
8	NFGSC Nashville CS	Hanover	Chautauqua	622,791	26	23,953
9	TGPC CS 233	York	Livingston	224,978	15	14,998
10	TGPC CS 241	LaFayette	Onondaga	3,039,661	218	13,943
11	TGPC CS 224	Clymer	Chautauqua	1,146,797	95	12,071
12	DTI Borger CS	Ithaca	Tompkins	780,159	92	8,479
13	DTI Utica Station	Frankfort	Herkimer	281,369	45	6,252
14	AG SE CS	Southeast	Putnam	1,688,814	287	5,884
15	AG Stony Point CS	Stony Point	Rockland	2,013,478	704	2,860
16	NFGSC Independence CS	Andover	Allegany	1,353,931	839	1,613
17	TGPC CS 245	Winfield	Herkimer	10,465,388	0	--
18	NFGSC Concord CS	Concord	Erie	1,733,171	0	--
				<b>40,192,726</b>	<b>2,660</b>	<b>15,110</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 10-Mile Radius, Pounds per capita

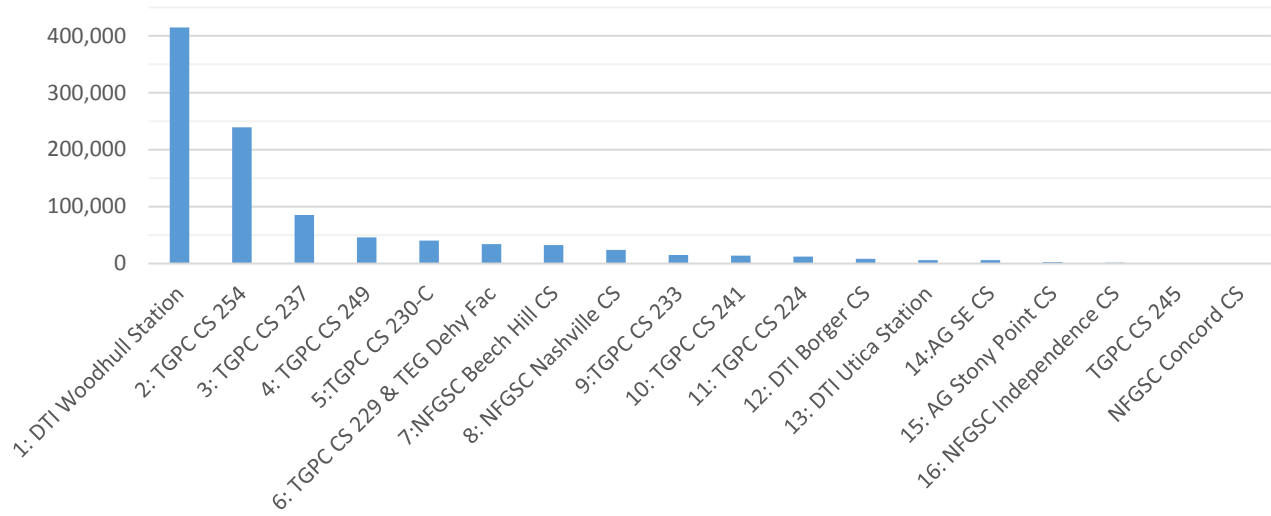


Table 2.5c.1c.

## Total Pounds by Facility: Circular Area Air Pollution Profile – 1-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	1-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	TGPC CS 245	Winfield	Herkimer	10,465,388	166	124,588
2	TGPC CS 249	Carlisle	Schoharie	4,323,285	154	28,073
3	NFGSC Concord CS	Concord	Erie	1,733,171	66	26,260
4	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	64	21,681
5	TGPC CS 254	Chatham	Columbia	2,393,661	137	17,472
6	DTI Woodhull Station	Woodhull	Steuben	829,223	57	14,548
7	TGPC CS 224	Clymer	Chautauqua	1,146,797	103	11,134
8	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	211	10,893
9	TGPC CS 229 & TEG DF	Eden	Erie	5,124,427	726	7,058
10	TGPC CS 241	LaFayette	Onondaga	3,039,661	460	6,608
11	NFGSC Nashville CS	Hanover	Chautauqua	622,791	177	3,519
12	TGPC CS 233	York	Livingston	224,978	109	2,064
13	AG SE CS	Southeast	Putnam	1,688,815	845	1,999
14	DTI Borger CS	Ithaca	Tompkins	780,159	396	1,970
15	TGPC CS 230-C	Lockport	Niagara	485,610	359	1,353
16	NFGSC Independence CS	Andover	Allegany	1,353,931	1080	1,254
17	DTI Utica Station	Frankfort	Herkimer	281,369	254	1,108
18	AG Stony Point CS	Stony Point	Rockland	2,013,478	2158	933
				<b>40,192,733</b>	<b>7,522</b>	<b>5,343</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 1-Mile Radius, Pounds per capita

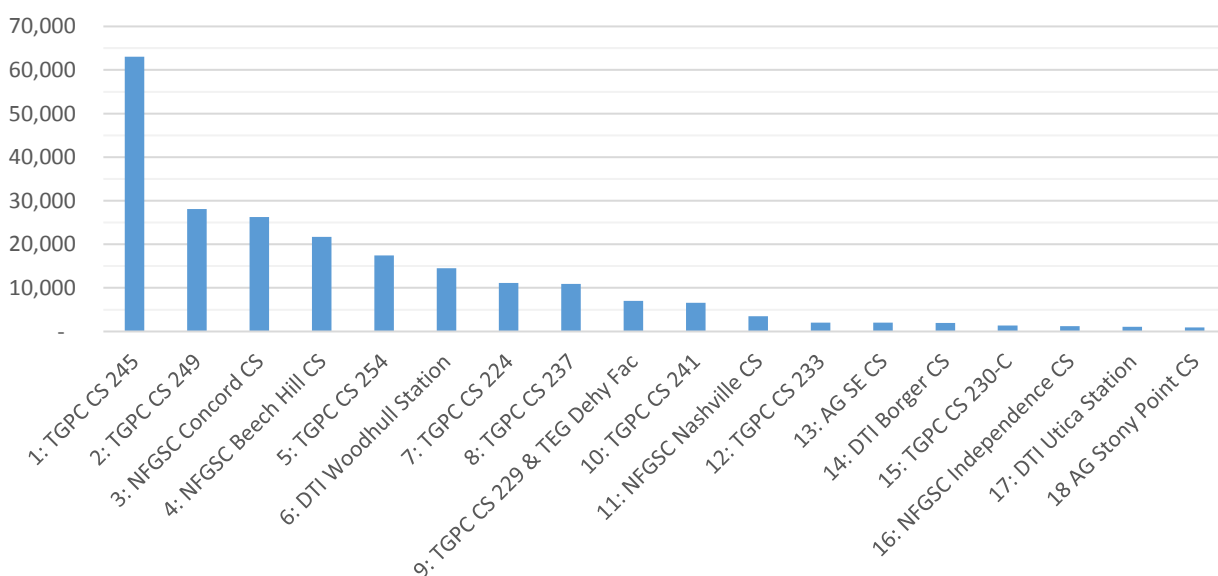


Table 2.5c.1d.

## Total Pounds by Facility: Circular Area Air Pollution Profile – 2-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	2-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	TGPC CS 245	Winfield	Herkimer	10,465,389	1,366	7,661
2	TGPC CS 249	Carlisle	Schoharie	4,323,285	675	6,405
3	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	329	4,218
4	TGPC CS 254	Chatham	Columbia	2,393,661	643	3,723
5	NFGSC Concord CS	Concord	Erie	1,733,171	529	3,276
6	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	796	2,887
7	DTI Woodhull Station	Woodhull	Steuben	829,223	371	2,235
8	TGPC CS 241	LaFayette	Onondaga	3,039,661	1,627	1,868
9	TGPC CS 224	Clymer	Chautauqua	1,146,797	622	1,844
10	NFGSC Nashville CS	Hanover	Chautauqua	622,791	383	1,626
11	TGPC CS 229 & TEG DF	Eden	Erie	5,124,427	3,803	1,347
12	NFGSC Independence CS	Andover	Allegany	1,353,931	1,377	983
13	DTI Borger CS	Ithaca	Tompkins	780,159	1,879	415
14	AG SE CS	Southeast	Putnam	1,688,815	4,307	392
15	TGPC CS 233	York	Livingston	224,978	841	268
16	TGPC CS 230-C	Lockport	Niagara	485,610	2,202	221
17	DTI Utica Station	Frankfort	Herkimer	281,369	1,406	200
18	AG Stony Point CS	Stony Point	Rockland	2,013,478	10,310	195
				<b>40,192,733</b>	<b>33,466</b>	<b>39,765</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 2-Mile Radius, Pounds per capita

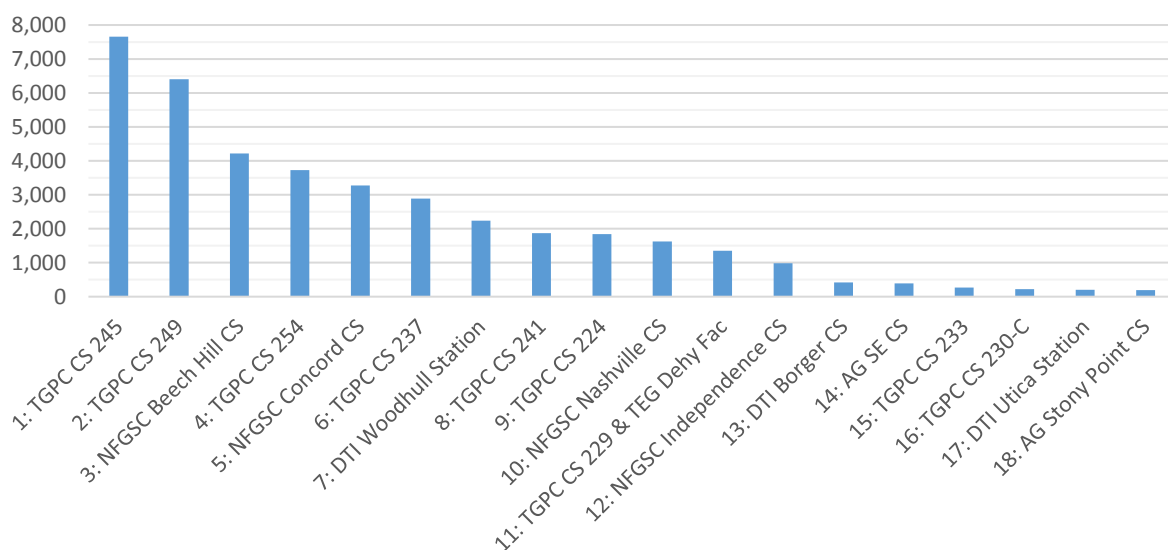


Table 2.5c.1d.

## Total Pounds by Facility: Circular Area Air Pollution Profile – 3-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	3-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	TGPC CS 245	Winfield	Herkimer	10,465,388	1,969	5,315
2	TGPC CS 249	Carlisle	Schoharie	4,323,285	1,266	3,414
3	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	687	2,019
4	TGPC CS 254	Chatham	Columbia	2,393,660	1,622	1,475
5	NFGSC Concord CS	Concord	Erie	1,733,171	1,297	1,336
6	NFGSC Nashville CS	Hanover	Chautauqua	622,791	675	922
7	DTI Woodhull Station	Woodhull	Steuben	829,223	950	872
8	NFGSC Independence CS	Andover	Allegany	1,353,931	1,639	826
9	TGPC CS 224	Clymer	Chautauqua	1,146,797	1,645	697
10	TGPC CS 241	LaFayette	Onondaga	3,039,661	4,484	677
11	TGPC CS 229 & TEG DF	Eden	Erie	5,124,426	11,106	461
12	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	5,815	395
13	DTI Borger CS	Ithaca	Tompkins	780,159	5,165	151
14	AG SE CS	Southeast	Putnam	1,688,814	13,824	122
15	TGPC CS 233	York	Livingston	224,978	2,140	105
16	TGPC CS 230-C	Lockport	Niagara	485,609	5,922	82
17	AG Stony Point CS	Stony Point	Rockland	2,013,478	24,626	81
18	DTI Utica Station	Frankfort	Herkimer	281,369	6,243	45
				<b>40,192,726</b>	<b>91,075</b>	<b>441</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 3-Mile Radius, Pounds per capita

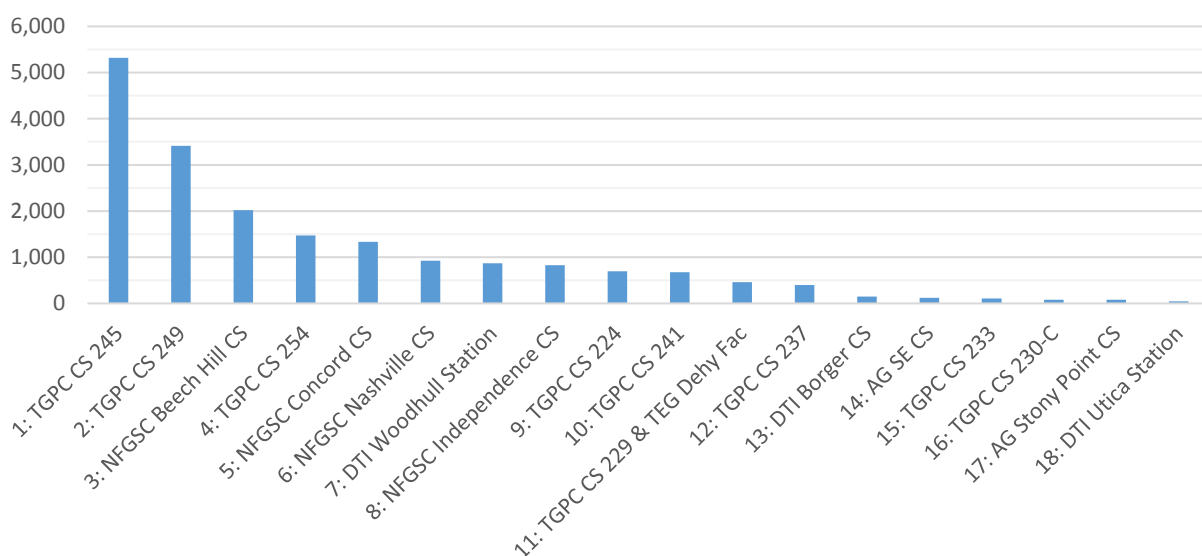


Table 2.5c.1f.

## Total Pounds by Facility: Circular Area Air Pollution Profile – 5-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	5-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	TGPC CS 245	Winfield	Herkimer	10,465,389	4,470	2,341
2	TGPC CS 249	Carlisle	Schoharie	4,323,285	3,668	1,179
3	NFGSC Independence CS	Andover	Allegany	1,353,931	2,638	513
4	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	2,999	463
5	NFGSC Concord CS	Concord	Erie	1,733,171	3,931	441
6	DTI Woodhull Station	Woodhull	Steuben	829,223	2,130	389
7	TGPC CS 254	Chatham	Columbia	2,393,661	7,455	321
8	TGPC CS 224	Clymer	Chautauqua	1,146,797	4,689	245
9	NFGSC Nashville CS	Hanover	Chautauqua	622,791	2,584	241
10	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	12,654	182
11	TGPC CS 229 & TEG DF	Eden	Erie	5,124,427	34,960	147
12	TGPC CS 241	LaFayette	Onondaga	3,039,661	25,469	119
13	TGPC CS 233	York	Livingston	224,978	4,538	50
14	AG Stony Point CS	Stony Point	Rockland	2,013,478	62,433	32
15	AG SE CS	Southeast	Putnam	1,688,815	66,671	25
16	DTI Borger CS	Ithaca	Tompkins	780,159	51,509	15
17	TGPC CS 230-C	Lockport	Niagara	485,610	39,624	12
18	DTI Utica Station	Frankfort	Herkimer	281,369	56,734	5
				<b>40,192,733</b>	<b>389,156</b>	<b>103</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 5-Mile Radius, Pounds per capita

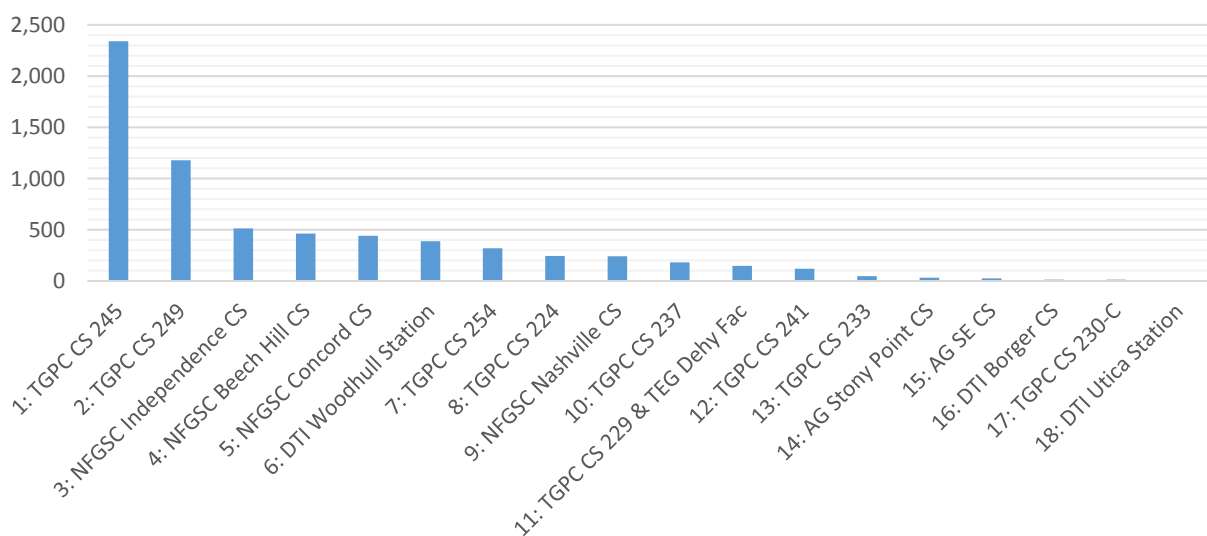


Table 2.5c.1g.

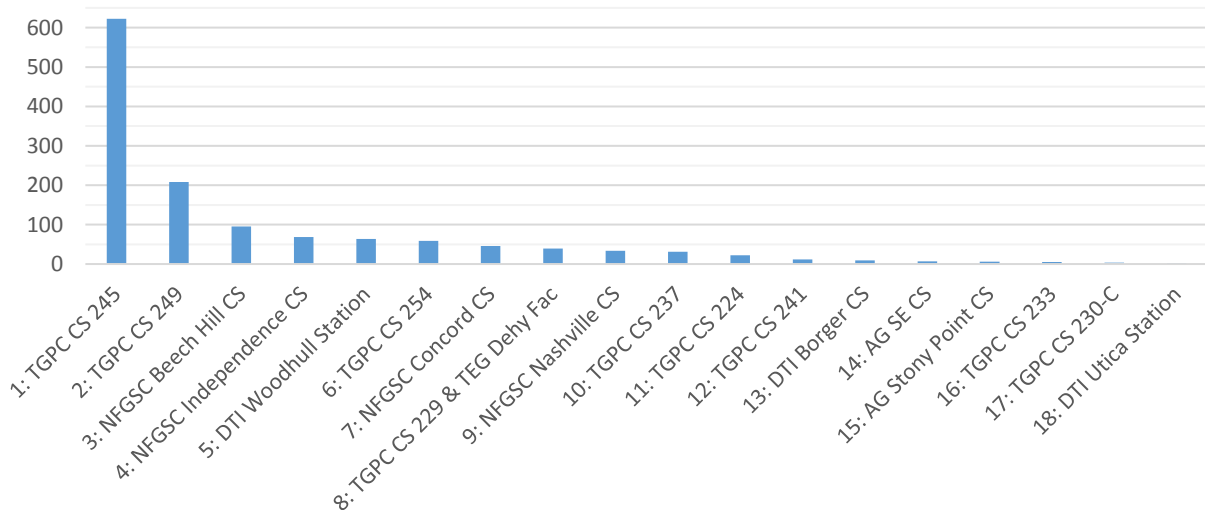
## Total Pounds by Facility: Circular Area Air Pollution Profile – 10-Mile Radius

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		Releases	10-Mile Radius	
	Facility Name (Short)	Town	County	7-Years	Population	Lbs. per capita
1	TGPC CS 245	Winfield	Herkimer	10,465,389	16,826	622
2	TGPC CS 249	Carlisle	Schoharie	4,323,285	20,745	208
3	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	14,592	95
4	NFGSC Independence CS	Andover	Allegany	1,353,931	19,772	68
5	DTI Woodhull Station	Woodhull	Steuben	829,223	12,947	64
6	TGPC CS 254	Chatham	Columbia	2,393,661	40,695	59
7	NFGSC Concord CS	Concord	Erie	1,733,171	37,974	46
8	TGPC CS 229 & TEG DF	Eden	Erie	5,124,427	131,667	39
9	NFGSC Nashville CS	Hanover	Chautauqua	622,791	18,661	33
10	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	72,831	32
11	TGPC CS 224	Clymer	Chautauqua	1,146,797	51,965	22
12	TGPC CS 241	LaFayette	Onondaga	3,039,661	257,224	12
13	DTI Borger CS	Ithaca	Tompkins	780,159	84,577	9
14	AG SE CS	Southeast	Putnam	1,688,815	235,473	7
15	AG Stony Point CS	Stony Point	Rockland	2,013,478	330,569	6
16	TGPC CS 233	York	Livingston	224,978	40,531	6
17	TGPC CS 230-C	Lockport	Niagara	485,610	145,809	3
18	DTI Utica Station	Frankfort	Herkimer	281,369	148,087	2
				<b>40,192,733</b>	<b>1,680,945</b>	<b>24</b>

## NYS Compressor Station Releases: 2008-2014

Total Pounds by Facility: Circular Area Profile, 10-Mile Radius, Pounds per capita





## 2.5c.2. Total Pounds by Facility: Annual, Monthly, Daily and Hourly Averages

Table 2.5c.

### Total Pounds by Facility: Annual, Monthly, Daily and Hourly Averages

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Identification	Location		7 Years Total	Annual Average	Monthly Average	Daily Average	Hourly Average
	Facility Name (Short)	Town	County					
1	TGPC CS 245	Winfield	Herkimer	10,465,388	1,495,055	124,588	4,096	171
2	TGPC 229 & TEG DF	Eden	Erie	5,124,426	732,061	61,005	2,006	84
3	TGPC CS 249	Carlisle	Schoharie	4,323,285	617,612	51,468	1,692	71
4	TGPC CS 241	LaFayette	Onondaga	3,039,661	434,237	36,186	1,190	50
5	TGPC CS 254	Chatham	Columbia	2,393,660	341,951	28,496	937	39
6	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	328,342	27,362	900	37
7	AGT Stony Point CS	Stony Point	Rockland	2,013,478	287,640	23,970	788	33
8	NFGSC Concord CS	Concord	Erie	1,733,171	247,596	20,633	678	28
9	AGT SOUTHEAST CS	Southeast	Putnam	1,688,814	241,259	20,105	661	28
10	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	198,227	16,519	543	23
11	NFGSC Independence CS	Andover	Allegany	1,353,931	193,419	16,118	530	22
12	TGPC CS 224	Clymer	Chautauqua	1,146,797	163,828	13,652	449	19
13	DTI Woodhull Station	Woodhull	Steuben	829,223	118,460	9,872	325	14
14	DTI Borger CS	Ithaca	Tompkins	780,159	111,451	9,288	305	13
15	NFGSC Nashville CS	Hanover	Chautauqua	622,791	88,970	7,414	244	10
16	TGPC CS 230-C	Lockport	Niagara	485,609	69,373	5,781	190	8
17	DTI Utica Station	Frankfort	Herkimer	281,369	40,196	3,350	110	5
18	TGPC CS 233	York	Livingston	224,978	32,140	2,678	88	4
				<b>40,192,726</b>	<b>5,741,818</b>	<b>478,485</b>	<b>15,731</b>	<b>655</b>

## 2.5d. Releases by NYS DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions.

Region 9, Western New York, ranked first with 11.6 million pounds (29.1%), closely followed by Region 6, Western Adirondacks/Eastern Lake Ontario (10.7 million pounds or 27%).

Region 4, Capital Region/Northern Catskills, ranked third with 6.7 million pounds (16.8%).

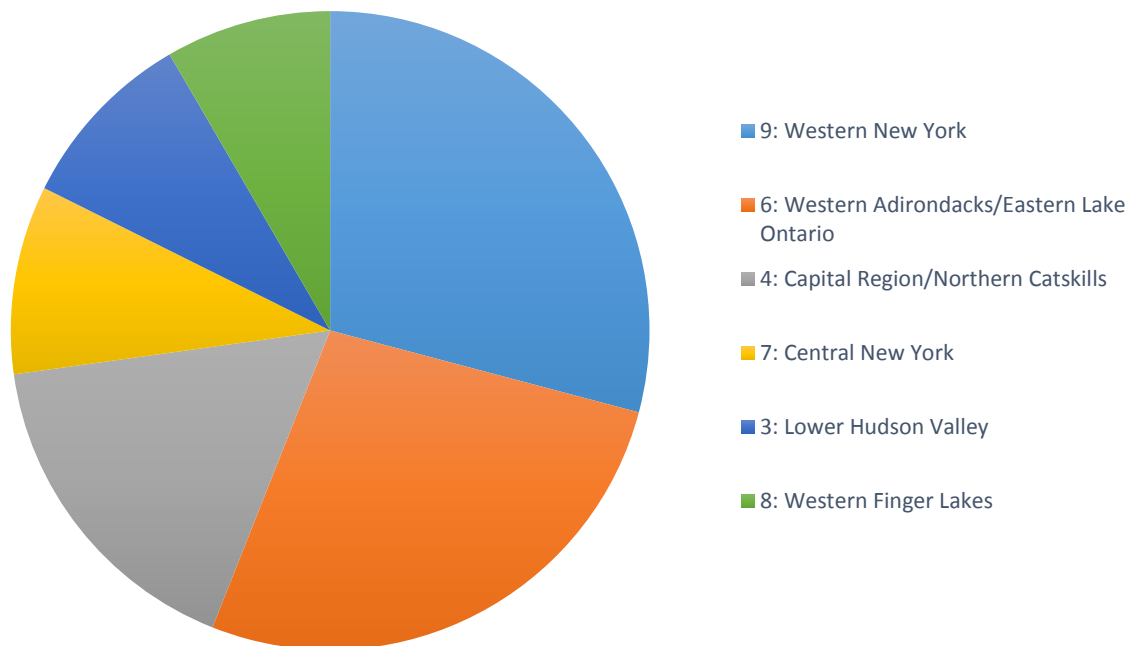
Table 2.5d.

### Total Releases by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 11, 14			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac	Ch	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	22	1,174,939	391,646	2,741,523	6.86
		Chautauqua	10	2	62	669,425	223,142	1,561,991	3.91
		Erie	2	2	55	2,938,971	979,657	6,857,598	17.15
		Niagara	13	1	27	208,118	69,373	485,610	1.21
				7	67	4,991,452	1,663,817	11,646,722	29.13
2	6: W. Adirondacks / E. Lake Ontario	Herkimer	1	2	67	4,605,753	1,535,251	10,746,758	26.88
3	4: Capital Region / N. Catskills	Columbia	6	1	27	1,025,855	341,952	2,393,661	5.99
		Schoharie	3	1	50	1,852,836	617,612	4,323,285	10.81
				2	57	2,878,691	959,564	6,716,946	16.80
4	7: Central New York	Onondaga	4	1	48	1,302,712	434,237	3,039,661	7.60
		Tompkins	12	1	47	334,354	111,451	780,159	1.95
				2	68	1,637,066	545,689	3,819,820	9.55
5	3: Lower Hudson Valley	Putnam	9	1	48	723,778	241,259	1,688,815	4.22
		Rockland	8	1	49	862,919	287,640	2,013,478	5.04
				2	63	1,586,697	528,899	3,702,293	9.26
6	8: Western Finger Lakes	Livingston	14	1	27	96,419	32,140	224,978	0.56
		Ontario	7	1	9	985,026	328,342	2,298,394	5.75
		Steuben	11	1	61	355,381	118,460	829,223	2.07
				3	61	1,436,827	478,942	3,352,596	8.38
				18	70	17,136,487	5,712,162	39,985,136	100%

**NYS Compressor Station NEI Releases by Region: 2008-2014**  
**Total Releases (Pounds)**



## 2.5e. Releases by County

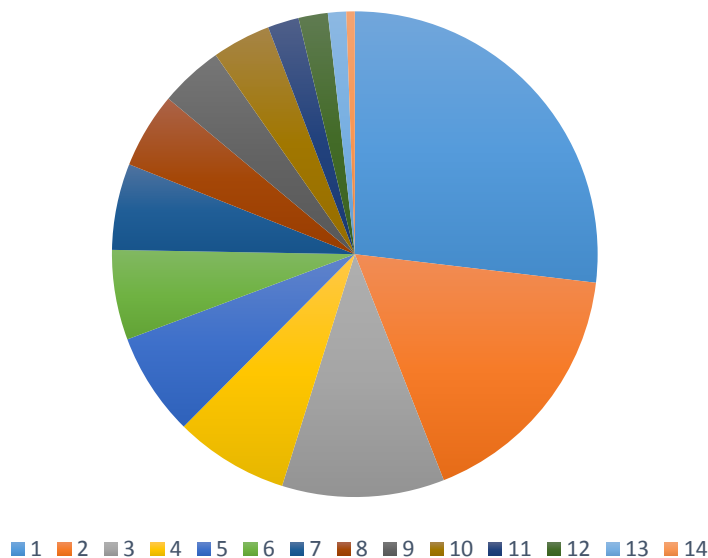
Table 2.5e.

### Total Pounds by County (ranked)

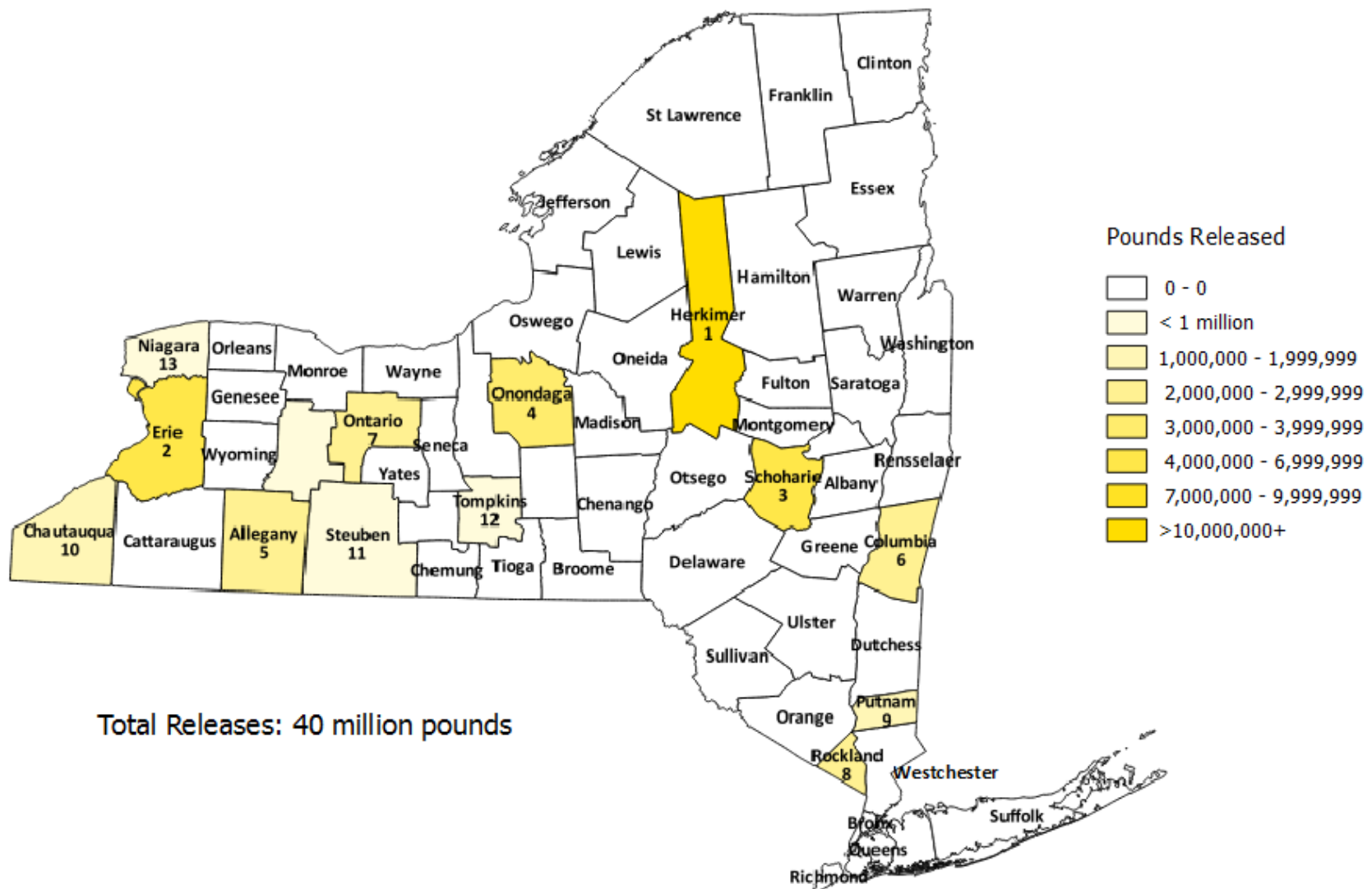
NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

Rank	Location				Pounds				7 Year Total	
	County	NY DEC Region	Fac	Ch	2008	2011	2014	3 Yr. Avg.	Pounds	%
1	Herkimer	6: W. Adirondacks/E. Lake Ontario	2	67	796,186	1,937,795	1,871,770	1,535,251	10,746,757	26.88
2	Erie	9: Western New York	2	55	864,493	1,161,927	912,550	979,656	6,857,598	17.15
3	Schoharie	4: Capital Region/N. Catskills	1	50	712,001	569,087	571,747	617,612	4,323,285	10.81
4	Onondaga	7: Central New York	1	48	297,484	574,213	431,013	434,237	3,039,661	7.60
5	Allegany	9: Western New York	2	22	235,166	413,713	526,058	391,646	2,741,523	6.86
6	Columbia	4: Capital Region/N. Catskills	1	27	288,373	260,769	476,711	341,951	2,393,660	5.99
7	Ontario	8: Western Finger Lakes	1	9	321,292	482,042	181,690	328,342	2,298,394	5.75
8	Rockland	3: Lower Hudson Valley	1	49	244,039	268,064	350,815	287,639	2,013,478	5.04
9	Putnam	3: Lower Hudson Valley	1	48	161,096	255,289	307,391	241,259	1,688,814	4.22
10	Chautauqua	9: Western New York	2	62	144,599	468,880	55,945	223,141	1,561,991	3.91
11	Steuben	8: Western Finger Lakes	1	61	104,802	209,129	41,449	118,460	829,223	2.07
12	Tompkins	7: Central New York	1	47	129,003	83,412	121,937	111,451	780,159	1.95
13	Niagara	9: Western New York	1	27	83,450	2,791	121,876	69,372	485,609	1.21
14	Livingston	8: Western Finger Lakes	1	27	55,594	31,315	9,509	32,139	224,978	0.56
			18		4,437,578	6,718,426	5,980,461	5,712,156	39,985,130	100%

**NYS Compressor Station NEI Releases by County: 2008-2014**  
Total Releases



## Total Releases by County



Total Releases: 40 million pounds

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## Chapter 3: Health Effects

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## Introduction

For most diseases discussed in this study, there is evidence of a relationship between specific **chemical exposures** and specific health outcomes.

An increase in certain vector borne infectious and parasitic diseases may result due to a warmer climate created by greenhouse gases, not because of chemical exposure per se. This is best described as an instance of “systemic causation.”

The WHO provides this definition of epidemiology:

Epidemiology is the study of the distribution and determinants of health-related states or events (including disease), and the application of this study to the control of diseases and other health problems. Various methods can be used to carry out epidemiological investigations: surveillance and descriptive studies can be used to study distribution; analytical studies are used to study determinants. (WHO)

The reader should be aware (1) that within science different conceptions of causality are employed, and (2) there is a difference between how science establishes proof of a relationship and that required in legal adjudication.

In mathematics, one can prove a theorem with absolute certainty of 100%.

However, in medicine, epidemiology (a subset of medicine) and biology, one can never absolutely prove “causation.” Therefore, science uses the “weight-of-the-evidence” and requires that multiple tests of association reach statistical significance at the 95% or 99% confidence interval—this is considered proof of associations so strong as to **imply** causation.

Because the subject matter of epidemiology is populations (not individuals), disease frequency (the rate of disease within a population), diseases patterns in time and place, credible scientific evidence is established when it reaches a 95% “Confidence Interval” (not 100% certainty). Epidemiologists describe the relationship between chemical exposure, co-morbidities and disease in terms of associations or probabilities, not in terms of cause and effect. To be more specific, the presence or absence of a property in a given population in terms of its exposure to a contaminant are expressed in epidemiology as an “odds ratio” (OR), “relative risk” or “risk ratio” (RR), or “hazard ratio” (HR).

When adjudicated in court, to prove harm from chemical exposure plaintiffs do not have to establish that the evidence of a relationship between a chemical exposure and a disease reaches 95% confidence (as it does in epidemiology) but rather that it is “more likely than not” that exposure caused the disease.

In these matters, an understanding of the principles of cognitive science are critical, specifically, the difference between “direct causation” and “systemic causation.”

George Lakoff, Richard and Rhoda Goldman Distinguished Professor of Cognitive Science and Linguistics at the University of California at Berkeley, describes the difference concisely:

Systemic causation is familiar. Smoking is a systemic cause of lung cancer. HIV is a systemic cause of AIDS. Working in coal mines is a systemic cause of black lung disease. Driving while drunk is a systemic cause of auto accidents. Sex without contraception is a systemic cause of unwanted pregnancies.

There is a difference between systemic and direct causation. Punching someone in the nose is direct causation. Throwing a rock through a window is direct causation. Picking up a glass of water and taking a drink is direct causation. Slicing bread is direct causation. Stealing your wallet is direct causation. Any application of force to something or someone that always produces an

immediate change to that thing or person is direct causation. When causation is direct, the word cause is unproblematic.

Systemic causation, because it is less obvious, is more important to understand. A systemic cause may be one of a number of multiple causes. It may require some special conditions. It may be indirect, working through a network of more direct causes. It may be probabilistic, occurring with a significantly high probability. It may require a feedback mechanism. In general, causation in ecosystems, biological systems, economic systems, and social systems tends not to be direct, but is no less causal. And because it is not direct causation, it requires all the greater attention if it is to be understood and its negative effects controlled.

Above all, it requires a name: systemic causation. (Lakoff 2017)

The only quibble we have is that Lakoff seems to suggest most people regard smoking as a systemic cause of lung cancer. We believe that if you asked most people, they would say “smoking causes lung cancer,” the direct implication being it is a “direct cause.” The term “systemic causation” is not in the vocabulary of the average person, and in our experience, it is rare to meet an epidemiologist who possess any familiarity with the concept per se (though their work generally assumes and sometimes expresses the idea). Equally important, most people don’t understand that epidemiology is not the study of individuals but of populations.

In this study we document the presence of 70 chemicals as airborne contaminants released by stationary combustion at natural gas compressor stations as reported by NEI, two additional stack released reported by GHGI (carbon dioxide and methane) not reported by NEI, and three chemicals from fugitive sources reported by GHGI (carbon dioxide, methane and nitrous oxide). In all, there is documented data for 73 chemicals.

In understanding how and under what circumstances these chemicals individually or collectively may adversely affect human health, the terms reviewed above should all be considered.

We have two concerns: (a) the direct and systemic effects of chemicals on human health and (b) the systemic health effects caused by greenhouse gases.

Acute chemical exposures may produce immediate and obvious health effects. Exposures to high levels of carbon monoxide is toxic to all hemoglobin animals, including human. In ordinary parlance we would say that when carbon monoxide poisoning occurs the acute chemical exposure was the direct (and immediate) cause of death.

High levels of air pollution result in asthmatic attacks, but they are not the cause of the patient’s underlying asthma. Because not everyone suffering from asthma has an asthmatic attack on days with particularly bad air pollution, the outcome is probabilistic, which is why an epidemiologist familiar with cognitive science would describe this as systemic causation.

Table 3a provides selected health effects for 6 compressor station pollutants indicating (a) the concern (chemical exposure or climate change) and (b) causation (direct or systemic).

Table 3b provide a list of all 70 stack pollutants and the major categories of disease they are positively associated with.

Table 3a.

## Natural Gas Pollutant: Cause for Concern and Causation for Selected Chemicals and Selected Diseases

## NYS Natural Gas Compressor Stations

Stack Rank	Chemical	Concern	Ch.	Title	Code	Disease description	Causation	Reference
1	Nitrogen oxides  Stack releases: 18,082,570 lbs.	Climate change	1.	Certain infectious and parasitic diseases		E.g., tick borne diseases	Systemic cause	Systemic cause of disease resulting from a warmer climate and spread of infectious and parasitic diseases
		Chemical exposure	2.	Neoplasms	C30-C39	Malignant neoplasms, respiratory system and intrathoracic organs	Systemic cause	Chen et al. 2014, Hamra et al. 2015, Han et al. 2016
		Chemical exposure	2.	Neoplasms	C54	breast (carcinomas)	Systemic cause	Chen et al. 2012, Jørgensen et al. 2016
		Chemical exposure	4.	Endocrine diseases	E11	diabetes mellitus	Systemic cause	Coogan et al. 2012, Eze et al. 2014
		Chemical exposure	10.	Diseases of the respiratory system	J45	asthma	Systemic cause	Di Giampaolo et al. 2011, van der Vliet 2011
2	Carbon monoxide  Stack releases: 12,359,731 lbs.	Chemical exposure	3.	Diseases of the blood, blood-forming organs, immune mechanism		Autoimmune disease	Systemic cause	Science Daily 6 September 2004, Nicholls 2001
		Chemical exposure	6.	Diseases of the nervous system	G30-G32	Other degenerative diseases of the nervous system	Systemic cause	Nicholls 2001
		Chemical exposure	9.	Diseases of the circulatory system	I20-I25	Ischemic heart diseases	Systemic cause	Alfted et al. 1989, Alfted et al. 1989, Nuvolone et al. 2011.
		Chemical exposure	10.	Respiratory system	J40-J47	Chronic lower respiratory diseases	Systemic cause	Sbihi et al. 2016, Tian et al. 2014
		Chemical exposure	20.	Poisoning and certain other consequences of external causes	T58	Toxic effect of carbon monoxide	Direct cause	NIOSH REL: TWA 35 ppm (40 mg/m <sup>3</sup> ) C 200 ppm (229 mg/m <sup>3</sup> ). Acute levels will without exception will sicken all exposed populations and at certain levels kill all people, so CO poisoning can be described as a direct cause poisoning and death. Approximately 40,000 people are treated for CO poisoning annually in the U.S. Signs and symptoms of high inhalation exposure include: headache, tachypnea, nausea, lassitude (weakness, exhaustion), dizziness, confusion, hallucinations; cyanosis; depressed S-T segment of electrocardiogram, angina, syncope (NIOSH Pocket Guide)
3	Volatile organic chemicals  Stack releases: 4,920,396 lbs.	Chemical exposure	4.	Endocrine, nutritional and metabolic diseases		endocrine system effects	Systemic cause	TEDX
		Chemical exposure	5.	Mental and behavioral disorders		coordination (loss) reduced cognitive capacity	Systemic cause	U.S. NIH ToxTown U.S. EPA, U.S. NIH
		Chemical exposure	6.	Nervous system diseases		CNS damage	Systemic cause	U.S. EPA, U.S. NIH
		Chemical exposure	9.	Circulatory system		cardiovascular disease	Systemic cause	Lin et al. 2013, Ye et al. 2017
4	Formaldehyde  Stack releases: 1,309,336 lbs.	Chemical exposure	2.	Neoplasms		Malignant neoplasms	Systemic cause	Known human carcinogen (IARC, State of California)
		Chemical exposure	2.	Neoplasms	C00	Malignant neoplasms, lip	Systemic cause	Meshkov 2014
		Chemical exposure	2.	Neoplasms	C06	Malignant neoplasms, mouth	Systemic cause	Meshkov 2014
		Chemical exposure	2.	Neoplasms	C06	Malignant neoplasms, oral cavity	Systemic cause	Merletti et al., 1991
		Chemical exposure	2.	Neoplasms	C11	Malignant neoplasms, nasopharyngeal	Systemic cause	Coggon et al. 2014, Hauptmann et al. 2004, IARC, Marsh et al. 2002, Puñal-Riobóo et al. 2010, Roush et al. 1987, U.S. NTP ROC 13th
		Chemical exposure	2.	Neoplasms	C15	Malignant neoplasms, esophagus	Systemic cause	Coggon et al. 2014
		Chemical exposure	2.	Neoplasms	C16	Malignant neoplasms, stomach	Systemic cause	Coggon et al. 2014
		Chemical exposure	2.	Neoplasms	C22	Malignant neoplasms, liver	Systemic cause	Coggon et al. 2014
		Chemical exposure	20	Symptoms and signs		Varied	Direct cause	NIOSH REL: Ca TWA 0.016 ppm C 0.1 ppm [15-minute]

Stack Rank	Chemical	Concern	Ch.	Title	Code	Disease description	Causation	Reference
								High exposure levels will typically result in: irritation eyes, nose, throat, respiratory system; lacrimation (discharge of tears); cough; wheezing (NIOSH Pocket Guide)
NA	Carbon dioxide	Climate change	1.	Certain infectious and parasitic diseases		E.g., tick borne diseases	Systemic cause	Systemic cause of disease resulting from a warmer climate and spread of infectious and parasitic diseases
	Stack releases: Amount TBD	Chemical exposure	10.	Diseases of the respiratory system			Systemic cause	Wong et al. 2011
	Fugitive releases: Amount TBD	Chemical exposure	20	Symptoms and signs		Varied	Direct cause	NIOSH REL: TWA 5000 ppm (9000 mg/m <sup>3</sup> ) ST 30,000 ppm (54,000 mg/m <sup>3</sup> ). At high exposure levels inhalation symptoms include: headache, dizziness, restlessness, paresthesia; dyspnea (breathing difficulty); sweating, malaise (vague feeling of discomfort); increased heart rate, cardiac output, blood pressure; coma; asphyxia; convulsions. (NIOSH Pocket Guide)
NA	Methane	Climate change	1.	Certain infectious and parasitic diseases		E.g., tick borne diseases	Systemic cause	Systemic cause of disease resulting from a warmer climate and spread of infectious and parasitic diseases
	Fugitive releases: Amount TBD	Chemical exposure	10.	Respiratory system	J80	acute respiratory distress syndrome (ARDS)	Direct cause	acute respiratory distress syndrome (ARDS)
NA	Nitrous oxides	Climate change	1.	Certain infectious and parasitic diseases		E.g., tick borne diseases	Systemic cause	Systemic cause of disease resulting from a warmer climate and spread of infectious and parasitic diseases
	Fugitive releases: amount TBD	Chemical exposure	15	Pregnancy, childbirth and the puerperium		Miscarriage or fetal death	Systemic cause	ILO 1996
		Chemical exposure	17.	Congenital malformations and deformations		Birth defects, mutations, fetal damage	Systemic cause	ILO 1996
		Chemical exposure	20.	Poisoning and certain other consequences of external causes	T58	Toxic effect of carbon nitrogen oxides	Direct cause	NIOSH REL: TWA 25 ppm (46 mg/m <sup>3</sup> ) (TWA over the time exposed) [*Note: REL for exposure to waste anesthetic gas.] At high exposure levels inhalation symptoms include: dyspnea (breathing difficulty); drowsiness, headache; asphyxia (NIOSH Pocket Guide)

Table 3b.

## Chemicals and Health Effects Ranked by Total Pounds

NYS Natural Gas Compressor Stations, Reported NEI Emissions: 2008 to 2014

				Neoplasms	Blood & immune system	Endocrine & related	Mental & Behavioral	Nervous system	Eye and adnexa	Ear and mastoid process	Circulatory	Respiratory	Digestive	Skin and subcutaneous	Musculoskeletal	Genitourinary	Genitourinary: Urinary	Genitourinary: Pelvis, genitals and breasts	Pregnancy, childbirth and the puerperium	Perinatal period	Congenital malformations & chrom. abnormalities	Symptoms, signs, abnormal clinical & lab. findings	Injury, poisoning	external causes
#	Chemical	Pounds	%	2	3	4	5	6	7	8	9	10	11	12	13	14	14a	14b	15	16	17	18	19	
				59	41	52	35	42	44	16	42	51	49	52	6	46	37	36	12	26	57	48	12	
1	Nitrogen oxides	18,082,571	45.22			✓	✓		✓		✓	✓		✓		✓		✓		✓	✓	✓		
2	Carbon monoxide	12,359,731	30.91		✓		✓	✓		✓	✓	✓	✓			✓		✓			✓	✓		
3	Volatile organic compounds	4,920,396	12.31	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓			✓	✓		
4	Formaldehyde	1,309,336	3.27	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	
5	PM10 Primary (Filt + Cond)	1,259,744	3.15	✓			✓				✓	✓		✓		✓		✓		✓	✓	✓		
6	PM 2.5 Primary (Filt + Cond)	1,106,198	2.77	✓			✓				✓	✓		✓		✓		✓		✓	✓	✓		
7	PM Condensable	540,267	1.35	✓		✓	✓							✓		✓		✓		✓	✓			
8	Sulfur dioxide	186,778	0.47	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓					✓	✓	✓	✓	
9	Acetaldehyde	65,969	0.16	✓	✓	✓	✓	✓	✓		✓	✓		✓		✓	✓	✓	✓	✓	✓	✓	✓	
10	Acrolein	52,723	0.13	✓				✓	✓		✓	✓	✓	✓					✓		✓	✓	✓	
11	Benzene	21,241	0.05	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		
12	Methanol	19,333	0.05	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓	✓		✓	✓	✓		
13	Toluene	19,308	0.05	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓		
14	Hexane	12,184	0.03	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓		
15	Xylene (mixed isomers)	8,394	0.02	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		
16	1,3-Butadiene	4,719	0.01	✓	✓	✓		✓	✓		✓	✓	✓	✓		✓	✓	✓			✓	✓		
17	2,2,4-Trimethylpentane	4,445	0.01					✓							✓		✓					✓		
18	Ethyl benzene	2,794	0.01	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓		✓	✓	✓		
19	Ammonia	1,573	0.00	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓					✓	✓	
20	Phenol	706	0.00	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓		✓	✓	✓	✓	
21	Naphthalene	696	0.00	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓		✓		✓	✓		
22	Nickel	692	0.00	✓	✓		✓	✓	✓		✓	✓	✓	✓		✓	✓	✓		✓	✓	✓		
23	Biphenyl	690	0.00	✓		✓		✓	✓		✓	✓	✓	✓		✓	✓				✓	✓		
24	Methane dichloride	629	0.00	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	
25	Propylene oxide	615	0.00	✓	✓		✓	✓	✓		✓	✓	✓	✓		✓	✓	✓			✓	✓	✓	
26	Manganese	350	0.00	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓		✓	✓	✓		
27	Ethylene dibromide	347	0.00	✓	✓	✓	✓	✓	✓			✓	✓	✓		✓	✓	✓			✓	✓		
28	1,1,2,2-Tetrachloroethane	309	0.00	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓				✓	✓		
29	Carbon tetrachloride	282	0.00	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓		
30	1,1,2-Trichloroethane	247	0.00	✓	✓		✓	✓			✓		✓	✓		✓	✓				✓			
31	Styrene	234	0.00	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓		
32	Chloroform	193	0.00	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓		✓	✓	✓		✓	✓	✓		

				Neoplasms	Blood & immune system	Endocrine & related	Mental & Behavioral	Nervous system	Eye and adnexa	Ear and mastoid process	Circulatory	Respiratory	Digestive	Skin and subcutaneous	Musculoskeletal	Genitourinary`	Genitourinary: Urinary	Genitourinary: Pelvis, genitals and breasts	Pregnancy, childbirth and the puerperium	Perinatal period	Congenital malformations & chrom. abnormalities	Symptoms, signs, abnormal clinical & lab. findings	Injury, poisoning . . . external causes
#	Chemical	Pounds	%	2	3	4	5	6	7	8	9	10	11	12	13	14	14a	14b	15	16	17	18	19
33	2-Methylnaphthalene	191	0.00	√	√		√		√			√										√	
34	Chlorobenzene	172	0.00	√	√	√	√	√	√		√	√	√	√	√	√	√				√	√	
35	Propylene dichloride	164	0.00	√	√			√				√	√			√	√				√	√	
36	1,3-Dichloropropene	161	0.00	√	√			√	√			√	√	√		√	√	√		√	√	√	
37	Ethylene dichloride	151	0.00	√	√	√	√	√	√		√	√	√	√		√	√	√			√	√	
38	Ethylidene dichloride	144	0.00	√				√	√		√	√	√			√	√	√					
39	Vinyl chloride	107	0.00	√	√		√	√	√	√	√	√	√	√		√		√			√	√	
40	Mercury	70	0.00	√	√	√	√	√	√	√	√	√	√	√		√	√	√	√		√	√	
41	Chromium III	56	0.00			√			√			√	√	√								√	
42	Phenanthrene	48	0.00	√		√			√			√	√	√						√	√	√	√
43	PAHs Total	35	0.00	√																			
44	Cadmium	30	0.00	√	√	√	√	√	√	√	√	√	√	√		√	√	√		√	√	√	√
45	Fluorene	28	0.00			√							√								√		
46	Benz[a]anthracene	19	0.00	√		√															√		
47	Benzo(j,k)fluorene	11	0.00			√							√								√		
48	Anthracene	10	0.00	√		√			√			√	√	√							√	√	√
49	Perchloroethylene	9	0.00	√	√	√	√	√	√		√	√	√	√		√	√	√	√	√	√	√	
50	Acenaphthene	8	0.00										√							√			
51	Pyrene	7	0.00			√		√						√							√		
52	Ethyl chloride	6	0.00	√	√		√	√	√		√	√	√	√	√	√	√	√			√	√	
53	Acenaphthylene	5	0.00			√						√											
54	Chrysene	4	0.00	√		√															√		
55	Chromium (VI)	2	0.00	√		√			√			√	√	√								√	
56	Benzo[g,h,i]perylene	2	0.00	√	√								√								√		
57	Benzo[b]fluoranthene	1	0.00	√		√															√		
58	Lead	1	0.00	√	√	√	√	√	√	√	√	√	√	√		√	√	√		√	√	√	√
59	Benzo[e]pyrene	0.09	0.00			√															√		
60	Arsenic	0.06	0.00	√	√	√		√			√	√	√	√		√	√			√	√	√	
61	Cobalt	0.03	0.00	√	√	√		√	√	√	√	√	√	√		√	√	√			√	√	
62	Indeno[1,2,3-cd]pyrene	0.02	0.00	√		√															√		
63	Benzo[a]pyrene	0.01	0.00	√	√	√						√	√	√		√					√		
64	Selenium	0.01	0.00	√	√	√	√	√	√		√	√	√	√	√	√	√	√			√	√	
65	Perylene	0.0039	0.00			√																	
66	Beryllium	0.0034	0.00	√			√		√		√	√	√								√	√	
67	7,12-Dimethylbenz[a]anthracene	0.0033	0.00	√	√	√								√		√		√					
68	Benzo[k]fluoranthene	0.0029	0.00	√		√															√		
69	3-Methylcholanthrene	0.0003	0.00	√		√					√			√									
70	Dibenz[a,h]anthracene	0.0001	0.00	√		√								√							√		

### 3.1 Certain infectious and parasitic diseases (A00-B99)

As indicated above, for certain infectious and parasitic diseases (Chapter 1 of ICD-10), adverse health effects are not the result of chemical exposures per se, but the result of a warmer climate created by greenhouse gases which lead to their spread and in many cases increased virulence.

The spread of a wide range of both human and animal infectious disease as a result of climate change is unavoidable, and some effects are already clear (Bouzid et al. 2014, Caminade et al. 2014, Confalonieri et al. 2015, Gislason 2014, Heffernan 2013, Medlock and Leach 2014, Parham et al. 2014, Ogden et al. 2014, Rodríguez-Morales 2013, Shuman 2011).

A wide variety of non-infectious and non-parasitic diseases will also increase in incidence as a result of climate change including: allergic disease (Barne et al. 2013, Behrendt and Ring 2012, Bielory et al. 2012), cardiopulmonary disorders (De Blois et al. 2015, Rice et al. 2014), respiratory disease (Barne et al. 2013, Gerardi and Kellerman 2014, Lin et al. 2012, Takaro et al. 2013), and skin diseases (Andersen 2011, Andersen et al. 2012, Balato et al. 2013).

Everyone will be affected by the impact of climate change on health--more vulnerable populations include the elderly (Gamble et al. 2013), children (Bernstein et al. 2011), and manual workers (Applebaum et al. 2016).

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## 3.2. Neoplasms (C00-D48)

### 3.2.1. Carcinogens by Evidence of Carcinogenicity

Fifty-nine of the 70 chemicals released by NYS natural gas compressor stations are associated with neoplastic diseases.

All 18 stations had carcinogenic releases. These totaled an estimated 9.5 million pounds from 2008 to 2014--an annual average of 1.4 million pounds.

Chemicals associated with cancer represented 23.7% of all compressor station releases.

Of the 59 chemicals linked to cancer, 22 chemicals are categorized as “known” human carcinogens by one or more authoritative sources:

1. International Agency for Research on Cancer (IARC) -- the specialized cancer agency of the World Health Organization (WHO),
2. U.S. National Toxicology Program (NTP) -- National Institute of Environmental Health Sciences, National Institutes of Health,
3. U.S. Environmental Protection Agency (EPA),
4. U.S. National Institute for Occupational Safety and Health (NIOSH),
5. U.S. Occupational Safety and Health Administration (OSHA), and
6. State of California, Office of Environmental Health Hazard Assessment (CA/OEHHA) -- part of the California Environmental Protection Agency (Cal/EPA).

Known human carcinogens account for 83% of total carcinogens.

Table 3.2.1a.

## Carcinogens by Evidence of Carcinogenicity

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Evidence of Carcinogenicity		Location				7 Years (estimate)		
		Ch	Fac	Cn	DEC	Average Annual Pounds	Total Pounds	%
1	Authority: known human carcinogen	23	18	14	6	1,129,164	7,904,153	82.87
2	Authority: probable human carcinogen	2	18	14	6	105	738	.01
3	Authority: possible human carcinogen	18	18	14	6	13,020	91,140	.96
4	Peer-reviewed: positive human and animal evidence of carcinogenicity	17	17	13	6	39,004	273,032	2.86
5	Peer-reviewed: positive human evidence of carcinogenicity	14	14	12	6	181,162	1,268,140	13.30
6	Peer-reviewed: positive animal evidence of carcinogenicity	3	11	9	6	152	1,069	.01
<b>Total</b>		<b>59</b>	<b>18</b>	<b>14</b>	<b>6</b>	<b>1,362,607</b>	<b>9,538,272</b>	<b>100%</b>

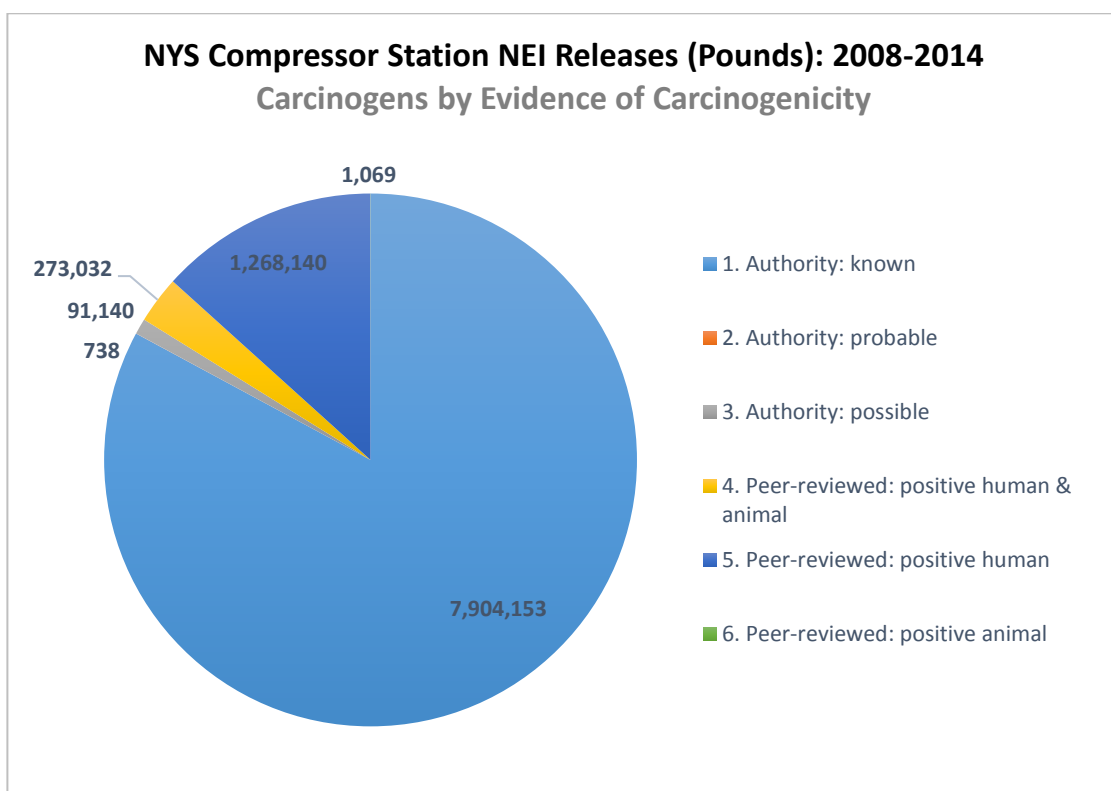


Table 3.2.1b.

## Neoplastic Diseases by Chemical (Top 20 Carcinogens)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

			Authors' Classification																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																			
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[illegible]

### 3.2a. Releases by Chemical

Volatile organic compounds (VOCs) as a group were responsible for 51.6% of all statewide carcinogenic releases, slightly more than 4.9 million pounds. In addition to VOCs as a group, NEI also identifies specific VOCs. Individual VOCs have different levels of evidence of human carcinogenicity. Formaldehyde, which ranks 2<sup>nd</sup>, is classified by IARC as a known human carcinogen. Acetaldehyde, which ranks 7<sup>th</sup>, is classified as a possible human carcinogen by IARC. Methanol, which ranks 10<sup>th</sup>, is classified by EU as having limited evidence of human carcinogenicity (R40). There is both animal and human evidence for the carcinogenicity of acrolein, but it has not been classified by IARC because of the limited amount of evidence available. Benzene, which ranks 9<sup>th</sup>, is universally considered a known human carcinogen (IARC, NTP, EPA, OSHA, NIOSH, CA/OEHHA). VOCs as a group undoubtedly contain a mixture of individuals VOCs, some of which are classified as known, probable or possible human carcinogens, as well as some that have not been classified by an authoritative agency for various reasons including the lack of available evidence upon which to make an assessment. While not every VOC is a carcinogen, many are. The emission reporting category VOCs is, in our opinion, reflective of known human carcinogenic activity and we have included it as a known human carcinogen in this report. Formaldehyde ranked second with 1,309,335 pounds (13.7%), followed by PM10 with 1,259,744 pounds (13.2%). These three chemicals were responsible for 78.5% of all carcinogens released by the state's natural gas compressor stations. The top 10 chemicals accounted for 99.4% of all carcinogenic releases.

Table 3.2a.

#### Neoplastic Diseases by Chemical (Top 20 Carcinogens)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Volatile organic compounds	18	14	6	2,108,741	702,913	4,920,395	51.59
2	Formaldehyde	18	14	6	561,143	187,047	1,309,335	13.73
3	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	13.21
4	PM2.5 Primary (Filt + Cond)	18	14	6	474,084	158,028	1,106,197	11.60
5	PM Condensable	18	14	6	231,543	77,181	540,267	5.66
6	Sulfur Dioxide	18	14	6	80,047	26,682	186,778	1.96
7	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.69
8	Acrolein	14	13	6	22,595	7,531	52,723	0.55
9	Benzene	16	13	6	9,103	3,034	21,240	0.22
10	Methanol	8	7	6	8,285	2,761	19,333	0.20
11	Toluene	16	13	6	8,274	2,758	19,307	0.20
12	Hexane	13	10	6	5,221	1,740	12,183	0.13
13	Xylenes (Mixed Isomers)	15	13	6	3,597	1,199	8,394	0.09
14	1,3-Butadiene	14	13	6	2,022	674	4,718	0.05
15	Ethyl Benzene	15	13	6	1,197	399	2,794	0.03
16	Ammonia	8	7	5	674	224	1,573	0.02
17	Phenol	11	10	6	302	100	706	0.01
18	Naphthalene	15	13	6	298	99	696	0.01
19	Nickel	11	11	6	296	98	691	0.01
20	Biphenyl	6	6	5	295	98	690	0.01
		18	14	6	4,085,879	1,361,953	9,533,733	99.97

### 3.2b. Releases by ICD Category

Neoplasms are subdivided into 3 major groups: malignant neoplasms (C00-C97), in situ neoplasms (D00-D09), benign neoplasms (D10-D36), and neoplasms of uncertain or unknown behavior (D37-D48). Chemicals released by natural gas compressor stations in NYS are positively associated with all four. It should be remembered, that a single chemical can be associated with more than one category of disease.

#### Malignant neoplasms (C00-C97)

Malignant neoplasms are sub-divided into 14 groups--the primary consideration for categorization being the effected organ or organ system. Fifty-six chemicals released by NYS compressor stations (2008-2014) are associated with malignant neoplasms.

All 18 NYS compressor stations had reported releases of chemicals associated with malignant neoplasms.

Four of the top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 1.7 million pounds or 18% of the total, followed by the company compressor in Carlisle (1.4 million pounds or 14.2%) and by its LaFayette facility (1.14 million pounds or 11.9%). These three sites were responsible for 4.2 million pounds or 44% of all statewide releases. The top 5 sites were responsible for 5.9 million pounds or slightly less than two-thirds (62.1%) of the state total. The facility average was 532,453 pounds. (Table 2c)

#### 1. Lip, oral cavity and pharynx (C10-C14)

Fourteen chemicals released by NYS natural gas compressor stations are associated with malignant neoplasms of the lip, oral cavity and pharynx. Specific organs affected include: lips (C00), tongue (C02), buccal, mouth, and oral cavity (C06), salivary gland (C08), nasopharynx (C11), hypopharyngeal (C13), and oral cavity and pharynx (C14).

Four of the top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 226,703 pounds or 15.7% of the total, followed by the company's compressor in LaFayette (186,512 pounds or 13%) and its Carlisle facility (159,281 pounds or 11.1%). These three sites were responsible for 572,496 pounds or 40% of all statewide releases. The top 5 sites were responsible for 852,720 pounds or more than one-half (59.2%) of the state total. The facility average was 80,029 pounds. (Table 2c.1)

#### 2. Digestive organs (C15-C26)

Thirty-eight chemicals released by NYS natural gas compressor stations are associated with digestive system malignancies. Specific organs affected include: esophagus (C15), stomach (C16), duodenum, small intestine (C17), colon (C18), rectum (C20), anus (C21), liver (C22), biliary tract (C24), pancreas (C25), and spleen (C26).

Four of the top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 187,951 pounds or 15.6% of the total, followed by the company's station in LaFayette (187,951 pounds or 12.9%) and its Carlisle facility (160,478 pounds or 11%). These three sites were responsible for 576,136 pounds or 39.4% of all statewide releases. The top 5 sites were responsible for 861,765 pounds or more than one-half (58.9%) of the state total. The facility average was 81,250 pounds. (Table 2c.2)

### 3. Respiratory system and intrathoracic organs (C30-C39)

Thirty-eight chemicals released by NYS natural gas compressor stations are associated with respiratory system and intrathoracic organ malignancies. Specific organs affected include: nasal cavity, nasal mucosa and paranasal sinus (C30), larynx, throat and trachea (C32), bronchus and lung (C34), cardiac and heart (C38), and respiratory tract (C39).

Four of the top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 1.7 million pounds or 18.1% of the total, followed by the company's station in Carlisle (1.3 million pounds or 14.2%) and its LaFayette facility (1.1 million pounds or 11.9%). These three sites were responsible for 4.1 million pounds or 44.2% of all statewide releases. The top 5 sites were responsible for 5.8 million pounds or slightly less than two-thirds (62.1%) of the state total. The facility average was 520,308 pounds. (Table 2c.3)

### 4. Bone and articular cartilage (C40-C41)

Thirty-five chemicals released by NYS natural gas compressor stations are associated with bone and articular cartilage malignancies, specifically, bone carcinoma and osteosarcoma subcutaneous tissue fibrosarcoma (C41).

Four of the top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 1.6 million pounds or 18.4% of the total, followed by the company's station in Carlisle (1.2 million pounds or 14.1%) and its LaFayette facility (1 million pounds or 12%). These three sites were responsible for 3.9 million pounds or 44.5% of all statewide releases. The top 5 sites were responsible for 5.4 million pounds or slightly less than two-thirds (62.1%) of the state total. The facility average was 487,068 pounds. (Table 2c.4)

### 5. Skin (C43-C44)

Fourteen chemicals released by NYS natural gas compressor stations are associated with skin carcinomas (C44).

TGPC's Compressor Station 229 in Eden ranked first with 13,750 pounds or slightly more than half (51.5%) of the total, followed by AG Stony Point compressor (4,553 pounds or 17.1%) and DTI's Woodhull Station (2,883 pounds or 10.8%). These three sites were responsible for 21,187 pounds or more than three-fourths (79.4%) of all statewide releases. The top 5 sites were responsible for 24,199 pounds or 90.1% the state total. The facility average was 1,483 pounds. (Table 2c.5)

### 6. Connective and soft tissue (C45-C49)

Seventeen chemicals released by NYS natural gas compressor stations are associated with connective and soft tissue malignancies, specifically, peritoneal cavity carcinoma and peritoneum mesothelioma (C48), and blood vessel angiosarcoma, carcinoma and hemangiosarcoma, connective tissue carcinoma and sarcoma, heart hemangiosarcoma, liver hemangiosarcoma, and muscle carcinoma (C49).

AG Stony Point Compressor Station ranked first with 7.516 pounds or 27.5% the total, followed by TGPC's compressor in Eden (6,843 pounds or 25%) and AG's Southeast Station (4,304 pounds or 16%). These three sites were responsible for 18,663 pounds or slightly more than two-thirds (68.3%) of all statewide releases. The top 5 sites were responsible for 22,680 pounds or 83% the state total. The facility average was 1,519 pounds. (Table 2c.6)

## 7. Breast and female genital organs (C50-C58)

Twenty-five chemicals released by NYS natural gas compressor stations are associated with breast and female genital organ malignancies. Specific organs affected include: breast adenocarcinoma, carcinoma and carcinosarcoma (C50), cervical carcinoma (C53), carcinoma of the uterus (C55), and ovarian carcinoma and granulosa cell carcinoma (C56).

The top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 766,684 pounds or 17.7% of the total, followed by the company's station in Carlisle (648,570 pounds or 15%) and its Eden facility (529,510 pounds or 12.2%). These three sites were responsible for 1.9 million pounds or 45% of all statewide releases. The top 5 sites were responsible for 2.8 million pounds or slightly less than two-thirds (63.5) of the state total. The facility average was 240,897 pounds. (Table 2c.7)

## 8. Male genital organs (C60-C63)

Thirteen chemicals released by NYS natural gas compressor stations are associated with male genital organ malignancies, specifically, prostate carcinomas (C61) and testes carcinomas (C62).

Four of the top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 222,045 pounds or 15.9% of the total, followed by NFGSC's station in Concord (188,88 pounds or 13.5%) and TGPC's LaFayette facility (179,381 pounds or 12.6%). These three sites were responsible for 590,314 pounds or 42.2% of all statewide releases. The top 5 sites were responsible for 866,213 pounds or slightly less than two-thirds (61.9%) of the state total. The facility average was 77,773 pounds. (Table 2c.8)

## 9. Urinary organs (C64-C68)

Twenty-five chemicals released by NYS natural gas compressor stations are associated with urinary tract malignancies, specifically, kidney carcinoma, cortical adenocarcinoma, renal cell carcinoma (C64), urinary bladder carcinoma and transitional cell carcinoma (C67), and urogenital carcinomas (C68).

The top 4 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 222,047 pounds or 15.8% of the total, followed by the company's station in LaFayette (186,990 pounds or 13%) and its Carlisle facility (159,689 pounds or 11.1%). These three sites were responsible for 575,865 pounds or 39% of all statewide releases. The top 5 sites were responsible for 862,150 pounds or 59% of the state total. The facility average was 80,063 pounds. (Table 2c.9)

## 10. Malignant neoplasms: Eye, brain and central nervous system (C69-C72)

Twenty chemicals released by NYS natural gas compressor stations are associated with eye, brain and central nervous system malignancies, specifically, retinoblastoma and uveal melanoma (C69), brain malignant astrocytoma, carcinoma and glioma (C71), and central nervous system carcinomas (C72).

The top 4 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 222,607 pounds or 15.6% of the total, followed by the company's station in LaFayette (187,876 pounds or 12.9%) and its Carlisle facility (160,382 pounds or 11%). These three sites were



responsible for 575,865 pounds or 39.4 %of all statewide releases. The top 5 sites were responsible for 862,150 or 59% of the state total. The facility average was 81,202 pounds. (Table 2c.10)

#### 11. Malignant neoplasms: Endocrine glands and related structures (C73-C75)

Ten chemicals released by NYS natural gas compressor stations are associated with endocrine glands and related structure malignancies, specifically, thyroid gland C-cell carcinoma, carcinomas and follicular cell carcinoma (C73) and adrenal gland malignant pheochromocytoma and carcinoma (C74), and pituitary gland carcinoma (C75).

Five of the top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company. TGPC's Compressor Station 245 in Winfield ranked first with 222,166 pounds or 18.2% of the total, followed by NPGSC's Concord station (189,058 pounds or 14.7%) and TGPC's LaFayette facility (179,540 pounds or 12.6%). These three sites were responsible for 590,765 pounds, or 42% of all statewide releases. The top 5 sites were responsible for 877,093 or slightly less than two-thirds (62.2%) of the state total. The facility average was 78,393 pounds. (Table 2c.11)

#### 12. Malignant neoplasms: Secondary and ill-defined (C76-C80)

Six chemicals released by NYS natural gas compressor stations are associated with secondary and ill-defined malignancies, specifically, head carcinoma (C76).

Algonquin Gas Transmission Company's facility in Stony Point, Rockland County, ranked first with 10,793 pounds or slightly less than one-third (32.4%) of the total, followed by the company's facility located in the village of Brewster in the town of Southeast (6,858 pounds or 20.6%). TCPC's station in Eden, Eire County, ranked third (5,172 pounds or 15.5%). These three sites were responsible for 22,823 or slightly more than two-thirds (68.5%) of the total. The top 5 sites were responsible for 28,724 or 86.2% of the state total. The facility average was 1,851 pounds. (Table 2c.12)

#### 13. Malignant Neoplasms, Stated or Presumed to be Primary, of Lymphoid, Haematopoietic and Related Tissue (C81-C96)

Thirty-one chemicals released by NYS natural gas compressor stations are associated with malignant neoplasms stated or presumed to be primary, of lymphoid, haematopoietic and related tissue. These diseases include: Hodgkin's disease (C81), other and unspecified types of non-Hodgkin's lymphoma, including histiocytic sarcomas, lymph sarcomas, lymph system carcinomas and reticulum cell sarcomas (C85), multiple myeloma and malignant plasma cell neoplasms (C90), lymphoid leukemia (C91), myeloid leukemia (C92), leukemia of unspecified cell type, including childhood leukemia (C95), and other and unspecified malignant neoplasms of lymphoid, haematopoietic and related tissue, including blood carcinoma, unspecified leukemia, hematologic and hematopoietic carcinoma (C96).

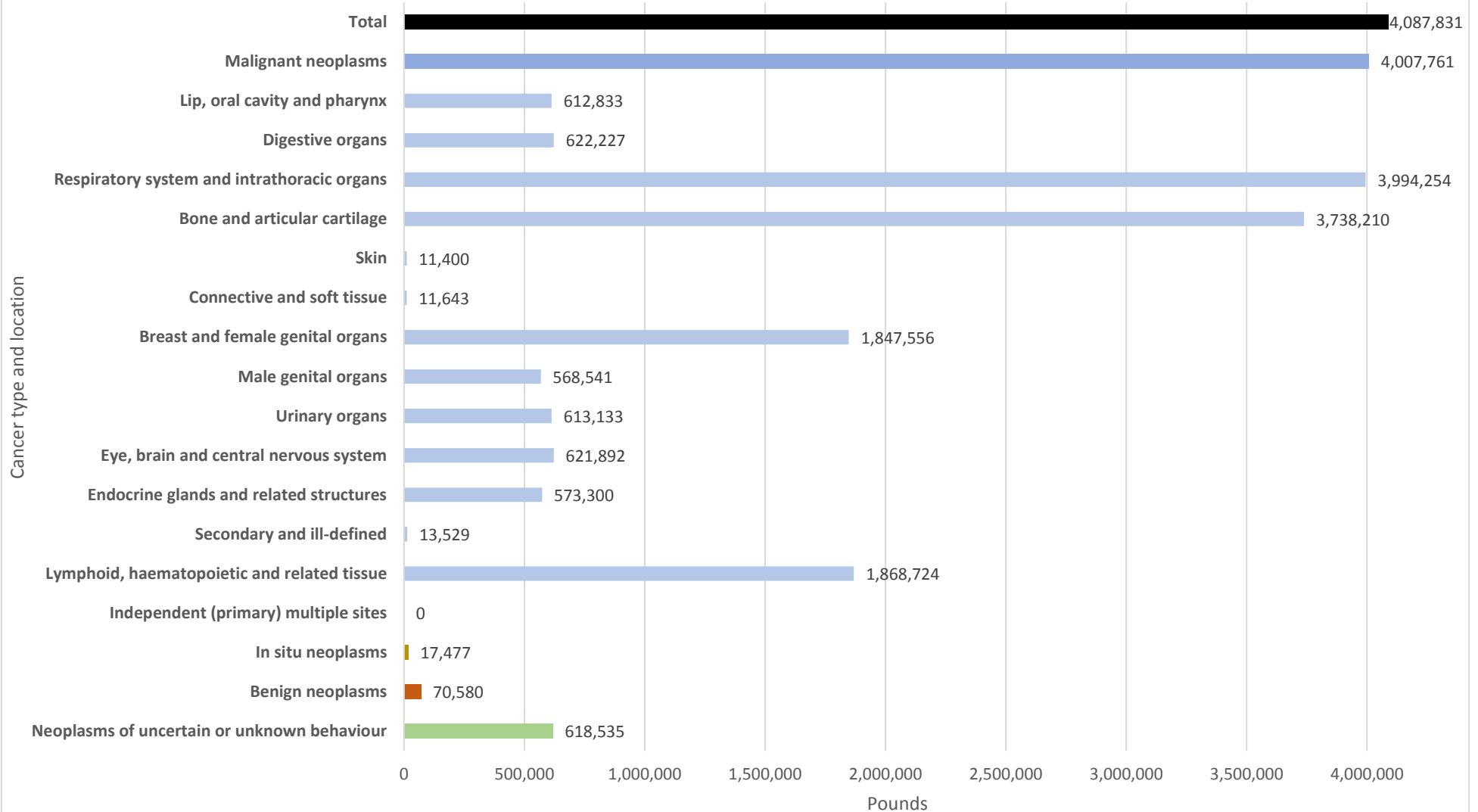
Table 3.b.

## Neoplastic Diseases by ICD Category

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Code	Description	'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	C00-C97	Malignant neoplasms	18	18	17	18	53	54	54	56	744,394	1,679,621	1,583,745	4,007,761
2	C00-C14	Lip, oral cavity and pharynx	18	18	16	18	12	14	14	14	118,992	254,897	238,943	612,833
3	C15-C26	Digestive organs	18	18	16	18	37	38	38	38	121,690	258,670	241,866	622,227
4	C30-C39	Respiratory system and intrathoracic organs	18	18	17	18	36	37	37	38	740,798	1,673,574	1,579,882	3,994,254
5	C40-C41	Bone and articular cartilage	18	18	17	18	33	34	34	35	694,106	1,551,399	1,492,704	3,738,210
6	C43-C44	Skin	16	15	13	16	12	12	12	14	2,362	5,008	4,029	11,400
7	C45-C49	Connective and soft tissue	17	17	15	17	17	17	17	17	1,929	5,074	4,639	11,643
8	C50-C58	Breast and female genital organs	18	18	16	18	23	25	25	25	361,015	823,303	663,237	1,847,556
9	C60-C63	Male genital organs	18	17	16	18	12	13	13	13	111,217	233,176	224,147	568,541
10	C64-C68	Urinary organs	18	18	16	18	24	24	24	25	119,062	255,474	238,596	613,133
11	C69-C72	Eye, brain and central nervous system	18	18	16	18	20	20	20	20	121,282	258,655	241,954	621,892
12	C73-C75	Endocrine glands and related structures	18	17	16	18	10	10	10	10	112,911	235,120	225,269	573,300
13	C76-C80	Secondary and ill-defined	17	16	14	17	6	6	6	6	2,054	5,690	5,771	13,516
14	C81-C96	Malignant neoplasms, stated or presumed to be primary, of lymphoid, haematopoietic and related tissue	18	18	16	18	31	31	31	31	364,338	833,140	671,245	1,868,724
15	C97	Malignant neoplasms of independent (primary) multiple sites	0	0	0	0	0	0	0	0	0	0	0	0
16	D00-D09	In situ neoplasms	16	15	13	16	3	3	3	3	3,313	7,557	6,606	17,477
17	D10-D36	Benign neoplasms	17	17	14	17	27	27	27	27	12,499	35,013	23,068	70,580
18	D37-D48	Neoplasms of uncertain or unknown behavior	18	18	16	18	39	40	40	41	121,277	257,142	240,115	618,535
	<b>C00-D48</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>56</b>	<b>57</b>	<b>57</b>	<b>59</b>	<b>751,985</b>	<b>1,693,810</b>	<b>1,642,034</b>	<b>4,087,831</b>

## C00-D58. Neoplastic Releases by ICD Category: 3 Year Total



### 3.2c. Releases by Facility: Malignant Neoplasms (ICD-10, C00-C97)

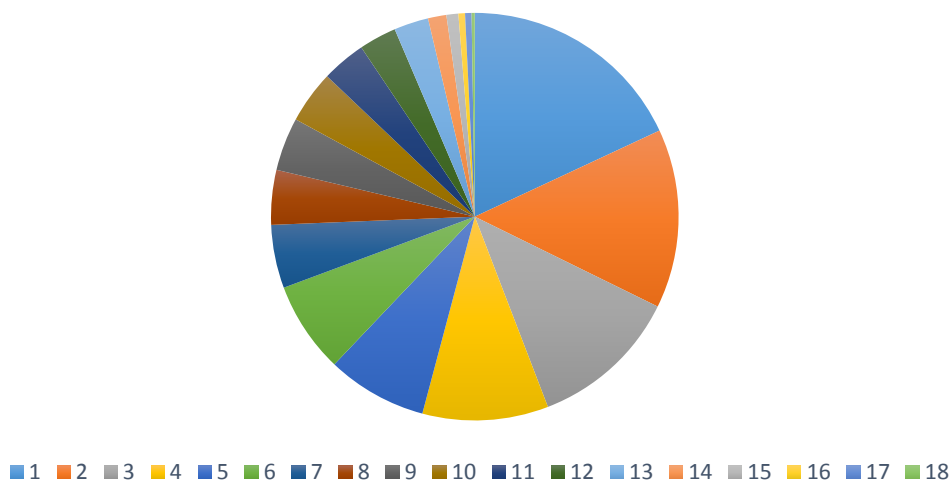
Table 3.2c.

#### Releases by Facility: Malignant Neoplasms (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	37	37	21	727,214	1,696,834	18.06
2	TGPC CS 249	Carlisle	Schoharie	37	23	22	572,367	1,335,523	14.21
3	TGPC CS 241	LaFayette	Onondaga	36	27	36	478,876	1,117,377	11.89
4	TGPC 229 & TEG DF	Eden	Erie	39	37	38	402,207	938,485	9.99
5	AGT Stony Point CS	Stony Point	Rockland	38	20	19	320,291	747,345	7.95
6	TGPC CS 237	Manchester, Phelps	Ontario	6	5	2	291,438	680,022	7.24
7	AGT SOUTHEAST CS	Southeast	Putnam	22	15	35	202,269	471,962	5.02
8	NFGSC Concord CS	Concord	Erie	8	7	9	174,647	407,511	4.34
9	TGPC CS 254	Chatham	Columbia	22	13	6	170,250	397,250	4.23
10	NFGSC Independence CS	Andover	Allegany	12	7	14	168,124	392,290	4.17
11	DTI Woodhull Station	Woodhull	Steuben	34	43	47	141,039	329,091	3.50
12	TGPC CS 224	Clymer	Chautauqua	37	35	37	119,806	279,548	2.97
13	NFGSC Beech Hill CS	Willing	Allegany	17	17	18	110,198	257,128	2.74
14	NFGSC Nashville CS	Hanover	Chautauqua	28	26	-	39,184	137,144	1.46
15	DTI Utica Station	Frankfort	Herkimer	28	33	46	37,981	88,622	0.94
16	TGPC CS 230-C	Lockport	Niagara	22	21	22	21,038	49,090	0.52
17	DTI Borger CS	Ithaca	Tompkins	34	35	16	20,642	48,166	0.51
18	TGPC CS 233	York	Livingston	22	13	1	10,183	23,762	0.25
				53	54	54	4,007,754	9,397,150	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
**Malignant Neoplasms**



### 3.2c.1. Releases by Facility: Malignant Neoplasms, Lip, Oral Cavity & Pharynx (ICD-10, C00-C14)

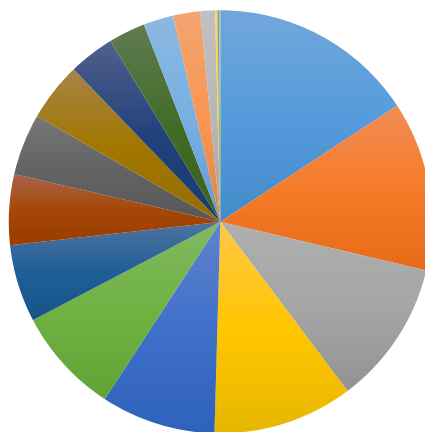
Table 3.3.2c.1.

#### Releases by Facility: Malignant Neoplasms, Lip, Oral Cavity and Pharynx (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	Facility Name (Short)	Location		Chemicals			7-Years (Pounds)		
		Town	County	'08	'11	'14	Average	Total	%
1	TGPC CS 245	Winfield	Herkimer	8	8	8	32,386	226,703	15.74
2	TGPC CS 241	LaFayette	Onondaga	8	8	8	26,645	186,512	12.95
3	TGPC CS 249	Carlisle	Schoharie	8	8	8	22,754	159,281	11.06
4	TGPC 229 & TEG DF	Eden	Erie	8	8	8	22,022	154,157	10.70
5	NFGSC Concord CS	Concord	Erie	2	1	3	18,010	126,067	8.75
6	TGPC CS 237	Manchester, Phelps	Ontario	1	1	1	16,708	116,956	8.12
7	AGT Stony Point CS	Stony Point	Rockland	10	7	8	12,190	85,332	5.92
8	TGPC CS 224	Clymer	Chautauqua	8	7	8	11,094	77,661	5.39
9	DTI Woodhull Station	Woodhull	Steuben	10	13	13	9,688	67,813	4.71
10	NFGSC Independence CS	Andover	Allegany	6	2	7	9,210	64,473	4.48
11	NFGSC Beech Hill CS	Willing	Allegany	7	7	7	7,182	50,271	3.49
12	AGT SOUTHEAST CS	Southeast	Putnam	7	6	10	5,789	40,520	2.81
13	TGPC CS 254	Chatham	Columbia	7	6	1	4,723	33,063	2.30
14	NFGSC Nashville CS	Hanover	Chautauqua	7	7	-	4,274	29,915	2.08
15	DTI Utica Station	Frankfort	Herkimer	7	11	13	2,295	16,063	1.12
16	TGPC CS 230-C	Lockport	Niagara	7	7	7	369	2,580	0.18
17	TGPC CS 233	York	Livingston	7	6	0	263	1,841	0.13
18	DTI Borger CS	Ithaca	Tompkins	10	12	6	189	1,322	0.09
				12	14	14	205,790	1,440,530	100%

#### NYS Compressor Station NEI Releases by Facility: 2008-2014 Malignant Neoplasms: Lip, Oral Cavity & Pharynx



1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

### 3.2c.2. Releases by Facility: Neoplasms, Digestive Organs

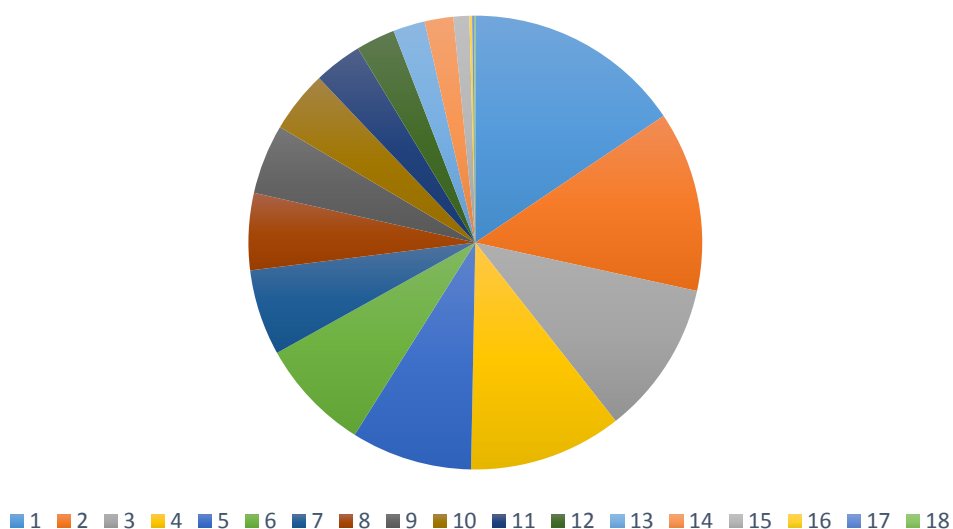
Table 3.2c.2.

#### Neoplastic Releases by Facility: Digestive Organs (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	Facility Name (Short)	Location		Chemicals			7-Years (Pounds)		
		Town	County	'08	'11	'14	Average	Total	%
1	TGPC CS 245	Winfield	Herkimer	29	29	14	32,530	227,707	15.57
2	TGPC CS 241	LaFayette	Onondaga	28	19	28	26,850	187,951	12.85
3	TGPC CS 249	Carlisle	Schoharie	29	16	15	22,925	160,478	10.97
4	TGPC 229 & TEG DF	Eden	Erie	28	27	28	22,795	159,562	10.91
5	NFGSC Concord CS	Concord	Erie	4	3	5	18,010	126,068	8.62
6	TGPC CS 237	Manchester, Phelps	Ontario	2	1	1	16,709	116,964	8.00
7	AGT Stony Point CS	Stony Point	Rockland	29	13	13	12,785	89,493	6.12
8	TGPC CS 224	Clymer	Chautauqua	29	27	29	11,454	80,175	5.48
9	DTI Woodhull Station	Woodhull	Steuben	21	28	31	10,400	72,802	4.98
10	NFGSC Independence CS	Andover	Allegany	8	3	10	9,211	64,478	4.41
11	NFGSC Beech Hill CS	Willing	Allegany	12	12	13	7,184	50,288	3.44
12	AGT SOUTHEAST CS	Southeast	Putnam	14	9	21	5,830	40,810	2.79
13	TGPC CS 254	Chatham	Columbia	14	7	1	4,732	33,124	2.26
14	NFGSC Nashville CS	Hanover	Chautauqua	16	16	-	4,285	29,993	2.05
15	DTI Utica Station	Frankfort	Herkimer	16	19	30	2,356	16,492	1.13
16	TGPC CS 230-C	Lockport	Niagara	14	13	14	399	2,792	0.19
17	TGPC CS 233	York	Livingston	14	7	0	278	1,945	0.13
18	DTI Borger CS	Ithaca	Tompkins	21	21	11	198	1,389	0.10
				37	37	38	208,930	1,462,509	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
Malignant Neoplasms: Digestive Organs



### 3.2c.3. Releases by Facility: Malignant Neoplasms, Respiratory System and Intrathoracic Organs (ICD-10, C30-C9)

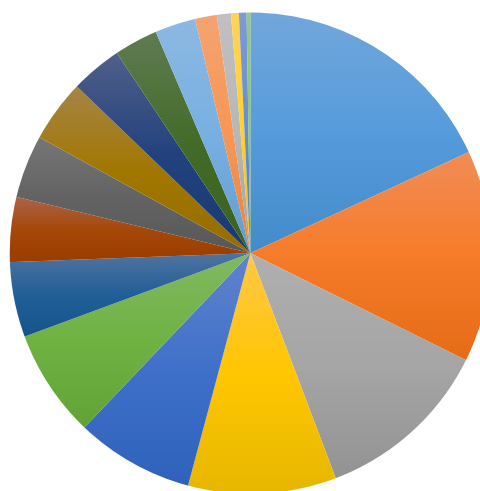
Table 3.2c.3.

#### Neoplastic Releases by Facility: Respiratory System and Intrathoracic Organs (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	Facility Name (Short)	Location		Chemicals			7-Years (Pounds)		
		Town	County	'08	'11	'14	Average	Total	%
1	TGPC CS 245	Winfield	Herkimer	25	25	17	242,124	1,694,870	18.10
2	TGPC CS 249	Carlisle	Schoharie	25	18	17	190,447	1,333,127	14.23
3	TGPC CS 241	LaFayette	Onondaga	24	21	24	159,204	1,114,430	11.90
4	TGPC 229 & TEG DF	Eden	Erie	27	26	27	133,041	931,287	9.94
5	AGT Stony Point CS	Stony Point	Rockland	27	17	17	106,158	743,107	7.93
6	TGPC CS 237	Manchester, Phelps	Ontario	5	5	2	97,145	680,015	7.26
7	AGT SOUTHEAST CS	Southeast	Putnam	20	13	26	67,392	471,742	5.04
8	NFGSC Concord CS	Concord	Erie	7	5	7	58,212	407,483	4.35
9	TGPC CS 254	Chatham	Columbia	20	12	5	56,703	396,923	4.24
10	NFGSC Independence CS	Andover	Allegany	10	6	11	55,998	391,984	4.19
11	DTI Woodhull Station	Woodhull	Steuben	27	33	33	46,203	323,418	3.45
12	TGPC CS 224	Clymer	Chautauqua	25	24	25	39,258	274,805	2.93
13	NFGSC Beech Hill CS	Willing	Allegany	15	15	15	36,636	256,454	2.74
14	NFGSC Nashville CS	Hanover	Chautauqua	21	20	0	19,553	136,871	1.46
15	DTI Utica Station	Frankfort	Herkimer	21	26	33	12,589	88,120	0.94
16	TGPC CS 230-C	Lockport	Niagara	20	19	20	7,007	49,046	0.52
17	DTI Borger CS	Ithaca	Tompkins	27	28	15	6,875	48,128	0.51
18	TGPC CS 233	York	Livingston	20	12	1	3,391	23,740	0.25
				36	37	37	1,337,936	9,365,551	100%

#### NYS Compressor Station NEI Releases by Facility: 2008-2014 Malignant Neoplasms: Respiratory & Intrathoracic Organs



1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

### 3.2c.4. Releases by Facility: Malignant Neoplasms, Bone and Articular Cartilage

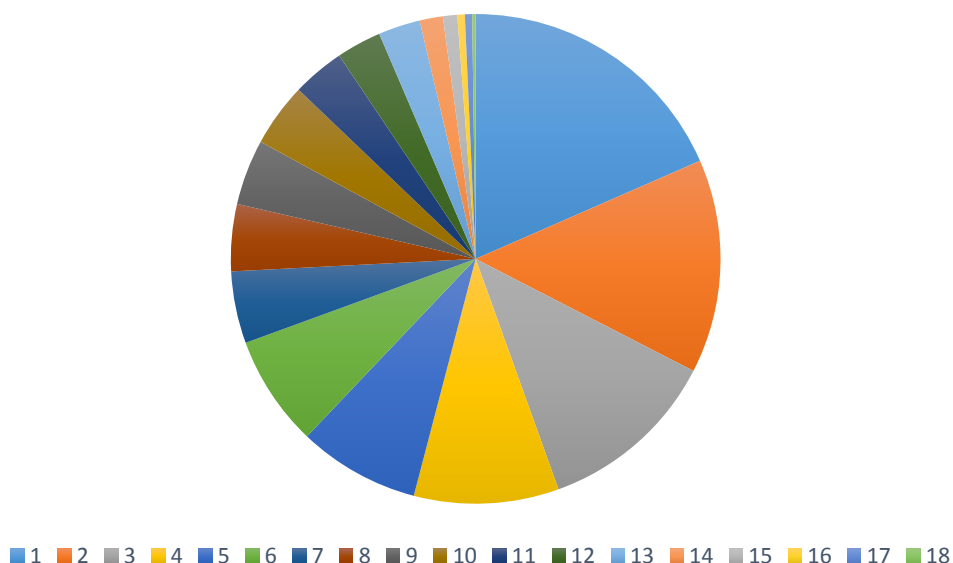
Table 3.2c.4.

#### Releases by Facility: Malignant Neoplasms, Bone and Articular Cartilage (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	Facility Name (Short)	Location		Chemicals			7-Years (Pounds)		
		Town	County	'08	'11	'14	Average	Total	%
1	TGPC CS 245	Winfield	Herkimer	22	22	14	230,920	1,616,443	18.44
2	TGPC CS 249	Carlisle	Schoharie	22	15	14	176,886	1,238,204	14.12
3	TGPC CS 241	LaFayette	Onondaga	21	18	21	149,626	1,047,383	11.95
4	TGPC 229 & TEG DF	Eden	Erie	24	23	24	119,758	838,307	9.56
5	AGT Stony Point CS	Stony Point	Rockland	25	14	14	100,508	703,556	8.02
6	TGPC CS 237	Manchester, Phelps	Ontario	4	4	2	91,916	643,411	7.34
7	AGT SOUTHEAST CS	Southeast	Putnam	18	11	24	59,530	416,713	4.75
8	NFGSC Independence CS	Andover	Allegany	9	5	10	55,385	387,695	4.42
9	NFGSC Concord CS	Concord	Erie	6	4	6	54,199	379,392	4.33
10	TGPC CS 254	Chatham	Columbia	18	10	4	52,602	368,212	4.20
11	DTI Woodhull Station	Woodhull	Steuben	25	30	30	43,265	302,855	3.45
12	TGPC CS 224	Clymer	Chautauqua	22	21	22	37,179	260,256	2.97
13	NFGSC Beech Hill CS	Willing	Allegany	13	13	13	34,633	242,428	2.77
14	NFGSC Nashville CS	Hanover	Chautauqua	20	19	0	19,171	134,194	1.53
15	DTI Utica Station	Frankfort	Herkimer	20	24	30	11,862	83,036	0.95
16	DTI Borger CS	Ithaca	Tompkins	25	26	13	6,195	43,362	0.49
17	TGPC CS 230-C	Lockport	Niagara	18	17	18	5,958	41,706	0.48
18	TGPC CS 233	York	Livingston	18	10	1	2,867	20,069	0.23
				33	33	34	1,252,460	8,767,222	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
Malignant Neoplasms: Bone and Articular Cartilage





### 3.2c.5. Releases by Facility: Malignant Neoplasms, Skin (ICD-10, C43-C44)

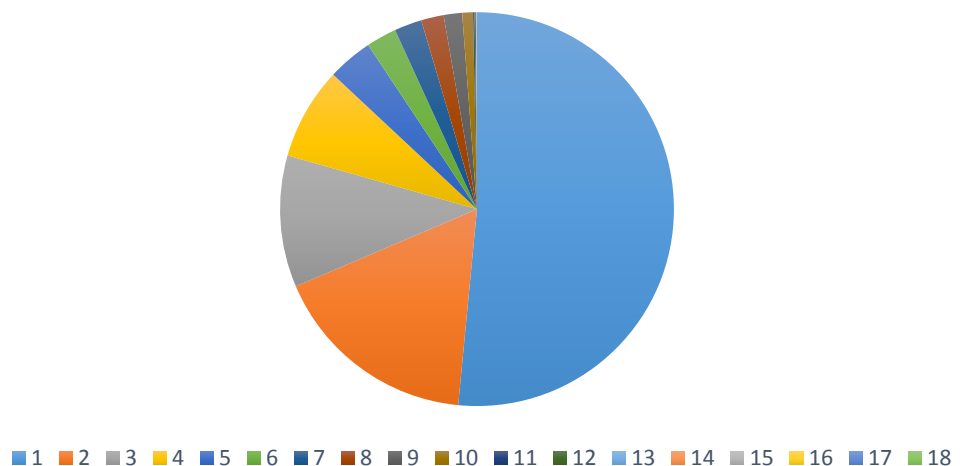
Table 3.2c.5.

#### Releases by Facility: Malignant Neoplasms, Skin (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Avg.	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	TGPC 229 & TEG DF	Eden	Erie	9	8	8	1,964	13,750	51.53
2	AGT Stony Point CS	Stony Point	Rockland	6	3	2	650	4,553	17.06
3	DTI Woodhull Station	Woodhull	Steuben	8	9	11	412	2,883	10.80
4	AGT SOUTHEAST CS	Southeast	Putnam	3	2	8	289	2,022	7.58
5	TGPC CS 224	Clymer	Chautauqua	7	7	7	141	990	3.71
6	DTI Utica Station	Frankfort	Herkimer	7	8	11	95	665	2.49
7	TGPC CS 241	LaFayette	Onondaga	7	5	7	86	602	2.26
8	TGPC CS 249	Carlisle	Schoharie	7	4	4	71	495	1.86
9	TGPC CS 245	Winfield	Herkimer	7	7	4	58	404	1.51
10	TGPC CS 254	Chatham	Columbia	3	1	0	33	234	0.88
11	TGPC CS 230-C	Lockport	Niagara	3	3	3	5	36	0.14
12	TGPC CS 233	York	Livingston	3	1	0	4	28	0.10
13	DTI Borger CS	Ithaca	Tompkins	8	8	2	2	16	0.06
14	NFGSC Beech Hill CS	Willing	Allegany	2	2	2	1	7	0.03
15	NFGSC Nashville CS	Hanover	Chautauqua	7	5	0	0.01	0.09	0.00
16	TGPC CS 237	Manchester, Phelps	Ontario	0	0	0	0	0.00	0.00
17	NFGSC Independence CS	Andover	Allegany	0	0	0	0	0.00	0.00
18	NFGSC Concord CS	Concord	Erie	0	0	0	0	0.00	0.00
				12	12	12	3,812	26,686	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
**Malignant Neoplasms: Skin**



### 3.2c.6. Releases by Facility: Malignant Neoplasms, Connective and Soft Tissue

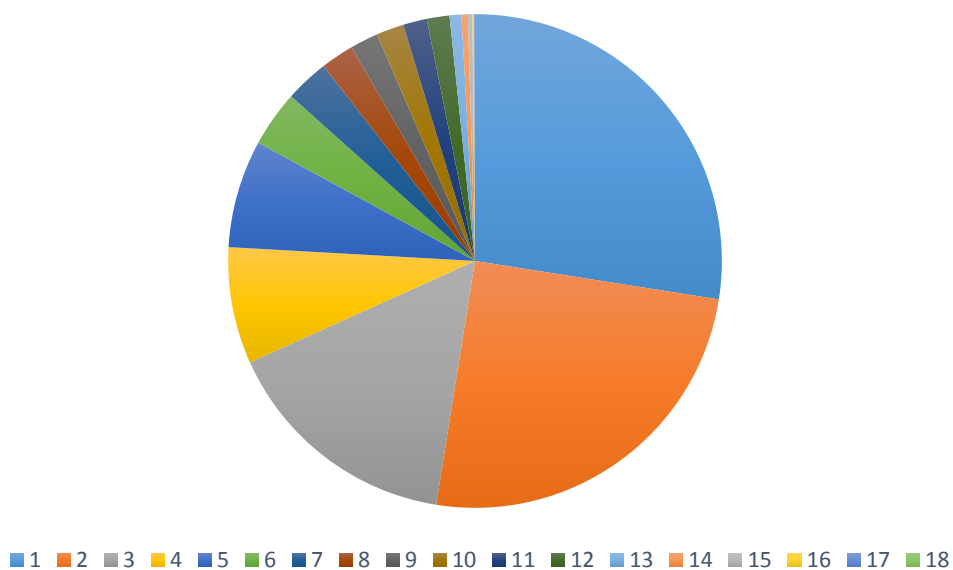
Table 3.2c.6.

#### Releases by Facility: Malignant Neoplasms, Connective and Soft Tissue (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	AGT Stony Point CS	Stony Point	Rockland	13	3	4	1,074	7,516	27.49
2	TGPC 229 & TEG DF	Eden	Erie	9	8	9	978	6,843	25.03
3	AGT SOUTHEAST CS	Southeast	Putnam	8	3	11	615	4,304	15.74
4	TGPC CS 254	Chatham	Columbia	8	2	1	298	2,088	7.64
5	DTI Woodhull Station	Woodhull	Steuben	12	13	14	276	1,930	7.06
6	TGPC CS 224	Clymer	Chautauqua	8	8	8	143	1,004	3.67
7	TGPC CS 230-C	Lockport	Niagara	8	7	8	112	783	2.86
8	TGPC CS 241	LaFayette	Onondaga	7	4	7	85	595	2.18
9	TGPC CS 233	York	Livingston	8	2	0	72	502	1.83
10	TGPC CS 249	Carlisle	Schoharie	8	4	4	70	493	1.80
11	DTI Utica Station	Frankfort	Herkimer	10	11	14	61	428	1.56
12	TGPC CS 245	Winfield	Herkimer	8	8	4	58	406	1.48
13	DTI Borger CS	Ithaca	Tompkins	12	12	4	30	211	0.77
14	NFGSC Concord CS	Concord	Erie	2	1	2	16	113	0.41
15	NFGSC Beech Hill CS	Willing	Allegany	4	4	4	12	81	0.30
16	NFGSC Independence CS	Andover	Allegany	3	1	3	4	30	0.11
17	NFGSC Nashville CS	Hanover	Chautauqua	10	10	0	2	15	0.06
18	TGPC CS 237	Manchester, Phelps	Ontario	0	0	0		0	0.00
				17	17	17	3,906	27,341	100 %

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
Malignant Neoplasms: Connective & Soft Tissue



### 3.2c.7. Releases by Facility: Malignant Neoplasms, Breast and Female Genital Organs

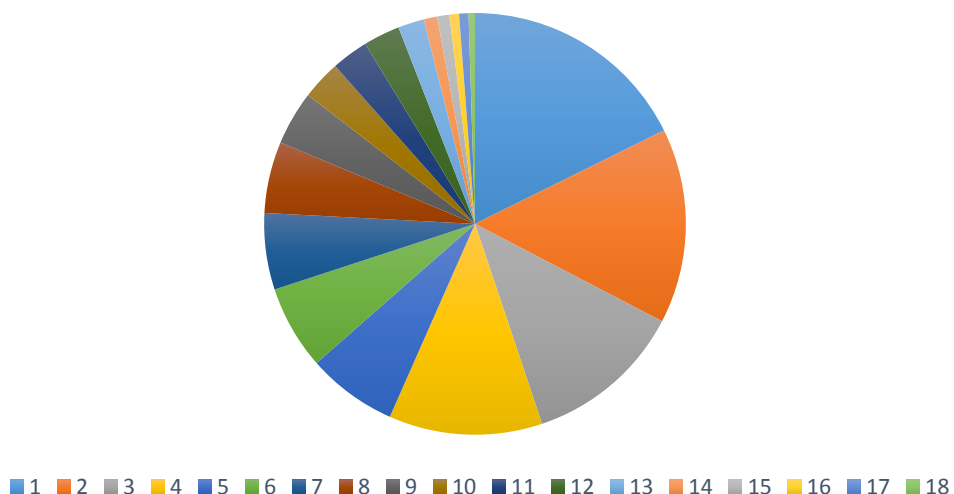
Table 3.2c.7.

#### Releases by Facility: Malignant Neoplasms, Breast and Female Genital Organs (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	19	19	12	109,526	766,684	17.68
2	TGPC CS 249	Carlisle	Schoharie	19	12	12	92,652	648,570	14.96
3	TGPC 229 & TEG DF	Eden	Erie	17	17	17	75,644	529,510	12.21
4	TGPC CS 241	LaFayette	Onondaga	19	13	19	72,893	510,251	11.77
5	TGPC CS 237	Manchester, Phelps	Ontario	5	4	1	42,836	299,858	6.92
6	NFGSC Concord CS	Concord	Erie	5	4	5	39,852	278,969	6.43
7	AGT SOUTHEAST CS	Southeast	Putnam	11	8	14	36,301	254,113	5.86
8	AGT Stony Point CS	Stony Point	Rockland	17	9	9	33,931	237,522	5.48
9	TGPC CS 254	Chatham	Columbia	11	7	5	25,753	180,276	4.16
10	DTI Woodhull Station	Woodhull	Steuben	12	18	19	18,612	130,284	3.00
11	NFGSC Beech Hill CS	Willing	Allegany	9	9	9	17,439	122,076	2.82
12	TGPC CS 224	Clymer	Chautauqua	19	17	19	17,399	121,793	2.81
13	NFGSC Independence CS	Andover	Allegany	6	5	6	12,417	86,924	2.00
14	NFGSC Nashville CS	Hanover	Chautauqua	9	9	0	6,339	44,373	1.02
15	TGPC CS 230-C	Lockport	Niagara	11	11	11	5,817	40,721	0.94
16	DTI Utica Station	Frankfort	Herkimer	9	13	19	4,536	31,755	0.73
17	TGPC CS 233	York	Livingston	11	7	0	4,454	31,181	0.72
18	DTI Borger CS	Ithaca	Tompkins	12	14	8	3,040	21,283	0.49
				23	25	24	619,441	4,336,143	100 %

#### NYS Compressor Station NEI Releases by Facility: 2008-2014 Malignant Neoplasms: Breast & Female Genital Organs



### 3.2c.8. Releases by Facility: Malignant Neoplasms, Male Genital Organs

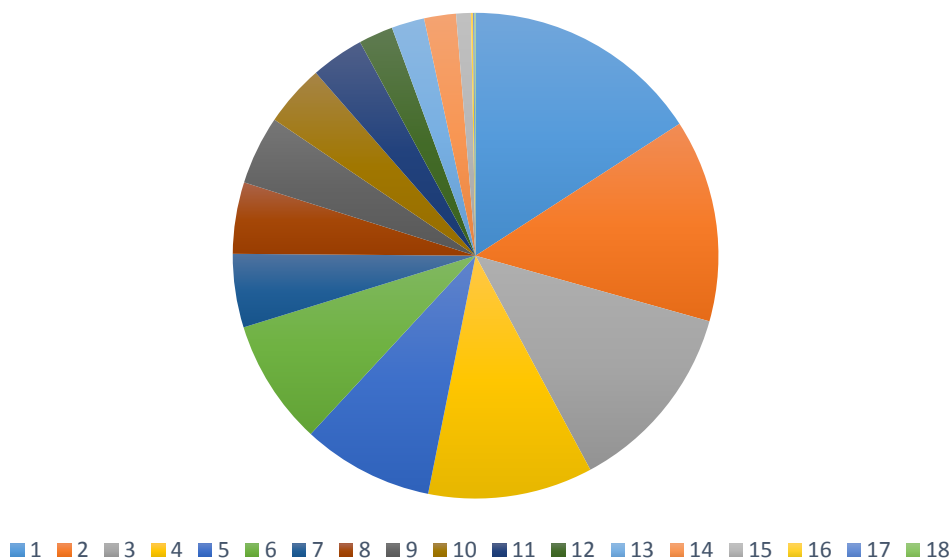
Table 3.2c.8.

#### Releases by Facility: Malignant Neoplasms, Male Genital Organs (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	9	9	7	31,720	222,045	15.86
2	NFGSC Concord CS	Concord	Erie	1	0	1	26,984	188,888	13.49
3	TGPC CS 241	LaFayette	Onondaga	9	9	9	25,625	179,381	12.81
4	TGPC CS 249	Carlisle	Schoharie	9	9	8	21,934	153,543	10.97
5	TGPC 229 & TEG DF	Eden	Erie	8	8	8	17,479	122,356	8.74
6	TGPC CS 237	Manchester, Phelps	Ontario	1	1	1	16,708	116,956	8.35
7	AGT Stony Point CS	Stony Point	Rockland	7	6	5	9,786	68,506	4.89
8	TGPC CS 224	Clymer	Chautauqua	9	8	9	9,516	66,614	4.76
9	NFGSC Independence CS	Andover	Allegany	1	1	3	9,162	64,140	4.58
10	DTI Woodhull Station	Woodhull	Steuben	1	10	10	8,215	57,511	4.11
11	NFGSC Beech Hill CS	Willing	Allegany	4	4	4	7,072	49,504	3.54
12	AGT SOUTHEAST CS	Southeast	Putnam	5	4	6	4,587	32,109	2.29
13	TGPC CS 254	Chatham	Columbia	5	3	1	4,381	30,670	2.19
14	NFGSC Nashville CS	Hanover	Chautauqua	4	4	0	4,243	29,704	2.12
15	DTI Utica Station	Frankfort	Herkimer	4	6	9	1,947	13,631	0.97
16	TGPC CS 230-C	Lockport	Niagara	5	5	5	273	1,915	0.14
17	TGPC CS 233	York	Livingston	5	3	0	194	1,363	0.10
18	DTI Borger CS	Ithaca	Tompkins	7	7	4	152	1,070	0.08
				12	13	13	199,978	1,399,906	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
Malignant Neoplasms: Male Genital Organs



### 3.2c.9. Releases by Facility: Urinary Organs

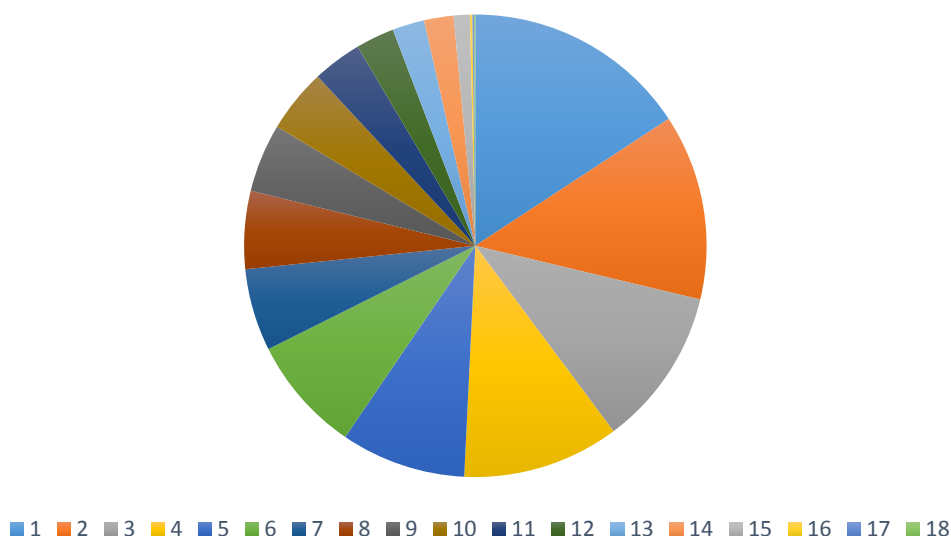
Table 3.2c.9.

#### Neoplastic Releases by Facility: Urinary Organs (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			Pounds	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	17	17	10	32,435	227,047	15.75
2	TGPC CS 241	LaFayette	Onondaga	16	13	16	26,713	186,990	12.98
3	TGPC CS 249	Carlisle	Schoharie	17	12	11	22,813	159,689	11.08
4	TGPC 229 & TEG DF	Eden	Erie	17	16	17	22,555	157,886	10.96
5	NFGSC Concord CS	Concord	Erie	4	2	4	18,006	126,040	8.75
6	TGPC CS 237	Manchester, Phelps	Ontario	2	1	1	16,709	116,964	8.12
7	AGT Stony Point CS	Stony Point	Rockland	17	9	8	11,843	82,900	5.75
8	TGPC CS 224	Clymer	Chautauqua	17	16	17	11,221	78,549	5.45
9	DTI Woodhull Station	Woodhull	Steuben	15	19	22	9,842	68,893	4.78
10	NFGSC Independence CS	Andover	Allegany	6	3	7	9,168	64,174	4.45
11	NFGSC Beech Hill CS	Willing	Allegany	8	8	8	7,081	49,565	3.44
12	AGT SOUTHEAST CS	Southeast	Putnam	12	6	15	5,538	38,766	2.69
13	TGPC CS 254	Chatham	Columbia	12	5	1	4,607	32,249	2.24
14	NFGSC Nashville CS	Hanover	Chautauqua	12	11	0	4,246	29,721	2.06
15	DTI Utica Station	Frankfort	Herkimer	12	13	21	2,324	16,269	1.13
16	TGPC CS 230-C	Lockport	Niagara	12	11	12	353	2,469	0.17
17	TGPC CS 233	York	Livingston	12	5	0	248	1,733	0.12
18	DTI Borger CS	Ithaca	Tompkins	15	14	7	175	1,227	0.09
				24	24	24	205,876	1,441,130	100%

**NYS Compressor Station NEI Releases by Facility: 2008-2014**  
Neoplastic Releases: Urinary Organs



### 3.2c.10. Releases by Facility: Malignant Neoplasms, Eye, Brain and Central Nervous System (C69-C72)

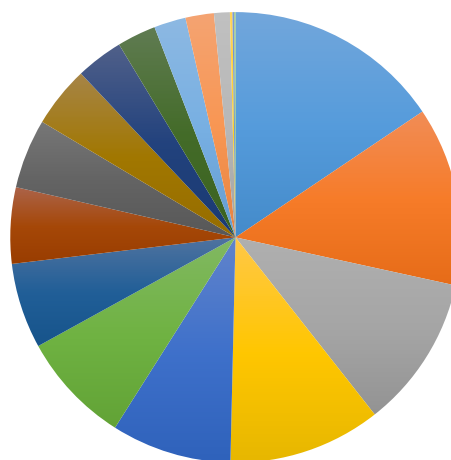
Table 3.2c.10.

#### Releases by Facility: Malignant Neoplasms, Eye, Brain and Central Nervous System (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			Pounds	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	18	18	14	32,515	227,607	15.57
2	TGPC CS 241	LaFayette	Onondaga	17	15	17	26,839	187,876	12.85
3	TGPC CS 249	Carlisle	Schoharie	18	14	14	22,912	160,382	10.97
4	TGPC 229 & TEG DF	Eden	Erie	15	14	15	22,892	160,246	10.96
5	NFGSC Concord CS	Concord	Erie	4	2	4	18,006	126,040	8.62
6	TGPC CS 237	Manchester, Phelps	Ontario	2	1	1	16,709	116,964	8.00
7	AGT Stony Point CS	Stony Point	Rockland	15	10	11	12,801	89,604	6.13
8	TGPC CS 224	Clymer	Chautauqua	18	16	18	11,403	79,821	5.46
9	DTI Woodhull Station	Woodhull	Steuben	12	16	18	10,419	72,931	4.99
10	NFGSC Independence CS	Andover	Allegany	7	3	9	9,168	64,177	4.39
11	NFGSC Beech Hill CS	Willing	Allegany	9	9	10	7,086	49,600	3.39
12	AGT SOUTHEAST CS	Southeast	Putnam	11	9	13	5,828	40,796	2.79
13	TGPC CS 254	Chatham	Columbia	11	7	2	4,770	33,391	2.28
14	NFGSC Nashville CS	Hanover	Chautauqua	8	8	0	4,257	29,798	2.04
15	DTI Utica Station	Frankfort	Herkimer	8	11	18	2,367	16,569	1.13
16	TGPC CS 230-C	Lockport	Niagara	11	10	11	375	2,626	0.18
17	TGPC CS 233	York	Livingston	11	7	0	268	1,874	0.13
18	DTI Borger CS	Ithaca	Tompkins	12	12	9	191	1,338	0.09
				20	20	20	208,806	1,461,640	100

#### NYS Compressor Station NEI Releases by Facility: 2008-2014 Malignant Neoplasms: Eye, Brain & CNS



1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

### 3.2c.11. Releases by Facility: Malignant Neoplasms, Endocrine Glands & Related Structures (C73-C75)

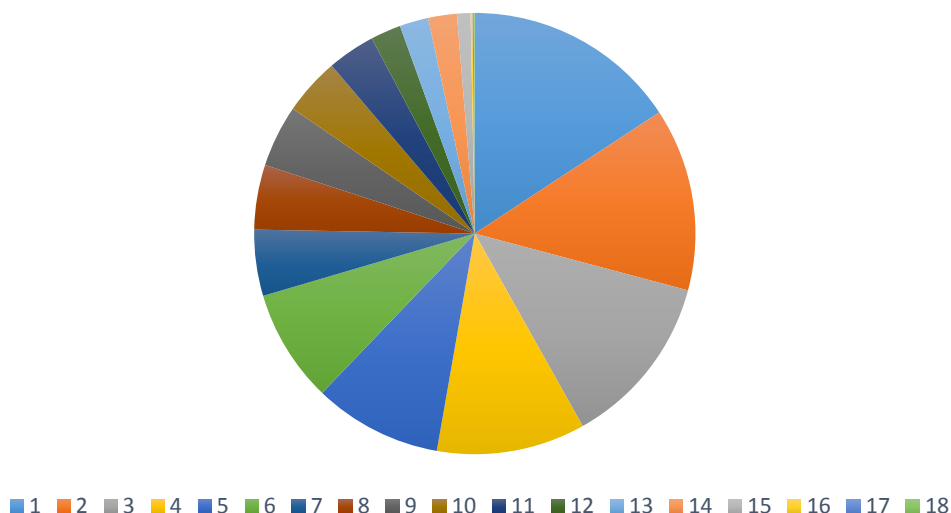
Table 3.2c.11.

#### Releases by Facility: Malignant Neoplasms, Endocrine Glands and Related Structures (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	7	7	3	31,738	222,166	18.18
2	NFGSC Concord CS	Concord	Erie	2	0	2	27,008	189,058	14.69
3	TGPC CS 241	LaFayette	Onondaga	7	3	7	25,649	179,540	12.58
4	TGPC CS 249	Carlisle	Schoharie	7	3	3	21,956	153,691	10.85
5	TGPC 229 & TEG DF	Eden	Erie	7	7	7	18,948	132,638	9.57
6	TGPC CS 237	Manchester, Phelps	Ontario	1	1	1	16,708	116,956	5.59
7	AGT Stony Point CS	Stony Point	Rockland	10	4	4	9,756	68,294	5.48
8	TGPC CS 224	Clymer	Chautauqua	7	7	7	9,565	66,955	5.25
9	NFGSC Independence CS	Andover	Allegany	3	2	3	9,167	64,169	4.83
10	DTI Woodhull Station	Woodhull	Steuben	6	10	9	8,441	59,084	4.05
11	NFGSC Beech Hill CS	Willing	Allegany	4	4	4	7,068	49,477	2.57
12	AGT SOUTHEAST CS	Southeast	Putnam	6	3	5	4,481	31,369	2.43
13	NFGSC Nashville CS	Hanover	Chautauqua	4	4	0	4,246	29,719	2.43
14	TGPC CS 254	Chatham	Columbia	6	2	1	4,234	29,641	1.15
15	DTI Utica Station	Frankfort	Herkimer	4	5	9	2,003	14,020	0.16
16	TGPC CS 230-C	Lockport	Niagara	6	6	6	282	1,972	0.11
17	TGPC CS 233	York	Livingston	6	2	0	189	1,320	0.08
18	DTI Borger CS	Ithaca	Tompkins	6	6	4	142	997	0.00
				10	10	10	201,581	1,411,067	100%

#### NYS Compressor Station NEI Releases by Facility: 2008-2014 Neplastic Releases: Endocrine and Related Stuctures



### 3.2c.12. Releases by Facility: Malignant Neoplasms, Secondary and Ill-defined (ICD-10, C76-C80)

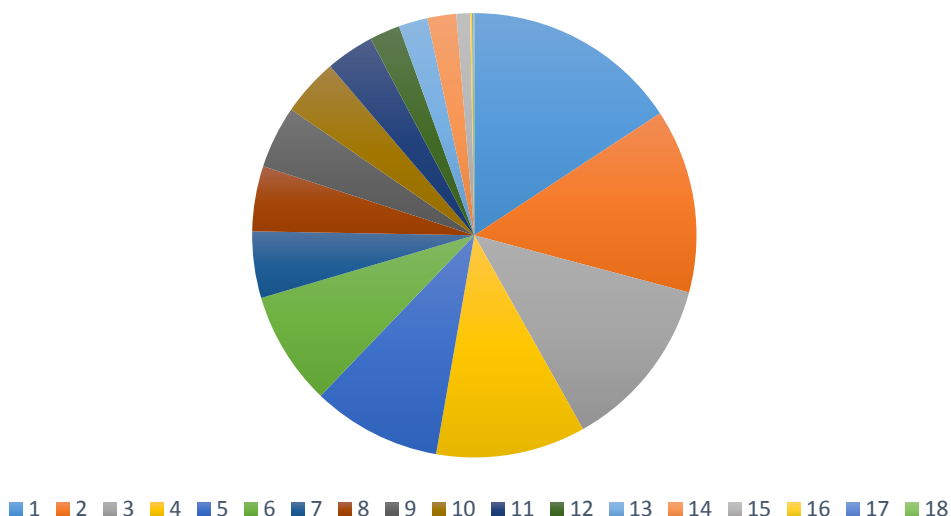
Table 3.2c.12.

#### Releases by Facility: Malignant Neoplasms, Secondary and Ill-defined (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	AGT Stony Point CS	Stony Point	Rockland	5	3	3	1,542	10,793	32.39
2	AGT SOUTHEAST CS	Southeast	Putnam	5	3	5	980	6,858	20.58
3	TGPC 229 & TEG DF	Eden	Erie	4	4	4	739	5,172	15.52
4	TGPC CS 254	Chatham	Columbia	5	3	0	642	4,497	13.50
5	DTI Woodhull Station	Woodhull	Steuben	5	6	6	201	1,405	4.22
6	TGPC CS 230-C	Lockport	Niagara	5	5	5	137	962	2.89
7	TGPC CS 224	Clymer	Chautauqua	3	3	3	112	787	2.36
8	TGPC CS 233	York	Livingston	5	3	0	94	659	1.98
9	TGPC CS 241	LaFayette	Onondaga	3	3	3	72	507	1.52
10	TGPC CS 249	Carlisle	Schoharie	3	3	3	58	409	1.23
11	TGPC CS 245	Winfield	Herkimer	3	3	3	47	331	0.99
12	DTI Utica Station	Frankfort	Herkimer	3	5	6	44	306	0.92
13	DTI Borger CS	Ithaca	Tompkins	5	6	3	43	300	0.90
14	NFGSC Concord CS	Concord	Erie	1	0	1	24	170	0.51
15	NFGSC Beech Hill CS	Willing	Allegany	3	3	3	17	116	0.35
16	NFGSC Independence CS	Andover	Allegany	3	1	4	5	32	0.10
17	NFGSC Nashville CS	Hanover	Chautauqua	3	3	0	2	15	0.05
18	TGPC CS 237	Manchester, Phelps	Ontario	0	0	0		0	0.00
				<b>6</b>	<b>6</b>	<b>6</b>	<b>4,760</b>	<b>33,319</b>	<b>100%</b>

**NYS Compressor Station Emissions by Facility: 2008-2014**  
Malignant Neoplasms, Secondary and Ill-defined





### 3.2c.13. Releases by Facility: Malignant Neoplasms, Stated or Presumed to be Primary, of Lymphoid, Hematopoietic and Related Tissue (ICD-10, C81-96)

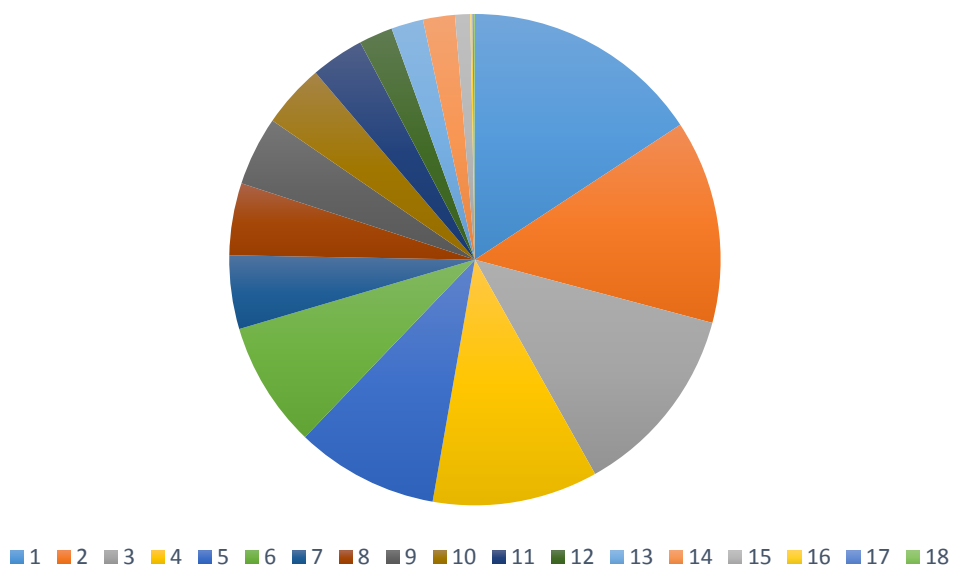
Table 3.2c.13.

Releases by Facility: Malignant Neoplasms, Malignant Neoplasms, Stated or Presumed to be Primary, of Lymphoid, Hematopoietic and Related Tissue (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			3-Yr Average	7 Years (estimate)	
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Pounds	Pounds	%
1	TGPC CS 245	Winfield	Herkimer	23	23	15	109,764	768,346	17.52
2	TGPC CS 249	Carlisle	Schoharie	23	16	16	92,946	650,622	14.83
3	TGPC 229 & TEG DF	Eden	Erie	25	24	25	77,334	541,341	12.34
4	TGPC CS 241	LaFayette	Onondaga	22	16	22	73,255	512,783	11.69
5	TGPC CS 237	Manchester, Phelps	Ontario	4	4	1	42,836	299,851	6.84
6	NFGSC Concord CS	Concord	Erie	5	5	6	39,857	278,996	6.36
7	AGT SOUTHEAST CS	Southeast	Putnam	16	11	19	37,129	259,905	5.93
8	AGT Stony Point CS	Stony Point	Rockland	26	16	15	35,662	249,635	5.69
9	TGPC CS 254	Chatham	Columbia	16	10	5	26,113	182,788	4.17
10	DTI Woodhull Station	Woodhull	Steuben	19	24	27	19,186	134,302	3.06
11	TGPC CS 224	Clymer	Chautauqua	23	22	23	17,967	125,770	2.87
12	NFGSC Beech Hill CS	Willing	Allegany	13	13	13	17,550	122,849	2.80
13	NFGSC Independence CS	Andover	Allegany	9	4	9	12,461	87,225	1.99
14	NFGSC Nashville CS	Hanover	Chautauqua	15	15	0	6,367	44,568	1.02
15	TGPC CS 230-C	Lockport	Niagara	16	15	16	5,882	41,174	0.94
16	DTI Utica Station	Frankfort	Herkimer	15	18	27	4,637	32,462	0.74
17	TGPC CS 233	York	Livingston	16	10	0	4,510	31,567	0.72
18	DTI Borger CS	Ithaca	Tompkins	19	19	12	3,079	21,553	0.49
				31	31	31	626,534	4,385,736	100%

**NYS Compressor Station Emissions by Facility: 2008-2014**  
Malignant Neoplasms, Lymphoid, Hematopoietic, Related Tissue



### 3.2d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of carcinogens.

DEC Region 9, Western New York, ranked first with 2.5 million pounds or 26% of the state total, followed by Region 6, Western Adirondacks/Eastern Lake Ontario (1,810,984 pounds or 19%) and Region 4, Capital Region/Northern Catskills (1,765,328 pounds or 18.5%). These three regions are responsible for slightly more than one-half (53%) of all toxic releases.

Table 3.2d.

#### C00-D58. Neoplastic Releases by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	NYS DEC Region	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	7	2	20	279,592	93,197	652,381	6.84
		Chautauqua	10	2	54	159,795	53,265	372,856	3.91
		Erie	2	2	45	583,207	194,402	1,360,816	14.27
		Niagara	12	1	23	29,917	9,972	69,806	0.73%
				7		1,052,511	350,837	2,455,859	25.75
2	6: W. Adirondacks/E. Lake Ontario	Herkimer	1	2	57	776,136	258,712	1,810,984	18.99
3	4: Capital Region/Northern Catskills	Columbia	9	1	23	173,569	57,856	404,994	4.25
		Schoharie	3	1	41	583,000	194,333	1,360,334	14.26
				2		756,569	252,190	1,765,328	18.51
4	7: Central New York	Onondaga	8	1	40	216,219	72,073	504,510	5.29
		Tompkins	5	1	42	331,393	110,464	773,250	8.11
				2		547,611	182,537	1,277,760	13.40
5	3: Lower Hudson Valley	Putnam	8	1	40	216,219	72,073	504,510	5.29
		Rockland	5	1	42	331,393	110,464	773,250	8.11
				2		547,611	182,537	1,277,760	13.40
6	8: Western Finger Lakes	Livingston	14	1	23	10,477	3,492	24,447	0.26
		Ontario	6	1	7	291,994	97,331	681,320	7.14
		Steuben	11	1	53	141,258	47,086	329,603	3.46
				3		443,730	147,910	1,035,369	10.85
					18	59	4,087,832	1,362,611	9,538,274

### 3.2e. Releases by County

Herkimer County ranked first with 1.8 million pounds or 19% of the state total, followed by Erie County (1,360,816 pounds or 14.27%) and Schoharie with a slightly smaller total (1,360,333 pounds or 14.26%). These three counties are responsible for nearly one-half (47.5%) of all carcinogenic releases.

The top five counties were responsible for slightly more than two-thirds (67.6%) of the state total.

The 14-county average was 681,304 pounds.

Table 3.2e.

#### C00-D58. Neoplastic Releases by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	57	776,135	258,711	1,810,983	18.99
2	Erie	9: Western New York	2	45	583,207	194,402	1,360,816	14.27
3	Schoharie	4: Capital Region/Northern Catskills	1	41	583,000	194,333	1,360,333	14.26
4	Onondaga	7: Central New York	1	40	488,561	162,853	1,139,975	11.95
5	Rockland	3: Lower Hudson Valley	1	42	331,392	110,464	773,249	8.11
6	Ontario	8: Western Finger Lakes	1	7	291,994	97,331	681,319	7.14
7	Allegany	9: Western New York	2	20	279,591	93,197	652,381	6.84
8	Putnam	3: Lower Hudson Valley	1	40	216,218	72,072	504,510	5.29
9	Columbia	4: Capital Region/Northern Catskills	1	23	173,569	57,856	404,994	4.25
10	Chautauqua	9: Western New York	2	54	159,795	53,265	372,855	3.91
11	Steuben	8: Western Finger Lakes	1	53	141,258	47,086	329,602	3.46
12	Niagara	9: Western New York	1	23	29,916	9,972	69,805	0.73
13	Tompkins	7: Central New York	1	40	22,713	7,571	52,997	0.56
14	Livingston	8: Western Finger Lakes	1	23	10,477	3,492	24,446	0.26
			<b>18</b>	<b>59</b>	<b>4,087,826</b>	<b>1,362,605</b>	<b>9,538,265</b>	<b>100</b>

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### 3.3. Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (D50-D89)

#### 3.3a. Releases by Chemical

Forty-one of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (ICD 10, Chapter 3). All 18 stations reported such releases. These totaled an estimated 18.7 million pounds from 2008 to 2014--an annual average of 2.7 million pounds.

Chemicals associated with blood and immune system diseases represented 47% of all reported natural gas compressor station releases.

Carbon monoxide was responsible for almost two-thirds (65.9%) of all statewide chemical releases. Volatile organic compounds as a group rank second with 4.9 million pounds (26.2%), followed by formaldehyde with 1.3 million pounds (7%). These three chemicals accounted for 99.1% of the state total.

Table 3.3a.

Diseases of the Blood and Blood-forming Organs and Certain Disorders Involving the Immune Mechanism by Chemical (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	65.91
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,395	26.24
3	Formaldehyde	18	14	6	561,144	187,048	1,309,335	6.98
4	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.35
5	Benzene	16	13	6	9,103	3,034	21,240	0.11
6	Methanol	8	7	6	8,286	2,762	19,333	0.10
7	Toluene	16	13	6	8,275	2,758	19,307	0.10
8	Hexane	13	10	6	5,222	1,741	12,183	0.06
9	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.04
10	1,3-Butadiene	14	13	6	2,022	674	4,718	0.03
		<b>18</b>	<b>14</b>	<b>6</b>	<b>8,031,690</b>	<b>2,677,230</b>	<b>18,740,605</b>	<b>99.94</b>

### 3.3b. Releases by ICD Category

Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism into 5 major groups. Chemicals released by natural gas compressor stations are positively associated with two of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**D70-D77:** Twenty-seven chemicals are associated with Coagulation defects, purpura and other hemorrhagic conditions These chemicals were released by all 18 stations.

**D80-D89:** Eighteen chemicals had effects broadly characterized as Other diseases of blood and blood-forming organs

Table 3.3b.

Diseases of the Blood and Blood-forming Organs and Certain Disorders Involving the Immune Mechanism by ICD Category

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	D50-D53	Nutritional anemias	0	0	0	0	0	0	0	0	0	0	0	0
2	D55-D64	Hemolytic anemias	0	0	0	0	0	0	0	0	0	0	0	0
3	D65-D69	Aplastic and other anemias	0	0	0	0	0	0	0	0	0	0	0	0
4	D70-D77	Coagulation defects, purpura and other hemorrhagic conditions	18	18	16	18	27	27	27	27	122,594	261,322	242,629	626,546
5	D80-D89	Other diseases of blood and blood-forming organs	18	18	17	18	17	18	18	18	1,534,607	2,284,799	2,088,789	5,908,195
	D50-D89	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>40</b>	<b>41</b>	<b>41</b>	<b>41</b>	<b>1,913,307</b>	<b>3,125,530</b>	<b>2,997,450</b>	<b>8,036,288</b>

### 3.3c. Releases by Facility

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 4.1 million pounds or slightly more than one-fifth of the total, followed by the Compressor 249 in Carlisle (2.1 million pounds or 11.1%) and by its LaFayette facility (2 million pounds or 10.3). These three sites were responsible for 8.1 million pounds or 43% of all statewide releases of chemicals associated with blood and immune system disorders.

The top 5 sites were responsible for 11 million pounds (58.5%) of the state total.

The facility average was 1,048,773 pounds.

Table 3.3c.

#### Diseases of the Blood and Blood-forming Organs and Certain Disorders Involving the Immune Mechanism by Facility

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	31	31	17	580,733	4,065,132	21.53
2	TGPC CS 249	Carlisle	Schoharie	31	18	18	299,205	2,094,438	11.09
3	TGPC CS 241	LaFayette	Onondaga	30	21	30	278,630	1,950,408	10.33
4	TGPC 229 & TEG DF	Eden	Erie	29	28	29	223,059	1,561,413	8.27
5	TGPC CS 237	Manchester, Phelps	Ontario	4	3	3	195,395	1,367,764	7.25
6	TGPC CS 254	Chatham	Columbia	17	10	4	161,398	1,129,784	5.98
7	AGT Stony Point CS	Stony Point	Rockland	33	15	16	154,988	1,084,914	5.75
8	NFGSC Independence CS	Andover	Allegany	10	5	12	147,748	1,034,239	5.48
9	NFGSC Beech Hill CS	Willing	Allegany	14	14	15	140,703	984,922	5.22
10	NFGSC Concord CS	Concord	Erie	6	5	7	133,625	935,372	4.95
11	TGPC CS 224	Clymer	Chautauqua	31	29	31	105,096	735,670	3.90
12	DTI Woodhull Station	Woodhull	Steuben	24	33	36	86,690	606,832	3.21
13	AGT SOUTHEAST CS	Southeast	Putnam	17	12	24	62,269	435,886	2.31
14	NFGSC Nashville CS	Hanover	Chautauqua	19	19	-	54,249	379,740	2.01
15	TGPC CS 230-C	Lockport	Niagara	17	16	17	22,498	157,488	0.83
16	DTI Utica Station	Frankfort	Herkimer	19	23	36	22,270	155,891	0.83
17	DTI Borger CS	Ithaca	Tompkins	24	25	13	21,652	151,564	0.80
18	TGPC CS 233	York	Livingston	17	10	2	6,638	46,465	0.25
				<b>40</b>	<b>41</b>	<b>41</b>	<b>2,696,846</b>	<b>18,877,920</b>	<b>100%</b>

### 3.3d Releases by DEC Region

Six of New York State's nine DEC regions reported releases of toxic chemicals associated with blood and immune system disorders.

DEC Region 9, Western New York, ranked first with an estimated 5.7 million pounds (30.2%) of releases from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, second with 4.2 million pounds (22.5%), followed by Region 4, Capital Region/Northern Catskills with 3.2 million pounds (17.2%).

Table 3.3d.

D50-D89: Diseases of the Blood and Blood-forming Organs and Certain Disorders Involving the Immune Mechanism by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	NYS DEC Region	County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
		Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	4	2	16	865,355	288,452	2,019,161	10.77
		Chautauqua	9	2	39	423,784	141,261	988,830	5.27
		Erie	2	2	32	1,070,051	356,684	2,496,785	13.32
		Niagara	12	1	17	67,495	22,498	157,488	0.84
				7	40	2,426,684	808,895	5,662,264	30.20
2	6: Adirondacks/E Lake Ontario	Herkimer	1	2	40	1,809,010	603,003	4,221,023	22.51
3	4: Capital Region/N. Catskills	Columbia	7	1	17	484,193	161,398	1,129,784	6.03
		Schoharie	3	1	31	897,616	299,205	2,094,438	11.17
				2	35	1,381,809	460,603	3,224,222	17.19
4	7: Central New York	Onondaga	5	1	30	835,889	278,630	1,950,408	10.40
		Tompkins	13	1	25	64,956	21,652	151,564	0.81
				2	41	900,845	300,282	2,101,972	11.21
5	8: Western Finger Lakes	Livingston	14	1	17	19,913	6,638	46,465	0.25
		Ontario	6	1	4	586,185	195,395	1,367,764	7.29
		Steuben	10	1	37	260,071	86,690	606,832	3.24
				3	37	866,169	288,723	2,021,060	10.78
6	3: Lower Hudson Valley	Livingston	11	1	26	186,808	62,269	435,886	2.32
		Ontario	8	1	33	464,963	154,988	1,084,914	5.79
				2	39	651,772	217,257	1,520,800	8.11
				18	41	8,036,289	2,678,763	18,751,340	100



### 3.3e. Releases by County

Herkimer County ranked first with 4.2 million pounds or 22.5% of the state total, followed by Erie County (2.5 million pounds or 13.3%) and Schoharie County (2.1 million pounds or 11.2%). These three counties are responsible for nearly one-half (48%) of all releases associated with blood and immune system diseases.

The top five counties were responsible for 12.8 million pounds or slightly more than two-thirds (68.2%) of the state total.

The 14-county average was 1,339,381 pounds.

Table 3.3e.

#### Diseases of the Blood and Blood-forming Organs and Certain Disorders Involving the Immune Mechanism by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	40	1,809,009	603,003	4,221,023	22.51
2	Erie	9: Western New York	2	32	1,070,050	356,684	2,496,785	13.32
3	Schoharie	4: Capital Region/Northern Catskills	1	31	897,616	299,205	2,094,438	11.17
4	Allegany	9: Western New York	2	16	865,354	288,452	2,019,161	10.77
5	Onondaga	7: Central New York	1	30	835,889	278,630	1,950,408	10.40
6	Ontario	8: Western Finger Lakes	1	4	586,184	195,395	1,367,764	7.29
7	Columbia	4: Capital Region/Northern Catskills	1	17	484,193	161,398	1,129,784	6.03
8	Rockland	3: Lower Hudson Valley	1	33	464,963	154,988	1,084,914	5.79
9	Chautauqua	9: Western New York	2	39	423,784	141,261	988,830	5.27
10	Steuben	8: Western Finger Lakes	1	37	260,070	86,690	606,832	3.24
11	Putnam	3: Lower Hudson Valley	1	26	186,808	62,269	435,886	2.32
12	Niagara	9: Western New York	1	17	67,494	22,498	157,488	0.84
13	Tompkins	7: Central New York	1	25	64,955	21,652	151,564	0.81
14	Livingston	8: Western Finger Lakes	1	17	19,913	6,638	46,465	0.25
			<b>18</b>	<b>41</b>	<b>8,036,282</b>	<b>2,678,763</b>	<b>18,751,340</b>	<b>100%</b>

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## 3.4. Endocrine, Nutritional and Metabolic Diseases (E00-E90)

### 3.4a. Releases by Chemical

Fifty-one of the 70 chemicals released by NYS natural gas compressor stations are associated with endocrine, nutritional and metabolic disorders (ICD 10, Chapter 4).

All 18 stations reported such releases. These totaled an estimated 7.1 million pounds from 2008 to 2014--an annual average of slightly more than 1 million pounds a year.

Chemicals associated with these effects accounted for 17.8% of all reported natural gas compressor station releases.

VOCs were responsible for 4.9 million pounds or slightly more than two-thirds (68.1%) of all statewide releases. Formaldehyde ranked second (1.3 million pounds or 18.4%), followed by PM Condensable (540,267 pounds or 7.6%). These three chemicals accounted for 95.1% of all releases.

The top 5 chemicals were responsible for 98.7% of the state total.

Table 3.4a.

#### E00-E90: Endocrine, Nutritional and Metabolic Diseases by Chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	69.13
2	Formaldehyde	18	14	6	561,144	187,048	1,309,336	18.40
3	PM Condensable	18	14	6	231,543	77,181	540,267	7.59
4	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	2.62
5	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.93
6	Benzene	16	13	6	9,103	3,034	21,241	0.30
7	Methanol	8	7	6	8,286	2,762	19,333	0.27
8	Toluene	16	13	6	8,275	2,758	19,308	0.27
9	Hexane	13	10	6	5,222	1,741	12,184	0.17
10	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.12
		18	14	6	3,044,231	1,014,744	7,103,205	99.80

### 3.4b. Releases by ICD Category

**E00-E35:** Forty-seven chemicals are associated with endocrine diseases. These include: adrenal weight change, androgen effects, delayed puberty, thymus weight changes, and thyroid hypofunction and other thyroid disorders.

**E70-E90:** Seventeen chemicals are associated with metabolic disorders. These include: biochemical and metabolic effects, homeostasis, hyperchloremic acidosis, and serum composition (changes: e.g. TP, bilirubin, cholesterol).

Table 3.04a.

#### E00-E90: Endocrine, Nutritional and Metabolic Diseases by ICD Category

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	E00-E35	Endocrine diseases	18	18	17	18	45	42	43	47	173,387	385,467	380,538	939,392
2	E40-E68	Nutritional diseases	0	0	0	0	0	0	0	0	0	0	0	0
3	E70-E90	Metabolic diseases	18	17	16	18	17	17	17	17	120,072	258,769	240,402	619,243
	<b>E00-E90</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>49</b>	<b>46</b>	<b>47</b>	<b>51</b>	<b>547,971</b>	<b>1,218,450</b>	<b>1,283,873</b>	<b>3,050,294</b>

### 3.4c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releasing these chemicals.

Four of the top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 1.3 million pounds (17.5%), followed by the Compressor Station 249 in Carlisle (948,972 pounds or 13.4%) and Compressor 241 in LaFayette (874,287 pounds or 12.2%). These three sites were responsible for 43% of all statewide releases.

The top 5 facilities were responsible for 61% of the total.

The facility average was 397,734 pounds.

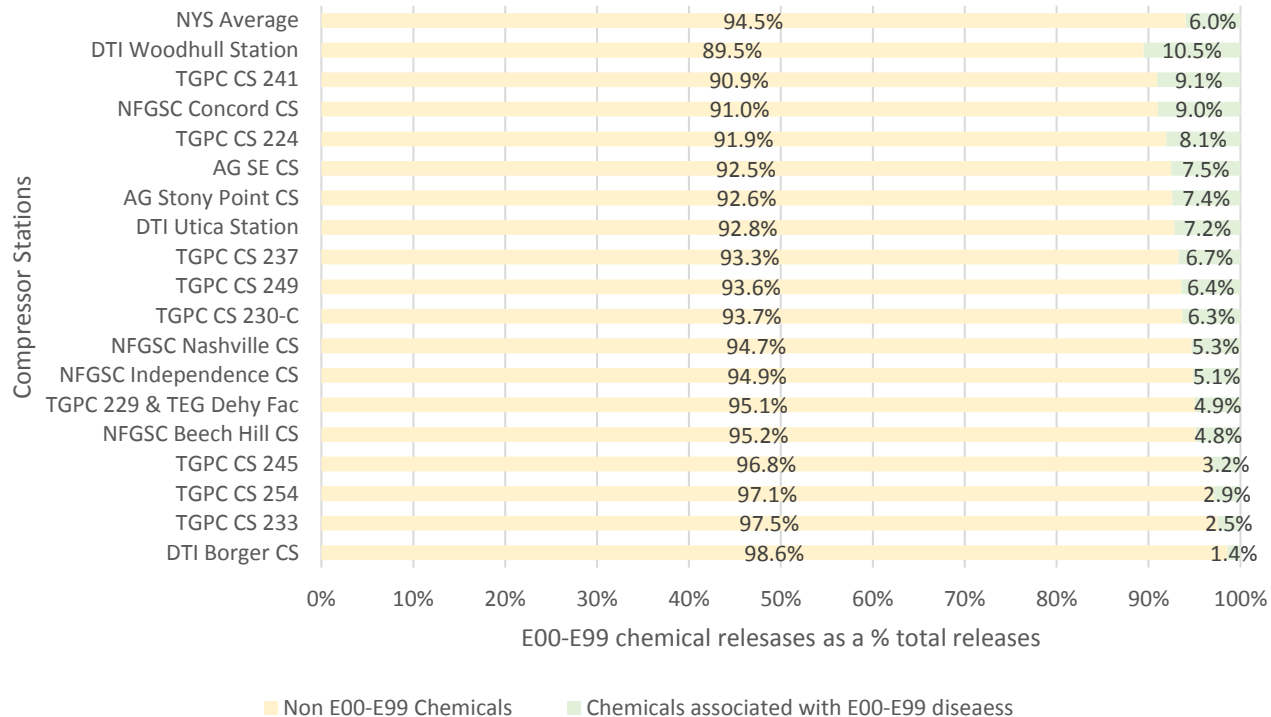
Table 3.4c.

#### Endocrine, Nutritional and Metabolic Diseases by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	34	32	17	179,267	1,254,870	17.53
2	TGPC CS 249	Carlisle	Schoharie	34	18	18	136,996	958,972	13.39
3	TGPC CS 241	LaFayette	Onondaga	32	25	31	124,938	874,562	12.22
4	AGT Stony Point CS	Stony Point	Rockland	33	17	16	90,941	636,585	8.89
5	TGPC 229 & TEG DF	Eden	Erie	38	33	34	89,691	627,840	8.77
6	TGPC CS 237	Manchester, Phelps	Ontario	5	4	3	76,433	535,028	7.47
7	NFGSC Independence CS	Andover	Allegany	10	5	12	53,498	374,487	5.23
8	AGT SOUTHEAST CS	Southeast	Putnam	20	13	37	48,166	337,165	4.71
9	TGPC CS 254	Chatham	Columbia	20	11	5	40,522	283,651	3.96
10	NFGSC Concord CS	Concord	Erie	6	6	7	40,512	283,584	3.96
11	DTI Woodhull Station	Woodhull	Steuben	33	41	44	38,601	270,204	3.77
12	TGPC CS 224	Clymer	Chautauqua	32	30	32	34,003	238,018	3.32
13	NFGSC Beech Hill CS	Willing	Allegany	14	14	15	28,685	200,796	2.80
14	NFGSC Nashville CS	Hanover	Chautauqua	29	27	--	17,947	125,632	1.75
15	DTI Utica Station	Frankfort	Herkimer	29	33	43	10,601	74,209	1.04
16	TGPC CS 230-C	Lockport	Niagara	20	19	20	5,433	38,028	0.53
17	DTI Borger CS	Ithaca	Tompkins	33	34	13	5,354	37,476	0.52
18	TGPC CS 233	York	Livingston	20	11	2	1,159	8,113	0.11
				<b>49</b>	<b>46</b>	<b>47</b>	<b>1,022,747</b>	<b>7,159,220</b>	<b>100%</b>

## E00-E99 Chemicals as % of Each Compressor Station's Total Releases



### 3.4d. Releases by DEC Region

Six of New York State's nine DEC regions reported releases of chemicals associated with endocrine and metabolic diseases.

DEC Region 9, Western New York, ranked first with 1.8 million pounds (26%) from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, ranked second with 1.3 million pounds (18.7%), closely followed by Region 3, Lower Hudson Valley, with 1.2 million pounds (17.5%).

Table 3.4d.

#### E00-E90: Endocrine, Nutritional and Metabolic Diseases by DEC Region (ranked)

##### NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	NYS DEC Region	County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
		Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	6	2	15	246,550	82,183	575,284	8.08
		Chautauqua	9	2	45	137,903	45,968	321,773	4.52
		Erie	3	2	40	390,611	130,204	911,425	12.81
		Niagara	12	1	20	16,298	5,433	38,028	0.53
				7	49	791,362	263,787	1,846,511	25.94
2	6: W Adirondacks/E Lake Ontario	Herkimer	1	2	50	569,606	189,869	1,329,080	18.67
3	4: Capital Region/N. Catskills	Columbia	10	1	20	121,565	40,522	283,651	3.99
		Schoharie	2	1	34	410,988	136,996	958,972	13.47
				2	39	532,553	177,518	1,242,624	17.46
6	3: Lower Hudson Valley	Putnam	8	1	37	144,499	48,166	337,165	4.74
		Rockland	5	1	35	272,822	90,941	636,585	8.94
				2	45	417,322	139,107	973,751	13.68
4	7: Central New York	Onondaga	4	1	32	374,813	124,938	874,563	12.29
		Tompkins	13	1	36	16,061	5,354	37,476	0.53
				2	49	390,874	130,291	912,039	12.81
5	8: Western Finger Lakes	Livingston	14	1	20	3,477	1,159	8,114	0.11
		Ontario	7	1	5	229,298	76,433	535,029	7.52
		Steuben	11	1	46	115,802	38,601	270,204	3.80
				3	46	348,577	116,192	813,347	11.43
				18	51	3,050,294	1,016,765	7,117,352	100

### 3.4e. Releases by County

All fourteen counties where compressor stations are located reported releases of chemicals linked to endocrine and metabolic disorders.

Herkimer County ranked first with 1.3 million pounds or 18.7% of the state total, followed by Schoharie County (958,972 pounds or 13.5%) and Erie County (911,425 pounds or 12.8%). These three counties are responsible for nearly one-half (45%) of all releases.

The top five counties were responsible for 12.8 million pounds or slightly less than two-thirds (66.2%) of the state total.

The 14-county average was 508,382 pounds.

Table 3.4e.

#### E00-E90: Endocrine, Nutritional and Metabolic Diseases by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	50	569,605	189,868	1,329,080	18.67
2	Schoharie	4: Capital Region/Northern Catskills	1	34	410,988	136,996	958,972	13.47
3	Erie	9: Western New York	2	40	390,610	130,203	911,425	12.81
4	Onondaga	7: Central New York	1	32	374,812	124,937	874,562	12.29
5	Rockland	3: Lower Hudson Valley	1	35	272,822	90,940	636,585	8.94
6	Allegany	9: Western New York	2	15	246,550	82,183	575,283	8.08
7	Ontario	8: Western Finger Lakes	1	5	229,298	76,432	535,028	7.52
8	Putnam	3: Lower Hudson Valley	1	37	144,499	48,166	337,165	4.74
9	Chautauqua	9: Western New York	2	45	137,902	45,967	321,773	4.52
10	Columbia	4: Capital Region/Northern Catskills	1	20	121,564	40,521	283,651	3.99
11	Steuben	8: Western Finger Lakes	1	46	115,801	38,600	270,204	3.80
12	Niagara	9: Western New York	1	20	16,297	5,432	38,028	0.53
13	Tompkins	7: Central New York	1	36	16,061	5,353	37,476	0.53
14	Livingston	8: Western Finger Lakes	1	20	3,477	1,159	8,113	0.11
			<b>18</b>	<b>50</b>	<b>3,050,286</b>	<b>1,016,757</b>	<b>7,117,345</b>	<b>100%</b>



## 3.5. Mental and Behavioral Disorders (F00-F99)

### 3.5a. Releases by Chemical

***Thirty-four of the 70 chemicals released by NYS natural gas compressor reported to NEI are associated with mental and behavioral disorders (ICD-10, Chapter 5).***

***All 18 stations reported such releases. These totaled an estimated 18.7 million pounds from 2008 to 2014--an annual average of 2.7 million pounds.***

Chemicals associated with mental and behavioral disorders represented 47% of all reported toxic releases reported from natural gas compressor stations reported to NEI.

Carbon monoxide was responsible for almost two-thirds (65.9%) of statewide releases of mental and behavioral system toxicants. Volatile organic compounds as a group ranked second (4.9 million pounds or 26.3%), followed by formaldehyde (1.3 million pounds or 7%). These three chemicals accounted for 99.96% of the state total.

Table 3.5a.

#### F00-F99: Mental and Behavioral Disorders by Chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	65.93
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	26.25
3	Formaldehyde	18	14	6	561,144	187,048	1,309,336	6.98
4	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.35
5	Benzene	16	13	6	9,103	3,034	21,241	0.11
6	Methanol	8	7	6	8,286	2,762	19,333	0.10
7	Toluene	16	13	6	8,275	2,758	19,308	0.10
8	Hexane	13	10	6	5,222	1,741	12,184	0.06
9	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.04
10	Ethyl Benzene	15	13	6	1,198	399	2,794	0.01
		18	14	6	8,030,865	2,676,955	18,738,685	99.96

### 3.5b. Releases by ICD Category

Mental and behavioral disorders are divided into 11 major groups.

Chemicals released by natural gas compressor stations are positively associated with 8 of them. It should be remembered that a single chemical can be associated with more than one disease group.

**F00-F09:** Ten chemicals are associated with organic, including symptomatic, mental disorders. Specific effects associated with these eight chemicals include euphoria, hallucinations, mood disturbance, and personality changes. These chemicals were released by all 18 facilities.

**F20-F29:** Two chemicals, carbon monoxide and mercury, are associated with schizophrenia, schizotypal and delusional disorders, including delusions and psychosis (manic depressive). These chemicals were released by all 18 facilities.

**F30-F39:** Thirteen chemicals are associated with mood (affective) disorders. Excitement and depression are the two specific effects found in the peer-reviewed literature. These chemicals were released by all 18 facilities.

**F40-F48:** Twelve chemicals are associated with neurotic, stress-related and somatoform disorders, including anxiety, incoordination, panic attacks and stupor. These chemicals were released by all 18 facilities.

**F50-F59:** Nineteen chemicals are connected to behavioral syndromes associated with physiological disturbances and physical factors, specifically, aimless wandering behavior, anorexia (loss of appetite), mental alertness and unspecified behavioral effects. These chemicals were released by all 18 facilities.

**F60-F69:** Two chemicals are associated with disorders of adult personality and behavior, specifically, aggression. Releases were reported by all 18 compressor stations.

**F80-F89:** Seven chemicals are connected to disorders of adult personality and behavior, including learning ability, decrease in manual dexterity and reduced cognitive capacity. These chemicals were released by all 18 facilities.

**F99:** Seven chemicals are associated with unspecified mental disorders.

Table 3.5b.

## Mental and Behavioral Disorders by ICD Category

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	F00-F09	Organic, including symptomatic, mental disorders	18	18	17	18	10	10	10	10	1,418,739	2,038,854	1,856,547	5,314,142
2	F10-F19	Mental and behavioral disorders due to psychoactive substance use	0	0	0	0	0	0	0	0	0	0	0	0
3	F20-F29	Schizophrenia, schizotypal and delusional disorders	18	18	17	18	2	2	2	2	1,416,012	2,030,636	1,850,408	5,297,057
4	F30-F39	Mood (affective) disorders	18	18	17	18	13	13	13	13	1,530,052	2,267,329	2,075,954	5,873,337
5	F40-F48	Neurotic, stress-related and somatoform disorders	18	18	17	18	12	12	12	12	1,792,142	2,868,481	2,758,797	7,419,420
6	F50-F59	Behavioral syndromes associated with physiological disturbances and physical factors	18	18	16	18	19	19	19	19	119,618	255,264	237,598	612,481
7	F60-F69	Disorders of adult personality and behavior	18	18	17	18	2	2	2	2	1,526,329	2,260,511	2,071,330	5,858,171
8	F70-F79	Mental retardation	0	0	0	0	0	0	0	0	0	0	0	0
9	F80-F89	Disorders of psychological development	18	18	17	18	7	7	7	7	377,778	839,518	909,277	2,126,574
10	F90-F98	Behavioral and emotional disorders with onset usually occurring in childhood and adolescence	0	0	0	0	0	0	0	0	0	0	0	0
11	F99	Unspecified mental disorder	18	18	17	18	7	7	7	7	377,778	839,518	909,277	2,126,574
	<b>F00-F99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>34</b>	<b>34</b>	<b>34</b>	<b>34</b>	<b>1,913,000</b>	<b>3,124,461</b>	<b>2,996,664</b>	<b>8,034,126</b>

### 3.5c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releasing chemicals associated with mental and behavioral disorders,

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 4.1 million pounds (22%), followed by Compressor Station 249 in Carlisle (2.1 million pounds or 11.1%) and Compressor 241 in LaFayette (2 million pounds or 10.3%). These three sites were responsible for 43% of all statewide releases.

The top 5 facilities were responsible for 58% of the total.

The facility average was 1,048,493 pounds.

Table 3.5c

#### Mental and Behavioral Disorders by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	27	27	16	580,710	4,064,973	21.54
2	TGPC CS 249	Carlisle	Schoharie	27	17	17	299,178	2,094,247	11.10
3	TGPC CS 241	LaFayette	Onondaga	26	19	26	278,597	1,950,179	10.33
4	TGPC 229 & TEG DF	Eden	Erie	24	23	24	222,733	1,559,128	8.26
5	TGPC CS 237	Manchester, Phelps	Ontario	4	3	3	195,395	1,367,764	7.25
6	TGPC CS 254	Chatham	Columbia	16	10	4	161,398	1,129,784	5.99
7	AGT Stony Point CS	Stony Point	Rockland	28	14	15	154,888	1,084,216	5.74
8	NFGSC Independ. CS	Andover	Allegany	10	5	12	147,748	1,034,239	5.48
9	NFGSC Beech Hill CS	Willing	Allegany	13	13	14	140,703	984,922	5.22
10	NFGSC Concord CS	Concord	Erie	6	5	7	133,625	935,372	4.96
11	TGPC CS 224	Clymer	Chautauqua	27	25	27	105,039	735,270	3.90
12	DTI Woodhull Station	Woodhull	Steuben	20	28	30	86,571	605,996	3.21
13	AGT SOUTHEAST CS	Southeast	Putnam	16	11	19	62,261	435,829	2.31
14	NFGSC Nashville CS	Hanover	Chautauqua	16	16	0	54,249	379,740	2.01
15	TGPC CS 230-C	Lockport	Niagara	16	15	16	22,498	157,487	0.83
16	DTI Utica Station	Frankfort	Herkimer	16	18	30	22,243	155,702	0.83
17	DTI Borger CS	Ithaca	Tompkins	20	20	12	21,652	151,563	0.80
18	TGPC CS 233	York	Livingston	16	10	2	6,638	46,464	0.25
				34	34	34	2,696,125	18,872,875	100%

Table 3.5c.2.

## Mental and Behavioral Disorders as a % of Each Station's Total Releases

## NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	Identification	Location		Pounds			Percent	
	Facility Name (Short)	Town	County	Total	Non-F00-F99	F00-F99	Non-F00-F99	F00-F99
1	DTI Woodhull Station	Woodhull	Steuben	829,223	559,019	270,204	67.4	32.6
2	AGT Stony Point CS	Stony Point	Rockland	2,013,478	1,376,893	636,585	68.4	31.6
3	TGPC CS 241	LaFayette	Onondaga	3,039,661	2,165,099	874,563	71.2	28.8
4	NFGSC Independence CS	Andover	Allegany	1,353,931	979,444	374,488	72.3	27.7
5	DTI Utica Station	Frankfort	Herkimer	281,369	207,160	74,209	73.6	26.4
6	TGPC CS 237	Manchester, Phelps	Ontario	2,298,394	1,763,365	535,029	76.7	23.3
7	TGPC CS 249	Carlisle	Schoharie	4,323,285	3,364,313	958,972	77.8	22.2
8	TGPC CS 224	Clymer	Chautauqua	1,146,797	908,779	238,018	79.2	20.8
9	NFGSC Nashville CS	Hanover	Chautauqua	622,791	497,159	125,632	79.8	20.2
10	AGT SOUTHEAST CS	Southeast	Putnam	1,688,815	1,351,650	337,165	80.0	20.0
11	NFGSC Concord CS	Concord	Erie	1,733,171	1,449,586	283,585	83.6	16.4
12	NFGSC Beech Hill CS	Willing	Allegany	1,387,592	1,186,796	200,796	85.5	14.5
13	TGPC 229 & TEG DF	Eden	Erie	5,124,427	4,496,586	627,840	87.7	12.3
14	TGPC CS 245	Winfield	Herkimer	10,465,389	9,210,518	1,254,871	88.0	12.0
15	TGPC CS 254	Chatham	Columbia	2,393,661	2,110,010	283,651	88.1	11.9
16	TGPC CS 230-C	Lockport	Niagara	485,610	447,581	38,028	92.2	7.8
17	DTI Borger CS	Ithaca	Tompkins	780,159	742,683	37,476	95.2	4.8
18	TGPC CS 233	York	Livingston	224,978	216,865	8,114	96.4	3.6
				<b>40,192,733</b>	<b>33,033,504</b>	<b>7,159,229</b>	<b>82.2</b>	<b>17.8</b>

### 3.5d. Releases by DEC Regions

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of chemicals associated with mental and behavioral disorders.

DEC Region 9, Western New York, ranked first with 5.7 million pounds or slightly less than one-third of total releases from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, second with 4.2 million pounds (22.5%), followed by Region 4, Capital Region/Northern Catskills, 3.2 million pounds (17.2%).

Table 3.5d.

#### Mental and Behavioral Disorders by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	NYS DEC Region	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	4	2	15	865,355	288,452	2,019,161	10.77
		Chautauqua	9	2	33	423,613	141,204	988,430	5.27
		Erie	2	2	27	1,069,071	356,357	2,494,500	13.31
		Niagara	12	1	16	67,494	22,498	157,487	0.84
				7	34	2,425,533	808,511	5,659,578	30.19
2	6: W. Adirondacks/E Lake Ontario	Herkimer	1	2	33	1,808,861	602,954	4,220,675	22.51
3	4: Capital Region/N. Catskills	Columbia	7	1	16	484,193	161,398	1,129,784	6.03
		Schoharie	3	1	27	897,534	299,178	2,094,247	11.17
				2	31	1,381,727	460,576	3,224,031	17.20
4	7: Central New York	Onondaga	5	1	26	835,791	278,597	1,950,179	10.40
		Tompkins	13	1	20	64,956	21,652	151,563	0.81
				2	34	900,747	300,249	2,101,742	11.21
5	8: Western Finger Lakes	Livingston	14	1	16	19,913	6,638	46,464	0.25
		Ontario	6	1	4	586,185	195,395	1,367,764	7.30
		Steuben	10	1	31	259,713	86,571	605,996	3.23
				3	31	865,810	288,603	2,020,224	10.78
6	3: Lower Hudson Valley	Putnam	11	1	21	186,784	62,261	435,829	2.32
		Rockland	8	1	28	464,664	154,888	1,084,216	5.78
				2	32	651,448	217,149	1,520,044	8.11
				18	34	8,034,126	2,678,042	18,746,295	100%

### 3.5e. Releases by County

All fourteen counties where compressor stations are located reported releases of chemicals linked to mental and behavioral disorders.

Herkimer County ranked first with 4.2 million pounds or 22.5% of the state total, followed by Erie County (2.5 million pounds or 13.3%) and Schoharie County (2 million pounds or 11.2%). These three counties are responsible for nearly one-half (47%) of all releases.

The top five counties were responsible for 12.8 million pounds or slightly more than two-thirds (68.1%) of the state total.

The 14-county average was 1,339,021 pounds.

Table 3.5e.

#### Mental and Behavioral Disorders by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	33	1,808,861	602,954	4,220,675	22.51
2	Erie	9: Western New York	2	27	1,069,071	356,357	2,494,500	13.31
3	Schoharie	4: Capital Region/Northern Catskills	1	27	897,534	299,178	2,094,247	11.17
4	Allegany	9: Western New York	2	15	865,355	288,452	2,019,161	10.77
5	Onondaga	7: Central New York	1	26	835,791	278,597	1,950,179	10.40
6	Ontario	8: Western Finger Lakes	1	4	586,185	195,395	1,367,764	7.30
7	Columbia	4: Capital Region/Northern Catskills	1	16	484,193	161,398	1,129,784	6.03
8	Rockland	3: Lower Hudson Valley	1	28	464,664	154,888	1,084,216	5.78
9	Chautauqua	9: Western New York	2	33	423,613	141,204	988,430	5.27
10	Steuben	8: Western Finger Lakes	1	31	259,713	86,571	605,996	3.23
11	Putnam	3: Lower Hudson Valley	1	21	186,784	62,261	435,829	2.32
12	Niagara	9: Western New York	1	16	67,494	22,498	157,487	0.84
13	Tompkins	7: Central New York	1	20	64,956	21,652	151,563	0.81
14	Livingston	8: Western Finger Lakes	1	16	19,913	6,638	46,464	0.25
			<b>18</b>	<b>34</b>	<b>8,034,126</b>	<b>2,678,042</b>	<b>18,746,295</b>	<b>100%</b>

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## 3.6. Diseases of the Nervous System (G00–G99)

### 3.6a. Releases by Chemical

***Forty-two of the 70 chemicals released by NYS natural gas compressor stations are associated with nervous system disorders (ICD-10, Chapter 6). All 18 stations reported such releases. These totaled an estimated 19 million pounds from 2008 to 2014--an annual average of 2.7 million pounds.***

Chemicals associated with nervous system diseases represent 47.6% of all reported toxic releases from NYS natural gas compressor stations reported to NEI.

Carbon monoxide was responsible for almost two-thirds (65.1%) of statewide releases of nervous system toxicants. Volatile organic compounds as a group ranked second (4.9 million pounds or 26%), followed by formaldehyde (1.3 million pounds or 6.9%). These three chemicals accounted for 97.9% of the state total.

Table 3.6a.

#### Diseases of the Nervous System by Chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	65.08
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	25.91
3	Formaldehyde	18	14	6	561,144	187,048	1,309,336	6.89
4	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.98
5	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.35
6	Acrolein	14	13	6	22,596	7,532	52,723	0.28
7	Benzene	16	13	6	9,103	3,034	21,241	0.11
8	Methanol	8	7	6	8,286	2,762	19,333	0.10
9	Toluene	16	13	6	8,275	2,758	19,308	0.10
10	Hexane	13	10	6	5,222	1,741	12,184	0.06
		18	14	6	8,128,714	2,709,571	18,966,998	99.87

### 3.6b. Releases by ICD Category

Diseases of the nervous system are subdivided into 11 major categories.

Chemicals released by natural gas compressor stations are positively associated with 6 of them. It should be remembered that a single chemical can be associated with more than one disease group.

**G00-G09:** Five chemicals are associated with inflammatory diseases of the central nervous system, specifically, amyotrophic lateral sclerosis. These chemicals were released by all 18 stations.

**G20-G26:** Two chemicals are associated with extrapyramidal and movement disorders, specifically, olfactory nerve changes. These chemicals were released by 8 stations.

**G40-G47:** Ten chemicals are associated with episodic and paroxysmal disorders, specifically, altered sleep time (including change in righting reflex), insomnia, peripheral nerve effects, sleep disorders, and sleepiness. These chemicals were released by 17 stations.

**G60-G64:** Two chemicals are associated with polyneuropathies and other disorders of the peripheral nervous system. These chemicals were released by all 18 stations.

**G80-G83:** One chemical, carbon monoxide, is associated with cerebral palsy and other paralytic syndromes. It is released by all stations.

**G90-G99:** Forty-two chemicals are associated with other disorders of the nervous system.

Table 3.6b.

#### G00-G99: Diseases of the Nervous System by ICD Code Group

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	G00-G09	Inflammatory diseases of the central nervous system	18	18	16	18	5	5	5	5	112,281	231,662	222,434	566,378
2	G10-G13	Systemic atrophies primarily affecting the central nervous system	0	0	0	0	0	0	0	0	0	0	0	0
3	G20-G26	Extrapyramidal and movement disorders	6	4	5	8	2	2	2	2	41	108	70	220
4	G30-G32	Other degenerative diseases of the nervous system	0	0	0	0	0	0	0	0	0	0	0	0
5	G40-G47	Episodic and paroxysmal disorders	17	16	14	17	10	10	10	10	7,391	23,902	15,437	46,732
6	G50-G59	Nerve, nerve root and plexus disorders	0	0	0	0	0	0	0	0	0	0	0	0
7	G60-G64	Polyneuropathies and other disorders of the peripheral nervous sys.	18	17	16	18	2	2	2	2	110,351	229,931	220,960	561,243
8	G70-G73	Diseases of myoneural junction and muscle	0	0	0	0	0	0	0	0	0	0	0	0
9	G80-G83	Cerebral palsy and other paralytic syndromes	18	18	17	18	1	1	1	1	1,415,995	2,030,629	1,850,402	5,297,027
10	G90-G99	Other disorders of the nervous system	18	18	17	18	42	42	42	42	1,924,189	3,151,601	3,063,418	8,139,210
	<b>G00-G99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>42</b>	<b>42</b>	<b>42</b>	<b>42</b>	<b>1,924,189</b>	<b>3,151,601</b>	<b>3,063,418</b>	<b>8,139,210</b>

### 3.6c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releasing chemicals associated with nervous system disorders.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 4.1 million pounds (22%), followed by Compressor Station 249 in Carlisle (2.1 million pounds or 11.1%) and Compressor 241 in LaFayette (2 million pounds or 10.3%). These three sites were responsible for 43% of all statewide releases.

The top 5 facilities were responsible for 58% of the total.

The facility average was 1,062,124 pounds.

Table 3.6c.

#### G00–G99: Diseases of the Nervous System by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	34	34	20	584,742	4,093,196	21.41
2	TGPC CS 249	Carlisle	Schoharie	34	21	21	303,242	2,122,694	11.10
3	TGPC CS 241	LaFayette	Onondaga	33	23	33	282,459	1,977,209	10.34
4	TGPC 229 & TEG DF	Eden	Erie	31	30	31	227,683	1,593,779	8.34
5	TGPC CS 237	Manchester, Phelps	Ontario	5	4	4	195,580	1,369,060	7.16
6	TGPC CS 254	Chatham	Columbia	19	12	5	162,516	1,137,610	5.95
7	AGT Stony Point CS	Stony Point	Rockland	36	17	18	159,633	1,117,432	5.84
8	NFGSC Independence CS	Andover	Allegany	11	6	13	147,851	1,034,958	5.41
9	NFGSC Beech Hill CS	Willing	Allegany	16	16	17	141,024	987,167	5.16
10	NFGSC Concord CS	Concord	Erie	7	6	8	133,787	936,510	4.90
11	TGPC CS 224	Clymer	Chautauqua	34	32	34	106,266	743,864	3.89
12	DTI Woodhull Station	Woodhull	Steuben	24	32	36	87,890	615,229	3.22
13	AGT SOUTHEAST CS	Southeast	Putnam	19	14	24	67,069	469,483	2.46
14	NFGSC Nashville CS	Hanover	Chautauqua	18	18	0	54,319	380,234	1.99
15	TGPC CS 230-C	Lockport	Niagara	19	18	19	25,460	178,217	0.93
16	DTI Utica Station	Frankfort	Herkimer	18	22	35	22,575	158,022	0.83
17	DTI Borger CS	Ithaca	Tompkins	24	24	15	22,343	156,403	0.82
18	TGPC CS 233	York	Livingston	19	12	3	6,737	47,157	0.25
				42	42	42	2,731,176	19,118,224	100%

### 3.6d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of chemicals associated with mental and nervous system disorders.

DEC Region 9, Western New York, ranked first with 5.7 million pounds or slightly less than one-third (30.2%) of total releases from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, second with 4.3 million pounds (22.4%), followed by Region 4, Capital Region/Northern Catskills, 3.3 million pounds (17.2%).

Table 3.6d.

#### G00–G99: Diseases of the Nervous System by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	NYS DEC Region	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	4	2	18	866,625	288,875	2,022,126	10.65
		Chautauqua	9	2	41	427,437	142,479	997,353	5.25
		Erie	2	2	34	1,084,410	361,470	2,530,290	13.32
		Niagara	12	1	19	76,379	25,459	178,217	0.94
				7	42	2,454,851	818,283	5,727,986	30.16
2	6: W Adirondacks/E Lake Ontario	Herkimer	1	2	41	1,821,950	607,316	4,251,218	22.38
3	4: Capital Region/N. Catskills	Columbia	7	1	19	487,547	162,515	1,137,610	5.99
		Schoharie	3	1	34	909,726	303,242	2,122,694	11.18
				2	38	1,397,273	465,757	3,260,304	17.17
4	7: Central New York	Onondaga	5	1	33	847,375	282,458	1,977,209	10.41
		Tompkins	13	1	24	67,030	22,343	156,403	0.82
				2	42	914,405	304,801	2,133,612	11.23
5	8: Western Finger Lakes	Livingston	14	1	19	20,210	6,736	47,157	0.25
		Ontario	6	1	5	586,740	195,580	1,369,060	7.21
		Steuben	10	1	37	263,669	87,889	615,229	3.24
				3	37	870,619	290,205	2,031,446	10.70
6	3: Lower Hudson Valley	Putnam	11	1	26	201,207	67,069	469,483	2.47
		Rockland	8	1	36	478,899	159,633	1,117,432	5.88
				2	39	680,106	226,702	1,586,915	8.36
				18	42	8,139,204	2,713,064	18,991,481	100%

### 3.6e. Releases by County

All fourteen counties where compressor stations are located reported releases of chemicals linked to nervous system disorders.

Herkimer County ranked first with 4.3 million pounds or 22.4% of the state total, followed by Erie County (2.5 million pounds or 13.3%) and Schoharie County (2.1 million pounds or 11.2%). These three counties are responsible for nearly one-half (48.9%) of all releases.

The top five counties were responsible for 12.8 million pounds or slightly more than two-thirds (68%) of the state total.

The 14-county average was 1,356,535 pounds.

Table 3.6e.

#### G00–G99: Diseases of the Nervous System by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	41	1,821,951	607,317	4,251,219	22.38
2	Erie	9: Western New York	2	34	1,084,410	361,470	2,530,291	13.32
3	Schoharie	4: Capital Region/Northern Catskills	1	34	909,726	303,242	2,122,695	11.18
4	Allegany	9: Western New York	2	18	866,625	288,875	2,022,126	10.65
5	Onondaga	7: Central New York	1	33	847,376	282,459	1,977,210	10.41
6	Ontario	8: Western Finger Lakes	1	5	586,740	195,580	1,369,061	7.21
7	Columbia	4: Capital Region/Northern Catskills	1	19	487,547	162,516	1,137,610	5.99
8	Rockland	3: Lower Hudson Valley	1	36	478,900	159,633	1,117,433	5.88
9	Chautauqua	9: Western New York	2	41	427,437	142,479	997,354	5.25
10	Steuben	8: Western Finger Lakes	1	37	263,670	87,890	615,230	3.24
11	Putnam	3: Lower Hudson Valley	1	26	201,207.04	67,069.01	469,483.08	2.47
12	Niagara	9: Western New York	1	19	76,379.14	25,459.71	178,217.98	0.94
13	Tompkins	7: Central New York	1	24	67,030.16	22,343.39	156,403.70	0.82
14	Livingston	8: Western Finger Lakes	1	19	20,210	6,737	47,158	0.25
			<b>18</b>	<b>42</b>	<b>8,139,210</b>	<b>2,713,070</b>	<b>18,991,490</b>	<b>100%</b>

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## 3.7. Diseases of the Eye and Adnexa (H00-H59)

### 3.7a. Releases by Chemical

***Forty-one of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the eye and adnexa (ICD-10, Chapter 7). All 18 stations reported such releases. These totaled an estimated 24.7 million pounds from 2008 to 2014--an annual average of 3.5 million pounds.***

Chemicals associated with eye and adnexa diseases represent 61.8% of all reported toxic releases from NYS natural gas compressor stations reported to NEI.

Nitrogen oxides were responsible for slightly less than three-fourths (73.2%) of statewide releases of chemicals linked to diseases of the eye and adnexa. Volatile organic compounds as a group ranked second (4.9 million pounds or 20%), followed by formaldehyde (1.3 million pounds or 5.3%). These three chemicals accounted for 98% of the state total.

Table 3.7a.

#### H00-H59. Diseases of the Eye and Adnexa by Chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	73.17
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	19.91
3	Formaldehyde	18	14	6	561,144	187,048	1,309,336	5.30
4	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.76
5	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.27
6	Acrolein	14	13	6	22,596	7,532	52,723	0.21
7	Benzene	16	13	6	9,103	3,034	21,241	0.09
8	Methanol	8	7	6	8,286	2,762	19,333	0.08
9	Toluene	16	13	6	8,275	2,758	19,308	0.08
10	Hexane	13	10	6	5,222	1,741	12,184	0.05
		18	14	6	10,581,359	3,527,120	24,689,838	99.90

### 3.7b. Releases by ICD Category

Diseases of the eye and adnexa system are subdivided into 12 major groups. Chemicals released by natural gas compressor stations are positively associated with 8 of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**H00-H06:** Nine chemicals are associated with disorders of eyelid, lacrimal system and orbit. Specific effects include: lacrimation and ptosis. These chemicals were released by all 18 stations.

**H10-H13:** Ten chemicals are associated with conjunctival disorders. Specific effects include: conjunctivitis and conjunctival irritation. These chemicals were released by all 18 stations.

**H15-H19:** Three chemicals are associated with disorders of sclera and cornea. Specific effects include: cornea damage and clouding. These chemicals were released by 13 stations.

**H20-H22:** A single chemical, propylene oxide, is associated with disorders of iris and ciliary body, specifically, iritis. Eight stations reported releases of this chemical.

**H30-H36:** Three chemicals are associated with glaucoma. Eighteen stations reported its release.

**H40-H42:** Two chemicals are associated with glaucoma. Fourteen stations reported its release.

**H53-H54:** Eleven chemicals are associated with visual disturbances and blindness. Eighteen stations reported their release.

**H55-H99:** Thirty-nine chemicals are associated with other disorders of eye and adnexa.

Table 3.7b

#### H00-H59. Diseases of the Eye and Adnexa by Chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	H00-H06	Disorders of eyelid, lacrimal system and orbit	18	17	16	18	9	9	9	9	112,005	235,242	224,320	571,568
2	H10-H13	Disorders of conjunctiva	18	18	17	18	10	10	10	10	487,727	1,070,740	1,130,225	2,688,693
3	H15-H19	Disorders of sclera and cornea	12	12	11	13	3	3	3	3	309	387	311	1,008
4	H20-H22	Disorders of iris & ciliary body	8	5	4	8	1	1	1	1	6	114	141	263
5	H25-H28	Disorders of lens	0	0	0	0	0	0	0	0	0	0	0	0
6	H30-H36	Disorders of choroid & retina	18	17	16	18	3	3	3	3	111,765	234,361	223,601	569,727
7	H40-H42	Glaucoma	14	10	10	14	2	2	2	2	40	117	124	282
8	H43-H45	Disorders of vitreous body and globe	0	0	0	0	0	0	0	0	0	0	0	0
9	H46-H48	Disorders of optic nerve and visual pathways	0	0	0	0	0	0	0	0	0	0	0	0
10	H49-H52	Disorders of ocular muscles, binocular movement, accommodation & refraction	0	0	0	0	0	0	0	0	0	0	0	0
11	H53-H54	Visual disturbances & blindness	18	18	16	18	11	11	11	11	115,165	240,009	228,723	583,898
12	H55-H59	Other disorders of eye and adnexa	18	18	17	18	39	39	39	39	2,777,490	4,113,923	3,700,237	10,591,651
	<b>H00-H59</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>41</b>	<b>41</b>	<b>41</b>	<b>41</b>	<b>2,777,500</b>	<b>4,113,949</b>	<b>3,700,250</b>	<b>10,591,700</b>



### 3.7c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releasing chemicals associated with diseases of the eye and adnexa.

The top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 7 million pounds (28.3%), followed by Compressor Station 229 in Eden (3.7 million pounds or 15%) and Compressor 249 in Carlisle (2.6 million pounds or 10.4%). These three sites were responsible for more than one-half (53.7%) of all statewide releases.

The top 5 facilities were responsible for slightly less than two-thirds (65.4%) of the total.

The facility average was 1,379,496 pounds.

Table 3.7c.

#### H00-H59: Diseases of the Eye and Adnexa by Facility

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	32	32	20	1,001,990	7,013,931	28.25
2	TGPC 229 & TEG DF	Eden	Erie	30	29	30	532,203	3,725,419	15.00
3	TGPC CS 249	Carlisle	Schoharie	32	21	21	368,835	2,581,844	10.40
4	TGPC CS 241	LaFayette	Onondaga	31	23	31	222,022	1,554,152	6.26
5	TGPC CS 254	Chatham	Columbia	19	12	5	194,478	1,361,349	5.48
6	AGT Stony Point CS	Stony Point	Rockland	33	18	18	192,160	1,345,120	5.42
7	AGT SOUTHEAST CS	Southeast	Putnam	19	14	25	183,354	1,283,480	5.17
8	TGPC CS 237	Manchester, Phelps	Ontario	5	4	4	177,838	1,244,864	5.01
9	NFGSC Concord CS	Concord	Erie	7	6	8	128,461	899,225	3.62
10	NFGSC Independ. CS	Andover	Allegany	11	6	13	95,203	666,418	2.68
11	DTI Borger CS	Ithaca	Tompkins	25	25	15	90,898	636,288	2.56
12	TGPC CS 224	Clymer	Chautauqua	32	30	32	84,987	594,909	2.40
13	NFGSC Beech Hill CS	Willing	Allegany	16	16	17	73,519	514,636	2.07
14	DTI Woodhull Station	Woodhull	Steuben	25	32	36	59,457	416,201	1.68
15	NFGSC Nashville CS	Hanover	Chautauqua	19	19	0	50,123	350,859	1.41
16	TGPC CS 230-C	Lockport	Niagara	19	18	19	42,774	299,420	1.21
17	DTI Utica Station	Frankfort	Herkimer	19	23	35	25,770	180,387	0.73
18	TGPC CS 233	York	Livingston	19	12	3	23,203	162,421	0.65
				<b>41</b>	<b>41</b>	<b>41</b>	<b>3,547,275</b>	<b>24,830,922</b>	<b>100%</b>

### 3.7d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of chemicals associated with mental and diseases of the eye and adnexa.

DEC Region 6, Western Adirondacks/Eastern Lake Ontario, ranked first with 7.2 million pounds or 20.3% of total releases from 2008 to 2014. Region 9, Western New York, was a close second with 6.9 million pounds (28.2%), followed by Region 4, Capital Region/Northern Catskills, 3.9 million pounds (16.1%).

Table 3.7d.

#### Diseases of the Eye and Adnexa by DEC Region

##### NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	No. \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	6: W. Adirondacks/E Lake Ontario	Herkimer	1	2	40	3,083,279	1,027,760	7,194,317	29.30
2	9: Western New York	Allegany	9	2	18	506,166	168,722	1,181,054	4.81
		Chautauqua	10	2	40	355,206	118,402	828,815	3.38
		Erie	2	2	33	1,981,990	660,663	4,624,644	18.84
		Niagara	13	1	19	128,323	42,774	299,420	1.22
				7	41	2,971,686	990,562	6,933,933	28.24
3	4: Capital Region/Northern Catskills	Columbia	5	1	19	583,435	194,478	1,361,349	5.54
		Schoharie	3	1	32	1,106,504	368,835	2,581,844	10.52
				2	36	1,689,940	563,313	3,943,192	16.06
4	3: Lower Hudson Valley	Putnam	7	1	27	550,063	183,354	1,283,480	5.23
		Rockland	6	1	34	576,480	192,160	1,345,120	5.48
				2	38	1,126,543	375,514	2,628,600	10.71
5	7: Central New York	Onondaga	4	1	31	666,065	222,022	1,554,152	6.33
		Tompkins	11	1	25	272,695	90,898	636,288	2.59
				2	41	938,760	312,920	2,190,439	8.92
6	8: Western Finger Lakes	Livingston	14	1	19	69,609	23,203	162,421	0.66
		Ontario	8	1	5	533,513	177,838	1,244,864	5.07
		Steuben	12	1	37	178,372	59,457	416,201	1.70
				2	37	711,885	237,295	1,661,065	6.77
				18	41	10,522,092	3,507,364	24,551,547	100%

### 3.7e. Releases by County

All fourteen counties where compressor stations are located reported releases of chemicals linked to diseases of the eye and adnexa.

Herkimer County ranked first with 7.2 million pounds or 29.1% of the state total, followed by Erie County (4.6 million pounds or 18.7%) and Schoharie County (2.6 million pounds or 10.5%). These three counties are responsible for more than one-half (58.3%) of all releases.

The top five counties were responsible for 17.3 million pounds or slightly more than two-thirds (70%) of the state total.

The 14-county average was 1,765,283 pounds.

Table 3.7e.

#### Diseases of the Eye and Adnexa by County

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	40	3,083,279	1,027,760	7,194,317	29.11
2	Erie	9: Western New York	2	33	1,981,990	660,663	4,624,644	18.71
3	Schoharie	4: Capital Region/Northern Catskills	1	32	1,106,504	368,835	2,581,844	10.45
4	Onondaga	7: Central New York	1	31	666,065	222,022	1,554,152	6.29
5	Columbia	4: Capital Region/Northern Catskills	1	19	583,435	194,478	1,361,349	5.51
6	Rockland	3: Lower Hudson Valley	1	34	576,480	192,160	1,345,120	5.44
7	Putnam	3: Lower Hudson Valley	1	27	550,063	183,354	1,283,480	5.19
8	Ontario	8: Western Finger Lakes	1	5	533,513	177,838	1,244,864	5.04
9	Allegany	9: Western New York	2	18	506,166	168,722	1,181,054	4.78
10	Chautauqua	9: Western New York	2	40	355,206	118,402	828,815	3.35
11	Tompkins	7: Central New York	1	25	272,695	90,898	636,288	2.57
12	Steuben	8: Western Finger Lakes	1	37	178,372	59,457	416,201	1.68
13	Niagara	9: Western New York	1	19	128,323	42,774	299,420	1.21
14	Livingston	8: Western Finger Lakes	1	19	69,609	23,203	162,421	0.66
			<b>18</b>	<b>41</b>	<b>10,591,701</b>	<b>3,530,567</b>	<b>24,713,969</b>	<b>100%</b>

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## 3.8. Diseases of the Ear and Mastoid Process (H60-H95)

### 3.8a. Releases by Chemical

Fifteen of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the ear and mastoid process (ICD-10, Chapter 8). All 18 stations reported such releases. These totaled an estimated 17.3 million pounds from 2008 to 2014—an annual average of 2.5 million pounds.

Chemicals associated with diseases of the ear and mastoid process represented 43.5% of all reported releases by natural gas compressor stations.

Two chemicals, carbon monoxide and volatile organic compounds, were responsible for 99.8% of all statewide releases.

Table 3.8a.

#### Diseases of the Ear and Mastoid Process by Chemical (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	71.26
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	28.37
3	Benzene	16	13	6	9,103	3,034	21,241	0.12
4	Toluene	16	13	6	8,275	2,758	19,308	0.11
5	Hexane	13	10	6	5,222	1,741	12,184	0.07
6	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.05
7	Ethyl Benzene	15	13	6	1,198	399	2,794	0.02
8	Methylene Chloride	8	7	6	269	90	629	0.00
9	Manganese	9	9	6	150	50	350	0.00
10	Styrene	8	7	6	100	33	234	0.00
11	Vinyl Chloride	8	7	6	46	15	107	0.00
12	Mercury	16	13	6	30	10	70	0.00
13	Cadmium	9	9	6	13	4	30	0.00
14	Lead	16	12	6	0	0	1	0.00
15	Cobalt	6	6	5	0	0	0	0.00
		18	14	6	7,433,772	2,477,924	17,345,468	100%



### 3.8b. Releases by ICD Category

Diseases of the ear and mastoid process are subdivided into 4 major groups. Chemicals released by natural gas compressor stations are positively associated with two of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**H80-H83:** Fifteen chemicals are associated with diseases of inner ear, including, change in cochlear structure or function, hearing deficits and hearing disturbance. These chemicals were released by all 18 stations.

**H90-H95:** These fifteen all had effects broadly characterized as other disorders of ear, characterized as changes in hearing acuity, hearing loss, and ototoxicity.

Table 3.8b.

#### Diseases of the Ear and Mastoid Process by ICD Code Group

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	H60-H62	Diseases of external ear	0	0	0	0	0	0	0	0	0	0	0	0
2	H65-H75	Diseases of middle ear and mastoid	0	0	0	0	0	0	0	0	0	0	0	0
3	H80-H83	Diseases of inner ear	16	15	13	16	5	5	5	5	1,810	5,743	5,661	13,215
4	H90-H95	Other disorders of ear	18	18	17	18	15	15	15	15	1,796,211	2,874,068	2,763,491	7,433,772
	H60-H95	Total	18	18	17	18	15	15	15	15	1,796,211	2,874,068	2,763,491	7,433,772

### 3.8c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releasing chemicals associated with diseases of the ear and mastoid process.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

TGPC's Compressor Station 245 in Winfield ranked first with 3.8 million pounds (22%), followed by Compressor Station 249 in Carlisle (1.9 million pounds or 11.1%) and Compressor 241 in Lafayette (1.8 million pounds or 10.1%). These three sites were responsible for 43.1% of all statewide releases.

The top 5 facilities were responsible for more than one-half (58.4%) of the total.

The facility average was 970,115 pounds.

Table 3.8c.

#### Diseases of the Ear and Mastoid Process by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	12	12	9	548,196	3,837,375	21.98
2	TGPC CS 249	Carlisle	Schoharie	12	9	9	276,272	1,933,902	11.07
3	TGPC CS 241	LaFayette	Onondaga	11	10	11	251,763	1,762,343	10.09
4	TGPC 229 & TEG DF	Eden	Erie	11	10	11	201,990	1,413,927	8.10
5	TGPC CS 237	Manchester, Phelps	Ontario	3	2	2	178,687	1,250,808	7.16
6	TGPC CS 254	Chatham	Columbia	10	6	2	157,064	1,099,446	6.30
7	AGT Stony Point CS	Stony Point	Rockland	14	8	9	144,424	1,010,968	5.79
8	NFGSC Independence CS	Andover	Allegany	7	3	9	138,539	969,770	5.55
9	NFGSC Beech Hill CS	Willing	Allegany	8	8	9	133,537	934,759	5.35
10	NFGSC Concord CS	Concord	Erie	4	4	4	115,615	809,305	4.63
11	TGPC CS 224	Clymer	Chautauqua	12	10	12	93,625	655,373	3.75
12	DTI Woodhull Station	Woodhull	Steuben	12	14	15	77,004	539,026	3.09
13	AGT SOUTHEAST CS	Southeast	Putnam	10	7	12	57,674	403,718	2.31
14	NFGSC Nashville CS	Hanover	Chautauqua	10	10	0	49,975	349,825	2.00
15	TGPC CS 230-C	Lockport	Niagara	10	9	10	22,205	155,432	0.89
16	DTI Borger CS	Ithaca	Tompkins	12	12	8	21,502	150,516	0.86
17	DTI Utica Station	Frankfort	Herkimer	10	11	15	20,007	140,050	0.80
18	TGPC CS 233	York	Livingston	10	6	2	6,505	45,534	0.26
				15	15	15	2,494,582	17,462,077	100%



### 3.8d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of chemicals associated with mental and diseases of the eye and adnexa.

Region 9, Western New York, ranked first with 5.2 million pounds or 29.9% of total releases from 2008 to 2014. DEC Region 6, Western Adirondacks/Eastern Lake Ontario, was second with 4 million pounds (23%), followed by Region 4, Capital Region/Northern Catskills, 3 million pounds (17.5%).

Table 3.8d.

#### Diseases of the Ear and Mastoid Process by DEC Region (ranked)

#### NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	NYS DEC Region	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	4	2	9	816,226	272,075	1,904,528	10.98
		Chautauqua	9	2	15	380,824	126,941	888,589	5.12
		Erie	2	2	12	952,814	317,605	2,223,232	12.82
		Niagara	12	1	10	66,614	22,205	155,432	0.90
				7	15	2,216,478	738,826	5,171,782	29.82
2	6: W. Adirondacks/E. Lake Ontario	Herkimer	1	2	15	1,704,611	568,204	3,977,425	22.93
3	4: Capital Region/N. Catskills	Columbia	7	1	10	471,191	157,064	1,099,446	6.34
		Schoharie	3	1	12	828,815	276,272	1,933,902	11.15
				2	14	1,300,006	433,335	3,033,348	17.49
4	7: Central New York	Onondaga	5	1	11	755,290	251,763	1,762,343	10.16
		Tompkins	13	1	12	64,507	21,502	150,516	0.87
				2	15	819,797	273,266	1,912,860	11.03
5	8: Western Finger Lakes	Livingston	14	1	10	19,515	6,505	45,534	0.26
		Ontario	6	1	3	536,060	178,687	1,250,808	7.21
		Steuben	10	1	15	231,011	77,004	539,026	3.11
				3	15	786,586	262,195	1,835,368	10.58
6	3: Lower Hudson Valley	Putnam	11	1	12	173,022	57,674	403,718	2.33
		Rockland	8	1	14	433,272	144,424	1,010,968	5.83
				2	15	606,294	202,098	1,414,686	8.16
				18	15	7,433,772	2,477,924	17,345,468	100%

### 3.8e. Releases by County

All fourteen counties where compressor stations are located reported releases of chemicals linked to diseases of the ear and mastoid process.

Herkimer County ranked first with 4 million pounds or 23% of the state total, followed by Erie County (2.2 million pounds or 12.8%) and Schoharie County (2 million pounds or 11.2%). These three counties are responsible for slightly less than one-half (47%) of all releases.

The top five counties were responsible for 11.8 million pounds or slightly more than two-thirds (68%) of the state total.

The 14-county average was 1,238,962 pounds.

Table 3.8e.

#### Diseases of the Ear and Mastoid Process by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	15	1,704,611	568,204	3,977,424	22.93
2	Erie	9: Western New York	2	12	952,814	317,605	2,223,231	12.82
3	Schoharie	4: Capital Region/Northern Catskills	1	12	828,815	276,272	1,933,901	11.15
4	Allegany	9: Western New York	2	9	816,226	272,075	1,904,528	10.98
5	Onondaga	7: Central New York	1	11	755,290	251,763	1,762,343	10.16
6	Ontario	8: Western Finger Lakes	1	3	536,060	178,687	1,250,807	7.21
7	Columbia	4: Capital Region/Northern Catskills	1	10	471,191	157,064	1,099,446	6.34
8	Rockland	3: Lower Hudson Valley	1	14	433,272	144,424	1,010,967	5.83
9	Chautauqua	9: Western New York	2	15	380,824	126,941	888,589	5.12
10	Steuben	8: Western Finger Lakes	1	15	231,011	77,004	539,026	3.11
11	Putnam	3: Lower Hudson Valley	1	12	173,022	57,674	403,718	2.33
12	Niagara	9: Western New York	1	10	66,614	22,205	155,432	0.90
13	Tompkins	7: Central New York	1	12	64,507	21,502	150,516	0.87
14	Livingston	8: Western Finger Lakes	1	10	19,515	6,505	45,534	0.26
			<b>18</b>	<b>15</b>	<b>7,433,772</b>	<b>2,477,924</b>	<b>17,345,462</b>	<b>100%</b>

## 3.9. Diseases of the Circulatory System (I00-I99)

### 3.9a. Releases by Chemical

Thirty-one of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the circulatory system (ICD-10 Chapter 9). All 18 stations reported such releases. These totaled an estimated 16.2 million pounds from 2008 to 2014--an annual average of 2.3 million pounds.

Eighteen of these 31 chemicals are categorized as known human circulatory system toxicants by U.S. ATSDR.

Carbon monoxide ranked first accounting for slightly more than three-fourths (76.5%) of the state total or 12.4 million pounds. Formaldehyde ranked second (1,309,335 pounds or 8.1%), closely followed by PM10 (1,259,744 pounds or 7.8%).

The average annual release was 5.6 million pounds.

Chemicals associated with circulatory system diseases represented 40.4% of releases by the state's natural gas compressor stations.

Table 3.9a.

#### Diseases of the Circulatory System by Chemical (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	76.53
2	Formaldehyde	18	14	6	561,144	187,048	1,309,336	8.11
3	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	7.80
4	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	6.85
5	Acrolein	14	13	6	22,596	7,532	52,723	0.33
6	Methanol	8	7	6	8,286	2,762	19,333	0.12
7	Toluene	16	13	6	8,275	2,758	19,308	0.12
8	Hexane	13	10	6	5,222	1,741	12,184	0.08
9	1,3-Butadiene	14	13	6	2,022	674	4,719	0.03
10	Ammonia	8	7	5	674	225	1,573	0.01
		18	14	6	6,919,221	2,306,407	16,144,849	99.97

### 3.9b. Releases by ICD Category

Circulatory system diseases are subdivided into 10 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with four of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**I10-I15:** One chemical released by 14 of the 18 compressor stations reporting to NEI has been associated with hypertensive disease.

**I10-I15:** One chemical released by 16 of the 18 compressor stations reporting to NEI has been associated with chronic rheumatic heart diseases.

**I30-I52:** Sixteen chemicals are associated with other forms of heart disease. Specific diseases cite in the literature include: cardiac arrhythmia, heart weight change, increased cardiovascular mortality, and acute pulmonary edema. These chemicals were in the emission inventories of all 18 NYS compressor stations reporting to NEI and totaled 1.6 million pounds.

**I70-I79:** Six chemicals are associated with diseases of arteries, arterioles and capillaries: blood vessels changes and regional, general arteriolar or venous dilation. All 18 stations reported release of these chemicals. Aggregate releases totaled 569,641 pounds.

**I95-I99:** Twenty-two chemicals are associated with other and unspecified disorders of the circulatory system. These totaled 5.3 million pounds.

Table 3.9b.

#### Diseases of the Circulatory System by ICD Code Group

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	I00-I02	Acute rheumatic fever	0	0	0	0	0	0	0	0	0	0	0	0
2	I05-I09	Chronic rheumatic heart diseases	13	12	12	14	1	1	1	1	273	998	750	2,022
3	I10-I15	Hypertensive diseases	16	8	11	16	1	1	1	1	0	0	0	0
4	I20-I25	Ischemic heart diseases	0	0	0	0	0	0	0	0	0	0	0	0
5	I26-I28	Pulmonary heart disease and diseases of pulmonary circulation	0	0	0	0	0	0	0	0	0	0	0	0
6	I30-I52	Other forms of heart disease	18	18	16	18	16	16	16	16	312,721	697,573	575,238	1,585,533
7	I60-I69	Cerebrovascular diseases	0	0	0	0	0	0	0	0	0	0	0	0
8	I70-I79	Diseases of arteries, arterioles and capillaries	18	17	16	18	6	6	6	6	111,642	233,625	224,373	569,641
9	I80-I89	Diseases of veins, lymphatic vessels and lymph nodes, not elsewhere classified	0	0	0	0	0	0	0	0	0	0	0	0
10	I95-I99	Other and unspecified disorders of the circulatory system	18	18	17	18	22	22	22	22	1,424,545	2,053,712	1,866,817	5,345,075
	I00-I99	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>31</b>	<b>31</b>	<b>31</b>	<b>31</b>	<b>1,735,766</b>	<b>2,747,361</b>	<b>2,438,330</b>	<b>6,921,459</b>

### 3.9c. Releases by Facility

All 18 natural gas compressor stations in NYS reported releases chemicals associated with circulatory system disease.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 3.6 million pounds or 22% of the state total, followed by Compressor Station 249 in Carlisle (1.8 million pounds or 11.1%) and Compressor Station 241 in LaFayette (1.6 million pounds or 9.9%). These three facilities were responsible for 43% of the state total.

The top 5 facilities were responsible for 59% of all releases.

The facility average was 902,768 pounds.

Table 3.9c.

#### Diseases of the Circulatory System by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	23	23	15	514,643	3,602,504	22.17
2	TGPC CS 249	Carlisle	Schoharie	23	16	16	258,460	1,809,220	11.13
3	TGPC CS 241	LaFayette	Onondaga	22	17	22	229,876	1,609,133	9.90
4	TGPC 229 & TEG DF	Eden	Erie	20	19	20	211,102	1,477,716	9.09
5	TGPC CS 237	Manchester, Phelps	Ontario	5	4	2	161,984	1,133,891	6.98
6	TGPC CS 254	Chatham	Columbia	15	8	5	147,900	1,035,300	6.37
7	NFGSC Concord CS	Concord	Erie	7	6	8	133,132	931,923	5.73
8	NFGSC Beech Hill CS	Willing	Allegany	12	12	13	129,878	909,148	5.59
9	NFGSC Independence CS	Andover	Allegany	9	6	10	106,814	747,699	4.60
10	AGT Stony Point CS	Stony Point	Rockland	26	13	14	102,540	717,779	4.42
11	TGPC CS 224	Clymer	Chautauqua	23	21	23	88,805	621,634	3.83
12	DTI Woodhull Station	Woodhull	Steuben	19	25	26	67,558	472,907	2.91
13	AGT SOUTHEAST CS	Southeast	Putnam	15	10	18	55,141	385,987	2.38
14	NFGSC Nashville CS	Hanover	Chautauqua	16	16	0	42,750	299,248	1.84
15	TGPC CS 230-C	Lockport	Niagara	15	14	15	25,902	181,314	1.12
16	DTI Borger CS	Ithaca	Tompkins	19	19	11	20,045	140,315	0.86
17	DTI Utica Station	Frankfort	Herkimer	16	18	26	16,299	114,095	0.70
18	TGPC CS 233	York	Livingston	15	8	1	8,572	60,007	0.37
				<b>31</b>	<b>31</b>	<b>31</b>	<b>2,321,403</b>	<b>16,249,821</b>	<b>100%</b>

### 3.9d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York's 9 DEC regions. All 6 regions had releases of chemicals associated with circulatory diseases.

DEC Region 9, Western New York, ranked first with an estimated 5.1 million pounds of toxic releases from 2008 to 2014. This represented nearly one-third (31.4%) of the state total. Region 6, Western Adirondacks/Eastern Lake Ontario, ranked second (3.7 million pounds or 23%), followed by Region 4, Capital Region/Northern Catskills (2.8 million pounds or 17.6%).

Table 3.9d.

#### Diseases of the Circulatory System by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	4	2	14	710,077	236,692	1,656,847	10.26
		Chautauqua	8	2	30	351,913	117,304	821,132	5.08
		Erie	2	2	23	1,032,702	344,234	2,409,639	14.92
		Niagara	12	1	15	77,705	25,901	181,313	1.12
				7	31	2,172,397	724,131	5,068,931	31.39
2	6: W. Adirondacks/E. Lake Ontario	Herkimer	1	2	30	1,592,828	530,942	3,716,599	23.01
3	4: Capital Region/N. Catskills	Columbia	7	1	15	443,699	147,899	1,035,299	6.41
		Schoharie	3	1	23	775,379	258,459	1,809,219	11.20
				2	27	1,219,078	406,358	2,844,518	17.61
4	7: Central New York	Onondaga	5	1	22	689,628	229,876	1,609,132	9.96
		Tompkins	13	1	19	60,135	20,045	140,315	0.87
				2	31	749,763	249,921	1,749,447	10.83
5	8: Western Finger Lakes	Livingston	14	1	15	25,717	8,572	60,006	0.37
		Ontario	6	1	5	485,953	161,984	1,133,891	7.02
		Steuben	10	1	27	202,674	67,558	472,907	2.93
				3	27	714,344	238,114	1,666,804	10.32
6	3: Lower Hudson Valley	Putnam	11	1	20	165,423	55,141	385,987	2.39
		Rockland	9	1	26	307,619	102,539	717,779	4.44
				2	29	473,042	157,680	1,103,766	6.83
				18	31	6,921,452	2,307,146	16,150,065	100%

### 3.9e. Releases by County

All 14 counties where compressor station are located reported releases of chemicals linked to circulatory system diseases.

Herkimer County ranked first with 3.7 million pounds or 23% of the state total, followed by Erie County (2.4 million pounds or 14.9%) and Schoharie (1.8 million pounds or 11.2%). These three counties are responsible for nearly one-half (49.1%) of all toxic releases.

The top five counties were responsible for 69%.

The 14-county average was 1,153,577 pounds.

Table 3.9e.

#### Diseases of the Circulatory System by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	30	1,592,828	530,943	3,716,599	23.01
2	Erie	9: Western New York	2	23	1,032,703	344,234	2,409,640	14.92
3	Schoharie	4: Capital Region/Northern Catskills	1	23	775,380	258,460	1,809,220	11.20
4	Allegany	9: Western New York	2	14	710,077	236,692	1,656,847	10.26
5	Onondaga	7: Central New York	1	22	689,628	229,876	1,609,133	9.96
6	Ontario	8: Western Finger Lakes	1	5	485,953	161,984	1,133,891	7.02
7	Columbia	4: Capital Region/Northern Catskills	1	15	443,700	147,900	1,035,300	6.41
8	Chautauqua	9: Western New York	2	30	351,914	117,305	821,133	5.08
9	Rockland	3: Lower Hudson Valley	1	26	307,620	102,540	717,779	4.44
10	Steuben	8: Western Finger Lakes	1	27	202,675	67,558	472,907	2.93
11	Putnam	3: Lower Hudson Valley	1	20	165,423	55,141	385,987	2.39
12	Niagara	9: Western New York	1	15	77,706	25,902	181,314	1.12
13	Tompkins	7: Central New York	1	19	60,135	20,045	140,315	0.87
14	Livingston	8: Western Finger Lakes	1	15	25,717	8,572	60,007	0.37
			<b>18</b>	<b>31</b>	<b>6,921,459</b>	<b>2,307,153</b>	<b>16,150,072</b>	<b>100%</b>

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## 3.10. Diseases of the Respiratory System (J00-J99)

### 3.10a. Releases by Chemical

***Fifty-one of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the respiratory system (ICD-10 Chapter 10). Releases of respiratory toxicants were reported by all 18 stations and totaled an estimated 39.6 million pounds from 2008 to 2014--an annual average of 5.7 million pounds.***

Chemicals associated with respiratory system diseases represented 98.6% of releases by the state's natural gas compressor stations.

Thirty-five of these 51 chemicals are categorized as known human respiratory toxicants by one or more authoritative sources (U.S. ATSDR, U.S. NIOSH, U.S. OSHA, State of California OEHHA, or the European Union).

Nitrogen oxides ranked first with 18.1 million pounds or 46% of the total, followed by carbon monoxide (12.4 million pounds or 31.3%) and volatile organic compounds (4.9 million pounds or 12.5%). These three chemicals accounted for 35.4 million pounds or 89.7% of all releases.

The top 10 chemicals were responsible for 99.8% of all respiratory toxicants.

Table 3.10a.

#### Diseases of the Respiratory System by Chemical (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Yearly Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	45.85
2	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	31.34
3	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	12.48
4	Formaldehyde	18	14	6	561,144	187,048	1,309,336	3.32
5	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	3.19
6	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	2.80
7	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.47
8	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.17
9	Acrolein	14	13	6	22,596	7,532	52,723	0.13
10	Benzene	16	13	6	9,103	3,034	21,241	0.05
		18	14	6	16,870,580	5,623,527	39,364,686	99.81

### 3.10b. Releases by ICD Category

Respiratory system diseases are subdivided into 10 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with four of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**J30-J39:** Eleven chemicals are associated with other diseases of upper respiratory tract, including: epithelial cell hyperplasia of the larynx, mucous membrane irritation, nasal irritation, nasal lesions, nasal septum deviation and ulceration, perforated septum, pharynx irritation, and throat irritation. These were released by 18 facilities.

**J40-J47:** Twenty-three chemicals are associated with lung diseases due to external agents, including asthma and asthma-like allergy, bronchiolar constriction, bronchitis, bronchospasm, emphysema, and changes in pulmonary vascular resistance.

**J68-J70:** Twenty-five chemicals are associated with lung diseases due to external agents, including sensitization by inhalation, breathing difficulty and irregularities, bronchial irritation and pneumonia, chemical pneumonitis, exacerbation of preexisting breathing problems, pneumonia, and shortness of breath. These were released by 18 facilities.

**J80-J84:** Nine chemicals are associated with other respiratory diseases principally affecting the interstitium were released by all 18 facilities.

**J95-J99:** All fifty-one chemicals are associated with other or unspecified diseases of the respiratory system.

Table 3.10b.

#### Diseases of the Respiratory System by ICD Category (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

ICD-10		Facilities				Chemicals				Pounds			
#	Description	'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	J00-J06 Acute upper respiratory infections	0	0	0	0	0	0	0	0	0	0	0	0
2	J09-J18 Influenza and Pneumonia	0	0	0	0	0	0	0	0	0	0	0	0
3	J20-J22 Other acute lower respiratory infections	0	0	0	0	0	0	0	0	0	0	0	0
4	J30-J39 Other diseases of upper respiratory tract	18	18	17	18	11	11	11	11	2,387,918	3,244,839	2,721,747	8,354,505
5	J40-J47 Chronic lower respiratory disease	18	18	17	18	25	25	25	25	4,386,826	6,587,700	5,886,833	16,861,360
6	J60-J70 Lung diseases due to external agents	18	18	17	18	23	23	23	23	1,546,400	2,309,453	2,153,249	6,009,103
7	J80-J84 Other respiratory diseases principally affecting the interstitium	18	18	17	18	9	9	9	9	2,384,131	3,238,346	2,717,314	8,339,793
8	J85-J86 Suppurative and necrotic conditions of lower respiratory tract	0	0	0	0	0	0	0	0	0	0	0	0
9	J90-J94 Other diseases of pleura	0	0	0	0	0	0	0	0	0	0	0	0
10	J95-J99 Other diseases of the respiratory system	18	18	17	18	51	50	51	51	4,394,088	6,607,931	5,900,863	16,902,883
	<b>J00-J99 Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>51</b>	<b>50</b>	<b>51</b>	<b>51</b>	<b>4,394,088</b>	<b>6,607,931</b>	<b>5,900,863</b>	<b>16,902,883</b>

### 3.10c. Releases by Facility

All natural gas compressor stations in NYS reported releases chemical associated with respiratory system disease.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 10.4 million pounds or 26.2% of the state total. Two other compressor station operated by that company ranked second and third: Compressor Station 229 in Eden (5 million pounds or 12.7%) and Compressor Station 249 in Carlisle (4.2 million pounds or 10.7%). These three facilities were responsible for slightly less than one-half (49.6%) of the state total.

The top 5 facilities were responsible for 63% of all releases.

The facility average was 2.2 million pounds.

Table 3.10c.

#### Diseases of the Respiratory System by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	38	37	23	1,484,234	10,389,639	26.21
2	TGPC 229 & TEG DF	Eden	Erie	37	35	36	721,422	5,049,951	12.74
3	TGPC CS 249	Carlisle	Schoharie	38	24	24	604,524	4,231,665	10.67
4	TGPC CS 241	LaFayette	Onondaga	37	27	36	425,243	2,976,701	7.51
5	TGPC CS 254	Chatham	Columbia	24	15	8	337,862	2,365,031	5.97
6	TGPC CS 237	Manchester, Phelps	Ontario	8	7	5	323,113	2,261,791	5.70
7	AGT Stony Point CS	Stony Point	Rockland	41	21	21	282,934	1,980,537	5.00
8	NFGSC Concord CS	Concord	Erie	10	9	11	243,583	1,705,081	4.30
9	AGT SOUTHEAST CS	Southeast	Putnam	24	17	33	233,542	1,634,796	4.12
10	NFGSC Beech Hill CS	Willing	Allegany	19	19	20	196,224	1,373,569	3.46
11	NFGSC Independence CS	Andover	Allegany	14	9	16	192,806	1,349,642	3.40
12	TGPC CS 224	Clymer	Chautauqua	37	35	37	162,657	1,138,602	2.87
13	DTI Woodhull Station	Woodhull	Steuben	33	40	44	116,642	816,493	2.06
14	DTI Borger CS	Ithaca	Tompkins	33	33	18	110,772	775,401	1.96
15	NFGSC Nashville CS	Hanover	Chautauqua	27	27	0	88,588	620,115	1.56
16	TGPC CS 230-C	Lockport	Niagara	24	23	24	68,325	478,274	1.21
17	DTI Utica Station	Frankfort	Herkimer	27	31	43	39,738	278,165	0.70
18	TGPC CS 233	York	Livingston	24	15	4	31,616	221,312	0.56
				<b>51</b>	<b>50</b>	<b>51</b>	<b>5,663,824</b>	<b>39,646,765</b>	<b>100%</b>

### 3.10d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with respiratory system diseases.

DEC Region 9, Western New York, ranked first with an estimated 29.2 million pounds (29.2%) of respiratory toxicants releases from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, was a close second with 10.7 million pounds (27.05%).

Table 3.10d.

#### Diseases of the Respiratory System by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	21	1,167,090	389,030	2,723,211	6.90
		Chautauqua	10	2	49	665,147	221,715	1,552,011	3.94
		Erie	2	2	40	2,895,013	965,004	6,755,032	17.13
		Niagara	13	1	24	204,974	68,324	478,274	1.21
				7	51	4,932,224	1,644,073	11,508,528	29.18
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	50	4,571,916	1,523,972	10,667,804	27.05
3	4: Capital Region / N. Catskills	Columbia	6	1	24	1,013,584	337,861	2,365,030	6.00
		Schoharie	3	1	38	1,813,570	604,523	4,231,665	10.73
				2	44	2,827,154	942,384	6,596,695	16.73
4	7: Central New York	Onondaga	4	1	37	1,275,728	425,242	2,976,700	7.55
		Tompkins	12	1	33	332,314	110,771	775,401	1.97
				2	51	1,608,042	536,013	3,752,101	9.51
6	3: Lower Hudson Valley	Putnam	9	1	35	700,626	233,542	1,634,795	4.15
		Rockland	8	1	42	848,801	282,933	1,980,536	5.02
				2	48	1,549,427	516,475	3,615,331	9.17
5	8: Western Finger Lakes	Livingston	14	1	24	94,848	31,616	221,312	0.56
		Ontario	7	1	8	969,338	323,112	2,261,790	5.73
		Steuben	11	1	45	349,925	116,641	816,492	2.07
				3	45	1,414,111	471,369	3,299,594	8.37
				18	51	16,902,874	5,634,286	39,440,053	100%

### 3.10e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to respiratory system diseases.

Herkimer County ranked first with 10.7 million pounds or 27% of the state total, followed by Erie County (6.8 million pounds or 17.1%) and Schoharie County (4.2 million pounds or 10.7%). These three counties are responsible for more than one-half (54.9%) of all toxic releases.

The top five counties were responsible for 69%.

Table 3.10e.

#### Diseases of the Respiratory System by County (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	50	4,571,916	1,523,972	10,667,804	27.05
2	Erie	9: Western New York	2	40	2,895,014	965,005	6,755,032	17.13
3	Schoharie	4: Capital Region/Northern Catskills	1	38	1,813,571	604,524	4,231,665	10.73
4	Onondaga	7: Central New York	1	37	1,275,729	425,243	2,976,701	7.55
5	Allegany	9: Western New York	2	21	1,167,091	389,030	2,723,212	6.90
6	Columbia	4: Capital Region/Northern Catskills	1	24	1,013,585	337,862	2,365,031	6.00
7	Ontario	8: Western Finger Lakes	1	8	969,339	323,113	2,261,791	5.73
8	Rockland	3: Lower Hudson Valley	1	42	848,801	282,934	1,980,537	5.02
9	Putnam	3: Lower Hudson Valley	1	35	700,627	233,542	1,634,796	4.15
10	Chautauqua	9: Western New York	2	49	665,148	221,716	1,552,012	3.94
11	Steuben	8: Western Finger Lakes	1	45	349,925	116,642	816,493	2.07
12	Tompkins	7: Central New York	1	33	332,315	110,772	775,401	1.97
13	Niagara	9: Western New York	1	24	204,975	68,325	478,274	1.21
14	Livingston	8: Western Finger Lakes	1	24	94,848	31,616	221,312	0.56
			<b>18</b>	<b>51</b>	<b>16,902,883</b>	<b>5,634,294</b>	<b>39,440,060</b>	<b>100%</b>

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## 3.11. Diseases of the Digestive System (K00-K93)

### 3.11a. Releases by Chemical

Fifty-two of the 70 chemicals released by NYS natural gas compressor stations are associated with digestive diseases (ICD-10 Chapter 11). Releases of digestive system toxicants were reported by all 18 stations and totaled an estimated 38.2 million pounds from 2008 to 2014--an annual average of 5.5 million pounds.

Chemicals associated with digestive system diseases represented 95.7% of releases by the state's natural gas compressor stations.

Nitrogen oxides ranked first with 18.1 million pounds or 47.3% of the total, followed by carbon monoxide (12.4 million pounds or 32.3%) and volatile organic compounds (4.9 million pounds or 12.9%). These three chemicals accounted for 35.4 million pounds or 92.4% of all releases.

The top 10 chemicals were responsible for 99.9% of all digestive toxicants.

The average annual release was 5.5 million pounds.

Table 3.11a.

#### Diseases of the Digestive System by Chemical (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	47.25
2	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	32.30
3	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	12.86
4	Formaldehyde	18	14	6	561,144	187,048	1,309,336	3.42
5	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	3.29
6	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.49
7	Acrolein	14	13	6	22,596	7,532	52,723	0.14
8	Benzene	16	13	6	9,103	3,034	21,241	0.06
9	Methanol	8	7	6	8,286	2,762	19,333	0.05
10	Toluene	16	13	6	8,275	2,758	19,308	0.05
		18	14	6	16,384,783	5,461,594	38,231,160	99.9

### 3.11b. Releases by ICD Category

Digestive system diseases are subdivided into 9 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with 8 of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**K00-K31:** Seven chemicals are associated with diseases of esophagus, stomach and duodenum, including esophagus (change in structure or function of the esophagus, esophageal inflammation and ulceration, gastritis, and stomach bleeding. All 18 facilities reported releases.

**K35-K38:** Two chemicals are associated with diseases of the appendix.

**K50-K52:** Three chemicals are associated with noninfective enteritis and colitis.

**K55-K63:** Three chemicals are associated with other diseases of intestines, including enteric disease and small intestine (ulceration or bleeding).

**K65-K67:** A single chemical is associated with diseases of peritoneum.

**K70-K77:** Twenty-four chemicals are associated with diseases of the liver, including cirrhosis, hepatitis, and liver damage, fatty degeneration, function impairment, injury, swelling, and weight changes.

**K80-K87:** Three chemicals are associated with disorders of gallbladder, biliary tract and pancreas.

**K90-K93:** Thirty-eight chemicals are associated with other diseases of the digestive system.

Table 3.11b.

#### K00-K93: Diseases of the Digestive System by ICD Code Group

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10		Facilities				Chemicals				Pounds			
#	Description	'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	K00-K14 Diseases of oral cavity, salivary glands and jaws												
2	K20-K31 Diseases of esophagus, stomach and duodenum	18	18	16	18	7	7	7	7	112,677	234,109	224,355	571,142
3	K35-K38 Diseases of appendix	18	18	17	18	2	2	2	2	2,377,287	3,235,327	2,676,948	8,289,563
4	K50-K52 Noninfective enteritis and colitis	18	18	17	18	3	3	3	3	2,276,943	3,007,254	2,545,586	7,829,785
5	K55-K63 Other diseases of intestines	18	18	17	18	3	3	3	3	1,416,050	2,030,806	1,850,560	5,297,417
6	K65-K67 Diseases of peritoneum	6	4	5	8	1	1	1	1	23	59	37	120
7	K70-K77 Diseases of liver	18	18	17	18	24	24	24	24	488,496	1,073,708	1,133,230	2,695,435
8	K80-K87 Disorders of gallbladder, biliary tract and pancreas	16	15	13	16	3	3	3	3	3,428	8,249	5,811	17,488
9	K90-K93 Other diseases of the digestive system	18	18	17	18	38	38	38	38	127,387	272,114	300,025	699,527
	<b>K00-K93 Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>45</b>	<b>45</b>	<b>45</b>	<b>45</b>	<b>4,297,063</b>	<b>6,371,787</b>	<b>5,731,539</b>	<b>16,400,390</b>



### 3.11c. Releases by Facility

All natural gas compressor stations in NYS reported releases chemical associated with diseases of the digestive system.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 10.2 million pounds or 26.5% of the state total. Two other compressor station operated by that company ranked second and third: Compressor Station 229 in Eden (4.9 million pounds or 12.7%) and Compressor Station 249 in Carlisle (4 million pounds or 10.5%). These three facilities were responsible for slightly less than one-half (49.6%) of the state total.

The top 5 facilities were responsible for 63% of all releases.

The facility average was 2.1 million pounds.

Table 3.11c.

#### K00-K93: Diseases of the Digestive System by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	35	35	21	1,453,762	10,176,331	26.45
2	TGPC 229 & TEG DF	Eden	Erie	34	33	34	698,204	4,887,431	12.70
3	TGPC CS 249	Carlisle	Schoharie	35	22	22	576,081	4,032,565	10.48
4	TGPC CS 241	LaFayette	Onondaga	34	26	34	406,862	2,848,035	7.40
5	TGPC CS 254	Chatham	Columbia	21	13	7	329,499	2,306,496	6.00
6	TGPC CS 237	Manchester, Phelps	Ontario	7	6	5	313,810	2,196,672	5.71
7	AGT Stony Point CS	Stony Point	Rockland	36	19	19	273,624	1,915,369	4.98
8	NFGSC Concord CS	Concord	Erie	9	8	10	234,664	1,642,645	4.27
9	AGT SOUTHEAST CS	Southeast	Putnam	21	15	29	222,553	1,557,873	4.05
10	NFGSC Beech Hill CS	Willing	Allegany	17	17	18	192,242	1,345,695	3.50
11	NFGSC Independence CS	Andover	Allegany	13	8	15	191,487	1,340,411	3.48
12	TGPC CS 224	Clymer	Chautauqua	35	33	35	158,556	1,109,894	2.88
13	DTI Woodhull Station	Woodhull	Steuben	29	37	41	111,868	783,073	2.04
14	DTI Borger CS	Ithaca	Tompkins	29	29	16	110,016	770,114	2.00
15	NFGSC Nashville CS	Hanover	Chautauqua	24	24	0	87,732	614,122	1.60
16	TGPC CS 230-C	Lockport	Niagara	21	20	21	66,076	462,535	1.20
17	DTI Utica Station	Frankfort	Herkimer	24	27	40	38,557	269,902	0.70
18	TGPC CS 233	York	Livingston	21	13	4	30,446	213,124	0.55
				<b>45</b>	<b>45</b>	<b>45</b>	<b>5,496,041</b>	<b>38,472,286</b>	<b>100%</b>

### 3.11d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with respiratory system diseases.

DEC Region 9, Western New York, ranked first with an estimated 11.2 million pounds (29.3%) of digestive toxicants releases from 2008 to 2014. Region 6, Western Adirondacks/Eastern Lake Ontario, was a close second with 10.5 million pounds (27.3%), followed by Region 4, Capital Region/Northern Catskills (6.4 million pounds or 16.5%).

Table 3.11d.

#### K00-K93: Diseases of the Digestive System by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	19	1,151,188	383,729	2,686,106	7.02
		Chautauqua	10	2	44	651,132	217,044	1,519,309	3.97
		Erie	2	2	37	2,798,604	932,868	6,530,076	17.06
		Niagara	13	1	21	198,229	66,076	462,535	1.21
				7	45	4,799,154	1,599,718	11,198,026	29.26
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	44	4,476,957	1,492,319	10,446,232	27.30
3	4: Capital Region / N Catskills	Columbia	6	1	21	988,498	329,499	2,306,496	6.03
		Schoharie	3	1	35	1,728,242	576,081	4,032,565	10.54
				2	39	2,716,740	905,580	6,339,060	16.57
4	7: Central New York	Onondaga	4	1	34	1,220,586	406,862	2,848,035	7.44
		Tompkins	12	1	29	330,049	110,016	770,114	2.01
				2	45	1,550,635	516,878	3,618,149	9.45
6	3: Lower Hudson Valley	Putnam	9	1	31	667,660	222,553	1,557,873	4.07
		Rockland	8	1	37	820,872	273,624	1,915,369	5.01
				2	45	1,488,532	496,177	3,473,242	9.08
5	8: Western Finger Lakes	Livingston	14	1	21	91,339	30,446	213,124	0.56
		Ontario	7	1	7	941,431	313,810	2,196,672	5.74
		Steuben	11	1	42	335,603	111,868	783,073	2.05
				3	42	1,368,372	456,124	3,192,869	8.34
				18	45	16,400,391	5,466,797	38,267,578	100%

### 3.11e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to digestive system diseases.

Herkimer County ranked first with 10.5 million pounds or 27.3% of the state total, followed by Erie County (6.5 million pounds or 17.1%) and Schoharie County (4 million pounds or 10.5%). These three counties are responsible for more than one-half (54.9%) of all toxic releases.

The top five counties were responsible for 69.4%.

The country average was 2.7 million pounds.

Table 3.11e.

#### K00-K93: Diseases of the Digestive System by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	44	4,476,956	1,492,318	10,446,232	27.30
2	Erie	9: Western New York	2	37	2,798,604	932,868	6,530,076	17.06
3	Schoharie	4: Capital Region/Northern Catskills	1	35	1,728,242	576,080	4,032,564	10.54
4	Onondaga	7: Central New York	1	34	1,220,586	406,862	2,848,034	7.44
5	Allegany	9: Western New York	2	19	1,151,188	383,729	2,686,105	7.02
6	Columbia	4: Capital Region/Northern Catskills	1	21	988,498	329,499	2,306,495	6.03
7	Ontario	8: Western Finger Lakes	1	7	941,430	313,810	2,196,671	5.74
8	Rockland	3: Lower Hudson Valley	1	37	820,872	273,624	1,915,369	5.01
9	Putnam	3: Lower Hudson Valley	1	31	667,659	222,553	1,557,872	4.07
10	Chautauqua	9: Western New York	2	44	651,132	217,044	1,519,309	3.97
11	Steuben	8: Western Finger Lakes	1	42	335,602	111,867	783,072	2.05
12	Tompkins	7: Central New York	1	29	330,048	110,016	770,113	2.01
13	Niagara	9: Western New York	1	21	198,229	66,076	462,534	1.21
14	Livingston	8: Western Finger Lakes	1	21	91,338	30,446	213,124	0.56
			<b>18</b>	<b>45</b>	<b>16,400,384</b>	<b>5,466,792</b>	<b>38,267,570</b>	<b>100%</b>

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## 3.12. Diseases of the Skin and Subcutaneous Tissue (L00-L99)

### 3.12a. Releases by Chemical

Forty-eight of the 70 chemicals released by NYS natural gas compressor stations are associated with skin and subcutaneous tissue diseases (ICD-10 Chapter 12). Releases of these toxicants were reported by all 18 stations and totaled an estimated 27.6 million pounds from 2008 to 2014--an annual average of 3.9 million pounds.

Chemicals associated with diseases of the skin and subcutaneous tissue represented 69% of releases by the state's natural gas compressor stations.

Nitrogen oxides ranked first with 18.1 million pounds or slightly less than two-thirds (65.6%) of the total, followed by volatile organic compounds (4.9 million pounds or 17.8%) and formaldehyde (1.3 million pounds or 4.8%). These three chemicals accounted for 24.3 million pounds or 88.2 of all releases.

The top 10 chemicals were responsible for 96.6% of all cutaneous and subcutaneous toxicants.

Table 3.12a.

#### Diseases of the Skin and Subcutaneous Tissue (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	65.56
2	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	17.84
3	Formaldehyde	18	14	6	561,144	187,048	1,309,336	4.75
4	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	4.57
5	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	4.01
6	PM Condensable	18	14	6	231,543	77,181	540,267	1.96
7	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.68
8	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.24
9	Acrolein	14	13	6	22,596	7,532	52,723	0.19
10	Benzene	16	13	6	9,103	3,034	21,241	0.08
		18	14	6	11,805,095	3,935,032	27,545,222	99.87

### 3.12b. Releases by ICD Category

Skin and subcutaneous diseases are subdivided into 8 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with 3 of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**L20-L30:** Seven chemicals are associated with dermatitis and eczema.

**L50-L54:** Five chemicals are associated with urticaria and erythema.

**L80-L99:** Forty-seven chemicals are associated with other disorders of the skin and subcutaneous tissue.

Table 3.12b.

#### L00-L99: Diseases of the Skin and Subcutaneous Tissue by ICD Category

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	L00-L08	Infections of the skin and subcutaneous tissue	0	0	0	0	0	0	0	0	0	0	0	0
2	L10-L14	Bullous disorders	0	0	0	0	0	0	0	0	0	0	0	0
3	L20-L30	Dermatitis and eczema	18	18	17	18	7	7	7	7	489,047	1,076,977	1,132,330	2,698,356
4	L40-L45	Papulosquamous disorders	0	0	0	0	0	0	0	0	0	0	0	0
5	L50-L54	Urticaria and erythema	18	18	17	18	5	5	5	5	376,503	833,995	904,262	2,114,761
6	L55-L59	Radiation-related disorders of the skin and subcutaneous tissue	0	0	0	0	0	0	0	0	0	0	0	0
7	L60-L75	Disorders of skin appendages	0	0	0	0	0	0	0	0	0	0	0	0
8	L80-L99	Other disorders of the skin and subcutaneous tissue	18	18	17	18	45	46	46	47	2,646,996	3,854,819	3,226,706	9,728,523
<b>L00-L99 Total</b>			<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>46</b>	<b>47</b>	<b>47</b>	<b>48</b>	<b>3,021,274</b>	<b>4,686,735</b>	<b>4,129,254</b>	<b>11,837,264</b>

### 3.12c. Releases by Facility

All natural gas compressor stations in NYS reported releases chemical associated with diseases of the skin and subcutaneous tissue diseases.

The top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 7.6 million pounds or 27.2% of the state total, followed by Compressor Station 229 in Eden (4.1 million pounds or 14.8%) and Compressor Station 249 in Carlisle (3.1 million pounds or 11.1%). These three facilities were responsible for slightly less than one-half (53.1%) of the state total.

The top 5 facilities were responsible for slightly less than two-thirds (65.3%) of all releases.

The facility average was 1.5 million pounds.

Table 3.12c.

#### L00-L99: Diseases of the Skin and Subcutaneous Tissue by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	35	35	22	1,079,145	7,554,017	27.23
2	TGPC 229 & TEG DF	Eden	Erie	35	34	35	586,143	4,103,000	14.79
3	TGPC CS 249	Carlisle	Schoharie	35	23	23	438,754	3,071,281	11.07
4	TGPC CS 241	LaFayette	Onondaga	34	26	34	268,300	1,878,097	6.77
5	TGPC CS 254	Chatham	Columbia	22	15	8	215,885	1,511,192	5.45
6	AGT Stony Point CS	Stony Point	Rockland	38	21	21	215,264	1,506,847	5.43
7	AGT SOUTHEAST CS	Southeast	Putnam	22	17	32	214,802	1,503,616	5.42
8	TGPC CS 237	Manchester, Phelps	Ontario	8	7	4	203,966	1,427,759	5.15
9	NFGSC Concord CS	Concord	Erie	10	9	11	150,324	1,052,268	3.79
10	NFGSC Independence CS	Andover	Allegany	14	9	16	98,457	689,200	2.48
11	DTI Borger CS	Ithaca	Tompkins	31	32	18	93,789	656,521	2.37
12	TGPC CS 224	Clymer	Chautauqua	35	33	35	91,319	639,232	2.30
13	NFGSC Beech Hill CS	Willing	Allegany	19	19	20	83,888	587,216	2.12
14	DTI Woodhull Station	Woodhull	Steuben	31	40	44	68,499	479,496	1.73
15	NFGSC Nashville CS	Hanover	Chautauqua	25	24	0	52,218	365,527	1.32
16	TGPC CS 230-C	Lockport	Niagara	22	21	22	48,326	338,285	1.22
17	DTI Utica Station	Frankfort	Herkimer	25	30	43	28,034	196,235	0.71
18	TGPC CS 233	York	Livingston	22	15	3	26,048	182,337	0.66
				<b>46</b>	<b>46</b>	<b>47</b>	<b>3,963,161</b>	<b>27,742,125</b>	<b>100%</b>

### 3.12d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with skin and subcutaneous tissue diseases.

DEC Region 6, Western Adirondacks/Eastern Lake Ontario, ranked first with 7,750,252 pounds (28.1%), closely followed by Region 9, Western New York (7,652,886 pounds or 27.7%). Region 4, Capital Region/Northern Catskills ranked third with 4.6 million pounds (16.6%).

Table 3.12d.

#### L00-L99: Diseases of the Skin and Subcutaneous Tissue by DEC Region (ranked)

##### NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	47	3,321,537	1,107,179	7,750,252	28.06
1	9: Western New York	Allegany	9	2	21	547,036	182,345	1,276,416	4.62
		Chautauqua	10	2	45	378,393	126,131	882,916	3.20
		Erie	2	2	38	2,209,400	736,467	5,155,268	18.66
		Niagara	13	1	22	144,979	48,326	338,285	1.22
				7	46	3,279,808	1,093,269	7,652,886	27.71
3	4: Capital Region / N Catskills	Columbia	5	1	22	647,654	215,885	1,511,192	5.47
		Schoharie	3	1	35	1,316,263	438,754	3,071,281	11.12
				2	39	1,963,917	654,639	4,582,474	16.59
6	3: Lower Hudson Valley	Putnam	7	1	34	644,407	214,802	1,503,616	5.44
		Rockland	6	1	39	645,792	215,264	1,506,847	5.46
				2	45	1,290,198	430,066	3,010,463	10.90
4	7: Central New York	Onondaga	4	1	34	804,899	268,300	1,878,097	6.80
		Tompkins	11	1	33	281,366	93,789	656,521	2.38
				2	48	1,086,265	362,088	2,534,617	9.18
5	8: Western Finger Lakes	Livingston	14	1	22	78,144	26,048	182,337	0.66
		Ontario	8	1	8	611,897	203,966	1,427,759	5.17
		Steuben	12	1	46	205,498	68,499	479,496	1.74
				3	46	895,539	298,513	2,089,592	7.57
				18	48	11,837,264	3,945,755	27,620,283	100%



### 3.12e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to cutaneous and subcutaneous diseases.

Herkimer County ranked first with 7.8 million pounds or 28.1% of the state total, followed by Erie County (5.2 million pounds or 18.7%) and Schoharie County (3.1 million pounds or 11.1%). These three counties are responsible for more than one-half (57.8%) of all toxic releases.

The top five counties were responsible for 70%.

The country average was 2 million pounds.

Table 3.12e

#### Diseases of the Skin and Subcutaneous Tissue by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	47	3,321,537	1,107,179	7,750,252	28.06
2	Erie	9: Western New York	2	38	2,209,400	736,467	5,155,268	18.66
3	Schoharie	4: Capital Region/Northern Catskills	1	35	1,316,263	438,754	3,071,281	11.12
4	Onondaga	7: Central New York	1	34	804,899	268,300	1,878,097	6.80
5	Columbia	4: Capital Region/Northern Catskills	1	22	647,654	215,885	1,511,192	5.47
6	Rockland	3: Lower Hudson Valley	1	39	645,792	215,264	1,506,847	5.46
7	Putnam	3: Lower Hudson Valley	1	34	644,407	214,802	1,503,616	5.44
8	Ontario	8: Western Finger Lakes	1	8	611,897	203,966	1,427,759	5.17
9	Allegany	9: Western New York	2	21	547,036	182,345	1,276,416	4.62
10	Chautauqua	9: Western New York	2	45	378,393	126,131	882,916	3.20
11	Tompkins	7: Central New York	1	33	281,366	93,789	656,521	2.38
12	Steuben	8: Western Finger Lakes	1	46	205,498	68,499	479,496	1.74
13	Niagara	9: Western New York	1	22	144,979	48,326	338,285	1.22
14	Livingston	8: Western Finger Lakes	1	22	78,144	26,048	182,337	0.66
			<b>18</b>	<b>48</b>	<b>11,837,264</b>	<b>3,945,755</b>	<b>27,620,283</b>	<b>100%</b>

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### 3.13. Diseases of the Musculoskeletal System and Connective Tissue (M00-M99)

#### 3.13a. Releases by Chemical

Seventeen of the 70 chemicals released by NYS natural gas compressor stations are associated with musculoskeletal system and connective tissue diseases (ICD-10 Chapter 13). Releases of these toxicants were reported by all 18 stations and totaled an estimated 1.2 million pounds from 2008 to 2014--an annual average of 3.9 million pounds.

Chemicals associated with musculoskeletal system and connective tissue diseases represented 3.1% of releases by the state's natural gas compressor stations.

PM 2.5 ranked first with 1.1 million pounds or 92.6% of the total, followed by benzene (21,241 pounds or 1.8%) and methanol (19,333 pounds or 1.6%). These three chemicals accounted for 96% of all releases.

Table 3.13a.

#### Diseases of the Musculoskeletal System and Connective Tissue by Chemical

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	92.62
2	Benzene	16	13	6	9,103	3,034	21,241	1.78
3	Methanol	8	7	6	8,286	2,762	19,333	1.62
4	Toluene	16	13	6	8,275	2,758	19,308	1.62
5	Hexane	13	10	6	5,222	1,741	12,184	1.02
6	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.70
7	1,3-Butadiene	14	13	6	2,022	674	4,719	0.40
8	Ammonia	8	7	5	674	225	1,573	0.13
9	Propylene Oxide	8	8	5	263	88	615	0.05
10	Carbon Tetrachloride	8	7	6	121	40	282	0.02
11	Chloroform	9	8	6	83	28	193	0.02
12	Chlorobenzene	9	8	6	74	25	172	0.01
13	Mercury	16	13	6	30	10	70	0.01
14	Cadmium	9	9	6	13	4	30	0.00
15	Ethyl Chloride	4	4	4	3	1	6	0.00
16	Lead	16	12	6	0	0	1	0.00
17	Selenium	5	5	5	0	0	0	0.00
		<b>18</b>	<b>14</b>	<b>6</b>	<b>511,850</b>	<b>170,617</b>	<b>1,194,318</b>	<b>100%</b>

### 3.13b. Releases by ICD Category

Musculoskeletal system and connective tissue diseases are subdivided into 7 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with 5 of them. It should be remembered, that a single chemical can be associated with more than one category of disease.

**M00-M25:** A single chemical released by 5 stations is associated with arthropathies, specifically, "Kashin-Beck disease".

**M30-M36:** Three chemicals released by all 18 sites are associated with systemic connective tissue disorders: undifferentiated connective tissue disease (UCTD) and connective tissue disease (CTD).

**M60-M79:** Twelve chemicals released by all 18 sites are associated with soft tissue disorders: muscle contractility, spasticity or weakness.

**M80-M90:** The heavy metal cadmium, released by 9 sites, is associated with osteopathies.

**M95-M99:** Three chemicals are associated with other disorders of the musculoskeletal system and connective tissue

Table 3.13a.

#### Diseases of the Musculoskeletal System and Connective Tissue by ICD Category

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	M00-M25	Arthropathies	4	4	3	5	1	1	1	1	0.0004	0.0017	0.0006	0.0027
2	M30-M36	Systemic connective tissue disorders	18	18	15	18	3	3	3	3	94,984	226,319	165,482	486,785
3	M40-M54	Dorsopathies	0	0	0	0	0	0	0	0	0	0	0	0
4	M60-M79	Soft tissue disorders	18	17	14	18	12	12	12	12	7,236	15,111	11,804	34,152
5	M80-M90	Osteopathies	9	5	4	9	1	1	1	1	8	0	4	13
6	M91-M94	Chondropathies	0	0	0	0	0	0	0	0	0	0	0	0
7	M95-M99	Other disorders of the musculoskeletal system and connective tissue	10	9	8	11	3	3	3	3	19	37	19	76
	<b>M00-M99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>15</b>	<b>18</b>	<b>17</b>	<b>17</b>	<b>17</b>	<b>17</b>	<b>100,200</b>	<b>237,557</b>	<b>174,092</b>	<b>511,850</b>

### 3.13c. Releases by Facility

All natural gas compressor stations in NYS reported releases chemical associated with musculoskeletal system and connective tissue diseases.

The top 5 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 211,513 pounds or 17.2% of the state total, followed by Compressor Station 249 in Carlisle (196,907 pounds or 16%) and Compressor Station 229 in Eden (170,674 pounds or 13.8%). These three facilities were responsible for one-half of the state total.

The top 5 facilities were responsible for slightly less than two-thirds (65.1%) of all releases.

The facility average was 68,510 million pounds over 7 years or 9,787 each year.

Table 3.13c.

#### Diseases of the Musculoskeletal System and Connective Tissue by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	13	13	8	30,216	211,513	17.15
2	TGPC CS 249	Carlisle	Schoharie	13	8	8	28,130	196,907	15.97
3	TGPC 229 & TEG DF	Eden	Erie	11	10	11	24,382	170,674	13.84
4	TGPC CS 241	LaFayette	Onondaga	12	9	12	17,983	125,878	10.21
5	TGPC CS 237	Manchester, Phelps	Ontario	2	1	0	13,956	97,690	7.92
6	AGT SOUTHEAST CS	Southeast	Putnam	9	6	12	11,741	82,189	6.66
7	AGT Stony Point CS	Stony Point	Rockland	14	8	10	11,115	77,803	6.31
8	NFGSC Concord CS	Concord	Erie	3	4	4	8,924	62,465	5.07
9	TGPC CS 254	Chatham	Columbia	9	4	1	8,668	60,676	4.92
10	DTI Woodhull Station	Woodhull	Steuben	12	15	15	5,165	36,154	2.93
11	NFGSC Beech Hill CS	Willing	Allegany	9	9	10	4,093	28,650	2.32
12	TGPC CS 224	Clymer	Chautauqua	13	12	13	3,494	24,460	1.98
13	TGPC CS 230-C	Lockport	Niagara	9	8	9	2,305	16,133	1.31
14	TGPC CS 233	York	Livingston	9	4	0	1,802	12,614	1.02
15	NFGSC Independence CS	Andover	Allegany	7	2	8	1,363	9,540	0.77
16	DTI Utica Station	Frankfort	Herkimer	9	11	15	1,140	7,978	0.65
17	NFGSC Nashville CS	Hanover	Chautauqua	9	9	0	895	6,266	0.51
18	DTI Borger CS	Ithaca	Tompkins	12	12	8	798	5,584	0.45
				<b>17</b>	<b>17</b>	<b>17</b>	<b>176,168</b>	<b>1,233,174</b>	<b>100%</b>

### 3.13d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with musculoskeletal system and connective tissue diseases.

Region 9, Western New York ranked first with 316,000 pounds (25.7%), followed by Region 6, Western Adirondacks/Eastern Lake Ontario (219,490 pounds or 17.8%) and Region 4, Capital Region/Northern Catskills (257,583 or 21%).

Table 3.13d.

#### Diseases of the Musculoskeletal System and Connective Tissue by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	9	2	10	16,367	5,456	38,191	3.10
		Chautauqua	11	2	16	12,273	4,091	28,637	2.33
		Erie	1	2	13	99,917	33,306	233,139	18.94
		Niagara	12	1	9	6,914	2,305	16,133	1.31
				7	17	135,472	45,157	316,100	25.68
2	6: W Adirondacks / E Lake Ontario	Herkimer	2	2	16	94,067	31,356	219,490	17.83
3	4: Capital Region / N Catskills	Columbia	8	1	9	26,004	8,668	60,676	4.93
		Schoharie	3	1	13	84,389	28,130	196,907	15.99
				2	15	110,393	36,798	257,583	20.92
6	3: Lower Hudson Valley	Putnam	6	1	13	35,224	11,741	82,189	6.68
		Rockland	7	1	14	33,344	11,115	77,803	6.32
				2	16	68,568	22,856	159,992	13.00
4	7: Central New York	Onondaga	4	1	12	53,948	17,983	125,878	10.22
		Tompkins	14	1	12	2,393	798	5,584	0.45
				2	17	56,341	18,780	131,462	10.68
5	8: Western Finger Lakes	Livingston	13	1	9	3,604	1,802	12,614	1.02
		Ontario	5	1	2	27,912	13,956	97,690	7.94
		Steuben	10	1	16	15,495	5,165	36,154	2.94
				3	16	47,010	20,923	146,459	11.90
				18	17	511,850	175,869	1,231,086	100%

### 3.13e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to musculoskeletal system and connective tissue diseases.

Erie County ranked first with 233,139 pounds or 19% of the state total, followed by Herkimer County (219,490 pounds or 17.8%) and Schoharie County (196,906 pounds or 16%). These three counties are responsible for slightly more than one-half (53%) of all toxic releases.

The top five counties were responsible for 71%.

The country average was 87,934 pounds over a 7-year period or 12,562 pounds annually.

Table 3.13e.

#### Diseases of the Musculoskeletal System and Connective Tissue by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Erie	9: Western New York	2	13	99,916	33,305	233,139	18.94
2	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	16	94,067	31,355	219,490	17.83
3	Schoharie	4: Capital Region/Northern Catskills	1	13	84,388	28,129	196,906	15.99
4	Onondaga	7: Central New York	1	12	53,947	17,982	125,878	10.23
5	Ontario	8: Western Finger Lakes	1	2	27,911	13,955	97,690	7.94
6	Putnam	3: Lower Hudson Valley	1	13	35,223	11,741	82,188	6.68
7	Rockland	3: Lower Hudson Valley	1	14	33,344	11,114	77,802	6.32
8	Columbia	4: Capital Region/Northern Catskills	1	9	26,003	8,667	60,675	4.93
9	Allegany	9: Western New York	2	10	16,367	5,455	38,190	3.10
10	Steuben	8: Western Finger Lakes	1	16	15,494	5,164	36,154	2.94
11	Chautauqua	9: Western New York	2	16	12,273	4,091	28,637	2.33
12	Niagara	9: Western New York	1	9	6,914	2,304	16,133	1.31
13	Livingston	8: Western Finger Lakes	1	9	3,604	1,802	12,614	1.02
14	Tompkins	7: Central New York	1	12	2,393	797	5,583	0.45
			<b>18</b>	<b>17</b>	<b>511,844</b>	<b>175,861</b>	<b>1,231,079</b>	<b>100%</b>

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## 3.14. Diseases of the Genitourinary System (N00-N99)

### 3.14a. Releases by Chemicals

Forty-three of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of the genitourinary system (ICD-10, Chapter 14).

Releases of genitourinary toxicants were reported by all 18 stations and totaled an estimated 39.7 million pounds from 2008 to 2014--an annual average of 5.7 million pounds.

Chemicals associated with genitourinary system diseases represented 99.4% of releases by the state's natural gas compressor stations.

Or, to put it differently, ***of the 40.2 million pounds of chemicals released by NYS's compressor stations, 98.9% had one or more effects on the genitourinary system.***

Nitrogen oxides ranked first with 18.1 million pounds or nearly one-half (45.5%) of the total, followed by carbon monoxide (12.4 million pounds or 31.1%) and volatile organic compounds (4.9 million pounds or 12.4%). These three chemicals accounted for 35.4 million pounds or 89% of all releases.

The top 10 chemicals accounted for 99.9% of the state total.

Table 3.14a.

#### Diseases of the Genitourinary System (Top 10 Chemicals by Pounds Released)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	45.50
2	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	31.10
3	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	12.38
4	Formaldehyde	18	14	6	561,144	187,048	1,309,336	3.29
5	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	3.17
6	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	2.78
7	PM Condensable	18	14	6	231,543	77,181	540,267	1.36
8	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.17
9	Benzene	16	13	6	9,103	3,034	21,241	0.05
10	Methanol	8	7	6	8,286	2,762	19,333	0.05
		18	14	6	17,007,765	5,669,255	39,684,785	99.86

### 3.14b. Releases by ICD Category

Genitourinary system diseases are subdivided into 2 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with both. It should be remembered, that a single chemical can be associated with more than one category of disease.

#### **N00-N39: Diseases of the urinary system**

Thirty-three chemicals are associated with diseases of the urinary system.

One chemical has been implicated in renal failure (N17-N19).

N25-N29: Thirty-two chemicals are associated with other disorders of kidney and ureter. Effects on kidneys include: changes in blood vessels or in circulation, permanent damage, depressed function, necrosis, stones, injury, lesions or weight change.

N30-N39: Six chemicals are associated with other diseases of urinary system, including damage to the Cowper's gland and bladder weight change.

#### **N40-N99: Diseases of the pelvis, genitals and breasts**

Thirty-seven chemicals are associated with diseases of the pelvis, genitals and breasts that effect reproduction.

Twenty-two chemicals are associated with diseases of male genital organs (N40-N51), including: epididymis, low hormone levels, male impotence, reduced fertility, semen (chemical contamination of semen, low amount of semen and low number of swimming semen), seminal vesicle injury, sperm (abnormalities, irregular shape and low number), and sterility.

A single chemical has been implicated in inflammatory diseases of female pelvic organs (N70-N77).

Nineteen have been connected to noninflammatory disorders of female genital tract (N80-N98): both primary infertility (infertility without any previous pregnancy) and secondary infertility (fertility problems occurring in a couple that has conceived on their own and had a child in the past), cervical erosion, effects on the ovaries (damage, weight changes and unspecified effects), menstrual problems including dysmenorrhea, endometrial stromal polyps, and vagina effects.

Table 3.14b

## Diseases of the Genitourinary System by ICD Code Group

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	N00-N39	Diseases of the genitourinary system: urinary system	18	18	17	18	33	33	33	33	495,206	1,091,088	1,143,810	2,730,105
1.1	N00-N08	Glomerular diseases	0	0	0	0	0	0	0	0	0	0	0	0
1.2	N10-N16	Renal tubulo-interstitial diseases	0	0	0	0	0	0	0	0	0	0	0	0
1.3	N17-N19	Renal failure	6	7	8	8	1	1	1	1	1,381	4,324	2,580	8,285
1.4	N20-N23	Urolithiasis	0	0	0	0	0	0	0	0	0	0	0	0
1.5	N25-N29	Other disorders of kidney and ureter	18	18	17	18	32	32	32	32	494,933	1,090,089	1,143,059	2,728,082
1.6	N30-N39	Other diseases of urinary system	18	17	16	18	6	6	6	6	112,062	235,167	225,555	572,785
2	N40-N99	Diseases of the genitourinary system: pelvis, genitals and breasts	18	18	17	18	36	37	37	37	4,426,090	6,690,914	5,913,394	17,030,399
2.1	N40-N51	Diseases of male genital organs	18	18	17	18	22	22	22	22	1,533,660	2,275,644	2,083,319	5,892,625
2.2	N60-N64	Disorders of breast	0	0	0	0	0	0	0	0	0	0	0	0
2.3	N70-N77	Inflammatory diseases of female pelvic organs	18	17	16	18	1	1	1	1	110,333	229,882	220,927	561,143
2.4	N80-N98	Noninflammatory disorders of female genital tract	18	18	17	18	19	19	19	19	738,279	1,664,712	1,573,589	3,976,581
2.5	N99	Other disorders of genitourinary tract	18	18	17	18	29	30	30	30	1,733,463	2,737,002	2,433,354	6,903,820
	<b>N00-N99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>42</b>	<b>43</b>	<b>43</b>	<b>43</b>	<b>4,426,468</b>	<b>6,691,459</b>	<b>5,913,756</b>	<b>17,031,684</b>

### 3.14c. Releases by Facility

All natural gas compressor stations in NYS reported releases chemical associated with genitourinary system diseases.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 10.4 pounds, more than one-half (26.1%) of the state total, followed by Compressor Station 229 in Eden (5.1 million or 12.7%) and Compressor Station 249 in Carlisle (4.3 pounds or 10.8%). In aggregate, these three facilities were responsible for 19.8 million pounds or slightly less than one-half (49.6%) of the state total.

The top 5 facilities were responsible for 25.2 million pounds, slightly less than two-thirds (63.1%) of all releases.

The facility average was 2.2 million pounds over 7 years or 317,048 pounds each year.

Table 3.14c.

#### Diseases of the Genitourinary System by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	34	34	22	1,491,035	10,437,248	26.13
2	TGPC 229 & TEG DF	Eden	Erie	31	30	31	727,142	5,089,991	12.74
3	TGPC CS 249	Carlisle	Schoharie	34	23	23	613,568	4,294,974	10.75
4	TGPC CS 241	LaFayette	Onondaga	33	24	33	430,379	3,012,652	7.54
5	TGPC CS 254	Chatham	Columbia	21	14	8	340,832	2,385,827	5.97
6	TGPC CS 237	Manchester, Phelps	Ontario	8	7	4	328,157	2,297,097	5.75
7	AGT Stony Point CS	Stony Point	Rockland	36	19	20	282,892	1,980,244	4.96
8	NFGSC Concord CS	Concord	Erie	10	9	11	247,433	1,732,032	4.34
9	AGT SOUTHEAST CS	Southeast	Putnam	21	16	26	236,452	1,655,166	4.14
10	NFGSC Beech Hill CS	Willing	Allegany	18	18	19	197,907	1,385,347	3.47
11	NFGSC Independence CS	Andover	Allegany	14	9	16	193,316	1,353,211	3.39
12	TGPC CS 224	Clymer	Chautauqua	34	32	34	162,635	1,138,443	2.85
13	DTI Woodhull Station	Woodhull	Steuben	25	34	38	117,138	819,966	2.05
14	DTI Borger CS	Ithaca	Tompkins	25	26	17	110,760	775,319	1.94
15	NFGSC Nashville CS	Hanover	Chautauqua	20	20	0	88,900	622,297	1.56
16	TGPC CS 230-C	Lockport	Niagara	21	20	21	66,406	464,840	1.16
17	DTI Utica Station	Frankfort	Herkimer	20	24	37	39,872	279,102	0.70
18	TGPC CS 233	York	Livingston	21	14	3	32,039	224,273	0.56
				<b>42</b>	<b>43</b>	<b>43</b>	<b>5,706,861</b>	<b>39,948,030</b>	<b>100%</b>

### 3.14d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with genitourinary system diseases.

Region 9, Western New York ranked first with 2.7 million pounds (29.1%), closely followed by Region 6, Western Adirondacks/Eastern Lake Ontario (10.7 million pounds or 27%). Region 4, Capital Region/Northern Catskills, ranked third with 6.7 million pounds (16.8%).

Table 3.14d.

#### Diseases of the Genitourinary System by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	20	1,173,668	391,223	2,738,558	6.89
		Chautauqua	10	2	41	665,703	221,901	1,553,307	3.91
		Erie	2	2	34	2,923,724	974,575	6,822,023	17.17
		Niagara	13	1	21	199,217	66,406	464,840	1.17
				7	42	4,962,312	1,654,104	11,578,729	29.14
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	42	4,592,722	1,530,907	10,716,351	26.97
3	4: Capital Region / N Catskills	Columbia	6	1	21	1,022,497	340,832	2,385,827	6.00
		Schoharie	3	1	34	1,840,703	613,568	4,294,974	10.81
				2	38	2,863,201	954,400	6,680,801	16.81
4	7: Central New York	Onondaga	4	1	33	1,291,137	430,379	3,012,652	7.58
		Tompkins	12	1	26	332,279	110,760	775,319	1.95
				2	43	1,623,416	541,139	3,787,971	9.53
6	3: Lower Hudson Valley	Putnam	9	1	28	709,357	236,452	1,655,166	4.16
		Rockland	8	1	36	848,676	282,892	1,980,244	4.98
				2	40	1,558,033	519,344	3,635,410	9.15
5	8: Western Finger Lakes	Livingston	14	1	21	96,117	32,039	224,273	0.56
		Ontario	7	1	8	984,470	328,157	2,297,097	5.78
		Steuben	11	1	39	351,414	117,138	819,966	2.06
				3	39	1,432,001	477,334	3,341,336	8.41
				18	43	17,031,685	5,677,228	39,740,598	100%

### 3.14e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to genitourinary system diseases.

Herkimer County ranked first with 10.7 million pounds, more than one-quarter (27%) of the state total, followed by Erie County (6.8 million pounds or 17.2%) and Schoharie County (4.3 million pounds or 10.8%). These three counties are responsible for slightly more than one-half (53%) of all toxic releases.

The top five counties were responsible for 21.8 million pounds or more than one-half (55%) of the state total.

The country average was 2.8 million pounds over a 7-year period or 405,516 pounds annually.

Table 3.14e.

#### Diseases of the Genitourinary System by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	42	4,592,722	1,530,907	10,716,350	26.97
2	Erie	9: Western New York	2	34	2,923,724	974,575	6,822,022	17.17
3	Schoharie	4: Capital Region/Northern Catskills	1	34	1,840,703	613,568	4,294,974	10.81
4	Onondaga	7: Central New York	1	33	1,291,137	430,379	3,012,651	7.58
5	Allegany	9: Western New York	2	20	1,173,668	391,223	2,738,558	6.89
6	Columbia	4: Capital Region/Northern Catskills	1	21	1,022,497	340,832	2,385,826	6.00
7	Ontario	8: Western Finger Lakes	1	8	984,470	328,157	2,297,097	5.78
8	Rockland	3: Lower Hudson Valley	1	36	848,676	282,892	1,980,244	4.98
9	Putnam	3: Lower Hudson Valley	1	28	709,357	236,452	1,655,165	4.16
10	Chautauqua	9: Western New York	2	41	665,703	221,901	1,553,307	3.91
11	Steuben	8: Western Finger Lakes	1	39	351,414	117,138	819,966	2.06
12	Tompkins	7: Central New York	1	26	332,279	110,760	775,318	1.95
13	Niagara	9: Western New York	1	21	199,217	66,406	464,840	1.17
14	Livingston	8: Western Finger Lakes	1	21	96,117	32,039	224,272	0.56
			<b>18</b>	<b>43</b>	<b>17,031,685</b>	<b>5,677,228</b>	<b>39,740,590</b>	<b>100%</b>

## 3.15. Pregnancy, Childbirth and the Puerperium (O00-O99)

### 3.15a. Releases by Chemical

Eighteen of the 70 chemicals released by NYS natural gas compressor stations are associated with diseases of pregnancy, childbirth and the puerperium (ICD-10, Chapter 15).

Releases of these toxicants were reported by all 18 stations and totaled an estimated 19.5 million pounds from 2008 to 2014--an annual average of 2.8 million pounds.

Chemicals associated with diseases of pregnancy, childbirth and the puerperium represented 48.8% of releases by the state's natural gas compressor stations.

Or, to put it differently, ***of the 40.2 million pounds of chemicals released by NYS's compressor stations, slightly less than one-half (48.62%) had adverse effects on pregnancy, childbirth and the puerperium.***

Nitrogen oxides ranked first with 18.1 million pounds or (92.5%) of the total. Formaldehyde was a distant second (1.3 million pounds or 6.7%), followed by acetaldehyde (65,969 pounds or 0.34%).

These two chemicals accounted for 19.4 million pounds or 99.2% of all releases.

Table 3.15a.

#### Pregnancy, Childbirth and the Puerperium by Chemical (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	92.54
2	Formaldehyde	18	14	6	561,144	187,048	1,309,336	6.70
3	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.34
4	Acrolein	14	13	6	22,596	7,532	52,723	0.27
5	Toluene	16	13	6	8,275	2,758	19,308	0.10
6	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.04
7	Naphthalene	15	13	6	298	99	696	0.00
8	Methylene Chloride	8	7	6	269	90	629	0.00
9	Ethylene Dibromide	8	7	6	149	50	347	0.00
10	Carbon Tetrachloride	8	7	6	121	40	282	0.00
11	Styrene	8	7	6	100	33	234	0.00
12	Ethylene Dichloride	6	6	5	65	22	151	0.00
13	Vinyl Chloride	8	7	6	46	15	107	0.00
14	Mercury	16	13	6	30	10	70	0.00
15	Cadmium	9	9	6	13	4	30	0.00
16	Tetrachloroethylene	4	4	4	4	1	9	0.00
17	Lead	16	12	6	0	0	1	0.00
18	Arsenic	6	6	5	0	0	0.1	0.00
		18	14	6	8,374,652	2,791,551	19,540,856	100%

### 3.15b. Releases by ICD Category

Diseases of pregnancy, childbirth and the puerperium are subdivided into 8 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with three. It should be remembered, that a single chemical can be associated with more than one category of disease.

O00-O08: Fourteen chemicals, released by all 18 sites, are associated with pregnancy with abortive outcome.

O30-O48: Five chemicals are associated with maternal care related to the fetus and amniotic cavity and possible delivery problems.

O85-O92: A single chemicals is implicated in complications predominantly related to the puerperium.

Table 3.15b.

#### Pregnancy, Childbirth and the Puerperium by ICD Code Group

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	O00-O08	Pregnancy with abortive outcome	18	18	17	18	14	14	14	14	2,389,023	3,255,109	2,729,996	8,374,129
2	O10-O16	Edema, proteinuria and hypertensive disorders in pregnancy, childbirth and the puerperium	0	0	0	0	0	0	0	0	0	0	0	0
3	O20-O29	Other maternal disorders predominantly related to pregnancy	0	0	0	0	0	0	0	0	0	0	0	0
4	O30-O48	Maternal care related to the fetus and amniotic cavity and possible delivery problems	15	15	12	15	5	5	5	5	4,477	15,355	8,962	28,795
5	O60-O75	Complications of labor and delivery	0	0	0	0	0	0	0	0	0	0	0	0
6	O80-O84	Delivery	0	0	0	0	0	0	0	0	0	0	0	0
7	O85-O92	Complications predominantly related to the puerperium	18	17	16	18	1	1	1	1	110,333	229,882	220,927	561,143
8	O95-O99	Other obstetric conditions, not elsewhere classified	0	0	0	0	0	0	0	0	0	0	0	0
	<b>O00-O99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>18</b>	<b>18</b>	<b>18</b>	<b>18</b>	<b>2,389,116</b>	<b>3,255,374</b>	<b>2,730,161</b>	<b>8,374,652</b>



### 3.15c. Releases by Facility

All natural gas compressor stations in NYS reported chemical releases associated with pregnancy, childbirth and the puerperium diseases.

The top 4 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 6.1 pounds (30.9%), followed by Compressor Station 229 in Eden (3.3 million or 16.9%) and Compressor Station 249 in Carlisle (1.9 million pounds or 9.5%). In aggregate, these three facilities were responsible for 11.2 million pounds, more than one-half (57.3%) of the state total.

The top 5 facilities were responsible for 13.4 million pounds, slightly more than two-thirds (68.4%) of all releases.

The facility average was 1.1 million pounds over 7 years or 155,768 pounds each year.

Table 3.15c.

#### Pregnancy, Childbirth and the Puerperium by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	16	16	10	865,884	6,061,190	30.88
2	TGPC 229 & TEG DF	Eden	Erie	14	13	14	473,307	3,313,147	16.88
3	TGPC CS 249	Carlisle	Schoharie	16	10	10	267,623	1,873,364	9.54
4	TGPC CS 254	Chatham	Columbia	10	7	2	162,679	1,138,751	5.80
5	AGT SOUTHEAST CS	Southeast	Putnam	10	7	11	148,254	1,037,778	5.29
6	TGPC CS 241	LaFayette	Onondaga	15	11	15	132,651	928,556	4.73
7	TGPC CS 237	Manchester, Phelps	Ontario	3	2	2	123,343	863,403	4.40
8	AGT Stony Point CS	Stony Point	Rockland	16	8	9	117,278	820,945	4.18
9	NFGSC Concord CS	Concord	Erie	4	3	4	109,935	769,544	3.92
10	DTI Borger CS	Ithaca	Tompkins	11	11	8	86,398	604,785	3.08
11	TGPC CS 224	Clymer	Chautauqua	16	14	16	63,140	441,983	2.25
12	NFGSC Beech Hill CS	Willing	Allegany	9	9	9	53,918	377,423	1.92
13	NFGSC Independence CS	Andover	Allegany	6	3	6	51,476	360,334	1.84
14	TGPC CS 230-C	Lockport	Niagara	10	9	10	38,680	270,758	1.38
15	NFGSC Nashville CS	Hanover	Chautauqua	8	8	0	36,799	257,594	1.31
16	DTI Woodhull Station	Woodhull	Steuben	11	15	16	31,911	223,376	1.14
17	TGPC CS 233	York	Livingston	10	7	1	22,714	158,999	0.81
18	DTI Utica Station	Frankfort	Herkimer	8	11	16	17,827	124,788	0.64
				<b>18</b>	<b>18</b>	<b>18</b>	<b>2,803,817</b>	<b>19,626,720</b>	<b>100%</b>

### 3.15d. Releases by DEC Regions

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with pregnancy, childbirth and the puerperium diseases.

Region 6, Western Adirondacks/Eastern Lake Ontario, ranked first with 6.2 million pounds, nearly one-half (46.3%) of the state total, closely followed by Region 9, Western New York (5.7 million pounds or 43.7%). Region 4, Capital Region/Northern Catskills, ranked third with 3 million pounds (14%).

Table 3.15d.

#### Pregnancy, Childbirth and the Puerperium by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	18	2,651,134	883,711	6,185,978	46.32
2	9: Western New York	Allegany	9	2	9	316,181	105,394	737,756	5.52
		Chautauqua	10	2	18	263,020	87,673	613,712	4.60
		Erie	2	2	15	1,749,724	583,241	4,082,690	30.57
		Niagara	12	1	10	116,039	38,680	270,758	2.03
				7	18	2,444,965	814,988	5,704,917	42.72
3	4: Capital Region / N Catskills	Columbia	4	1	10	488,036	162,679	1,138,751	8.53
		Schoharie	3	1	16	802,870	267,623	1,873,364	14.03
				2	17	1,290,907	430,302	3,012,116	22.55
4	3: Lower Hudson Valley	Putnam	5	1	11	444,762	148,254	1,037,778	7.77
		Rockland	8	1	16	351,834	117,278	820,945	6.15
				2	17	796,596	265,532	1,858,724	13.92
4	7: Central New York	Onondaga	6	1	15	397,953	132,651	928,556	6.95
		Tompkins	11	1	11	259,194	86,398	604,785	4.53
				2	18	657,146	219,049	1,533,342	11.48
5	8: Western Finger Lakes	Livingston	14	1	10	68,143	22,714	158,999	1.19
		Ontario	7	1	3	370,030	123,343	863,403	6.47
		Steuben	13	1	16	95,733	31,911	223,376	1.67
				3	18	533,905	177,968	1,245,779	9.33
				16	18	5,723,519	1,907,840	13,354,877	100%

### 3.15e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to genitourinary system diseases.

Herkimer County ranked first with 6.2 million pounds, slightly less than one-third (31.7%) of the state total, followed by Erie County (4.1 million pounds or 20.9%) and Schoharie County (1.9 million pounds or 9.6%). These three counties are responsible for 12.1 million pounds (62.1%) of all toxic releases.

The top five counties were responsible for 14.3 million pounds or nearly three-fourths (73.3%) of the state total.

The country average was 1.4 million pounds over a 7-year period or 199,396 pounds annually.

Table 3.15e.

#### Pregnancy, Childbirth and the Puerperium by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	18	2,651,134	883,711	6,185,978	31.66
2	Erie	9: Western New York	2	15	1,749,724	583,241	4,082,690	20.89
3	Schoharie	4: Capital Region/Northern Catskills	1	16	802,870	267,623	1,873,364	9.59
4	Columbia	4: Capital Region/Northern Catskills	1	10	488,036	162,679	1,138,751	5.83
5	Putnam	3: Lower Hudson Valley	1	11	444,762	148,254	1,037,778	5.31
6	Onondaga	7: Central New York	1	15	397,953	132,651	928,556	4.75
7	Ontario	8: Western Finger Lakes	1	3	370,030	123,343	863,403	4.42
8	Rockland	3: Lower Hudson Valley	1	16	351,834	117,278	820,945	4.20
9	Allegany	9: Western New York	2	9	316,181	105,394	737,756	3.78
10	Chautauqua	9: Western New York	2	18	263,020	87,673	613,712	3.14
11	Tompkins	7: Central New York	1	11	259,194	86,398	604,785	3.09
12	Niagara	9: Western New York	1	10	116,039	38,680	270,758	1.39
13	Steuben	8: Western Finger Lakes	1	16	95,733	31,911	223,376	1.14
14	Livingston	8: Western Finger Lakes	1	10	68,143	22,714	158,999	0.81
			<b>18</b>	<b>18</b>	<b>8,374,652</b>	<b>2,791,551</b>	<b>19,540,856</b>	<b>100%</b>

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## 3.16. Certain Conditions Originating in the Perinatal Period (P00-P96)

### 3.16a. Releases by Chemical

Twenty of the 70 chemicals released by NYS natural gas compressor stations are associated with Certain Conditions Originating in the Perinatal Period (ICD-10, Chapter 16).

Releases of these toxicants were reported by all 18 stations and totaled an estimated 22.4 million pounds from 2008 to 2014--an annual average of 3.2 million pounds.

Or, to put it differently, ***of the 40.2 million pounds of chemicals released by NYS's compressor stations, 56% have been associated with certain conditions originating in the perinatal period.***

Nitrogen oxides ranked first with 18.1 million pounds, more than three-fourths (80.7%) of the total. Formaldehyde was a distant second (1,309,336 pounds or 5.8%), followed by PM 10 (1,259,744 million pounds or 3.8%). These three chemicals accounted for 19.4 million pounds or 92% of all releases.

The top 10 chemicals were responsible for virtually all releases (99.5%).

The annual average release was 3.2 million pounds.

Table 3.16a.

#### Certain Conditions Originating in the Perinatal Period by Chemical (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	80.67
2	Formaldehyde	18	14	6	561,144	187,048	1,309,336	5.84
3	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	5.62
4	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	4.93
5	PM Condensable	18	14	6	231,543	77,181	540,267	2.41
6	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.29
7	Benzene	16	13	6	9,103	3,034	21,241	0.09
8	Toluene	16	13	6	8,275	2,758	19,308	0.09
9	Xylenes (Mixed Isomers)	15	13	6	3,598	1,199	8,394	0.04
10	Phenol	11	10	6	303	101	706	0.00
					9,605,886	3,201,962	22,413,733	99.99
11	Nickel	11	11	6	296	99	692	0.00
12	Manganese	9	9	6	150	50	350	0.00
13	Carbon Tetrachloride	8	7	6	121	40	282	0.00
14	Styrene	8	7	6	100	33	234	0.00
15	Chloroform	9	8	6	83	28	193	0.00
16	Phenanthrene	11	9	6	21	7	48	0.00
17	Cadmium	9	9	6	13	4	30	0.00
18	Acenaphthene	10	8	6	4	1	8.3	0.00
19	Lead	16	12	6	0	0	1	0.00
20	Arsenic	6	6	5	0	0	0	0.00
					788	263	1,838	0.01
					9,606,673	3,202,224	22,415,571	100%

### 3.16b. Releases by ICD Category

Certain Conditions Originating in the Perinatal Period are subdivided into 10 major groups. Chemicals released by natural gas compressor stations in NYS are positively associated with three. It should be remembered, that a single chemical can be associated with more than one category of disease.

**P05-P08:** Twenty-one chemicals are associated with disorders related to length of gestation and fetal growth: birth weight (low or extremely low), growth statistics (e.g., reduced weight gain), preterm birth, and small for gestational age.

**P50-P61:** A single chemical has been connected to hemorrhagic and hematological disorders of fetus and newborn.

Table 3.16b.

#### Certain Conditions Originating in the Perinatal Period by ICD Code Group

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	P00-P04 Fetus and newborn affected by maternal factors and by complications of pregnancy, labor and delivery		0	0	0	0	0	0	0	0	0	0	0	0
2	P05-P08 Disorders related to length of gestation and fetal growth		18	18	17	18	19	19	19	19	2,631,857	3,820,078	3,154,716	9,606,652
3	P10-P15 Birth trauma		0	0	0	0	0	0	0	0	0	0	0	0
4	P20-P29 Respiratory and cardiovascular disorders specific to the perinatal period		0	0	0	0	0	0	0	0	0	0	0	0
5	P35-P39 Infections specific to the perinatal period		0	0	0	0	0	0	0	0	0	0	0	0
6	P50-P61 Hemorrhagic and hematological disorders of fetus and newborn		10	8	6	11	1	1	1	1	3	14	2	20
7	P70-P74 Transitory endocrine and metabolic disorders specific to fetus and newborn		0	0	0	0	0	0	0	0	0	0	0	0
8	P75-P78 Digestive system disorders of fetus and newborn		0	0	0	0	0	0	0	0	0	0	0	0
9	P80-P83 Conditions involving the integument and temperature regulation of fetus and newborn		0	0	0	0	0	0	0	0	0	0	0	0
10	P90-P96 Other disorders originating in the perinatal period		18	18	15	18	1	1	1	1	92,594	220,983	160,506	474,084
	<b>P00-P96 Total</b>		<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>20</b>	<b>20</b>	<b>20</b>	<b>20</b>	<b>2,631,861</b>	<b>3,820,092</b>	<b>3,154,718</b>	<b>9,606,673</b>

### 3.16c. Releases by Facility

All natural gas compressor stations in NYS reported chemical releases associated with certain conditions originating in the perinatal period.

Six of the 7 top polluters were facilities operated by the Tennessee Gas Pipeline Company, including the top 4.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 6.6 million pounds (29.3%), followed by Compressor Station 229 in Eden (3.7 million pounds or 16.4%) and Compressor Station 249 in Carlisle (2.4 million pounds or 10.5%). In aggregate, these three facilities were responsible for 17.9 million pounds, slightly more than one-half (50.9%) of the state total.

The top 5 facilities were responsible for 22.5 million pounds, slightly less than two-thirds (66%) of all releases.

The facility average was 2 million pounds over 7 years or 278,879 pounds each year.

Table 3.16c.

#### Certain Conditions Originating in the Perinatal Period by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	16	16	10	942,678	6,598,747	29.32
2	TGPC 229 & TEG DF	Eden	Erie	16	15	16	526,098	3,682,688	16.36
3	TGPC CS 249	Carlisle	Schoharie	16	10	10	337,100	2,359,704	10.48
4	TGPC CS 254	Chatham	Columbia	14	10	6	184,150	1,289,055	5.73
5	AGT SOUTHEAST CS	Southeast	Putnam	14	10	17	179,875	1,259,125	5.59
6	TGPC CS 241	LaFayette	Onondaga	15	12	15	178,380	1,248,661	5.55
7	TGPC CS 237	Manchester, Phelps	Ontario	5	5	2	149,469	1,046,289	4.65
8	AGT Stony Point CS	Stony Point	Rockland	18	9	10	139,970	979,796	4.35
9	NFGSC Concord CS	Concord	Erie	7	5	7	131,814	922,698	4.10
10	DTI Borger CS	Ithaca	Tompkins	16	16	10	89,289	625,023	2.78
11	TGPC CS 224	Clymer	Chautauqua	16	16	16	68,616	480,315	2.13
12	NFGSC Beech Hill CS	Willing	Allegany	10	10	10	64,286	450,005	2.00
13	NFGSC Independence CS	Andover	Allegany	10	6	10	54,734	383,143	1.70
14	TGPC CS 230-C	Lockport	Niagara	14	13	14	44,297	310,080	1.38
15	DTI Woodhull Station	Woodhull	Steuben	16	19	20	40,062	280,440	1.25
16	NFGSC Nashville CS	Hanover	Chautauqua	14	14	0	38,896	272,276	1.21
17	TGPC CS 233	York	Livingston	14	10	1	25,581	179,072	0.80
18	DTI Utica Station	Frankfort	Herkimer	14	16	20	19,886	139,202	0.62
				20	20	20	3,215,181	22,506,319	100%

### 3.16d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with certain conditions originating in the perinatal period.

Region 6, Western Adirondacks/Eastern Lake Ontario, ranked first with 6.7 million pounds (30.1%), closely followed by Region 9, Western New York (6.4 million pounds or 28.6%). Region 4, Capital Region/Northern Catskills, ranked third with 3.6 million pounds (16.3%).

Table 3.16d.

#### Certain Conditions Originating in the Perinatal Period by County by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	20	2,887,693	962,564	6,737,950	30.06
2	9: Western New York	Allegany	9	2	11	357,064	119,021	833,149	3.72
		Chautauqua	10	2	20	283,643	94,548	661,833	2.95
		Erie	2	2	17	1,973,737	657,912	4,605,387	20.55
		Niagara	12	1	14	132,892	44,297	310,080	1.38
				7	20	2,747,335	915,778	6,410,449	28.60
3	4: Capital Region / N Catskills	Columbia	4	1	14	552,453	184,151	1,289,056	5.75
		Schoharie	3	1	16	1,011,302	337,101	2,359,705	10.53
				2	19	1,563,754	521,251	3,648,760	16.28
4	3: Lower Hudson Valley	Putnam	5	1	18	539,625	179,875	1,259,126	5.62
		Rockland	8	1	18	419,913	139,971	979,796	4.37
				2	20	959,538	319,846	2,238,922	9.99
5	7: Central New York	Onondaga	6	1	15	535,141	178,380	1,248,662	5.57
		Tompkins	11	1	16	267,867	89,289	625,024	2.79
				2	20	803,008	267,669	1,873,686	8.36
6	8: Western Finger Lakes	Livingston	14	1	14	76,745	25,582	179,073	0.80
		Ontario	7	1	5	448,410	149,470	1,046,290	4.67
		Steuben	13	1	20	120,189	40,063	280,441	1.25
				3	20	645,344	215,115	1,505,803	6.72
				18	20	9,606,673	3,202,224	22,415,571	100%



### 3.16e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to certain conditions originating in the perinatal period.

Herkimer County ranked first with 9.8 million pounds, (28%) of the state total, followed by Erie County (6.3 million pounds or 18.1%) and Schoharie County (3.6 million pounds or 10.4%). These three counties are responsible for 19.7 million pounds (56.4%) of all toxic releases.

The top five counties were responsible for 24.5 million pounds or 70% of the state total.

The country average was 2.5 million pounds over a 7-year period or 356,756 pounds annually.

Table 3.16e.

#### Certain Conditions Originating in the Perinatal Period by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	20	2,887,693	962,564	6,737,950	30.06
2	Erie	9: Western New York	2	17	1,973,737	657,912	4,605,387	20.55
3	Schoharie	4: Capital Region/Northern Catskills	1	16	1,011,301	337,100	2,359,704	10.53
4	Columbia	4: Capital Region/Northern Catskills	1	14	552,452	184,150	1,289,055	5.75
5	Putnam	3: Lower Hudson Valley	1	18	539,625	179,875	1,259,125	5.62
6	Onondaga	7: Central New York	1	15	535,140	178,380	1,248,661	5.57
7	Ontario	8: Western Finger Lakes	1	5	448,409	149,469	1,046,289	4.67
8	Rockland	3: Lower Hudson Valley	1	18	419,912	139,970	979,796	4.37
9	Allegany	9: Western New York	2	11	357,063	119,021	833,148	3.72
10	Chautauqua	9: Western New York	2	20	283,642	94,547	661,832	2.95
11	Tompkins	7: Central New York	1	16	267,867	89,289	625,023	2.79
12	Niagara	9: Western New York	1	14	132,891	44,297	310,080	1.38
13	Steuben	8: Western Finger Lakes	1	20	120,188	40,062	280,440	1.25
14	Livingston	8: Western Finger Lakes	1	14	76,745	25,581	179,072	0.80
			<b>18</b>	<b>22</b>	<b>9,606,665</b>	<b>3,202,217</b>	<b>22,415,562</b>	<b>100%</b>

Table 3.16f.

## Certain Conditions Originating in the Perinatal Period by chemical

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

#	Chemical name	U.S. EPA National Emissions Inventory Pounds	Congenital malformations, deformations and chromosomal abnormalities	Congenital malformations and deformations	Nervous system	Eye, ear, face and neck	Circulatory system	Respiratory system	Digestive system	Genital organs	Urinary system	Musculoskeletal system	Other	Chromosomal abnormalities, nec	California Proposition 65 Status
			Q00-Q99	Q00-Q89	Q00-Q07	Q10-Q18	Q20-Q28	Q30-Q34	Q35-Q45	Q50-Q56	Q60-Q64	Q65-Q79	Q80-Q89	Q90-Q99	CA P65
				1	1.1	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2	
			59	57	16	4	9	4	19	2	9	19	55	32	
1	Acetaldehyde	65,969.0555	Y	Y	Y	Y			Y		Y	Y	Y	Y	
2	Acrolein	52,723.4283	Y	Y									Y		
3	Anthracene	9.9008	Y	Y									Y	Y	
4	Arsenic	0.0631	Y	Y	Y		Y	Y	Y				Y	Y	
5	Benz[a]anthracene	19.4738	Y	Y									Y	Y	
6	Benzene	21,240.5186	Y	Y	Y		Y		Y			Y	Y	Y	Known
7	Benzo[j,k]fluorene	10.6660	Y	Y									Y		
8	Benzo[a]pyrene	0.0133	Y	Y									Y	Y	
9	Benzo[b]fluoranthene	0.6858	Y	Y									Y		
10	Benzo[e]pyrene	0.0581	Y	Y									Y		
11	Benzo[g,h,i]perylene	1.7487	Y	Y									Y	Y	
12	Benzo[k]fluoranthene	0.0019	Y	Y									Y		
13	Beryllium	0.0034	Y	Y									Y		
14	Biphenyl	690.3042	Y	Y									Y		
15	Butadiene, 1,3-	4,718.8745	Y	Y								Y	Y	Y	Known
16	Cadmium	30.4906	Y	Y	Y	Y	Y		Y		Y	Y	Y	Y	Known
17	Carbon monoxide	12,359,731.3420	Y	Y	Y		Y		Y				Y		Known
18	Carbon tetrachloride	281.6842	Y	Y	Y				Y			Y	Y		
19	Chlorobenzene	171.9160	Y	Y				Y	Y			Y	Y	Y	
20	Chloroethane (Ethyl chloride)	5.9185	Y	Y									Y		
21	Chloroform	192.8606	Y	Y	Y				Y		Y	Y	Y	Y	Known
22	Chrysene	4.0913	Y	Y									Y	Y	
23	Cobalt	0.0264	Y	Y									Y	Y	
24	Dibenz[a,h]anthracene	0.0000	Y	Y									Y	Y	
25	Dichloropropene, 1,3-	160.9530	Y	Y									Y	Y	
26	Dimethylbenz[a]anthracene, 7,12-	0.0033	Y											Y	
27	Ethylbenzene	2,794.3829	Y	Y								Y	Y	Y	
28	Ethylene dibromide	346.9677	Y	Y				Y					Y	Y	Known
29	Ethylene dichloride	150.8763	Y	Y									Y	Y	
30	Fluorene	28.0605	Y	Y									Y		
31	Formaldehyde	1,309,335.5542	Y	Y					Y		Y	Y	Y	Y	
32	Hexane, n-	12,183.8539	Y	Y							Y	Y	Y	Y	
33	Indeno[1,2,3-cd]pyrene	0.0240	Y	Y									Y		
34	Lead	0.5586	Y	Y	Y		Y					Y	Y	Y	Known

#	Chemical name	U.S. EPA National Emissions Inventory Pounds	Congenital malformations, deformations and chromosomal abnormalities	Congenital malformations and deformations	Nervous system	Eye, ear, face and neck	Circulatory system	Respiratory system	Digestive system	Genital organs	Urinary system	Musculoskeletal system	Other	Chromosomal abnormalities, nec	California Proposition 65 Status
			Q00-Q99	Q00-Q89	Q00-Q07	Q10-Q18	Q20-Q28	Q30-Q34	Q35-Q45	Q50-Q56	Q60-Q64	Q65-Q79	Q80-Q89	Q90-Q99	CA P65
				1	1.1	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2	
			59	57	16	4	9	4	19	2	9	19	55	32	
35	Manganese	350.2412	Y	Y	Y			Y	Y				Y		
36	Mercury	70.0315	Y	Y	Y								Y		
37	Methane dichloride [1910.1052]	628.7595	Y	Y							Y	Y	Y	Y	
38	Methanol	19,333.1517	Y	Y	Y	Y			Y	Y	Y	Y	Y		
39	Methylcholanthrene, 3-	0.0003	Y											Y	
40	Naphthalene	696.4536	Y	Y		Y						Y	Y	Y	
41	Nickel	691.7926	Y	Y									Y		Candidate
42	Nitrogen oxides (NO2)	18,082,570.5018	Y	Y	Y				Y						
43	Perchloroethylene	8.9808	Y	Y					Y			Y	Y	Y	
44	Phenanthrene	47.9187	Y	Y									Y		
45	Phenol	706.0520	Y	Y					Y			Y	Y	Y	
46	PM 2.5 Primary (Filt + Cond)	1,106,197.8579	Y	Y			Y		Y				Y		
47	PM10 Primary (Filt + Cond)	1,259,744.3362	Y	Y			Y		Y				Y		
48	Propylene dichloride	163.9497	Y	Y									Y		
49	Propylene oxide	614.7228	Y	Y					Y			Y	Y	Y	
50	Pyrene	7.0711	Y	Y									Y		
51	Selenium	0.0064	Y	Y									Y		
52	Styrene	233.7242	Y	Y						Y	Y		Y	Y	
53	Sulfur dioxide	186,778.1614	Y	Y			Y		Y				Y		
54	Tetrachloroethane, 1,1,2,2-	308.7690	Y	Y									Y	Y	
55	Toluene	19,307.6774	Y	Y	Y						Y	Y	Y	Y	Known
56	Trichloroethane, 1,1,2-	247.4703	Y	Y	Y										
57	Vinyl chloride	106.6165	Y	Y	Y								Y	Y	
58	Volatile organic compounds (VOCs)	4,920,395.6676	Y	Y	Y		Y		Y				Y		
59	Xylene (mixed isomers)	8,394.2111	Y	Y								Y	Y		

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## 17. Congenital Malformations, Deformations & Chromosomal Abnormalities (Q00-Q99)

### 3.17a. Releases by Chemical

The relationship between chemical exposures in utero and during the early postnatal period and adverse health effects has been well documented.

“Epidemiological studies have shown that children's exposure to air pollutants during fetal development and early postnatal life is associated with many types of health problems including abnormal development (low birth weight [LBW], very low birth weight [VLBW], preterm birth [PTB], intrauterine growth restriction [IUGR], congenital defects, and intrauterine and infant mortality), decreased lung growth, increased rates of respiratory tract infections, childhood asthma, behavioral problems, and neurocognitive decrements.” (Wang and Pinkerton 2007).

Fifty-nine of the 70 chemicals released by NYS natural gas compressor stations are associated with congenital malformations, deformations and chromosomal abnormalities (ICD-10, Chapter 17).

Releases of these toxicants were reported by all 18 stations and totaled an estimated 39.4 million pounds from 2008 to 2014--an annual average of 5.6 million pounds.

Or, to put it differently, ***of the 40.2 million pounds of chemicals released by NYS's compressor stations, 98% have been associated with congenital malformations, deformations and chromosomal abnormalities.*** Nitrogen oxides ranked first with 18.1 million pounds, slightly less than one-half (45.9%) of the state total. Carbon monoxide was second (12.4 million pounds or 31.3%), followed by volatile organic compounds (4.9 million pounds or 12.5%). These three chemicals accounted for 35.4 million pounds or 89.7 of all releases.

The top 10 chemicals were responsible for virtually all releases (99.8%). The annual average release was 5.6 million pounds.

Table 3.17a.

#### Congenital Malformations, Deformations & Chromosomal Abnormalities

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	45.85
2	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	31.34
3	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	12.48
4	Formaldehyde	18	14	6	561,144	187,048	1,309,336	3.32
5	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	3.19
6	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	2.80
7	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.47
8	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.17
9	Acrolein	14	13	6	22,596	7,532	52,723	0.13
10	Benzene	16	13	6	9,103	3,034	21,241	0.05
		18	14	6	16,870,580	5,623,527	39,364,686	99.81

### 3.17b. Releases by ICD Category

Congenital malformations, deformations & chromosomal abnormalities into 2 major disease categories. Chemicals released by natural gas compressor stations in NYS are positively associated with both.

It should be remembered, that a single chemical can be associated with more than one subcategory of disease within an ICD disease group (chapter) as well as with more than one disease group.

#### Q00-Q89: Congenital Malformation and Deformations

Fifty-seven chemicals are associated with congenital malformation and deformations. This includes:

Q00-Q07: Sixteen chemicals associated with nervous system malformations and deformations, including: brain abnormalities and defects (anencephaly, holoprosencephaly, microcephaly), CNS abnormalities and defects, cognitive developmental delay with greater language impairment, cognitive function, lower IQ, neural tube defects (opening to the spinal cord at the base of the brain), neurological impairment, spatial memory function impairment, and spina bifida.

Q10-Q18: Four chemicals are associated with eye, ear, face and neck malformations and deformations: ear abnormalities, eye abnormalities (anophthalmia and cataracts), facial clefts, and gross facial agenesis.

Q20-Q28: Ten chemicals are associated with circulatory system malformations and deformations, including

Table 3.17b.

#### Congenital Malformations, Deformations & Chromosomal Abnormalities by ICD Code Group

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	Q00-Q89	Congenital malformations and deformations	18	18	17	18	57	54	54	57	4,393,806	6,607,676	5,900,691	16,902,175
1.1	Q00-Q07	Nervous system	18	18	17	18	16	16	16	16	4,068,877	5,882,704	5,258,344	15,209,926
1.2	Q10-Q18	Eye, ear, face and neck	15	15	12	15	4	4	4	4	5,825	19,569	11,475	36,869
1.3	Q20-Q28	Circulatory system	18	18	17	18	10	10	10	10	4,269,779	6,336,905	5,651,896	16,258,581
1.4	Q30-Q34	Respiratory system	14	8	7	14	4	4	4	4	150	107	113	372
1.5	Q35-Q45	Digestive system	18	18	17	18	17	17	17	17	4,386,043	6,586,345	5,884,324	16,856,713
1.6	Q50-Q56	Genital organs	6	7	8	8	2	2	2	2	1,399	4,373	2,612	8,385
1.7	Q60-Q64	Urinary system	18	17	16	18	9	9	9	9	119,382	254,922	237,359	611,663
1.8	Q65-Q79	Musculoskeletal system	18	18	16	18	19	19	19	19	122,314	262,300	243,932	628,547
1.9	Q80-Q89	Other	18	18	17	18	55	52	52	55	2,124,445	3,614,575	3,413,375	9,152,395
2	Q90-Q99	Chromosomal abnormalities, nec	18	18	16	18	30	31	31	32	120,669	256,739	239,709	617,118
	Q00-Q99	Total	18	18	17	18	57	56	56	59	4,393,806	6,607,676	5,900,691	16,902,175

### 3.17c. Releases by Facility

All natural gas compressor stations in NYS reported chemical releases associated with congenital malformations, deformations & chromosomal abnormalities.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 10.4 million pounds, more than one-quarter (26.2%) of the state total. Compressor Station 229 in Eden ranked second (5 million pounds or 12.7%) followed by Compressor Station 249 in Carlisle (4.2 million pounds or 10.7%). In aggregate, these three facilities were responsible for 19.7 million pounds, slightly less than one-half (49.6%) of the state total.

The top 5 facilities were responsible for 25 million pounds, slightly less than two-thirds (63.1%) of all releases.

The facility average was 2.1 million pounds over 7 years or 314,643 pounds each year.

Table 3.17c.

#### Congenital Malformations, Deformations & Chromosomal Abnormalities by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	43	42	23	1,484,232	10,389,625	26.21
2	TGPC 229 & TEG DF	Eden	Erie	45	42	43	721,420	5,049,941	12.74
3	TGPC CS 249	Carlisle	Schoharie	43	24	24	604,523	4,231,660	10.67
4	TGPC CS 241	LaFayette	Onondaga	41	32	41	425,236	2,976,652	7.51
5	TGPC CS 254	Chatham	Columbia	24	15	8	337,861	2,365,027	5.97
6	TGPC CS 237	Manchester, Phelps	Ontario	8	7	5	323,113	2,261,791	5.71
7	AGT Stony Point CS	Stony Point	Rockland	40	22	20	282,935	1,980,542	5.00
8	NFGSC Concord CS	Concord	Erie	10	8	10	243,579	1,705,053	4.30
9	AGT SOUTHEAST CS	Southeast	Putnam	24	17	40	233,541	1,634,787	4.12
10	NFGSC Beech Hill CS	Willing	Allegany	18	18	19	196,128	1,372,896	3.46
11	NFGSC Independence CS	Andover	Allegany	13	9	15	192,763	1,349,341	3.40
12	TGPC CS 224	Clymer	Chautauqua	42	40	42	162,655	1,138,582	2.87
13	DTI Woodhull Station	Woodhull	Steuben	38	47	51	116,594	816,160	2.06
14	DTI Borger CS	Ithaca	Tompkins	38	39	18	110,770	775,390	1.96
15	NFGSC Nashville CS	Hanover	Chautauqua	32	30	0	88,560	619,919	1.56
16	TGPC CS 230-C	Lockport	Niagara	24	23	24	68,322	478,255	1.21
17	DTI Utica Station	Frankfort	Herkimer	32	37	50	39,732	278,121	0.70
18	TGPC CS 233	York	Livingston	24	15	4	31,615	221,306	0.56
				<b>57</b>	<b>56</b>	<b>56</b>	<b>5,663,578</b>	<b>39,645,048</b>	<b>100%</b>

### 3.17d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with congenital malformations, deformations and chromosomal abnormalities.

Region 9, Western New York, ranked first with 11.5 million pounds (29.2%), closely followed by Region 6, Western Adirondacks/Eastern Lake Ontario (10.7 million pounds or 27.1%). Region 4, Capital Region/Northern Catskills, ranked third with 6.7 million pounds (16.7%).

Table 3.17d.

#### Congenital Malformations, Deformations & Chromosomal Abnormalities by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	20	1,166,673	388,891	2,722,237	6.90
		Chautauqua	10	2	54	665,084	221,695	1,551,861	3.93
		Erie	2	2	47	2,894,998	964,999	6,754,994	17.13
		Niagara	13	1	24	204,966	68,322	478,254	1.21
				7	57	4,931,721	1,643,907	11,507,346	29.18
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	58	4,571,891	1,523,964	10,667,746	27.05
3	4: Capital Region / N Catskills	Columbia	6	1	24	1,013,583	337,861	2,365,026	6.00
		Schoharie	3	1	43	1,813,569	604,523	4,231,659	10.73
				2	48	2,827,151	942,384	6,596,685	16.73
4	7: Central New York	Onondaga	4	1	41	1,275,708	425,236	2,976,651	7.55
		Tompkins	12	1	41	332,310	110,770	775,390	1.97
				2	58	1,608,018	536,006	3,752,041	9.51
6	3: Lower Hudson Valley	Putnam	9	1	42	700,623	233,541	1,634,787	4.15
		Rockland	8	1	42	848,804	282,935	1,980,541	5.02
				2	53	1,549,427	516,476	3,615,328	9.17
5	8: Western Finger Lakes	Livingston	14	1	24	94,845	31,615	221,305	0.56
		Ontario	7	1	8	969,339	323,113	2,261,790	5.73
		Steuben	11	1	54	349,783	116,594	816,160	2.07
				3	54	1,413,967	471,322	3,299,255	8.37
				18	59	16,902,175	5,634,058	39,438,401	100%



### 3.17e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals linked to congenital malformations, deformations & chromosomal abnormalities.

Herkimer County ranked first with 10.7 million pounds or (27%) of the state total, followed by Erie County (6.8 million pounds or 17.1%) and Schoharie County (4.2 million pounds or 10.7%). These three counties are responsible for 21.7 million pounds (56.4%) of all toxic releases.

The top five counties were responsible for 27.4 million pounds or more than two-thirds (69.34) of the state total.

The country average was 2.8 million pounds over a 7-year period or 402,433 pounds annually.

Table 3.17e.

#### Congenital Malformations, Deformations & Chromosomal Abnormalities by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	58	4,571,891	1,523,964	10,667,747	27.05
2	Erie	9: Western New York	2	47	2,894,998	964,999	6,754,994	17.13
3	Schoharie	4: Capital Region/Northern Catskills	1	43	1,813,569	604,523	4,231,660	10.73
4	Onondaga	7: Central New York	1	41	1,275,708	425,236	2,976,652	7.55
5	Allegany	9: Western New York	2	20	1,166,673	388,891	2,722,237	6.90
6	Columbia	4: Capital Region/Northern Catskills	1	24	1,013,583	337,861	2,365,027	6.00
7	Ontario	8: Western Finger Lakes	1	8	969,339	323,113	2,261,791	5.73
8	Rockland	3: Lower Hudson Valley	1	42	848,804	282,935	1,980,542	5.02
9	Putnam	3: Lower Hudson Valley	1	42	700,623	233,541	1,634,787	4.15
10	Chautauqua	9: Western New York	2	54	665,084	221,695	1,551,862	3.93
11	Steuben	8: Western Finger Lakes	1	54	349,783	116,594	816,160	2.07
12	Tompkins	7: Central New York	1	41	332,310	110,770	775,390	1.97
13	Niagara	9: Western New York	1	24	204,966	68,322	478,255	1.21
14	Livingston	8: Western Finger Lakes	1	24	94,845	31,615	221,306	0.56
			<b>18</b>	<b>59</b>	<b>16,902,175</b>	<b>5,634,058</b>	<b>39,438,408</b>	<b>100%</b>

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## 3.18. Symptoms, Signs and Abnormal Clinical and Laboratory Findings (R00-R99)

### 3.18a. Releases by Chemicals

Forty-three of the 70 chemicals released by NYS natural gas compressor stations are associated with congenital malformations, deformations and chromosomal abnormalities (ICD-10, Chapter 18).

Releases of these toxicants were reported by all 18 stations and totaled an estimated 39.4 million pounds from 2008 to 2014--an annual average of 5.6 million pounds.

Or, to put it differently, ***of the 40.2 million pounds of chemicals released by NYS's compressor stations, 98% have been associated with symptoms, signs and abnormal clinical and laboratory findings.***

Nitrogen oxides ranked first with 18.1 million pounds, slightly less than one-half (45.9%) of the state total. Carbon monoxide was second (12.4 million pounds or 31.3%), followed by volatile organic compounds (4.9 million pounds or 12.5%). These three chemicals accounted for 35.4 million pounds or 89.7 of all releases.

The top 10 chemicals were responsible for virtually all releases (99.8%).

The annual average release was 5.6 million pounds.

Table 3.18a.

#### Symptoms, Signs and Abnormal Clinical and Laboratory Findings by Chemical (Top 10)

NYS Natural Gas Compressor Stations, 2008-2014

Chemical		Location			3 Years	7 Year Estimate: 2008 to 2014		
Rank	Name	Fac's	Cn's	Reg's	Pounds	Average	Pounds	%
1	Nitrogen Oxides	18	14	6	7,749,673	2,583,224	18,082,571	45.85
2	Carbon Monoxide	18	14	6	5,297,028	1,765,676	12,359,731	31.34
3	Volatile Organic Compounds	18	14	6	2,108,741	702,914	4,920,396	12.48
4	Formaldehyde	18	14	6	561,144	187,048	1,309,336	3.32
5	PM10 Primary (Filt + Cond)	18	14	6	539,890	179,963	1,259,744	3.19
6	PM2.5 Primary (Filt + Cond)	18	14	6	474,085	158,028	1,106,198	2.80
7	Sulfur Dioxide	18	14	6	80,048	26,683	186,778	0.47
8	Acetaldehyde	14	13	6	28,272	9,424	65,969	0.17
9	Acrolein	14	13	6	22,596	7,532	52,723	0.13
10	Benzene	16	13	6	9,103	3,034	21,241	0.05
		18	14	6	16,870,580	5,623,527	39,364,686	99.81

### 3.18b. Releases by ICD Category

Symptoms, signs and abnormal clinical and laboratory findings into 3 major disease categories. Chemicals released by natural gas compressor stations in NYS are positively associated with two.

It should be remembered, that a single chemical can be associated with more than one subcategory of disease within an ICD disease group (chapter) as well as with more than one disease group.

#### **R00-R69: Symptoms and signs**

Forty-two chemicals are associated with symptoms, signs and abnormal clinical and laboratory findings. This includes:

**R00-R09:** Thirty chemicals are associated with circulatory and respiratory system symptoms: changes in blood pressure, chest discomfort (burning sensation, constriction, pain), dyspnea, epistaxis, heart palpitations, heart rate (bradycardia, tachycardia), phlegm, pulse rate decrease without fall in blood pressure, pulse rate increase without fall in blood pressure, and wheezing.

**R10-R19:** Twenty-eight chemicals associated with digestive system and abdomen symptoms: abdomen (colic, cramps and pain), nausea or vomiting, and unspecified liver effects.

**R20-R23:** Thirty-two chemicals are associated with skin and subcutaneous tissue symptoms: skin (burning sensation, cracking, discoloration, pain, paresthesia, and rash), numbness in extremities, and a tingling sensation.

**R30-R39:** One chemical is associated with urinary system symptoms.

**R40-R46:** Thirty-four chemicals are associated with cognition, perception, emotional state and behavior: R40 (drowsiness, dizziness, somnolence), R41 (amnesia, confusion, memory disturbances, impairment and loss, mental confusion), R42 (dizziness, giddiness), R42 (lightheadedness and vertigo), R43 (anosmia, olfactory fatigue and unspecified effects), R43 (metallic or unpleasant taste in mouth), R45 (agitation or irritability), and R53 (lethargy).

**R47-R49:** Four chemicals are associated with speech and voice symptoms: R47 (speech (difficulties, disorders and impairment), and R49 (voice loss and disturbances).

**R50-R69:** Thirty-five chemicals are associated with general symptoms and signs: R50 (fever), R51 (headache), R53 (asthenia, alteration of classical conditioning, fatigue, listlessness, malaise, weakness), R55 (lowered consciousness), R56 (convulsions), R61 (diaphoresis), R63 (loss or decreased weight gain), R63 (altered fluid and food intake), R68 (chills), and R68 (decreased libido).

#### **R70-R94: Abnormal clinical and laboratory findings, not elsewhere classified**

Five chemicals are associated with abnormal clinical and laboratory findings, not elsewhere classified.

Table 3.18b.

## Symptoms, Signs and Abnormal Clinical and Laboratory Findings by ICD Code Category

NYS Natural Gas Compressor Stations, 2008-2014

ICD-10			Facilities				Chemicals				Pounds			
#	Description		'08	'11	'14	Tot	'08	'11	'14	Tot	2008	2011	2014	Total
1	R00-R69	Symptoms and signs	18	18	17	18	42	42	42	42	4,301,427	6,386,827	5,740,302	16,428,557
1.1	R00-R09	Circulatory and respiratory systems	18	18	17	18	30	30	30	30	2,775,402	4,111,790	3,698,584	10,585,778
1.2	R10-R19	Digestive system and abdomen	18	18	17	18	28	28	28	28	4,296,286	6,369,501	5,729,821	16,395,609
1.3	R20-R23	Skin and subcutaneous tissue	18	18	16	18	32	32	32	32	233,958	516,707	441,385	1,192,051
1.4	R25-R29	Nervous and musculoskeletal systems	18	18	16	18	26	26	26	26	122,751	262,577	244,109	629,437
1.5	R30-R39	Urinary system	6	7	8	8	1	1	1	1	1,381	4,324	2,580	8,285
1.6	R40-R46	Cognition, perception, emotional state and behavior	18	18	17	18	34	34	34	34	1,913,213	3,125,300	2,997,323	8,035,836
1.7	R47-R49	Speech and voice	18	17	16	18	4	4	4	4	111,704	233,516	224,349	569,571
1.8	R50-R69	General symptoms and signs	18	18	17	18	35	35	35	35	4,293,722	6,372,351	5,681,897	16,347,971
2	R70-R94	Abnormal clinical and laboratory findings, not elsewhere classified	18	18	17	18	5	5	5	5	1,508,969	2,253,122	2,012,719	5,774,810
3	R95-R99	Ill-defined and unknown causes of mortality	0	0	0	0	0	0	0	0	0	0	0	0
	<b>R00-R99</b>	<b>Total</b>	<b>18</b>	<b>18</b>	<b>17</b>	<b>18</b>	<b>43</b>	<b>43</b>	<b>43</b>	<b>43</b>	<b>4,394,022</b>	<b>6,607,810</b>	<b>5,900,809</b>	<b>16,902,642</b>

### 3.18c. Releases by Facility

All natural gas compressor stations in NYS reported chemical releases associated with symptoms, signs and abnormal clinical and laboratory findings.

The top 6 polluters were facilities operated by the Tennessee Gas Pipeline Company.

Tennessee Gas Pipeline Company Compressor Station 245, ranked first with 10.4 million pounds, more than one-quarter (26.2%) of the state total. Compressor Station 229 in Eden ranked second (5. million pounds or 12.7%) followed by Compressor Station 249 in Carlisle (4.2 million pounds or 10.7%). In aggregate, these three facilities were responsible for 19.7 million pounds, slightly less than one-half (49.6%) of the state total.

The top 5 facilities were responsible for 25 million pounds, slightly less than two-thirds (63.1%) of all releases.

The facility average was 2.1 million pounds over 7 years or 314,652 pounds each year.

Table 3.18c.

#### Symptoms, Signs and Abnormal Clinical and Laboratory Findings by Facility (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

Facility		Location		Chemicals			7 Years (estimate)		
Rank	Facility Name (Short)	Town	County	'08	'11	'14	Average	Tot. Lbs.	%
1	TGPC CS 245	Winfield	Herkimer	34	34	23	1,484,228	10,389,599	26.21
2	TGPC 229 & TEG DF	Eden	Erie	32	31	32	721,386	5,049,701	12.74
3	TGPC CS 249	Carlisle	Schoharie	34	24	24	604,520	4,231,638	10.67
4	TGPC CS 241	LaFayette	Onondaga	33	26	33	425,231	2,976,619	7.51
5	TGPC CS 254	Chatham	Columbia	22	15	8	337,861	2,365,025	5.97
6	TGPC CS 237	Manchester, Phelps	Ontario	8	7	5	323,113	2,261,791	5.70
7	AGT Stony Point CS	Stony Point	Rockland	35	21	21	282,934	1,980,537	5.00
8	NFGSC Concord CS	Concord	Erie	10	9	11	243,583	1,705,081	4.30
9	AGT SOUTHEAST CS	Southeast	Putnam	22	17	28	233,540	1,634,783	4.12
10	NFGSC Beech Hill CS	Willing	Allegany	19	19	20	196,224	1,373,569	3.46
11	NFGSC Independence CS	Andover	Allegany	14	9	16	192,806	1,349,642	3.40
12	TGPC CS 224	Clymer	Chautauqua	34	32	34	162,642	1,138,497	2.87
13	DTI Woodhull Station	Woodhull	Steuben	28	35	39	116,642	816,492	2.06
14	DTI Borger CS	Ithaca	Tompkins	28	28	18	110,772	775,401	1.96
15	NFGSC Nashville CS	Hanover	Chautauqua	22	22	0	88,588	620,114	1.56
16	TGPC CS 230-C	Lockport	Niagara	22	21	22	68,321	478,245	1.21
17	DTI Utica Station	Frankfort	Herkimer	22	26	38	39,738	278,165	0.70
18	TGPC CS 233	York	Livingston	22	15	4	31,615	221,303	0.56
				<b>43</b>	<b>43</b>	<b>43</b>	<b>5,663,743</b>	<b>39,646,203</b>	<b>100%</b>

### 3.18d. Releases by DEC Region

The 18 compressor stations analyzed are in 6 of New York State's 9 DEC regions. All 6 regions had releases of chemicals associated with symptoms, signs and abnormal clinical and laboratory findings.

Region 9, Western New York, ranked first with 11.5 million pounds (29.2%), closely followed by Region 6, Western Adirondacks/Eastern Lake Ontario (10.7 million pounds or 27.1%). Region 4, Capital Region/Northern Catskills, ranked third with 6.7 million pounds (16.7%).

Table 3.18e.

#### Symptoms, Signs and Abnormal Clinical and Laboratory Findings by DEC Region (ranked)

NYS Natural Gas Compressor Station NEI Emissions, 2008 to 2011

NYS DEC Region		County		3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
Rank	Number \ Name	Name	Rank	Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	9: Western New York	Allegany	5	2	21	1,167,091	389,030	2,723,211	6.90
		Chautauqua	10	2	42	665,103	221,701	1,551,907	3.93
		Erie	2	2	35	2,894,907	964,969	6,754,782	17.13
		Niagara	13	1	22	204,962	68,321	478,244	1.21
				7	43	4,932,063	1,644,021	11,508,144	29.18
2	6: W Adirondacks / E Lake Ontario	Herkimer	1	2	42	4,571,899	1,523,966	10,667,764	27.05
3	4: Capital Region / N Catskills	Columbia	6	1	22	1,013,582	337,861	2,365,024	6.00
		Schoharie	3	1	34	1,813,559	604,520	4,231,637	10.73
				2	38	2,827,141	942,380	6,596,661	16.73
4	7: Central New York	Onondaga	4	1	33	1,275,694	425,231	2,976,619	7.55
		Tompkins	12	1	28	332,315	110,772	775,401	1.97
				2	43	1,608,009	536,003	3,752,020	9.51
6	3: Lower Hudson Valley	Putnam	9	1	30	700,621	233,540	1,634,782	4.15
		Rockland	8	1	36	848,801	282,934	1,980,536	5.02
				2	40	1,549,423	516,474	3,615,318	9.17
5	8: Western Finger Lakes	Livingston	14	1	22	94,844	31,615	221,302	0.56
		Ontario	7	1	8	969,339	323,113	2,261,790	5.73
		Steuben	11	1	40	349,925	116,642	816,492	2.07
				3	40	1,414,108	471,369	3,299,584	8.37
				18	43	16,902,642	5,634,214	39,439,491	100%

### 3.18e. Releases by County

All 14 counties where compressor stations are located reported releases of chemicals associated with symptoms, signs and abnormal clinical and laboratory findings.

Herkimer County ranked first with 10.7 million pounds or (27%) of the state total, followed by Erie County (6.8 million pounds or 17.1%) and Schoharie County (4.2 million pounds or 10.7%). These three counties are responsible for 21.7 million pounds (54.9%) of all toxic releases.

The top five counties were responsible for 27.4 million pounds or more than two-thirds (69.4%) of the state total.

The country average was 2.8 million pounds over a 7-year period or 402,444 pounds annually.

Table 3.18e.

#### Symptoms, Signs and Abnormal Clinical and Laboratory Findings by County (ranked)

NYS Natural Gas Compressor Stations, 2008-2014

Rank	County	NYS DEC Region	3 Years: 2008, 2011, 2014			7-Year Estimate: 2008-2014		
			Fac's	Ch's	Total Pounds	Average Pounds	Total Pounds	%
1	Herkimer	6: Western Adirondacks/E. Lake Ontario	2	42	4,571,899	1,523,966	10,667,764	27.05
2	Erie	9: Western New York	2	35	2,894,907	964,969	6,754,782	17.13
3	Schoharie	4: Capital Region/Northern Catskills	1	34	1,813,559	604,520	4,231,637	10.73
4	Onondaga	7: Central New York	1	33	1,275,694	425,231	2,976,619	7.55
5	Allegany	9: Western New York	2	21	1,167,091	389,030	2,723,211	6.90
6	Columbia	4: Capital Region/Northern Catskills	1	22	1,013,582	337,861	2,365,024	6.00
7	Ontario	8: Western Finger Lakes	1	8	969,339	323,113	2,261,790	5.73
8	Rockland	3: Lower Hudson Valley	1	36	848,801	282,934	1,980,536	5.02
9	Putnam	3: Lower Hudson Valley	1	30	700,621	233,540	1,634,782	4.15
10	Chautauqua	9: Western New York	2	42	665,103	221,701	1,551,907	3.93
11	Steuben	8: Western Finger Lakes	1	40	349,925	116,642	816,492	2.07
12	Tompkins	7: Central New York	1	28	332,315	110,772	775,401	1.97
13	Niagara	9: Western New York	1	22	204,962	68,321	478,244	1.21
14	Livingston	8: Western Finger Lakes	1	22	94,844	31,615	221,302	0.56
			<b>18</b>	<b>43</b>	<b>16,902,642</b>	<b>5,634,214</b>	<b>39,439,491</b>	<b>100%</b>



## Chapter 4. Facility Profiles

Sources:

New York State Department of Environmental Conservation, U.S. Energy Information Administration, U.S. EPA Envirofacts, U.S. EPA National Emissions Inventory

\* System Configuration - natural gas pipeline system design layout. Some systems are a combination of the trunk and grid. Where two are shown, the first represents the predominant system design.

Trunk - systems are large-diameter long-distance trunklines that generally tie supply areas to natural gas market areas.

Grid - systems are usually a network of many interconnections and delivery points that operate in and serve major natural gas market areas

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## 4.1. Algonquin Gas Southeast Compressor Station (Putnam, New York)

### 4.1a. Facility Profile

Table 4.1a.

#### Algonquin Gas Southeast Compressor Station: Facility Profile

Putnam NY

Facility name, short	AGT SOUTHEAST CS Southeast
Facility name, full	Algonquin Gas Southeast Compressor Station
EIS Facility ID	8474311
DEC Region	3 -- Lower Hudson Valley
County	Putnam
Town	Southeast
Village \ Hamlet	Brewster
Address	142 Tulip Rd
Zip	10509
DEC Facility ID	3373000060
DEC Permit Type	Air State Facility
DEC Permit ID	3-3730-00060/00013
DEC Permit Effective Date	7/15/2015
DEC Permit Description	
DEC Permit Review Report	
Company	Algonquin Gas Transmission LLC
Project	Algonquin Incremental Market (AIM)
Pipeline	Algonquin
Principal Supply Source	Interstate System
System Configuration (Primary / Secondary) *	Trunk/Grid
Status	Operational
Horsepower, existing	10,302
Horsepower, modifications\expansion	43,640

## 4.1b. Health Effects of Facility Releases

Table 4.1b.

### Algonquin Gas Southeast Compressor Station: Health Effects of Releases by ICD-10 Chapter & Group

Putnam NY

International Classification of Disease, 10 <sup>th</sup> edition			Ch #	2008-14 Estimated Lbs.		State Rank	% of State
Ch.	Description	Code		Average	Total		
2	Neoplasms	C00-D48	40	72,072	504,510	7	5.26
2a	Malignant neoplasms	C00-C97	37	67,423	471,962	7	5.02
2a.1	Lip, oral cavity and pharynx	C00-C14	11	5,788	40,519	12	2.81
2a.2	Digestive organs	C15-C26	23	5,830	40,810	12	2.79
2a.3	Respiratory system and intrathoracic organs	C30-C39	28	67,391	471,742	7	5.04
2a.4	Bone and articular cartilage	C40-C41	26	59,530	416,713	7	4.75
2a.5	Skin	C43-C44	8	288	2,021	4	7.58
2a.6	Connective and soft tissue	C45-C49	13	614	4,303	3	15.74
2a.07	Breast and female genital organs	C50-C58	15	36,301	254,113	7	5.86
2a.07.50	Female breast	C50	13	28,590	200,134	8	5.28
2a.07.55	Uterus	C55	3	9	64	9	1.31
2a.07.56	Ovary	C56	3	289	2,025	4	7.77
2a.08	Male genital organs	C60-C63	8	4,587	32,109	11	2.65
2a.09	Urinary organs	C64-C68	16	5,538	38,766	12	2.69
2a.10	Eye, brain and central nervous system	C69-C72	14	5,828	40,796	12	2.79
2a.11	Endocrine glands and related structures	C73-C75	7	4,481	31,369	11	2.57
2a.12	Secondary and ill-defined	C76-C80	6	979	6,858	2	20.69
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic and related tissue	C81-C96	20	37,129	259,904	7	5.93
2a.14	Malignant neoplasms of independent (primary) multiple sites	C97	0	0	0	0	0
2b	In situ neoplasms	D00-D09	2	834	5,841	3	13.98
2c	Benign neoplasms	D10-D36	17	967	6,771	8	4.09
2d	Neoplasms of uncertain or unknown behavior	D37-D48	27	5,582	39,074	12	2.69
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	24	62,269	435,885	13	2.31
4	Endocrine, nutritional and metabolic diseases	E00-E90	37	48,166	337,165	8	4.71
5	Mental and behavioral disorders	F00-F99	21	62,261	435,828	13	2.31
6	Diseases of the nervous system	G00-G99	26	67,069	469,483	13	2.46
7	Diseases of the eye and adnexa	H00-H59	27	183,354	1,283,479	7	5.17
8	Diseases of the ear and mastoid process	H60-H95	12	57,674	403,718	13	2.31
9	Diseases of the circulatory system	I00-I99	20	55,141	385,987	13	2.38
10	Diseases of the respiratory system	J00-J99	35	233,542	1,634,795	9	4.12
11	Diseases of the digestive system	K00-K93	31	222,553	1,557,872	9	4.05
12	Diseases of the skin and subcutaneous tissue	L00-L99	34	214,802	1,503,615	7	5.42
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	13	11,741	82,188	6	6.66
14	Diseases of the genitourinary system	N00-N99	28	236,452	1,655,165	9	4.14
14a	Diseases of the genitourinary system: urinary system	N00-N39	20	35,528	248,697	10	3.88
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	25	236,445	1,655,115	9	4.14
15	Pregnancy, childbirth and the puerperium	O00-O99	11	148,254	1,037,778	5	5.29
16	Certain conditions originating in the perinatal period	P00-P96	18	179,875	1,259,125	5	5.59
17	Congenital malformations, deformations, chromosomal abnormalities	Q00-Q99	42	233,541	1,634,787	9	4.12
18	Symptoms, signs and abnormal cl. and laboratory findings, nec	R00-R99	30	233,540	1,634,782	9	4.12
<b>Total Releases</b>			<b>48</b>	<b>241,259</b>	<b>1,688,814</b>	<b>9</b>	<b>4.20</b>

## 4.2. Algonquin Gas Stony Point Compressor Station (Stony Point, New York)

### 4.2a. Facility Profile

Table 4.2a.

#### Algonquin Gas Stony Point Compressor Station: Facility Profile

Stony Point NY

Facility name, short	AGT Stony Point CS
Facility name, full	Algonquin Gas Stony Point Compressor Station
EIS Facility ID	7952911
DEC Region	3 -- Lower Hudson Valley
County	Rockland
Town	Stony Point
Village \ Hamlet	
Address	1 Lindberg Rd
Zip	10980
DEC Permit Type	Air Title V Facility
DEC Facility ID	3392800001
DEC Permit ID	3-3928-00001/00027
DEC Permit Effective Date	12/21/2015
Company	Algonquin Gas Transmission LLC
Project	Algonquin Incremental Market (AIM)
Pipeline	Algonquin
Principal Supply Source	Interstate System
System Configuration (Primary / Secondary) *	Trunk/Grid
Facility Status	Operational \ Expansion under review
Facility Status Dates	
Horsepower, existing	12,000
Horsepower, modifications\expansion	One new compressor to be added at this site.

## 4.2b. Health Effects of Facility Releases

Table 4.2b.

### Algonquin Gas Stony Point Compressor Station: Health Effects of Facility Releases

Stony Point NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	5	42	110,464	773,249	8.07
2a	Malignant neoplasms	C00-C97	5	40	106,763	747,345	7.95
2a.1	Lip, oral cavity and pharynx	C00-C14	7	10	12,190	85,332	5.92
2a.2	Digestive organs	C15-C26	7	30	12,784	89,492	6.12
2a.3	Respiratory system and intrathoracic organs	C30-C39	5	29	106,158	743,106	7.93
2a.4	Bone and articular cartilage	C40-C41	5	26	100,507	703,555	8.02
2a.5	Skin	C43-C44	2	7	650	4,553	17.06
2a.6	Connective and soft tissue	C45-C49	1	13	1,073	7,515	27.49
2a.07	Breast and female genital organs	C50-C58	8	17	33,931	237,522	5.48
2a.07.50	Female breast	C50	7	16	29,326	205,287	5.41
2a.07.55	Uterus	C55	3	3	99	698	14.27
2a.07.56	Ovary	C56	2	3	649	4,548	17.44
2a.08	Male genital organs	C60-C63	6	10	9,786	68,506	5.66
2a.09	Urinary organs	C64-C68	7	19	11,842	82,899	5.75
2a.10	Eye, brain and central nervous system	C69-C72	7	16	12,800	89,604	6.13
2a.11	Endocrine glands and related structures	C73-C75	6	10	9,756	68,294	5.59
2a.12	Secondary and ill-defined	C76-C80	1	5	1,541	10,792	32.56
2a.13	Malignant neoplasms, lymphoid, haematopoietic, related	C81-C96	8	28	35,662	249,634	5.69
2a.14	Malignant neoplasms of independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	2	3	1,444	10,109	24.19
2c	Benign neoplasms	D10-D36	2	22	3,270	22,896	13.84
2d	Neoplasms of uncertain or unknown behavior	D37-D48	7	30	12,248	85,739	5.90
3	Diseases of the blood and blood-forming organs, immune mechanism	D50-D89	7	29	154,987	1,084,914	5.75
4	Endocrine, nutritional and metabolic diseases	E00-E90	4	35	90,940	636,585	8.89
5	Mental and behavioral disorders	F00-F99	7	28	154,887	1,084,215	5.74
6	Diseases of the nervous system	G00-G99	7	36	159,633	1,117,432	5.84
7	Diseases of the eye and adnexa	H00-H59	6	33	192,160	1,345,120	5.42
8	Diseases of the ear and mastoid process	H60-H95	7	14	144,423	1,010,967	5.79
9	Diseases of the circulatory system	I00-I99	10	26	102,539	717,779	4.42
10	Diseases of the respiratory system	J00-J99	7	42	282,933	1,980,536	5.00
11	Diseases of the digestive system	K00-K93	7	37	273,624	1,915,369	4.98
12	Diseases of the skin and subcutaneous tissue	L00-L99	6	39	215,263	1,506,847	5.43
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	7	14	11,114	77,802	6.31
14	Diseases of the genitourinary system	N00-N99	7	36	282,892	1,980,244	4.96
14a	Diseases of the genitourinary system: urinary system	N00-N39	4	27	82,163	575,141	8.97
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	7	30	282,888	1,980,219	4.96
15	Pregnancy, childbirth and the puerperium	O00-O99	8	16	117,277	820,945	4.18
16	Certain conditions originating in the perinatal period	P00-P96	8	18	139,970	979,796	4.35
17	Congenital malformations, deformations and chromosomal ab.	Q00-Q99	7	42	282,934	1,980,541	5.00
18	Symptoms, signs and abnormal clinical, laboratory findings, nec	R00-R99	7	36	282,933	1,980,536	5.00
<b>Total Releases</b>			<b>7</b>	<b>49</b>	<b>287,639</b>	<b>2,013,478</b>	<b>5.01</b>

## 4.3. DTI E.M. Borger Compressor Station (Ithaca NY)

### 4.3a. Facility Profile

Table 4.3a.

#### DTI E.M. Borger Compressor Station

Ithaca NY

Facility name, short	DTI Borger CS
Facility name, full	DTI E.M. Borger Compressor Station
EIS Facility ID	8542411
DEC Region	7 -- Central New York
County	Tompkins
Town	Ithaca
Village \ Hamlet	
Address	219 Ellis Hollow Creek
Zip	14850
DEC Permit Type	Air State Facility
DEC Facility ID	7502400007
DEC Permit ID	7-5024-00007/00004
DEC Permit Effective Date	01/08/2014
Company	Dominion Transportation Inc.
Project	New Market Project
Pipeline	Dominion
Principal Supply Source	
System Configuration (Primary / Secondary) *	
Facility Status	Operational
Facility Status Dates	
Horsepower, existing	18,430 HP
Horsepower, modifications\expansion	(1) Dresser Clark DC 990 5800 HP ngfsct, (1) Dresser Clark DC 990 5800 HP ngfsct, (1) Dresser Clark DC 990 5800 HP ngfsct, (1) Solar Turbines Inc. Taurus 70-1030S HP ngfsct



## 4.3b. Health Effects of Facility Releases

Table 4.2b.

### DTI E.M. Borger Compressor Station: Health Effects of Facility Releases

Ithaca NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	17	40	7,571	52,998	0.55
2a	Malignant neoplasms	C00-C97	17	37	6,881	48,166	0.51
2a.1	Lip, oral cavity and pharynx	C00-C14	18	12	189	1,322	0.09
2a.2	Digestive organs	C15-C26	18	21	198	1,389	0.10
2a.3	Respiratory system and intrathoracic organs	C30-C39	17	29	6,875	48,128	0.51
2a.4	Bone and articular cartilage	C40-C41	16	27	6,195	43,362	0.49
2a.5	Skin	C43-C44	13	10	2	16	0.06
2a.6	Connective and soft tissue	C45-C49	13	12	30	211	0.77
2a.07	Breast and female genital organs	C50-C58	18	14	3,040	21,283	0.49
2a.07.50	Female breast	C50	18	12	2,361	16,525	0.44
2a.07.55	Uterus	C55	11	3	0	1	0.01
2a.07.56	Ovary	C56	13	3	2	16	0.06
2a.08	Male genital organs	C60-C63	18	7	153	1,070	0.08
2a.09	Urinary organs	C64-C68	18	15	175	1,227	0.09
2a.10	Eye, brain and central nervous system	C69-C72	18	12	191	1,338	0.09
2a.11	Endocrine glands and related structures	C73-C75	18	6	142	997	0.07
2a.12	Secondary and ill-defined	C76-C80	13	6	43	300	0.90
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	18	19	3,079	21,553	0.49
2a.14	Neoplasms of independent (primary) multiple sites	C97	0	0	0	0	0.00
2b	In situ neoplasms	D00-D09	13	2	27	188	0.45
2c	Benign neoplasms	D10-D36	13	15	27	187	0.11
2d	Neoplasms of uncertain or unknown behavior	D37-D48	18	26	186	1,305	0.09
3	Diseases of the blood, blood-forming organs, immune mechanism	D50-D89	17	22	21,652	151,564	0.80
4	Endocrine, nutritional and metabolic diseases	E00-E90	17	36	5,354	37,476	0.52
5	Mental and behavioral disorders	F00-F99	17	20	21,652	151,563	0.80
6	Diseases of the nervous system	G00-G99	17	24	22,343	156,404	0.82
7	Diseases of the eye and adnexa	H00-H59	11	25	90,898	636,288	2.56
8	Diseases of the ear and mastoid process	H60-H95	16	12	21,502	150,516	0.86
9	Diseases of the circulatory system	I00-I99	16	19	20,045	140,315	0.86
10	Diseases of the respiratory system	J00-J99	14	33	110,772	775,401	1.96
11	Diseases of the digestive system	K00-K93	14	29	110,016	770,114	2.00
12	Diseases of the skin and subcutaneous tissue	L00-L99	11	33	93,789	656,521	2.37
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	18	12	798	5,584	0.45
14	Diseases of the genitourinary system	N00-N99	14	26	110,760	775,319	1.94
14a	Diseases of the genitourinary system: urinary system	N00-N39	16	18	3,987	27,909	0.44
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	14	23	110,758	775,306	1.94
15	Pregnancy, childbirth and the puerperium	O00-O99	10	11	86,398	604,785	3.08
16	Certain conditions originating in the perinatal period	P00-P96	10	16	89,289	625,024	2.78
17	Congenital malformations, deformations, chromosomal abnormalities	Q00-Q99	14	41	110,770	775,390	1.96
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	14	28	110,772	775,401	1.96
<b>Total Releases</b>			<b>14</b>	<b>47</b>	<b>111,451</b>	<b>780,159</b>	<b>1.94</b>

## 4.4. DTI Utica Station (Frankfurt NY)

### 4.4a. Facility Profile

Table 4.4a.

#### DTI Utica Station

#### Frankfurt NY

Facility name, short	DTI Utica Station
Facility name, full	DTI Utica Station
EIS Facility ID	8035211
DEC Region	6 -- Western Adirondacks/Eastern Lake Ontario
County	Herkimer
Town	Frankfort
Village \ Hamlet	
Address	1103 Higby Rd
Zip	13340
DEC Permit Type	Air Title V Facility
DEC Facility ID	6212600037
DEC Permit ID	6-2126-00037/00025
DEC Permit Effective Date	5/25/2016
DEC Permit Description	Application for renewal of Air Title V Facility.
DEC Permit Review Report	
Company	Dominion Transmission Inc.
Project	New Market Project
Pipeline	Dominion
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	5,550
	(5) 1,100 hp Cooper Bessemer GMVC-6 compressor units

## 4.4b. Health Effects of Facility Releases

Table 4.2b.

### DTI Utica Station: Health Effects of Facility Releases

Frankfurt NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	15	51	12,696	88,873	0.93
2a	Malignant neoplasms	C00-C97	15	48	12,660	88,622	0.94
2a.1	Lip, oral cavity and pharynx	C00-C14	15	13	2,295	16,063	1.12
2a.2	Digestive organs	C15-C26	15	30	2,356	16,491	1.13
2a.3	Respiratory system and intrathoracic organs	C30-C39	15	34	12,589	88,120	0.94
2a.4	Bone and articular cartilage	C40-C41	15	31	11,862	83,035	0.95
2a.5	Skin	C43-C44	6	13	95	665	2.49
2a.6	Connective and soft tissue	C45-C49	11	14	61	427	1.56
2a.07	Breast and female genital organs	C50-C58	16	19	4,537	31,755	0.73
2a.07.50	Female breast	C50	16	17	4,098	28,687	0.76
2a.07.55	Uterus	C55	6	3	27	189	3.86
2a.07.56	Ovary	C56	6	3	94	660	2.53
2a.08	Male genital organs	C60-C63	15	11	1,947	13,631	0.97
2a.09	Urinary organs	C64-C68	15	22	2,324	16,269	1.13
2a.10	Eye, brain and central nervous system	C69-C72	15	18	2,367	16,568	1.13
2a.11	Endocrine glands and related structures	C73-C75	15	9	2,003	14,019	0.99
2a.12	Secondary and ill-defined	C76-C80	12	6	44	306	0.92
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	16	27	4,637	32,462	0.74
2a.14	Independent (primary) multiple sites	C97	0	0	0	0	0
2b	In situ neoplasms	D00-D09	7	3	100	702	1.68
2c	Benign neoplasms	D10-D36	9	20	669	4,682	2.83
2d	Neoplasms of uncertain or unknown behavior	D37-D48	15	35	2,347	16,430	1.13
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	16	31	22,270	155,891	0.83
4	Endocrine, nutritional and metabolic diseases	E00-E90	15	45	10,601	74,209	1.04
5	Mental and behavioral disorders	F00-F99	16	30	22,243	155,702	0.83
6	Diseases of the nervous system	G00-G99	16	35	22,575	158,022	0.83
7	Diseases of the eye and adnexa	H00-H59	17	34	25,770	180,386	0.73
8	Diseases of the ear and mastoid process	H60-H95	17	15	20,007	140,049	0.80
9	Diseases of the circulatory system	I00-I99	17	26	16,299	114,095	0.70
10	Diseases of the respiratory system	J00-J99	17	43	39,738	278,165	0.70
11	Diseases of the digestive system	K00-K93	17	40	38,557	269,901	0.70
12	Diseases of the skin and subcutaneous tissue	L00-L99	17	44	28,034	196,235	0.71
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	16	15	1,140	7,977	0.65
14	Diseases of the genitourinary system	N00-N99	17	37	39,872	279,102	0.70
14a	Diseases of the genitourinary system: urinary system	N00-N39	15	28	10,060	70,421	1.10
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	17	31	39,864	279,049	0.70
15	Pregnancy, childbirth and the puerperium	O00-O99	18	16	17,827	124,788	0.64
16	Certain conditions originating in the perinatal period	P00-P96	18	20	19,886	139,202	0.62
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	17	52	39,732	278,121	0.70
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	17	38	39,738	278,165	0.70
<b>Total Releases</b>			<b>17</b>	<b>59</b>	<b>40,196</b>	<b>281,369</b>	<b>0.70</b>

## 4.5. DTI Woodhull Station (Woodhull NY)

### 4.5a. Facility Profile

Table 4.5a.

#### DTI Woodhull Station

#### Woodhull NY

Facility name, short	DTI Woodhull Station
Facility name, full	DTI Woodhull Station
EIS Facility ID	8437611
DEC Region	8
County	Steuben
Town	Woodhull
Village \ Hamlet	
Address	974 Co Rte 99
Zip	14898
DEC Permit Type	Air Title V Facility
DEC Facility ID	468200006
DEC Permit ID	8-4682-00006/00034
DEC Permit Effective Date	7/10/2014
DEC Permit Description	Title V Facility Permit renewal
DEC Permit Review Report	
Company	Dominion Transmission Inc.
Project	New Market Project
Pipeline	Dominion
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	14,700 HP
	(5) 2,000 HP reciprocating ngfce (2) 1,800 HP reciprocating ngfce (1) 1,100 HP reciprocating ngfce

## 4.5b. Health Effects of Facility Releases

Table 4.2b.

### DTI Woodhull Station: Health Effects of Facility Releases

Woodhull NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	11	53	47,086	329,602	3.44
2a	Malignant neoplasms	C00-C97	11	50	47,013	329,091	3.50
2a.1	Lip, oral cavity and pharynx	C00-C14	9	13	9,688	67,813	4.71
2a.2	Digestive organs	C15-C26	9	32	10,400	72,801	4.98
2a.3	Respiratory system and intrathoracic organs	C30-C39	11	35	46,203	323,418	3.45
2a.4	Bone and articular cartilage	C40-C41	11	32	43,265	302,854	3.45
2a.5	Skin	C43-C44	3	13	412	2,883	10.80
2a.6	Connective and soft tissue	C45-C49	5	15	276	1,929	7.06
2a.07	Breast and female genital organs	C50-C58	10	20	18,612	130,284	3.00
2a.07.50	Female breast	C50	10	18	16,924	118,465	3.12
2a.07.55	Uterus	C55	2	3	119	835	17.06
2a.07.56	Ovary	C56	3	3	402	2,811	10.78
2a.08	Male genital organs	C60-C63	10	11	8,216	57,511	4.11
2a.09	Urinary organs	C64-C68	9	23	9,842	68,892	4.78
2a.10	Eye, brain and central nervous system	C69-C72	9	18	10,419	72,931	4.99
2a.11	Endocrine glands and related structures	C73-C75	10	10	8,441	59,084	4.19
2a.12	Secondary and ill-defined	C76-C80	5	6	201	1,404	4.22
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	10	28	19,186	134,302	3.06
2a.14	Independent (primary) multiple sites	C97	0	0	0	0	0
2b	In situ neoplasms	D00-D09	4	3	431	3,015	7.22
2c	Benign neoplasms	D10-D36	3	22	3,270	22,892	13.84
2d	Neoplasms of uncertain or unknown behavior	D37-D48	9	36	10,353	72,472	4.98
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	12	32	86,683	606,778	3.21
4	Endocrine, nutritional and metabolic diseases	E00-E90	11	46	38,601	270,204	3.77
5	Mental and behavioral disorders	F00-F99	12	31	86,571	605,996	3.21
6	Diseases of the nervous system	G00-G99	12	37	87,890	615,229	3.22
7	Diseases of the eye and adnexa	H00-H59	14	36	59,457	416,201	1.68
8	Diseases of the ear and mastoid process	H60-H95	12	15	77,004	539,026	3.09
9	Diseases of the circulatory system	I00-I99	12	27	67,558	472,907	2.91
10	Diseases of the respiratory system	J00-J99	13	45	116,642	816,492	2.06
11	Diseases of the digestive system	K00-K93	13	42	111,868	783,072	2.04
12	Diseases of the skin and subcutaneous tissue	L00-L99	14	46	68,499	479,496	1.73
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	10	16	5,165	36,154	2.93
14	Diseases of the genitourinary system	N00-N99	13	39	117,138	819,966	2.05
14a	Diseases of the genitourinary system: urinary system	N00-N39	8	30	36,570	255,987	3.99
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	13	33	117,052	819,363	2.05
15	Pregnancy, childbirth and the puerperium	O00-O99	16	16	31,911	223,376	1.14
16	Certain conditions originating in the perinatal period	P00-P96	15	20	40,063	280,440	1.25
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	13	54	116,594	816,160	2.06
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	13	40	116,642	816,492	2.06
<b>Total Releases</b>			<b>13</b>	<b>61</b>	<b>118,460</b>	<b>829,223</b>	<b>2.06</b>

## 4.6. NFGSC Beech Hill Compressor Station (Willing NY)

### 4.6a. Facility Profile

Table 4.6a.

#### NFGSC Beech Hill Compressor Station

Willing NY

Facility name, short	NFGSC Beech Hill CS
Facility name, full	NFGSC Beech Hill Compressor Station
EIS Facility ID	8377711
DEC Region	9
County	Allegany
Town	Willing
Village \ Hamlet	
Address	1161 Peet Rd
Zip	14895
DEC Permit Type	Air Title V Facility
DEC Facility ID	9027400004
DEC Permit ID	9-0274-00004/00015
DEC Permit Effective Date	4/8/2013
DEC Permit Description	
DEC Permit Review Report	
Company	National Fuel Gas Supply Corp.
Project	Part of the Niagara Expansion Project and the Northern Access 2015 Project which are joint projects undertaken by National Fuel Gas Supply Corporation and Tennessee Gas Pipeline Company.
Pipeline	Empire (AKA "National Fuel")
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	8,350 HP
	(2) 2,750 HP reciprocating ngfce (1) 2,850 HP reciprocating ngfce
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.6b. Health Effects of Facility Releases

Table 4.6b.

### NFGSC Beech Hill Compressor Station: Health Effects of Facility Releases

Willing NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	13	19	37,053	259,370	2.71
2a	Malignant neoplasms	C00-C97	13	18	36,733	257,128	2.74
2a.1	Lip, oral cavity and pharynx	C00-C14	11	7	7,182	50,270	3.49
2a.2	Digestive organs	C15-C26	11	13	7,184	50,287	3.44
2a.3	Respiratory system and intrathoracic organs	C30-C39	13	15	36,636	256,453	2.74
2a.4	Bone and articular cartilage	C40-C41	13	13	34,633	242,427	2.77
2a.5	Skin	C43-C44	14	2	1	7	0.03
2a.6	Connective and soft tissue	C45-C49	15	4	12	80	0.30
2a.07	Breast and female genital organs	C50-C58	11	9	17,440	122,076	2.82
2a.07.50	Female breast	C50	12	8	15,436	108,053	2.85
2a.07.55	Uterus	C55	12	1	0	0	0.00
2a.07.56	Ovary	C56	14	2	1	7	0.03
2a.08	Male genital organs	C60-C63	11	4	7,072	49,504	3.54
2a.09	Urinary organs	C64-C68	11	8	7,081	49,564	3.44
2a.10	Eye, brain and central nervous system	C69-C72	11	10	7,086	49,599	3.39
2a.11	Endocrine glands and related structures	C73-C75	11	4	7,068	49,476	3.51
2a.12	Secondary and ill-defined	C76-C80	15	3	17	115	0.35
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	12	13	17,550	122,849	2.80
2a.14	Independent (primary) multiple sites	C97	0	0	0	0	0
2b	In situ neoplasms	D00-D09	14	2	10	72	0.17
2c	Benign neoplasms	D10-D36	16	9	9	64	0.04
2d	Neoplasms of uncertain or unknown behavior	D37-D48	11	12	7,179	50,255	3.46
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	9	15	140,703	984,922	5.22
4	Endocrine, nutritional and metabolic diseases	E00-E90	13	15	28,685	200,796	2.80
5	Mental and behavioral disorders	F00-F99	9	14	140,703	984,921	5.22
6	Diseases of the nervous system	G00-G99	9	17	141,024	987,167	5.16
7	Diseases of the eye and adnexa	H00-H59	13	17	73,519	514,635	2.07
8	Diseases of the ear and mastoid process	H60-H95	9	9	133,537	934,758	5.35
9	Diseases of the circulatory system	I00-I99	8	13	129,878	909,148	5.59
10	Diseases of the respiratory system	J00-J99	10	20	196,224	1,373,569	3.46
11	Diseases of the digestive system	K00-K93	10	18	192,242	1,345,694	3.50
12	Diseases of the skin and subcutaneous tissue	L00-L99	13	20	83,888	587,215	2.12
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	11	10	4,093	28,650	2.32
14	Diseases of the genitourinary system	N00-N99	10	19	197,907	1,385,347	3.47
14a	Diseases of the genitourinary system: urinary system	N00-N39	13	13	26,363	184,538	2.88
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	10	17	197,810	1,384,673	3.47
15	Pregnancy, childbirth and the puerperium	O00-O99	12	9	53,918	377,422	1.92
16	Certain conditions originating in the perinatal period	P00-P96	12	10	64,287	450,005	2.00
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	10	19	196,128	1,372,896	3.46
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	10	20	196,224	1,373,569	3.46
<b>Total Releases</b>			<b>10</b>	<b>21</b>	<b>198,227</b>	<b>1,387,592</b>	<b>3.45</b>

## 4.7. NFGSC Concord Compressor Station (Concord NY)

### 4.7a. Facility Profile

Table 4.7a.

#### NFGSC Concord Compressor Station

Concord NY

Facility name, short	NFGSC Concord Compressor Station
Facility name, full	NFGSC Concord CS
EIS Facility ID	8503411
DEC Region	9
County	Erie
Town	Concord
Village \ Hamlet	Springville
Address	5510 Genesee Rd
Zip	14141
DEC Permit Type	Air Title V Facility
DEC Facility ID	9143800044
DEC Permit ID	9-1438-00044/00014
DEC Permit Effective Date	3/31/2015
DEC Permit Description	
DEC Permit Review Report	
Company	National Fuel Gas Supply Corp.
Project	Part of the Niagara Expansion Project and the Northern Access 2015 Project which are joint projects undertaken by National Fuel Gas Supply Corporation and Tennessee Gas Pipeline Company.
Pipeline	Empire (AKA "National Fuel")
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	
Horsepower, existing	
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	



## 4.7b. Health Effects of Facility Releases

Table 4.7b.

### NFGSC Concord Compressor Station: Health Effects of Facility Releases

Concord NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Ch.	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	8	10	58,379	408,650	4.26
2a	Malignant neoplasms	C00-C97	8	9	58,216	407,511	4.34
2a.1	Lip, oral cavity and pharynx	C00-C14	5	3	18,010	126,066	8.75
2a.2	Digestive organs	C15-C26	5	5	18,010	126,067	8.62
2a.3	Respiratory system and intrathoracic organs	C30-C39	8	7	58,212	407,482	4.35
2a.4	Bone and articular cartilage	C40-C41	9	6	54,199	379,392	4.33
2a.5	Skin	C43-C44	--	--	0	0	0.00
2a.6	Connective and soft tissue	C45-C49	14	2	16	113	0.41
2a.07	Breast and female genital organs	C50-C58	6	5	39,853	278,969	6.43
2a.07.50	Female breast	C50	6	4	35,840	250,878	6.61
2a.07.55	Uterus	C55	--	--	0	0	0.00
2a.07.56	Ovary	C56	--	--	0	0	0.00
2a.08	Male genital organs	C60-C63	2	1	26,984	188,888	13.49
2a.09	Urinary organs	C64-C68	5	4	18,006	126,039	8.75
2a.10	Eye, brain and central nervous system	C69-C72	5	4	18,006	126,039	8.62
2a.11	Endocrine glands and related structures	C73-C75	2	2	27,008	189,058	13.40
2a.12	Secondary and ill-defined	C76-C80	14	1	24	170	0.51
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	6	6	39,857	278,996	6.36
2a.14	Independent (primary) multiple sites	C97	--	--	0	0	0.00
2b	In situ neoplasms	D00-D09	--	--	0	0	0.00
2c	Benign neoplasms	D10-D36	14	2	16	113	0.07
2d	Neoplasms of uncertain or unknown behavior	D37-D48	5	5	18,010	126,067	8.67
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	10	7	133,625	935,371	4.96
4	Endocrine, nutritional and metabolic diseases	E00-E90	10	7	40,512	283,584	3.96
5	Mental and behavioral disorders	F00-F99	10	7	133,625	935,371	4.96
6	Diseases of the nervous system	G00-G99	10	8	133,787	936,510	4.90
7	Diseases of the eye and adnexa	H00-H59	9	8	128,461	899,225	3.62
8	Diseases of the ear and mastoid process	H60-H95	10	4	115,615	809,305	4.63
9	Diseases of the circulatory system	I00-I99	7	8	133,132	931,923	5.73
10	Diseases of the respiratory system	J00-J99	8	11	243,583	1,705,081	4.30
11	Diseases of the digestive system	K00-K93	8	10	234,664	1,642,645	4.27
12	Diseases of the skin and subcutaneous tissue	L00-L99	9	11	150,324	1,052,268	3.79
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	8	4	8,924	62,464	5.07
14	Diseases of the genitourinary system	N00-N99	8	11	247,433	1,732,031	4.34
14a	Diseases of the genitourinary system: urinary system	N00-N39	9	6	36,353	254,468	3.97
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	8	10	247,429	1,732,003	4.34
15	Pregnancy, childbirth and the puerperium	O00-O99	9	4	109,935	769,543	3.92
16	Certain conditions originating in the perinatal period	P00-P96	9	7	131,814	922,698	4.10
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	8	10	243,579	1,705,053	4.30
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	8	11	243,583	1,705,081	4.30
<b>Total Releases</b>			<b>8</b>	<b>12</b>	<b>247,596</b>	<b>1,733,171</b>	<b>4.31</b>

## 4.8. NFGSC Independence Compressor Station (Andover NY)

### 4.8a. Facility Profile

Table 4.8a.

#### NFGSC Independence Compressor Station

Andover NY

Facility name, short	NFGSC Independence Compressor Station
Facility name, full	NFGSC Independence CS
EIS Facility ID	8377611
DEC Region	9
County	Allegany
Town	Andover
Village \ Hamlet	
Address	2210 County Road 22
Zip	14806
DEC Permit Type	Air Title V Facility
DEC Facility ID	9026000009
DEC Permit ID	9-0260-00009/00016
DEC Permit Effective Date	4/9/2013
DEC Permit Description	
DEC Permit Review Report	
Company	National Fuel Gas Supply Corp.
Project	Part of the Niagara Expansion Project and the Northern Access 2015 Project which are joint projects undertaken by National Fuel Gas Supply Corporation and Tennessee Gas Pipeline Company.
Pipeline	Empire (AKA "National Fuel")
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	5,000
	(2) 1,000 HP reciprocating ngfce (2) 1,500 HP reciprocating ngfce
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.8b. Health Effects of Facility Releases

Table 4.8b.

### NFGSC Independence Compressor Station: Facility Releases by Health Effects (2008-2014)

Andover NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	10	15	56,144	393,010	4.10
2a	Malignant neoplasms	C00-C97	10	14	56,041	392,290	4.17
2a.1	Lip, oral cavity and pharynx	C00-C14	10	7	9,210	64,473	4.48
2a.2	Digestive organs	C15-C26	10	10	9,211	64,477	4.41
2a.3	Respiratory system and intrathoracic organs	C30-C39	10	11	55,997	391,984	4.19
2a.4	Bone and articular cartilage	C40-C41	8	10	55,385	387,695	4.42
2a.5	Skin	C43-C44	17	1	0	1	0.00
2a.6	Connective and soft tissue	C45-C49	16	3	4	30	0.11
2a.07	Breast and female genital organs	C50-C58	13	6	12,417	86,924	2.00
2a.07.50	Female breast	C50	13	5	11,805	82,636	2.18
2a.07.55	Uterus	C55	--	--	0	0	0.00
2a.07.56	Ovary	C56	17	1	0	1	0.00
2a.08	Male genital organs	C60-C63	9	3	9,162	64,140	4.58
2a.09	Urinary organs	C64-C68	10	7	9,167	64,173	4.45
2a.10	Eye, brain and central nervous system	C69-C72	10	9	9,168	64,176	4.39
2a.11	Endocrine glands and related structures	C73-C75	9	3	9,166	64,168	4.55
2a.12	Secondary and ill-defined	C76-C80	16	4	4	32	0.10
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	13	9	12,460	87,225	1.99
2a.14	Independent (primary) multiple sites	C97	--	--	0	0	0.00
2b	In situ neoplasms	D00-D09	15	2	.4	3	0.01
2c	Benign neoplasms	D10-D36	17	5	4	33	0.02
2d	Neoplasms of uncertain or unknown behavior	D37-D48	10	9	9,211	64,477	4.43
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	8	12	147,748	1,034,238	5.48
4	Endocrine, nutritional and metabolic diseases	E00-E90	7	12	53,498	374,487	5.23
5	Mental and behavioral disorders	F00-F99	8	12	147,748	1,034,238	5.48
6	Diseases of the nervous system	G00-G99	8	13	147,851	1,034,958	5.41
7	Diseases of the eye and adnexa	H00-H59	10	13	95,202	666,418	2.68
8	Diseases of the ear and mastoid process	H60-H95	8	9	138,538	969,769	5.55
9	Diseases of the circulatory system	I00-I99	9	10	106,814	747,699	4.60
10	Diseases of the respiratory system	J00-J99	11	16	192,806	1,349,642	3.40
11	Diseases of the digestive system	K00-K93	11	15	191,487	1,340,411	3.48
12	Diseases of the skin and subcutaneous tissue	L00-L99	10	16	98,457	689,200	2.48
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	15	8	1,362	9,540	0.77
14	Diseases of the genitourinary system	N00-N99	11	16	193,315	1,353,211	3.39
14a	Diseases of the genitourinary system: urinary system	N00-N39	7	10	52,786	369,507	5.76
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	11	15	193,272	1,352,909	3.39
15	Pregnancy, childbirth and the puerperium	O00-O99	13	6	51,476	360,333	1.84
16	Certain conditions originating in the perinatal period	P00-P96	13	10	54,734	383,143	1.70
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	11	15	192,762	1,349,340	3.40
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	11	16	192,806	1,349,642	3.40
<b>Total Releases</b>			<b>11</b>	<b>17</b>	<b>193,418</b>	<b>1,353,931</b>	<b>3.37</b>

## 4.9. NFGSC Nashville Compressor Station (Hanover NY)

### 4.9a. Facility Profile

Table 4.9a.

#### NFGSC Nashville Compressor Station

Hanover NY

<b>Facility name, short</b>	NFGSC Nashville Compressor Station
<b>Facility name, full</b>	NFGSC Nashville CS
<b>EIS Facility ID</b>	7806511
<b>DEC Region</b>	9
<b>County</b>	Chautauqua
<b>Town</b>	Hanover
<b>Village \ Hamlet</b>	Forestville
<b>Address</b>	11413 Allegany Rd
<b>Zip</b>	14062
<b>DEC Permit Type</b>	Air State Facility
<b>DEC Facility ID</b>	9064600048
<b>DEC Permit ID</b>	9-0646-00048/00019
<b>DEC Permit Effective Date</b>	7/25/2014
<b>DEC Permit Description</b>	Permit modification was made to correct two administrative errors
<b>DEC Permit Review Report</b>	
<b>Company</b>	National Fuel Gas Supply Corp.
<b>Project</b>	Part of the Niagara Expansion Project and the Northern Access 2015 Project which are joint projects undertaken by National Fuel Gas Supply Corporation and Tennessee Gas Pipeline Company.
<b>Pipeline</b>	Empire (AKA "National Fuel")
<b>Principal Supply Source</b>	
<b>System Configuration (Primary/Secondary)</b>	
<b>Status</b>	Operational
<b>Horsepower, existing</b>	1,028 HP
	(2) 660HP, (1) 225 HP, (1) 203 HP
<b>Total estimated releases (2008-2014): pounds</b>	
<b>Total estimated releases (2008-2014): rank</b>	

## 4.9b. Health Effects of Facility Releases

Table 4.9b.

### NFGSC Nashville Compressor Station: Facility Releases by Health Effects (2008-2014)

Hanover NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	14	31	19,663	137,639	1.44
2a	Malignant neoplasms	C00-C97	14	28	19,592	137,144	1.46
2a.1	Lip, oral cavity and pharynx	C00-C14	14	7	4,274	29,915	2.08
2a.2	Digestive organs	C15-C26	14	16	4,285	29,993	2.05
2a.3	Respiratory system and intrathoracic organs	C30-C39	14	21	19,553	136,871	1.46
2a.4	Bone and articular cartilage	C40-C41	14	20	19,171	134,194	1.53
2a.5	Skin	C43-C44	15	7	0	0	0.00
2a.6	Connective and soft tissue	C45-C49	17	10	2	15	0.06
2a.07	Breast and female genital organs	C50-C58	14	9	6,339	44,374	1.02
2a.07.50	Female breast	C50	14	7	5,957	41,697	1.10
2a.07.55	Uterus	C55	13	2	0	0	0.00
2a.07.56	Ovary	C56	15	2	0	0	0.00
2a.08	Male genital organs	C60-C63	14	4	4,243	29,704	2.12
2a.09	Urinary organs	C64-C68	14	12	4,246	29,721	2.06
2a.10	Eye, brain and central nervous system	C69-C72	14	8	4,257	29,798	2.04
2a.11	Endocrine glands and related structures	C73-C75	13	4	4,246	29,719	2.11
2a.12	Secondary and ill-defined	C76-C80	17	3	2	15	0.05
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	14	15	6,367	44,568	1.02
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	16	2	0	0	0.00
2c	Benign neoplasms	D10-D36	15	10	13	92	0.06
2d	Neoplasms of uncertain or unknown behavior	D37-D48	14	20	4,285	29,993	2.06
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	14	16	54,249	379,740	2.01
4	Endocrine, nutritional and metabolic diseases	E00-E90	14	29	17,947	125,632	1.75
5	Mental and behavioral disorders	F00-F99	14	16	54,249	379,740	2.01
6	Diseases of the nervous system	G00-G99	14	18	54,319	380,235	1.99
7	Diseases of the eye and adnexa	H00-H59	15	19	50,123	350,859	1.41
8	Diseases of the ear and mastoid process	H60-H95	14	10	49,975	349,825	2.00
9	Diseases of the circulatory system	I00-I99	14	16	42,750	299,248	1.84
10	Diseases of the respiratory system	J00-J99	15	27	88,588	620,115	1.56
11	Diseases of the digestive system	K00-K93	15	24	87,732	614,122	1.60
12	Diseases of the skin and subcutaneous tissue	L00-L99	15	25	52,218	365,527	1.32
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	17	9	895	6,266	0.51
14	Diseases of the genitourinary system	N00-N99	15	20	88,900	622,297	1.56
14a	Diseases of the genitourinary system: urinary system	N00-N39	14	13	17,497	122,476	1.91
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	15	17	88,872	622,101	1.56
15	Pregnancy, childbirth and the puerperium	O00-O99	15	8	36,799	257,594	1.31
16	Certain conditions originating in the perinatal period	P00-P96	16	14	38,897	272,276	1.21
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	15	32	88,560	619,919	1.56
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	15	22	88,588	620,114	1.56
<b>Total Releases</b>			<b>15</b>	<b>38</b>	<b>88,970</b>	<b>622,791</b>	<b>1.55</b>

## 4.10. TGPC Compressor Station 224

### 4.10a. Facility Profile

Table 4.10a.

#### TGPC Compressor Station 224

Clymer NY

<b>Facility name, short</b>	TGPC Compressor Station 224
<b>Facility name, full</b>	TGPC CS 224
<b>EIS Facility ID</b>	7806411
<b>DEC Region</b>	9
<b>County</b>	Chautauqua
<b>Town</b>	Clymer
<b>Village \ Hamlet</b>	
<b>Address</b>	9766 Ravlin Hill Rd
<b>Zip</b>	14724
<b>DEC Permit Type</b>	Air Title V Facility
<b>DEC Facility ID</b>	9064200016
<b>DEC Permit ID</b>	9-0642-00016/00017
<b>DEC Permit Effective Date</b>	5/21/2013
<b>DEC Permit Description</b>	
<b>DEC Permit Review Report</b>	
<b>Company</b>	Tennessee Gas Pipeline Company
<b>Project</b>	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
<b>Pipeline</b>	Tennessee Gas Pipeline
<b>Principal Supply Source</b>	
<b>System Configuration (Primary/Secondary)</b>	
<b>Status</b>	Operational
<b>Horsepower, existing</b>	8,000 HP
	(4) 2000 HP4-cycle lean burn reciprocating engine
<b>Total estimated releases (2008-2014): pounds</b>	
<b>Total estimated releases (2008-2014): rank</b>	

## 4.10b. Health Effects of Facility Releases

Table 4.10b.

### TGPC Compressor Station 224: Facility Releases by Health Effects (2008-2014)

Clymer NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	12	40	40,157	281,096	2.93
2a	Malignant neoplasms	C00-C97	12	37	39,935	279,548	2.97
2a.1	Lip, oral cavity and pharynx	C00-C14	8	8	11,094	77,661	5.39
2a.2	Digestive organs	C15-C26	8	29	11,454	80,175	5.48
2a.3	Respiratory system and intrathoracic organs	C30-C39	12	25	39,258	274,805	2.93
2a.4	Bone and articular cartilage	C40-C41	12	22	37,179	260,256	2.97
2a.5	Skin	C43-C44	5	7	141	990	3.71
2a.6	Connective and soft tissue	C45-C49	6	8	143	1,004	3.67
2a.07	Breast and female genital organs	C50-C58	12	19	17,399	121,793	2.81
2a.07.50	Female breast	C50	11	17	16,280	113,960	3.00
2a.07.55	Uterus	C55	4	3	52	365	7.44
2a.07.56	Ovary	C56	5	2	126	881	3.38
2a.08	Male genital organs	C60-C63	8	9	9,516	66,614	4.76
2a.09	Urinary organs	C64-C68	8	17	11,221	78,549	5.45
2a.10	Eye, brain and central nervous system	C69-C72	8	18	11,403	79,821	5.46
2a.11	Endocrine glands and related structures	C73-C75	8	7	9,565	66,955	4.75
2a.12	Secondary and ill-defined	C76-C80	7	3	112	787	2.36
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	11	23	17,967	125,770	2.87
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	6	3	155	1,086	2.60
2c	Benign neoplasms	D10-D36	4	21	2,829	19,804	11.97
2d	Neoplasms of uncertain or unknown behavior	D37-D48	8	28	11,383	79,684	5.48
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	11	25	105,076	735,534	3.90
4	Endocrine, nutritional and metabolic diseases	E00-E90	12	32	34,003	238,018	3.32
5	Mental and behavioral disorders	F00-F99	11	27	105,039	735,270	3.90
6	Diseases of the nervous system	G00-G99	11	34	106,266	743,864	3.89
7	Diseases of the eye and adnexa	H00-H59	12	31	84,984	594,890	2.40
8	Diseases of the ear and mastoid process	H60-H95	11	12	93,625	655,373	3.75
9	Diseases of the circulatory system	I00-I99	11	23	88,805	621,634	3.83
10	Diseases of the respiratory system	J00-J99	12	37	162,657	1,138,602	2.87
11	Diseases of the digestive system	K00-K93	12	35	158,556	1,109,894	2.88
12	Diseases of the skin and subcutaneous tissue	L00-L99	12	35	91,319	639,232	2.30
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	12	13	3,494	24,460	1.98
14	Diseases of the genitourinary system	N00-N99	12	34	162,635	1,138,443	2.85
14a	Diseases of the genitourinary system: urinary system	N00-N39	12	26	32,594	228,156	3.56
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	12	30	162,603	1,138,223	2.85
15	Pregnancy, childbirth and the puerperium	O00-O99	11	16	63,140	441,983	2.25
16	Certain conditions originating in the perinatal period	P00-P96	11	16	68,616	480,315	2.13
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	12	42	162,655	1,138,582	2.87
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	12	34	162,642	1,138,497	2.87
<b>Total Releases</b>			<b>12</b>	<b>47</b>	<b>163,828</b>	<b>1,146,797</b>	<b>2.85</b>

## 4.11. TGPC Compressor Station 229 & TEG Dehydration Facility (Eden NY)

### 4.11a. Facility Profile

Table 4.11a.

#### TGPC Compressor Station 229 & TEG Dehydration Facility

Eden NY

Facility name, short	TGPC Compressor Station 229 & TEG Dehydration Facility
Facility name, full	TGPC 229 & TEG DF
EIS Facility ID	8503511
DEC Region	9
County	Erie
Town	Eden
Village \ Hamlet	
Address	7586 East Eden Road
Zip	14057
DEC Permit Type	Air Title V Facility
DEC Facility ID	9143800044
DEC Permit ID	9-1440-00034/00021
DEC Permit Effective Date	7/31/2013
DEC Permit Description	
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	9,714
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	



## 4.11b. Health Effects of Facility Releases

Table 4.11b.

### TGPC Compressor Station 229 & TEG Dehydration: Facility Releases by Health Effects (2008-2014)

Eden NY

International Classification of Disease, 10 <sup>th</sup> edition			State Rank	Ch #	2008-14 Estimated Lbs.		% of State
Chapter	Description	Code			Average	Total	
2	Neoplasms	C00-D48	12	40	40,157	281,096	2.93
2a	Malignant neoplasms	C00-C97	12	37	39,935	279,548	2.97
2a.1	Lip, oral cavity and pharynx	C00-C14	8	8	11,094	77,661	5.39
2a.2	Digestive organs	C15-C26	8	29	11,454	80,175	5.48
2a.3	Respiratory system and intrathoracic organs	C30-C39	12	25	39,258	274,805	2.93
2a.4	Bone and articular cartilage	C40-C41	12	22	37,179	260,256	2.97
2a.5	Skin	C43-C44	5	7	141	990	3.71
2a.6	Connective and soft tissue	C45-C49	6	8	143	1,004	3.67
2a.07	Breast and female genital organs	C50-C58	12	19	17,399	121,793	2.81
2a.07.50	Female breast	C50	11	17	16,280	113,960	3.00
2a.07.55	Uterus	C55	4	3	52	365	7.44
2a.07.56	Ovary	C56	5	2	126	881	3.38
2a.08	Male genital organs	C60-C63	8	9	9,516	66,614	4.76
2a.09	Urinary organs	C64-C68	8	17	11,221	78,549	5.45
2a.10	Eye, brain and central nervous system	C69-C72	8	18	11,403	79,821	5.46
2a.11	Endocrine glands and related structures	C73-C75	8	7	9,565	66,955	4.75
2a.12	Secondary and ill-defined	C76-C80	7	3	112	787	2.36
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	11	23	17,967	125,770	2.87
2a.14	Independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	6	3	155	1,086	2.60
2c	Benign neoplasms	D10-D36	4	21	2,829	19,804	11.97
2d	Neoplasms of uncertain or unknown behavior	D37-D48	8	28	11,383	79,684	5.48
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	11	25	105,076	735,534	3.90
4	Endocrine, nutritional and metabolic diseases	E00-E90	12	32	34,003	238,018	3.32
5	Mental and behavioral disorders	F00-F99	11	27	105,039	735,270	3.90
6	Diseases of the nervous system	G00-G99	11	34	106,266	743,864	3.89
7	Diseases of the eye and adnexa	H00-H59	12	31	84,984	594,890	2.40
8	Diseases of the ear and mastoid process	H60-H95	11	12	93,625	655,373	3.75
9	Diseases of the circulatory system	I00-I99	11	23	88,805	621,634	3.83
10	Diseases of the respiratory system	J00-J99	12	37	162,657	1,138,602	2.87
11	Diseases of the digestive system	K00-K93	12	35	158,556	1,109,894	2.88
12	Diseases of the skin and subcutaneous tissue	L00-L99	12	35	91,319	639,232	2.30
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	12	13	3,494	24,460	1.98
14	Diseases of the genitourinary system	N00-N99	12	34	162,635	1,138,443	2.85
14a	Diseases of the genitourinary system: urinary system	N00-N39	12	26	32,594	228,156	3.56
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	12	30	162,603	1,138,223	2.85
15	Pregnancy, childbirth and the puerperium	O00-O99	11	16	63,140	441,983	2.25
16	Certain conditions originating in the perinatal period	P00-P96	11	16	68,616	480,315	2.13
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	12	42	162,655	1,138,582	2.87
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	12	34	162,642	1,138,497	2.87
Total Releases			12	47	163,828	1,146,797	2.85

## 4.12. TGPC Compressor Station 230-C (Lockport NY)

### 4.12a. Facility Profile

Table 4.12a.

#### TGPC Compressor Station 230-C

Lockport NY

Facility name, short	TGPC Compressor Station 230-C
Facility name, full	TGPC CS 230-C
EIS Facility ID	7417311
DEC Region	9
County	Niagara
Town	Lockport
Village \ Hamlet	
Address	5186 Lockport Junction Rd
Zip	14094
DEC Permit Type	Air State Facility
DEC Facility ID	9292000008
DEC Permit ID	9-2920-00008/00015
DEC Permit Effective Date	12/2/2014
DEC Permit Description	
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	18,000
	(4) 4,500 HP Solar Centaur H compressor turbines
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.12b. Health Effects of Facility Releases

Table 4.12b.

### TGPC Compressor Station 230-C: Facility Releases by Health Effects

Lockport NY

International Classification of Disease, 10 <sup>th</sup> edition			State Rank	Ch #	2008-14 Estimated Lbs.		% of State
Chapter	Description	Code			Average	Total	
2	Neoplasms	C00-D48	16	23	9,972	69,806	0.73
2a	Malignant neoplasms	C00-C97	16	22	7,013	49,091	0.52
2a.1	Lip, oral cavity and pharynx	C00-C14	16	7	369	2,580	0.18
2a.2	Digestive organs	C15-C26	16	14	399	2,792	0.19
2a.3	Respiratory system and intrathoracic organs	C30-C39	16	20	7,007	49,046	0.52
2a.4	Bone and articular cartilage	C40-C41	17	18	5,958	41,706	0.48
2a.5	Skin	C43-C44	11	3	5	36	0.14
2a.6	Connective and soft tissue	C45-C49	7	8	112	783	2.86
2a.07	Breast and female genital organs	C50-C58	15	11	5,817	40,722	0.94
2a.07.50	Female breast	C50	15	10	4,771	33,396	0.88
2a.07.55	Uterus	C55	10	2	2	16	0.33
2a.07.56	Ovary	C56	11	3	6	44	0.17
2a.08	Male genital organs	C60-C63	16	5	274	1,915	0.14
2a.09	Urinary organs	C64-C68	16	12	353	2,469	0.17
2a.10	Eye, brain and central nervous system	C69-C72	16	11	375	2,626	0.18
2a.11	Endocrine glands and related structures	C73-C75	16	6	282	1,972	0.14
2a.12	Secondary and ill-defined	C76-C80	6	5	137	962	2.89
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	15	16	5,882	41,174	0.94
2a.14	Independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	11	2	48	333	0.80
2c	Benign neoplasms	D10-D36	11	11	70	487	0.29
2d	Neoplasms of uncertain or unknown behavior	D37-D48	16	14	382	2,671	0.18
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	15	17	22,498	157,488	0.83
4	Endocrine, nutritional and metabolic diseases	E00-E90	16	20	5,433	38,028	0.53
5	Mental and behavioral disorders	F00-F99	15	16	22,498	157,487	0.83
6	Diseases of the nervous system	G00-G99	15	19	25,460	178,218	0.93
7	Diseases of the eye and adnexa	H00-H59	16	19	42,774	299,420	1.21
8	Diseases of the ear and mastoid process	H60-H95	15	10	22,205	155,432	0.89
9	Diseases of the circulatory system	I00-I99	15	15	25,902	181,314	1.12
10	Diseases of the respiratory system	J00-J99	16	24	68,325	478,274	1.21
11	Diseases of the digestive system	K00-K93	16	21	66,076	462,535	1.20
12	Diseases of the skin and subcutaneous tissue	L00-L99	16	22	48,326	338,285	1.22
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	13	9	2,305	16,133	1.31
14	Diseases of the genitourinary system	N00-N99	16	21	66,406	464,840	1.16
14a	Diseases of the genitourinary system: urinary system	N00-N39	17	14	1,428	9,997	0.16
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	16	20	66,405	464,837	1.16
15	Pregnancy, childbirth and the puerperium	O00-O99	14	10	38,680	270,758	1.38
16	Certain conditions originating in the perinatal period	P00-P96	14	14	44,297	310,080	1.38
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	16	24	68,322	478,255	1.21
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	16	22	68,321	478,245	1.21
<b>Total Releases</b>			<b>16</b>	<b>27</b>	<b>69,373</b>	<b>485,610</b>	<b>1.21</b>

## 4.13. TGPC Compressor Station 233 (York NY)

### 4.13a. Facility Profile

Table 4.13a.

#### TGPC Compressor Station 233

York NY

Facility name, short	TGPC Compressor Station 233
Facility name, full	TGPC CS 233
EIS Facility ID	8471211
DEC Region	8
County	Livingston
Town	York
Village \ Hamlet	Piffard
Address	2262 Dow Rd
Zip	14533
DEC Permit Type	Air Title V Facility
DEC Facility ID	8245200008
DEC Permit ID	8-2452-00008/00007
DEC Permit Effective Date	10/28/2015
DEC Permit Description	
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	9,000
	(2) 4,500 HP compressor engines
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.13b. Health Effects of Facility Releases

Table 4.13b.

### TGPC Compressor Station 233: Facility Releases by Health Effects

York NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	18	23	3,492	24,447	0.26
2a	Malignant neoplasms	C00-C97	18	22	3,395	23,762	0.25
2a.1	Lip, oral cavity and pharynx	C00-C14	17	7	263	1,841	0.13
2a.2	Digestive organs	C15-C26	17	14	278	1,945	0.13
2a.3	Respiratory system and intrathoracic organs	C30-C39	18	20	3,391	23,740	0.25
2a.4	Bone and articular cartilage	C40-C41	18	18	2,867	20,069	0.23
2a.5	Skin	C43-C44	12	3	4	28	0.10
2a.6	Connective and soft tissue	C45-C49	9	8	72	502	1.83
2a.07	Breast and female genital organs	C50-C58	17	11	4,455	31,182	0.72
2a.07.50	Female breast	C50	17	10	3,669	25,686	0.68
2a.07.55	Uterus	C55	18	2			0.00
2a.07.56	Ovary	C56	12	3	5	32	0.12
2a.08	Male genital organs	C60-C63	17	5	195	1,363	0.10
2a.09	Urinary organs	C64-C68	17	12	248	1,733	0.12
2a.10	Eye, brain and central nervous system	C69-C72	17	11	268	1,874	0.13
2a.11	Endocrine glands and related structures	C73-C75	17	6	189	1,320	0.09
2a.12	Secondary and ill-defined	C76-C80	8	5	94	659	1.98
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	17	16	4,510	31,567	0.72
2a.14	Independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	12	2	40	283	0.68
2c	Benign neoplasms	D10-D36	12	11	45	314	0.19
2d	Neoplasms of uncertain or unknown behavior	D37-D48	17	14	263	1,843	0.13
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	18	17	6,638	46,465	0.25
4	Endocrine, nutritional and metabolic diseases	E00-E90	18	20	1,159	8,114	0.11
5	Mental and behavioral disorders	F00-F99	18	16	6,638	46,464	0.25
6	Diseases of the nervous system	G00-G99	18	19	6,737	47,158	0.25
7	Diseases of the eye and adnexa	H00-H59	18	19	23,203	162,421	0.65
8	Diseases of the ear and mastoid process	H60-H95	18	10	6,505	45,534	0.26
9	Diseases of the circulatory system	I00-I99	18	15	8,572	60,007	0.37
10	Diseases of the respiratory system	J00-J99	18	24	31,616	221,312	0.56
11	Diseases of the digestive system	K00-K93	18	21	30,446	213,124	0.55
12	Diseases of the skin and subcutaneous tissue	L00-L99	18	22	26,048	182,337	0.66
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	14	9	1,802	12,614	1.02
14	Diseases of the genitourinary system	N00-N99	18	21	32,039	224,273	0.56
14a	Diseases of the genitourinary system: urinary system	N00-N39	18	14	537	3,762	0.06
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	18	20	32,039	224,271	0.56
15	Pregnancy, childbirth and the puerperium	O00-O99	17	10	22,714	158,999	0.81
16	Certain conditions originating in the perinatal period	P00-P96	17	14	25,582	179,073	0.80
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	18	24	31,615	221,306	0.56
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	18	22	31,615	221,303	0.56
	Total Releases		18	27	32,140	224,978	0.56

2	Neoplasms	C00-D48	18	23	3,492	24,447	0.26
2a	Malignant neoplasms	C00-C97	18	22	3,395	23,762	0.25
2a.1	Lip, oral cavity and pharynx	C00-C14	17	7	263	1,841	0.13
2a.2	Digestive organs	C15-C26	17	14	278	1,945	0.13
2a.3	Respiratory system and intrathoracic organs	C30-C39	18	20	3,391	23,740	0.25
2a.4	Bone and articular cartilage	C40-C41	18	18	2,867	20,069	0.23
2a.5	Skin	C43-C44	12	3	4	28	0.10
2a.6	Connective and soft tissue	C45-C49	9	8	72	502	1.83
2a.07	Breast and female genital organs	C50-C58	17	11	4,455	31,182	0.72
2a.07.50	Female breast	C50	17	10	3,669	25,686	0.68
2a.07.55	Uterus	C55	18	2			0.00
2a.07.56	Ovary	C56	12	3	5	32	0.12
2a.08	Male genital organs	C60-C63	17	5	195	1,363	0.10
2a.09	Urinary organs	C64-C68	17	12	248	1,733	0.12
2a.10	Eye, brain and central nervous system	C69-C72	17	11	268	1,874	0.13
2a.11	Endocrine glands and related structures	C73-C75	17	6	189	1,320	0.09
2a.12	Secondary and ill-defined	C76-C80	8	5	94	659	1.98
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	17	16	4,510	31,567	0.72
2a.14	Independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	12	2	40	283	0.68
2c	Benign neoplasms	D10-D36	12	11	45	314	0.19
2d	Neoplasms of uncertain or unknown behavior	D37-D48	17	14	263	1,843	0.13
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	18	17	6,638	46,465	0.25
4	Endocrine, nutritional and metabolic diseases	E00-E90	18	20	1,159	8,114	0.11
5	Mental and behavioral disorders	F00-F99	18	16	6,638	46,464	0.25
6	Diseases of the nervous system	G00-G99	18	19	6,737	47,158	0.25
7	Diseases of the eye and adnexa	H00-H59	18	19	23,203	162,421	0.65
8	Diseases of the ear and mastoid process	H60-H95	18	10	6,505	45,534	0.26
9	Diseases of the circulatory system	I00-I99	18	15	8,572	60,007	0.37
10	Diseases of the respiratory system	J00-J99	18	24	31,616	221,312	0.56
11	Diseases of the digestive system	K00-K93	18	21	30,446	213,124	0.55
12	Diseases of the skin and subcutaneous tissue	L00-L99	18	22	26,048	182,337	0.66
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	14	9	1,802	12,614	1.02
14	Diseases of the genitourinary system	N00-N99	18	21	32,039	224,273	0.56
14a	Diseases of the genitourinary system: urinary system	N00-N39	18	14	537	3,762	0.06
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	18	20	32,039	224,271	0.56
15	Pregnancy, childbirth and the puerperium	O00-O99	17	10	22,714	158,999	0.81
16	Certain conditions originating in the perinatal period	P00-P96	17	14	25,582	179,073	0.80
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	18	24	31,615	221,306	0.56
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	18	22	31,615	221,303	0.56
	Total Releases		18	27	32,140	224,978	0.56

## 4.14. TGPC Compressor Station 237 (Manchester, Phelps NY)

### 4.14a. Facility Profile

Table 4.14a.

#### TGPC Compressor Station 237

Manchester, Phelps NY

Facility name, short	TGPC Compressor Station 237
Facility name, full	TGPC CS 237
EIS Facility ID	7210411
DEC Region	8 -- Western Finger Lakes
County	Ontario
Town	Manchester, Phelps
Village \ Hamlet	Clifton Springs
Address	2001 Archer Road
Zip	14432
DEC Permit Type	Air Title V Facility
DEC Facility ID	323400013
DEC Permit ID	8-3234-00013/00011
DEC Permit Effective Date	6/14/2016
DEC Permit Description	Renewal of the Title V Facility Permit originally issued November 23, 1999 and previously renewed October 4, 2010.
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	8,000
	(1) 4,000 HP reciprocating ngfce, (2) 2,000 HP reciprocating ngfce

## 4.14b. Health Effects of Facility Releases

Table 4.14b.

### TGPC Compressor Station 237: Facility Releases by Health Effects

Manchester, Phelps NY

International Classification of Disease, 10 <sup>th</sup> edition			State Rank	Ch #	2008-14 Estimated Lbs.		% of State
Chapter	Description	Code			Average	Total	
2	Neoplasms	C00-D48	6	7	97,331	681,320	7.11
2a	Malignant neoplasms	C00-C97	6	6	97,146	680,023	7.24
2a.1	Lip, oral cavity and pharynx	C00-C14	6	1	16,708	116,956	8.12
2a.2	Digestive organs	C15-C26	6	2	16,709	116,964	8.00
2a.3	Respiratory system and intrathoracic organs	C30-C39	6	5	97,145	680,015	7.26
2a.4	Bone and articular cartilage	C40-C41	6	4	91,916	643,411	7.34
2a.5	Skin	C43-C44	--	0	0	0	0.00
2a.6	Connective and soft tissue	C45-C49	--	0	0	0	0.00
2a.07	Breast and female genital organs	C50-C58	5	5	42,837	299,859	6.92
2a.07.50	Female breast	C50	5	4	37,608	263,255	6.94
2a.07.55	Uterus	C55	--	0	0	0	0.00
2a.07.56	Ovary	C56	--	0	0	0	0.00
2a.08	Male genital organs	C60-C63	6	1	16,708	116,956	8.35
2a.09	Urinary organs	C64-C68	6	2	16,709	116,964	8.12
2a.10	Eye, brain and central nervous system	C69-C72	6	2	16,709	116,964	8.00
2a.11	Endocrine glands and related structures	C73-C75	6	1	16,708	116,956	8.29
2a.12	Secondary and ill-defined	C76-C80	--	0	0	0	0.00
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	5	4	42,836	299,851	6.84
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	--	0	0	0	0.00
2c	Benign neoplasms	D10-D36	--	0	0	0	0.00
2d	Neoplasms of uncertain or unknown behavior	D37-D48	6	2	16,709	116,964	8.05
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	5	4	195,395	1,367,764	7.25
4	Endocrine, nutritional and metabolic diseases	E00-E90	6	5	76,433	535,029	7.47
5	Mental and behavioral disorders	F00-F99	5	4	195,395	1,367,764	7.25
6	Diseases of the nervous system	G00-G99	5	5	195,580	1,369,061	7.16
7	Diseases of the eye and adnexa	H00-H59	8	5	177,838	1,244,864	5.01
8	Diseases of the ear and mastoid process	H60-H95	5	3	178,687	1,250,808	7.16
9	Diseases of the circulatory system	I00-I99	5	5	161,984	1,133,891	6.98
10	Diseases of the respiratory system	J00-J99	6	8	323,113	2,261,791	5.70
11	Diseases of the digestive system	K00-K93	6	7	313,810	2,196,672	5.71
12	Diseases of the skin and subcutaneous tissue	L00-L99	8	8	203,966	1,427,759	5.15
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	5	2	13,956	97,690	7.92
14	Diseases of the genitourinary system	N00-N99	6	8	328,157	2,297,097	5.75
14a	Diseases of the genitourinary system: urinary system	N00-N39	6	3	71,018	497,128	7.75
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	6	8	328,157	2,297,097	5.75
15	Pregnancy, childbirth and the puerperium	O00-O99	7	3	123,343	863,403	4.40
16	Certain conditions originating in the perinatal period	P00-P96	7	5	149,470	1,046,290	4.65
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	6	8	323,113	2,261,791	5.71
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	6	8	323,113	2,261,791	5.70
<b>Total Releases</b>			<b>6</b>	<b>9</b>	<b>328,342</b>	<b>2,298,394</b>	<b>5.72</b>



## 4.15. TGPC Compressor Station 241 (LaFayette NY)

### 4.15a. Facility Profile

Table 4.15a.

#### TGPC Compressor Station 241

LaFayette NY

Facility name, short	TGPC Compressor Station 241
Facility name, full	TGPC CS 241
EIS Facility ID	7436111
DEC Region	7 -- Central New York
County	Onondaga
Town	LaFayette
Village \ Hamlet	
Address	3447 Sentinel Heights Rd
Zip	13084
DEC Permit Type	Air Title V Facility
DEC Facility ID	7313400022
DEC Permit ID	7-3134-00022/00011
DEC Permit Effective Date	1/23/2012
DEC Permit Description	Title V Renewal and a modification to revise to the condition requiring that TGP comply with 6 NYCRR Part 212.
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	6,800 HP

## 4.15b. Health Effects of Facility Releases

Table 4.15b.

### TGPC Compressor Station 241: Facility Releases by Health Effects

LaFayette NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	3	40	162,854	1,139,976	11.89
2a	Malignant neoplasms	C00-C97	3	37	159,625	1,117,378	11.89
2a.1	Lip, oral cavity and pharynx	C00-C14	2	8	26,645	186,512	12.95
2a.2	Digestive organs	C15-C26	2	29	26,850	187,951	12.85
2a.3	Respiratory system and intrathoracic organs	C30-C39	3	25	159,204	1,114,430	11.90
2a.4	Bone and articular cartilage	C40-C41	3	22	149,626	1,047,383	11.95
2a.5	Skin	C43-C44	7	7	86	602	2.26
2a.6	Connective and soft tissue	C45-C49	8	7	85	595	2.18
2a.07	Breast and female genital organs	C50-C58	4	19	72,893	510,251	11.77
2a.07.50	Female breast	C50	4	17	63,931	447,517	11.80
2a.07.55	Uterus	C55	5	3	32	222	4.54
2a.07.56	Ovary	C56	7	2	82	572	2.19
2a.08	Male genital organs	C60-C63	3	10	25,626	179,381	12.81
2a.09	Urinary organs	C64-C68	2	17	26,713	186,990	12.98
2a.10	Eye, brain and central nervous system	C69-C72	2	17	26,839	187,876	12.85
2a.11	Endocrine glands and related structures	C73-C75	3	7	25,649	179,540	12.72
2a.12	Secondary and ill-defined	C76-C80	9	3	72	507	1.52
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	4	22	73,255	512,783	11.69
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	8	3	99	690	1.65
2c	Benign neoplasms	D10-D36	5	20	1,801	12,605	7.62
2d	Neoplasms of uncertain or unknown behavior	D37-D48	2	27	26,809	187,665	12.91
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	3	24	278,616	1,950,313	10.33
4	Endocrine, nutritional and metabolic diseases	E00-E90	3	32	124,938	874,563	12.22
5	Mental and behavioral disorders	F00-F99	3	26	278,597	1,950,179	10.33
6	Diseases of the nervous system	G00-G99	3	33	282,459	1,977,210	10.34
7	Diseases of the eye and adnexa	H00-H59	4	30	222,020	1,554,140	6.26
8	Diseases of the ear and mastoid process	H60-H95	3	11	251,763	1,762,343	10.09
9	Diseases of the circulatory system	I00-I99	3	22	229,876	1,609,133	9.90
10	Diseases of the respiratory system	J00-J99	4	37	425,243	2,976,701	7.51
11	Diseases of the digestive system	K00-K93	4	34	406,862	2,848,035	7.40
12	Diseases of the skin and subcutaneous tissue	L00-L99	4	34	268,300	1,878,097	6.77
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	4	12	17,983	125,878	10.21
14	Diseases of the genitourinary system	N00-N99	4	33	430,379	3,012,652	7.54
14a	Diseases of the genitourinary system: urinary system	N00-N39	3	25	112,696	788,872	12.30
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	4	29	430,367	3,012,569	7.54
15	Pregnancy, childbirth and the puerperium	O00-O99	6	15	132,651	928,556	4.73
16	Certain conditions originating in the perinatal period	P00-P96	6	15	178,380	1,248,662	5.55
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	4	41	425,236	2,976,652	7.51
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	4	33	425,231	2,976,619	7.51
<b>Total Releases</b>			<b>4</b>	<b>48</b>	<b>434,237</b>	<b>3,039,661</b>	<b>7.56</b>

## 4.16. TGPC Compressor Station 245 (Winfield NY)

### 4.16a. Facility Profile

Table 4.16a.

#### TGPC Compressor Station 245

Winfield NY

Facility name, short	TGPC Compressor Station 245
Facility name, full	TGPC CS 245
EIS Facility ID	8035411
DEC Region	6 -- Western Adirondacks / Eastern Lake Ontario
County	Herkimer
Town	Winfield
Village \ Hamlet	West Winfield
Address	457 Burrows Rd
Zip	13491
DEC Permit Type	Air Title V Facility
DEC Facility ID	6215600018
DEC Permit ID	6-2156-00018/00021
DEC Permit Effective Date	4/1/2015
DEC Permit Description	
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	7,000 (5) 1,400 hp Worthington UTC-165, 2-Stroke Lean Burn(2SLB) compressor engine
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.16b. Health Effects of Facility Releases

Table 4.16b.

### TGPC Compressor Station 245: Facility Releases by Health Effects

Winfield NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	3	40	162,854	1,139,976	11.89
2a	Malignant neoplasms	C00-C97	3	37	159,625	1,117,378	11.89
2a.1	Lip, oral cavity and pharynx	C00-C14	2	8	26,645	186,512	12.95
2a.2	Digestive organs	C15-C26	2	29	26,850	187,951	12.85
2a.3	Respiratory system and intrathoracic organs	C30-C39	3	25	159,204	1,114,430	11.90
2a.4	Bone and articular cartilage	C40-C41	3	22	149,626	1,047,383	11.95
2a.5	Skin	C43-C44	7	7	86	602	2.26
2a.6	Connective and soft tissue	C45-C49	8	7	85	595	2.18
2a.07	Breast and female genital organs	C50-C58	4	19	72,893	510,251	11.77
2a.07.50	Female breast	C50	4	17	63,931	447,517	11.80
2a.07.55	Uterus	C55	5	3	32	222	4.54
2a.07.56	Ovary	C56	7	2	82	572	2.19
2a.08	Male genital organs	C60-C63	3	10	25,626	179,381	12.81
2a.09	Urinary organs	C64-C68	2	17	26,713	186,990	12.98
2a.10	Eye, brain and central nervous system	C69-C72	2	17	26,839	187,876	12.85
2a.11	Endocrine glands and related structures	C73-C75	3	7	25,649	179,540	12.72
2a.12	Secondary and ill-defined	C76-C80	9	3	72	507	1.52
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	4	22	73,255	512,783	11.69
2a.14	Independent (primary) multiple sites	C97					
2b	In situ neoplasms	D00-D09	8	3	99	690	1.65
2c	Benign neoplasms	D10-D36	5	20	1,801	12,605	7.62
2d	Neoplasms of uncertain or unknown behavior	D37-D48	2	27	26,809	187,665	12.91
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	3	24	278,616	1,950,313	10.33
4	Endocrine, nutritional and metabolic diseases	E00-E90	3	32	124,938	874,563	12.22
5	Mental and behavioral disorders	F00-F99	3	26	278,597	1,950,179	10.33
6	Diseases of the nervous system	G00-G99	3	33	282,459	1,977,210	10.34
7	Diseases of the eye and adnexa	H00-H59	4	30	222,020	1,554,140	6.26
8	Diseases of the ear and mastoid process	H60-H95	3	11	251,763	1,762,343	10.09
9	Diseases of the circulatory system	I00-I99	3	22	229,876	1,609,133	9.90
10	Diseases of the respiratory system	J00-J99	4	37	425,243	2,976,701	7.51
11	Diseases of the digestive system	K00-K93	4	34	406,862	2,848,035	7.40
12	Diseases of the skin and subcutaneous tissue	L00-L99	4	34	268,300	1,878,097	6.77
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	4	12	17,983	125,878	10.21
14	Diseases of the genitourinary system	N00-N99	4	33	430,379	3,012,652	7.54
14a	Diseases of the genitourinary system: urinary system	N00-N39	3	25	112,696	788,872	12.30
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	4	29	430,367	3,012,569	7.54
15	Pregnancy, childbirth and the puerperium	O00-O99	6	15	132,651	928,556	4.73
16	Certain conditions originating in the perinatal period	P00-P96	6	15	178,380	1,248,662	5.55
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	4	41	425,236	2,976,652	7.51
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	4	33	425,231	2,976,619	7.51
<b>Total Releases</b>			<b>4</b>	<b>48</b>	<b>434,237</b>	<b>3,039,661</b>	<b>7.56</b>

## 4.17. TGPC Compressor Station 249 (Carlisle NY)

### 4.17a. Facility Profile

Table 4.17a.

#### TGPC Compressor Station 249

Carlisle NY

Facility name, short	TGPC Compressor Station 249
Facility name, full	TGPC CS 249
EIS Facility ID	8435311
DEC Region	4
County	Schoharie
Town	Carlisle
Village \ Hamlet	
Address	2480 US Route 20
Zip	12031
DEC Permit Type	Air Title V Facility
DEC Facility ID	4432400005
DEC Permit ID	4-4324-00005/00007
DEC Permit Effective Date	11/6/2015
DEC Permit Description	Renewal of the Title V permit.
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	9,100 HP
Total estimated releases (2008-2014): pounds	
Total estimated releases (2008-2014): rank	

## 4.17b. Health Effects of Facility Releases

Table 4.17b.

### TGPC Compressor Station 249: Facility Releases by Health Effects

Carlisle NY

International Classification of Disease, 10 <sup>th</sup> edition			State Rank	Ch #	2008-14 Estimated Lbs.		% of State
Chapter	Description	Code			Average	Total	
2	Neoplasms	C00-D48	2	41	194,333	1,360,334	14.19
2a	Malignant neoplasms	C00-C97	2	38	190,789	1,335,524	14.21
2a.1	Lip, oral cavity and pharynx	C00-C14	3	8	22,754	159,281	11.06
2a.2	Digestive organs	C15-C26	3	30	22,925	160,478	10.97
2a.3	Respiratory system and intrathoracic organs	C30-C39	2	26	190,447	1,333,127	14.23
2a.4	Bone and articular cartilage	C40-C41	2	23	176,886	1,238,204	14.12
2a.5	Skin	C43-C44	8	7	71	495	1.86
2a.6	Connective and soft tissue	C45-C49	10	8	70	493	1.80
2a.07	Breast and female genital organs	C50-C58	2	19	92,653	648,571	14.96
2a.07.50	Female breast	C50	2	17	79,589	557,125	14.69
2a.07.55	Uterus	C55	7	3	26	183	3.73
2a.07.56	Ovary	C56	8	2	66	461	1.77
2a.08	Male genital organs	C60-C63	4	10	21,935	153,543	10.97
2a.09	Urinary organs	C64-C68	3	18	22,813	159,689	11.08
2a.10	Eye, brain and central nervous system	C69-C72	3	18	22,912	160,382	10.97
2a.11	Endocrine glands and related structures	C73-C75	4	7	21,956	153,691	10.89
2a.12	Secondary and ill-defined	C76-C80	10	3	58	409	1.23
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	2	23	92,946	650,622	14.83
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	9	3	80	560	1.34
2c	Benign neoplasms	D10-D36	6	21	1,454	10,181	6.15
2d	Neoplasms of uncertain or unknown behavior	D37-D48	3	28	22,890	160,231	11.02
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	2	25	299,200	2,094,397	11.09
4	Endocrine, nutritional and metabolic diseases	E00-E90	2	34	136,996	958,972	13.39
5	Mental and behavioral disorders	F00-F99	2	27	299,178	2,094,247	11.10
6	Diseases of the nervous system	G00-G99	2	34	303,242	2,122,695	11.10
7	Diseases of the eye and adnexa	H00-H59	3	31	368,833	2,581,834	10.40
8	Diseases of the ear and mastoid process	H60-H95	2	12	276,272	1,933,902	11.07
9	Diseases of the circulatory system	I00-I99	2	23	258,460	1,809,220	11.13
10	Diseases of the respiratory system	J00-J99	3	38	604,524	4,231,665	10.67
11	Diseases of the digestive system	K00-K93	3	35	576,081	4,032,565	10.48
12	Diseases of the skin and subcutaneous tissue	L00-L99	3	35	438,754	3,071,281	11.07
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	2	13	28,130	196,907	15.97
14	Diseases of the genitourinary system	N00-N99	3	34	613,568	4,294,974	10.75
14a	Diseases of the genitourinary system: urinary system	N00-N39	2	26	120,350	842,447	13.14
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	3	30	613,556	4,294,893	10.75
15	Pregnancy, childbirth and the puerperium	O00-O99	3	16	267,623	1,873,364	9.54
16	Certain conditions originating in the perinatal period	P00-P96	3	16	337,101	2,359,705	10.48
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	3	43	604,523	4,231,660	10.67
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	3	34	604,520	4,231,638	10.67
<b>Total Releases</b>			<b>3</b>	<b>50</b>	<b>617,612</b>	<b>4,323,285</b>	<b>10.76</b>

## 4.18. TGPC Compressor Station 254 (Chatham NY)

### 4.18a. Facility Profile

Table 4.18a.

#### TGPC Compressor Station 254

Chatham NY

Facility name, short	TGPC Compressor Station 254
Facility name, full	TGPC CS 254
EIS Facility ID	8525311
DEC Region	4
County	Columbia
Town	Chatham
Village \ Hamlet	Riders-Mills
Address	ST Rte 66 -- E Side S of County Line
Zip	12123
DEC Facility ID	4102600037
DEC Permit Type	Air Title V Facility
DEC Permit ID	4-1026-00037/00029
DEC Permit Effective Date	8/11/2014
DEC Permit Description	
DEC Permit Review Report	
Company	Tennessee Gas Pipeline Company
Project	Part of the Niagara Expansion Project by TGP/Kinder Morgan, which is related to National Fuel's Northern Access Project.
Pipeline	Tennessee Gas Pipeline
Principal Supply Source	
System Configuration (Primary/Secondary)	
Status	Operational
Horsepower, existing	10,475
	(1) gas turbine, (6) reciprocating engines

## 4.18b. Health Effects of Facility Releases

Table 4.18b.

### TGPC Compressor Station 254: Facility Releases by Health Effects

Chatham NY

International Classification of Disease, 10 <sup>th</sup> edition			State	Ch	2008-14 Estimated Lbs.		% of
Chapter	Description	Code	Rank	#	Average	Total	State
2	Neoplasms	C00-D48	9	23	57,856	404,994	4.23
2a	Malignant neoplasms	C00-C97	9	22	56,750	397,251	4.23
2a.1	Lip, oral cavity and pharynx	C00-C14	13	7	4,723	33,063	2.30
2a.2	Digestive organs	C15-C26	13	14	4,732	33,124	2.26
2a.3	Respiratory system and intrathoracic organs	C30-C39	9	20	56,703	396,923	4.24
2a.4	Bone and articular cartilage	C40-C41	10	18	52,602	368,212	4.20
2a.5	Skin	C43-C44	10	3	33	234	0.88
2a.6	Connective and soft tissue	C45-C49	4	8	298	2,088	7.64
2a.07	Breast and female genital organs	C50-C58	9	11	25,754	180,276	4.16
2a.07.50	Female breast	C50	9	10	21,664	151,648	4.00
2a.07.55	Uterus	C55	15	2			0.00
2a.07.56	Ovary	C56	10	3	34	237	0.91
2a.08	Male genital organs	C60-C63	13	5	4,382	30,671	2.19
2a.09	Urinary organs	C64-C68	13	12	4,607	32,249	2.24
2a.10	Eye, brain and central nervous system	C69-C72	13	11	4,770	33,391	2.28
2a.11	Endocrine glands and related structures	C73-C75	14	6	4,234	29,641	2.10
2a.12	Secondary and ill-defined	C76-C80	4	5	642	4,497	13.50
2a.13	Stated or presumed to be primary, of lymphoid, haematopoietic, related	C81-C96	9	16	26,113	182,788	4.17
2a.14	Independent (primary) multiple sites	C97	--	0	0	0	0.00
2b	In situ neoplasms	D00-D09	5	2	392	2,743	6.56
2c	Benign neoplasms	D10-D36	10	11	265	1,854	1.12
2d	Neoplasms of uncertain or unknown behavior	D37-D48	13	14	4,660	32,623	2.24
3	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	D50-D89	6	17	161,398	1,129,784	5.98
4	Endocrine, nutritional and metabolic diseases	E00-E90	9	20	40,522	283,651	3.96
5	Mental and behavioral disorders	F00-F99	6	16	161,398	1,129,784	5.99
6	Diseases of the nervous system	G00-G99	6	19	162,516	1,137,610	5.95
7	Diseases of the eye and adnexa	H00-H59	5	19	194,478	1,361,349	5.48
8	Diseases of the ear and mastoid process	H60-H95	6	10	157,064	1,099,446	6.30
9	Diseases of the circulatory system	I00-I99	6	15	147,900	1,035,300	6.37
10	Diseases of the respiratory system	J00-J99	5	24	337,862	2,365,031	5.97
11	Diseases of the digestive system	K00-K93	5	21	329,499	2,306,496	6.00
12	Diseases of the skin and subcutaneous tissue	L00-L99	5	22	215,885	1,511,192	5.45
13	Diseases of the musculoskeletal system and connective tissue	M00-M99	9	9	8,668	60,676	4.92
14	Diseases of the genitourinary system	N00-N99	5	21	340,832	2,385,827	5.97
14a	Diseases of the genitourinary system: urinary system	N00-N39	11	14	35,305	247,133	3.85
14b	Diseases of the genitourinary system: pelvis, genitals and breasts	N40-N99	5	20	340,830	2,385,810	5.97
15	Pregnancy, childbirth and the puerperium	O00-O99	4	10	162,679	1,138,751	5.80
16	Certain conditions originating in the perinatal period	P00-P96	4	14	184,151	1,289,056	5.73
17	Congenital malformations, deformations and chromosomal abnormalities	Q00-Q99	5	24	337,861	2,365,027	5.97
18	Symptoms, signs and abnormal clinical and laboratory findings, nec	R00-R99	5	22	337,861	2,365,025	5.97
<b>Total Releases</b>			<b>5</b>	<b>27</b>	<b>341,952</b>	<b>2,393,661</b>	<b>5.96</b>



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# Low-Concentration PM<sub>2.5</sub> and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study

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**BACKGROUND:** Both short- and long-term exposures to fine particulate matter ( $\leq 2.5 \mu\text{m}$ ; PM<sub>2.5</sub>) are associated with mortality. However, whether the associations exist at levels below the new U.S. Environmental Protection Agency (EPA) standards (12  $\mu\text{g}/\text{m}^3$  of annual average PM<sub>2.5</sub>, 35  $\mu\text{g}/\text{m}^3$  daily) is unclear. In addition, it is not clear whether results from previous time series studies (fit in larger cities) and cohort studies (fit in convenience samples) are generalizable.

**OBJECTIVES:** We estimated the effects of low-concentration PM<sub>2.5</sub> on mortality.

**METHODS:** High resolution (1 km  $\times$  1 km) daily PM<sub>2.5</sub> predictions, derived from satellite aerosol optical depth retrievals, were used. Poisson regressions were applied to a Medicare population ( $\geq 65$  years of age) in New England to simultaneously estimate the acute and chronic effects of exposure to PM<sub>2.5</sub>, with mutual adjustment for short- and long-term exposure, as well as for area-based confounders. Models were also restricted to annual concentrations  $< 10 \mu\text{g}/\text{m}^3$  or daily concentrations  $< 30 \mu\text{g}/\text{m}^3$ .

**RESULTS:** PM<sub>2.5</sub> was associated with increased mortality. In the study cohort, 2.14% (95% CI: 1.38, 2.89%) and 7.52% (95% CI: 1.95, 13.40%) increases were estimated for each 10- $\mu\text{g}/\text{m}^3$  increase in short- (2 day) and long-term (1 year) exposure, respectively. The associations held for analyses restricted to low-concentration PM<sub>2.5</sub> exposure, and the corresponding estimates were 2.14% (95% CI: 1.34, 2.95%) and 9.28% (95% CI: 0.76, 18.52%). Penalized spline models of long-term exposure indicated a larger effect for mortality in association with exposures  $\geq 6 \mu\text{g}/\text{m}^3$  versus those  $< 6 \mu\text{g}/\text{m}^3$ . In contrast, the association between short-term exposure and mortality appeared to be linear across the entire exposure distribution.

**CONCLUSIONS:** Using a mutually adjusted model, we estimated significant acute and chronic effects of PM<sub>2.5</sub> exposure below the current U.S. EPA standards. These findings suggest that improving air quality with even lower PM<sub>2.5</sub> than currently allowed by U.S. EPA standards may benefit public health.

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## Introduction

Many studies have found associations between fine particulate matter [PM with aerodynamic diameter  $\leq 2.5 \mu\text{m}$  (PM<sub>2.5</sub>)] and increased mortality (Dockery et al. 1993; Franklin et al. 2007; Pope et al. 2002; Schwartz 1994; Zanobetti and Schwartz 2009). Biological evidence has been established for plausible mechanisms between PM<sub>2.5</sub> and mortality, such as increased risk of ventricular arrhythmia and thrombotic processes, increased system inflammation and oxidative stress, increased blood pressure, decreased plaque stability, and reduced lung function, among others (Brook et al. 2009; Gauderman et al. 2004; Gurgueira et al. 2002; Suwa et al. 2002; Yue et al. 2007). Based on evidence from epidemiological and toxicological studies (Chen and Nadziejko 2005; Furuyama et al. 2006; Ohtoshi et al. 1998), National Ambient Air Quality Standards (NAAQS) were implemented for fine particulate matter. For example, the U.S. Environmental Protection Agency (EPA) revised the fine particle NAAQS in 1997, 2006, and 2012 in order to protect public

health (U.S. EPA 1997, 2006, 2013). Further changes in the standards require additional studies to elucidate whether health effects occur at levels below the current annual and daily U.S. EPA NAAQS of 12 and 35  $\mu\text{g}/\text{m}^3$ , respectively. The Clean Air Act Amendments of 1990 require the U.S. EPA to review national air quality standards every 5 years to determine whether they should be retained or revised; thus, whether health effects can be observed below the current standards is of great interest and importance.

Previous studies have generally focused on either long-term (Hart et al. 2011; Jerrett et al. 2005; Puett et al. 2009; Schwartz 2000) or short-term (Dominici et al. 2006; Katsouyanni et al. 1997; Samoli et al. 2008; Schwartz and Dockery 1992) exposures across the entire range of PM<sub>2.5</sub> concentrations. In the case of time series analyses of short-term exposures, the need to ensure the relevance of the monitoring data as well as the need to have a study population of a size for sufficient power has limited analyses to large cities; hence, exurbs, small cities, and

rural areas are not generally represented in the literature, which may compromise the generalizability of the results. In addition, there is spatial variability in PM<sub>2.5</sub> concentrations within cities that time series studies generally do not take into account, which can introduce exposure measurement error (Laden et al. 2006; Lepeule et al. 2012).

Chronic effects studies began using comparisons across cities of mortality experiences of cohorts living in various communities and the monitored air pollutant concentrations in those communities (Dockery et al. 1993; Pope et al. 1995). Again, these studies suffered from exposure error due to failure to capture within-city spatial variability in exposure. Because the geographic exposure gradient is the exposure contrast in these studies, the failure to capture within-city contrasts leads to classical measurement error with expected downward bias. Studies with, for example, land use regression estimates of exposure have generally reported larger effect sizes (Miller et al. 2007; Puett et al. 2009). Previous cohort studies have not controlled for the acute effects of particles when estimating chronic effects, raising the question of whether there are independent chronic effects that represent more than the cumulative effects of acute responses.

In general, existing study cohorts are not representative of the overall population. For example, the American Cancer Society (ACS) cohort has a higher level of education than the U.S. population as a whole (Stellman

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and Garfinkel 1986). Hence, few population-based cohort studies have been conducted until recently (Kloog et al. 2013).

Several time series studies examined the concentration–response relationship between PM<sub>2.5</sub> and mortality below concentrations of 100 µg/m<sup>3</sup>; these studies generally reported a linear concentration–response relationship (Samoli et al. 2008; Schwartz and Zanobetti 2000). However, there have been few studies focusing on exposures below the current daily U.S. EPA standard of 35 µg/m<sup>3</sup>.

Many studies have examined the shape of the concentration–response curve for long-term exposure versus short-term exposure, but in general, they have not covered population-based cohorts, or have only included very low exposures (Schwartz et al. 2008; Crouse et al. 2012).

We recently presented a new hybrid method of assessing temporally and spatially resolved PM<sub>2.5</sub> exposure for epidemiological studies by combining 1 km × 1 km resolution satellite-retrieved aerosol optical depth (AOD) measurements with traditional land use terms, meteorological variables, and their interactions (Kloog et al. 2014a). This approach allows for predicting daily PM<sub>2.5</sub> concentrations at a 1 km × 1 km spatial resolution throughout the New England area of the northeastern United States. We also validated our model's performance in rural areas: 10-fold cross-validation (CV) of our model in rural areas (using the IMPROVE stations)

resulted in a CV  $R^2$  of 0.92. Further details have been published (Kloog et al. 2014a).

The present study aimed to simultaneously estimate acute and chronic health effects of PM<sub>2.5</sub> in a population-based Medicare cohort (≥ 65 years of age) encompassing the New England region. We used high-spatial-resolution exposure estimates based on satellite measurements that are available across the region and not just in limited locations. To make this study relevant to future assessments of current U.S. EPA standards, we repeated the analysis after restricting the data to long-term exposures (365-day moving average) < 10 µg/m<sup>3</sup> and repeated the time series analysis of short-term exposures after restricting the data to 2-day average exposures < 30 µg/m<sup>3</sup>.

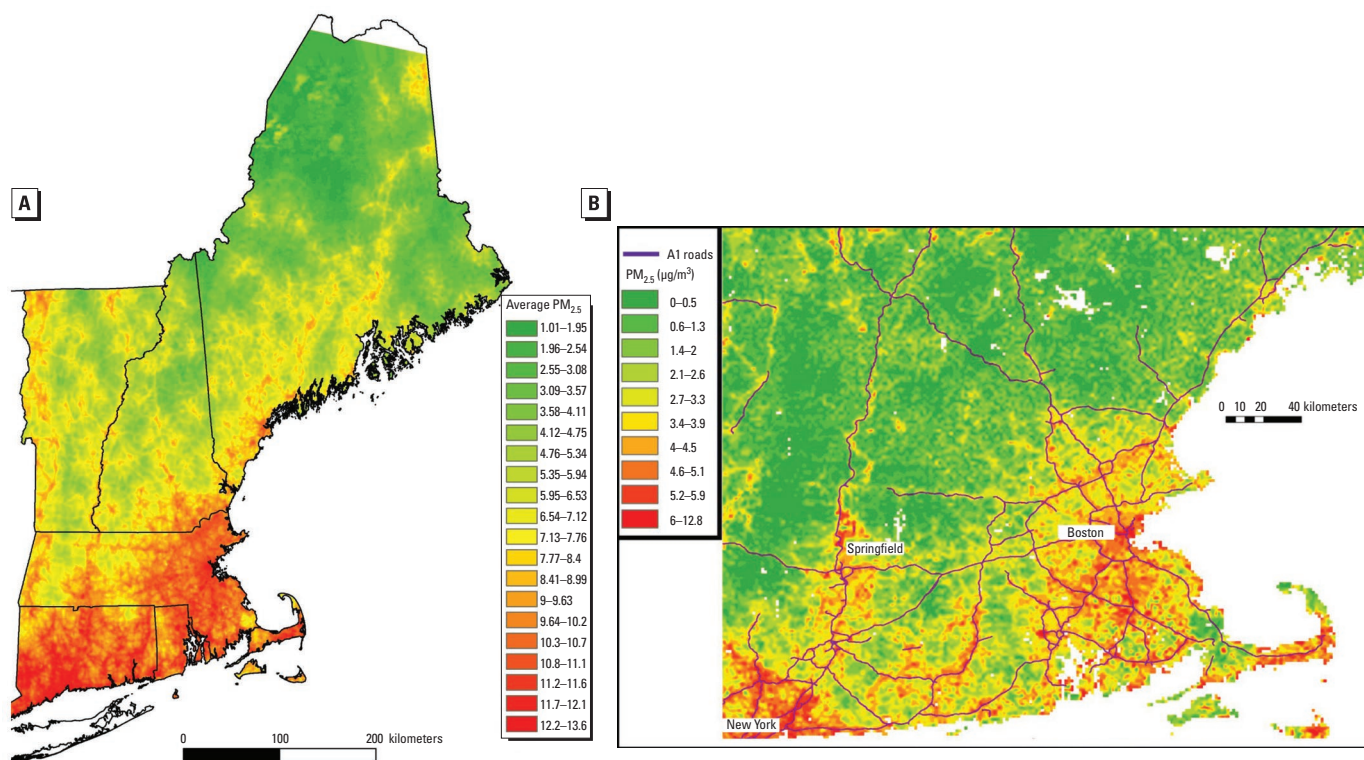
## Methods

**Study domain.** The spatial domain of our study included the New England area, comprising the states of Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont (Figure 1A).

**Exposure data.** A 3-stage statistical modeling approach for predicting daily PM<sub>2.5</sub> was previously reported incorporating AOD and land use data for the New England region (Kloog et al. 2011). Previous studies have shown that using actual physical measurements in our prediction models improved predictive accuracy over that of comparable land use or spatial smoothing models

(Kloog et al. 2011). With AOD retrieved by the multi-angle implementation of atmospheric correction (MAIAC) algorithm, a similar approach was applied for estimating daily PM<sub>2.5</sub> exposures in New England at a spatial resolution of 1 km × 1 km (Kloog et al. 2014a). In this study, the same PM<sub>2.5</sub> exposure predictions were employed.

Briefly, we calibrated the AOD–PM<sub>2.5</sub> relationship on each day of the study period (2003–2008) using data from grid cells with both ground PM<sub>2.5</sub> monitors and AOD measurements (stage 1), and we used inverse probability weighting to address selection bias due to nonrandom missingness patterns in the AOD measurements. We then used the AOD–PM<sub>2.5</sub> relationship to predict PM<sub>2.5</sub> concentrations for grid cells that lacked monitors but had available AOD measurement data (stage 2). Finally, we used a generalized additive mixed model (GAMM) with spatial smoothing and a random intercept for each 1 km × 1 km grid cell to impute data for grid cells/days for which AOD measurements were not available (stage 3). The performance of the estimated PM<sub>2.5</sub> was validated by 10-fold cross-validation. High out-of-sample  $R^2$  ( $R^2 = 0.89$ , year-to-year variation 0.88–0.90 for the years 2003–2008) was found for days with available AOD data. Excellent performance held even in cells/days with no available AOD ( $R^2 = 0.89$ , year-to-year variation 0.87–0.91 for the years 2003–2008). The 1-km model had better spatial (0.87)



**Figure 1.** (A) Mean PM<sub>2.5</sub> concentrations in 2004 at a high resolution (1 km × 1 km) across New England predicted by the AOD models. (B) Predicted PM<sub>2.5</sub> concentrations at a 1 km × 1 km grid for 15 November 2003.

and temporal (0.87) out-of-sample  $R^2$  than the previous 10-km model (0.78 and 0.84, respectively). Details of the  $PM_{2.5}$  prediction models are in Kloog et al. (2014a).

Figure 1A shows an example of mean  $PM_{2.5}$  concentrations in 2004 at a 1 km  $\times$  1 km spatial resolution across New England. By averaging the estimated daily exposures at each location, we generated long-term exposures.

Figure 1B (a subset of the study area) shows that spatial variability existed even for daily data and was not identical to the long-term pattern shown in Figure 1A. That is, there was space–time variation in the  $PM_{2.5}$  exposure captured in this analysis, but not in previous time-series analyses.

Because the deaths were coded at the ZIP code level, both long- and short-term predictions were matched to ZIP codes by using ArcGIS (ESRI, Redlands, CA) and SAS (SAS Institute Inc., Cary, NC) to link the ZIP code centroid to the nearest  $PM_{2.5}$  grid.

Traditionally, studies of acute air pollution effects have controlled for temperature using values taken from the nearest airport. This approach is not feasible for the entire region because many residences are distant from airports. In addition, there is spatiotemporal variation in temperature. We have applied a similar 3-stage statistical modeling approach to estimate daily ambient temperature at 1 km  $\times$  1 km resolution in New England using satellite-derived surface temperature (Kloog et al. 2014b). To our knowledge, such fine control for temperature has not previously been used in air pollution epidemiology.

**Mortality data.** Individual mortality records were obtained from the U.S. Medicare program for all residents  $\geq 65$  years of age for all available years during 2003–2008 (CMS 2013b). The Medicare cohort was used because of the availability of ZIP code of residence data, whereas National Center for Health Statistics mortality data are only available at the county level. Additionally, previous studies found that elderly people are highly susceptible to the effects of particulate matter (Pope 2000). The Medicare beneficiary denominator file from the Centers for Medicare and Medicaid services (CMS 2013a) lists all beneficiaries enrolled in the Medicare fee-for-service (FFS) program and contains information on beneficiaries' eligibility and enrollment in Medicare and the date of death. The Medicare Provider Analysis and Review (MEDPAR) file includes information on age, sex, race, ZIP code of residence, and one record for each hospital admission (CMS 2013c).

Daily mortality was first aggregated by ZIP code and then matched with the corresponding  $PM_{2.5}$  exposure. We summarized the mortality data by ZIP code and day because that was the finest resolution we could obtain for addresses. Because the mortality data sets

did not include changes of residence, we assumed that the subjects lived at their current address over the entire study period.

**Covariates.** We used daily 1-km temperature data estimated from surface temperature measured by satellites (Kloog et al. 2014b). All socioeconomic variables were obtained through the U.S. Census Bureau 2000 Census Summary File 3, which includes social, economic, and housing characteristics (U.S. Census Bureau 2000). ZIP code tabulation area–level socioeconomic variables, including race, education, and median household income, were used. The county-level percentage of people who currently smoke every day, obtained from the CDC Behavioral Risk Factor Surveillance survey for the entire country, was also adjusted (CDC 2013). Dummy variables were used to control for day of the week.

**Statistical models.** Conventionally, the acute effects of air pollution are estimated by Poisson log-linear models, and the chronic effects of air pollution are estimated by Cox proportional hazard models (Kloog et al. 2013; Laden et al. 2006). Laird and Olivier (1981) noted the equivalence of the likelihood of a proportional hazard model with piecewise constant hazard for each year of follow-up and a Poisson regression with a dummy variable for each year of follow-up. We have taken advantage of this equivalence to generalize from dummy variables for each year to a spline of time to represent the baseline hazard and to aggregate subjects into counts per person time at risk, and we obtained a mixed Poisson regression model (Kloog et al. 2012). This approach allows the rate of death as a function of both long- and short-term exposures to be modeled simultaneously. By doing so, we achieved the equivalence of a separate time series analysis for each ZIP code, greatly reducing the exposure error in that part of the model, while simultaneously conducting a survival analysis on the participants, and we were also able to estimate the independent effects of both exposures.

Most time series studies have reported stronger associations with acute exposures when exposures were defined as the mean  $PM_{2.5}$  on the day of death and the previous day (lag01) than when they were defined as the mean  $PM_{2.5}$  on the current day only, or for exposures with longer lags (Schwartz et al. 1996; Schwartz 2004). We used the lag01 average for our main analysis but performed a sensitivity analysis on that choice. Long-term exposure was calculated as the 365-day moving average ending on the date of death so that our results were comparable with those of previous studies (Lepeule et al. 2012; Schwartz et al. 2008). Short-term exposure was defined as the difference between the 2-day average and the long-term average, ensuring that acute and chronic effects were

independent. We subtracted the long-term average from the short-term average to avoid collinearity issues and to ensure that differences between ZIP codes in  $PM_{2.5}$  at a given time did not contribute to the short-term effect estimate. Thus, the short-term effect could not be confounded by variables that differed across ZIP codes.

Specifically, we fit a Poisson survival analysis with a logarithmic link function and a log (population) offset term and modeled the expected daily death counts ( $\mu_{it}$ ) in the  $i$ th ZIP code on the  $t$ th day as follows:

$$\log(\mu_{it}) = \lambda_i + \beta_1 PM_{it} + \beta_2 \Delta PM_{it} + \lambda(t) + \text{temporal covariates} + \text{spatial covariates} + \text{offset}, \quad [1]$$

where  $\lambda_i$  is a random intercept for each ZIP code,  $PM_{it}$  is the 365-day moving average ending on day  $t$  in ZIP code  $i$ ,  $\Delta PM_{it}$  is the deviation of the 2-day average from its long-term average ( $PM_{it}$ ) in ZIP code  $i$ ,  $\lambda(t)$  is a smooth function of time, temporal covariates are temperature and day of the week, and spatial covariates are socioeconomic factors defined at the ZIP code level (percent of people without high school education, percent of white people, median household income) and smoking data at the county level. Additionally, a quasi-Poisson model was used to control for possible overdispersion (Ver Hoef and Boveng 2007).

We estimated  $\lambda(t)$  with a natural cubic spline with 5 degrees of freedom (df) per year to control for time and season trends. The specific temporal and spatial covariates that we used were a natural cubic spline for temperature with 3 df in total; a categorical variable for day of the week; linear variables for percent of people without high school education, percent of white people, median household income, and percent of people who currently smoke every day.

The number of deaths per ZIP code area over the study period (2003–2008) averaged 319 with a standard deviation of 430. Because the outcome was counts, we could not adjust for age and sex as in a Cox model. Instead, we adjusted for variables that varied by ZIP code. The analyses were repeated without mutual adjustment for short- and long-term  $PM_{2.5}$ .

We modeled the association between all-cause mortality and  $PM_{2.5}$  at low doses in which the person-time at risk in each year of follow-up in each ZIP code was used as the offset. We also conducted effect modification by population size by choosing the median (4,628) of the ZIP code–level total population as the cutoff between urban and rural areas.

**Estimating the effects of low-level  $PM_{2.5}$ .** For full cohort analyses with 10,938,852 person-years of follow-up, all observed deaths were used. To estimate effects at low



levels of exposure, we performed restricted analyses: we conducted one analysis restricted to annual exposures < 10 µg/m<sup>3</sup>, below the current annual PM<sub>2.5</sub> NAAQS of 12 µg/m<sup>3</sup>, and another restricted to observations with short-term exposure < 30 µg/m<sup>3</sup>, below the current daily PM<sub>2.5</sub> NAAQS of 35 µg/m<sup>3</sup>. After these exclusions, the chronic analyses were restricted to 268,050 deaths out of 551,024 deaths in total, and the acute analyses were restricted to 422,637 deaths.

**Assessing the dose–response relationship.** For both the acute and chronic analyses, we fit penalized regression splines in the restricted analyses to estimate the shape of the dose–response curve below current U.S. EPA standards. The degrees of freedom of the penalized splines for PM<sub>2.5</sub> were estimated by generalized cross-validation (GCV).

## Results

Table 1 presents a summary of the predicted exposures for both short- and long-term PM<sub>2.5</sub> exposure across all grid cells in the study area.

Table 2 presents the estimated percent change in all-cause mortality with 95% CIs for a 10-µg/m<sup>3</sup> increase in both short- and long-term PM<sub>2.5</sub> in the restricted and full cohort. In the restricted population, we found an estimated 9.28% increase in mortality (95% CI: 0.76, 18.52%) for every 10-µg/m<sup>3</sup> increase in long-term PM<sub>2.5</sub> exposure. A 2.14% increase in mortality (95% CI: 1.34, 2.95%) was observed for every 10-µg/m<sup>3</sup> increase in short-term PM<sub>2.5</sub> exposure. For long-term exposure, the effect estimates were smaller when higher pollution days were included (7.52%; 95% CI: 1.95, 13.40%), suggesting larger effects between low-concentration long-term PM<sub>2.5</sub> and mortality.

Without mutual adjustment, lower estimates were found for both acute and chronic

effects than for those with mutual adjustment. In full-cohort analyses, a 2.08% (95% CI: 1.32, 2.84%) and a 6.46% (95% CI: 0.93, 12.30%) increase in mortality was found for each 10-µg/m<sup>3</sup> increase in short- and long-term PM<sub>2.5</sub>, respectively. In restricted analyses, the corresponding effect estimates were 2.07% (95% CI: 1.27, 2.89%) and 7.16% (95% CI: –1.23, 16.27%), respectively.

Our results were robust to the choice of lag period for acute exposure. We analyzed different averaging periods (Figure 2): for example, lag0 (day of death exposure) and lag04 (a moving average of day of death exposure and previous 4-day exposure). For the acute effects, we found a significant but smaller association for lag0 PM<sub>2.5</sub> (1.71%; 95% CI: 1.09, 2.34%) and lag04 PM<sub>2.5</sub> (1.76%; 95% CI: 0.72, 2.81%) than for lag01 analysis. The lag period used for short-term exposure did not affect estimates of chronic effects. For example, estimated increases in mortality with a 10-µg/m<sup>3</sup> increase in long-term PM<sub>2.5</sub> were 7.35% (95% CI: 1.79, 13.21%) and 7.25% (95% CI: 1.69, 13.12%) when short-term PM<sub>2.5</sub> was classified using lag0 or lag04, respectively.

We also examined effect modification by population size. In the full cohort, a significant interaction was found for chronic effects ( $p < 0.01$ ), with a larger effect of 12.56% (95% CI: 5.71, 19.85%) in urban areas compared with 3.21% (95% CI: –2.92, 9.72%) in rural areas. Such a significant interaction, however, was not observed in the restricted analysis ( $p = 0.16$ ). Estimates were 14.27% (95% CI: 3.19, 26.53%) and 5.48% (95% CI: –4.21, 16.16%) in urban and rural areas, respectively. For short-term exposure, population size did not modify the acute effects in either the full cohort or the restricted analysis ( $p = 0.74$  and 0.46, respectively).

**Table 1.** Descriptive statistics for PM<sub>2.5</sub> exposure and temperature in New England, 2003–2008.

Covariate	Mean	SD	Minimum	Median	Maximum	Range	Q1	Q3	IQR
Lag01 PM <sub>2.5</sub> (µg/m <sup>3</sup> )	8.21	5.10	0.00	7.10	53.98	53.98	4.60	10.65	6.05
1-year PM <sub>2.5</sub> (µg/m <sup>3</sup> )	8.12	2.28	0.08	8.15	20.22	20.14	6.22	10.00	3.78
Temperature (°C)	9.24	6.50	–36.79	9.81	41.51	78.30	4.90	14.39	9.49

**Table 2.** Percent increase in mortality (95% CI) for a 10-µg/m<sup>3</sup> increase for both short-term and long-term PM<sub>2.5</sub>.

PM <sub>2.5</sub> exposure	Model	Percent increase	p-Value
With mutual adjustment			
Short-term PM <sub>2.5</sub>	Low daily exposure <sup>a</sup>	2.14 ± 0.81	< 0.001
	Full cohort	2.14 ± 0.75	< 0.001
Long-term PM <sub>2.5</sub>	Low chronic exposure <sup>b</sup>	9.28 ± 8.88	0.032
	Full cohort	7.52 ± 5.73	0.007
Without mutual adjustment			
Short-term PM <sub>2.5</sub>	Low daily exposure <sup>a</sup>	2.07 ± 0.80	< 0.001
	Full cohort	2.08 ± 0.76	< 0.001
Long-term PM <sub>2.5</sub>	Low chronic exposure <sup>b</sup>	7.16 ± 8.75	0.109
	Full cohort	6.46 ± 5.69	0.026

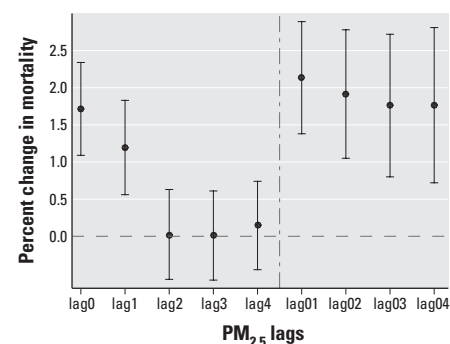
The full cohort analysis had 551,024 deaths.

<sup>a</sup>The analysis was restricted only to person time with daily PM<sub>2.5</sub> < 30 µg/m<sup>3</sup> (422,637 deaths). <sup>b</sup>The analysis was restricted only to person time with chronic PM<sub>2.5</sub> < 10 µg/m<sup>3</sup> (268,050 deaths).

In our penalized spline model for long-term exposure below the cutoff of 10 µg/m<sup>3</sup> (Figure 3A), we found a nonlinear relationship between long-term PM<sub>2.5</sub> and mortality. The association was linear with evidence of a smaller effect < 6 µg/m<sup>3</sup>. However, a large confidence interval was observed; hence, we could not be confident whether the slope of the dose–response curve changed for long-term exposures < 6 µg/m<sup>3</sup>. When examining the shape of the dose–response curve for chronic effects, both a linear term for short-term exposure (the difference) and a penalized spline for long-term average exposure were included in the model, resulting in a penalized spline with a df of 1.71. In contrast, we only included the 2-day average in the penalized spline model of acute effects in order to provide an interpretable dose–response relationship (Figure 3B). The results of this analysis indicated a linear association across the exposure distribution, but we could not be certain about the shape of the slope for acute effects < 3 µg/m<sup>3</sup>.

## Discussion

When we applied the predicted daily PM<sub>2.5</sub> with 1-km spatial resolution from our novel hybrid models, we observed that both short- and long-term PM<sub>2.5</sub> exposure were significantly associated with all-cause mortality among residents of New England ≥ 65 years of age, even when restricted to ZIP codes and times with annual exposures < 10 µg/m<sup>3</sup> or with daily exposure < 30 µg/m<sup>3</sup>. Hence, the association of particle exposure with mortality exists for concentrations below the current standards established by the United States, the World Health Organization (WHO) (10 µg/m<sup>3</sup> of annual average PM<sub>2.5</sub>, 25 µg/m<sup>3</sup> daily), and the European Union (EU) (25 µg/m<sup>3</sup> of annual average PM<sub>2.5</sub>) (EU 2013; WHO 2013). Notably, this analysis includes all areas in New England and all Medicare enrollees ≥ 65 years of age in this region, and it provides chronic effect estimates that are independent of acute effects. Based



**Figure 2.** Percent change in mortality per 10-µg/m<sup>3</sup> increase in short-term PM<sub>2.5</sub> with different lags with mutual adjustment. Error bars indicate the 95% CIs.

on a penalized spline model, the positive dose–response relationship between chronic exposure and mortality appears to be linear for  $\text{PM}_{2.5}$  concentrations  $\geq 6 \mu\text{g}/\text{m}^3$ , with a positive (though smaller and less precise) dose–response slope continuing below this level. This lack of power is likely due to the small exposed population in areas with annual  $\text{PM}_{2.5} < 6 \mu\text{g}/\text{m}^3$ , which were quite rural.

For acute effects, we found a 2.14% (95% CI: 1.38, 2.89%) increase in all-cause mortality per  $10\text{-}\mu\text{g}/\text{m}^3$  increment in  $\text{PM}_{2.5}$  for the full cohort of our study, which is higher than the effect size of most studies using city averages obtained from monitors. For instance, in a U.S. national study by Zanobetti and Schwartz (2009), the effect size was 0.98% (95% CI: 0.75, 1.22%). Similar results were also obtained in a systematic review, where researchers determined that the overall summary estimate was 1.04% (95% CI: 0.52, 1.56%) per  $10\text{-}\mu\text{g}/\text{m}^3$  increment in  $\text{PM}_{2.5}$  (Atkinson et al. 2014). The exposure data used in most previous studies had low spatial resolution (citywide average, not ZIP code), which introduced exposure measurement error and likely resulted in a downward bias in estimates; our results (for the acute effect) are consistent with such a phenomenon. Our restricted study estimated a 2.14% (95% CI: 1.34, 2.95%) increase in all-cause mortality per  $10\text{-}\mu\text{g}/\text{m}^3$  increment in  $\text{PM}_{2.5}$ , which was close to the effect size of the full cohort study, possibly because the sample size of the restricted study for acute effects was close to that of the full cohort. Furthermore, the U.S. EPA daily standard ( $35 \mu\text{g}/\text{m}^3$ ) was almost never exceeded in this study. In addition, lower effect estimates for short-term exposure were observed with mutual adjustment for both full cohort and restricted analyses. This finding has important implications for the interpretation of previous studies without such mutual adjustment.

For chronic effects, the effect estimate in our full cohort study was consistent with findings of previous studies with comparable sample sizes (Hoek et al. 2013; Laden et al. 2006; Lepeule et al. 2012). For example, an ACS study comprising 500,000 adults from 51 U.S. cities reported a 6% (95% CI: 2, 11%) increase in all-cause mortality for each  $10\text{-}\mu\text{g}/\text{m}^3$  increment in  $\text{PM}_{2.5}$  (Pope et al. 2002). A study of 13.2 million elderly Medicare recipients across the eastern United States found a 6.8% (95% CI: 4.9, 8.7%) increase in all-cause mortality for each  $10\text{-}\mu\text{g}/\text{m}^3$  increment in  $\text{PM}_{2.5}$  (Zeger et al. 2008). When we restricted our analysis to annual concentrations  $< 10 \mu\text{g}/\text{m}^3$ , a larger slope of 9.28% (95% CI: 0.76, 18.52%) increase per  $10 \mu\text{g}/\text{m}^3$  was observed. Our findings suggest a larger effect at low concentrations among those  $\geq 65$  years of age, which may also reflect particle

composition. The sources and composition of the particles may differ between low-pollution days and high-pollution days, which are likely more affected by secondary aerosols. Compared with the effect estimate for the full cohort, the effect estimate from the restricted analysis was closer to estimates published in the literature that reported larger effect estimates, such as those reported by the ESCAPE (European Study of Cohorts for Air Pollution Effects) study, the Harvard Six Cities study, and the Women's Health Initiative study (Beelen et al. 2014; Puett et al. 2008). Smaller effect estimates were also observed for chronic effects without mutual adjustment.

To the best of our knowledge, this study is the first of its kind to restrict exposure and to explore the dose–response relationship between  $\text{PM}_{2.5}$  below the current U.S. EPA standards ( $12 \mu\text{g}/\text{m}^3$  of annual average  $\text{PM}_{2.5}$ ,  $35 \mu\text{g}/\text{m}^3$  daily) and mortality. Moreover, the use of the Medicare cohort means that we studied the entire population of Medicare enrollees  $\geq 65$  years of age and not a convenience sample. In addition, temperature was controlled on a  $1 \text{ km} \times 1 \text{ km}$  fine geographic scale. The acute and chronic effects observed in analyses restricted to low  $\text{PM}_{2.5}$  exposure were similar to or even higher than those of the full cohort analyses. These results indicate that the adverse health effects of  $\text{PM}_{2.5}$  are at least retained, if not strengthened, at low levels of exposure. However, the findings from the penalized spline model did not support a strong association at the lowest range of  $\text{PM}_{2.5}$  concentrations. This finding provides epidemiological evidence for the reevaluation of U.S. EPA guidelines and standards, although more evidence is needed to confirm the association  $< 6 \mu\text{g}/\text{m}^3$ .

The Poisson survival analysis applied in this study provided a novel method of simultaneously assessing acute and chronic effects. As shown in our analysis, the chronic effect estimate was much larger than the acute effect estimate after controlling for the acute

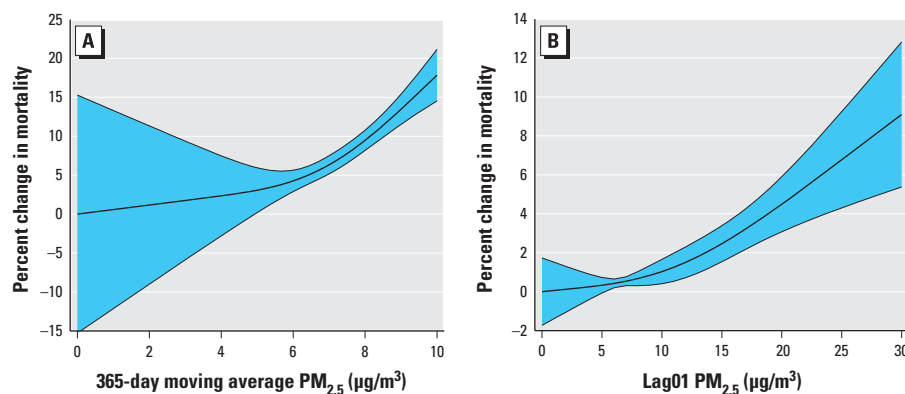
estimate, indicating that there were chronic effects of  $\text{PM}_{2.5}$ , which cannot be solely explained by the short-term exposure.

Another key component of this study is that the application of high spatial ( $1 \text{ km} \times 1 \text{ km}$ ) and temporal (daily) resolution of  $\text{PM}_{2.5}$  concentrations reduced exposure error to a certain extent. The out-of-sample  $R^2$  was higher than that for the predictions with  $10 \text{ km} \times 10 \text{ km}$  spatial resolution.

A potential limitation is the limited availability of individual-level confounders, such as smoking status, which could bias the health effect estimates. We were able to control for ZIP code–level education, median income, race, and county-level smoking data. However, Brochu et al. (2011) reported that census tract–level socioeconomic indicators were uncorrelated with  $\text{PM}_{2.5}$  on the subregional and local scale, providing some assurance that confounding by socioeconomic status may not be much of an issue. The results reported by Brochu et al. (2011) suggest that those variables may not confound the association, but the inability to control for them remains an issue. Another limitation is that we did not examine other pollutants such as ozone ( $\text{O}_3$ ) or nitrogen dioxide ( $\text{NO}_2$ ) owing to a lack of data at the same spatial level as that of  $\text{PM}_{2.5}$ .

## Conclusions

In conclusion, the acute and chronic effects of low-concentration  $\text{PM}_{2.5}$  were examined for a Medicare population using a comprehensive exposure data set obtained from a satellite-based prediction model. Our findings show that both short- and long-term exposure to  $\text{PM}_{2.5}$  were associated with all-cause mortality, even for exposure levels not exceeding the newly revised U.S. EPA standards, suggesting that adverse health effects occur at low levels of fine particles. The policy implication of these findings is that improving the air quality at even lower levels of  $\text{PM}_{2.5}$  than presently allowed by the U.S. EPA standards can yield health benefits.



**Figure 3.** The dose–response relationship between long-term  $\text{PM}_{2.5}$  and mortality at low doses with mutual adjustment (A) and the dose–response relationship between short-term  $\text{PM}_{2.5}$  and mortality at low doses without mutual adjustment (B). Shaded areas indicate the 95% CIs.

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# Estimating Causal Effects of Local Air Pollution on Daily Deaths: Effect of Low Levels

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**BACKGROUND:** Although many time-series studies have established associations of daily pollution variations with daily deaths, there are fewer at low concentrations, or focused on locally generated pollution, which is becoming more important as regulations reduce regional transport. Causal modeling approaches are also lacking.

**OBJECTIVE:** We used causal modeling to estimate the impact of local air pollution on mortality at low concentrations.

**METHODS:** Using an instrumental variable approach, we developed an instrument for variations in local pollution concentrations that is unlikely to be correlated with other causes of death, and examined its association with daily deaths in the Boston, Massachusetts, area. We combined height of the planetary boundary layer and wind speed, which affect concentrations of local emissions, to develop the instrument for particulate matter  $\leq 2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ), black carbon (BC), or nitrogen dioxide ( $\text{NO}_2$ ) variations that were independent of year, month, and temperature. We also used Granger causality to assess whether omitted variable confounding existed.

**RESULTS:** We estimated that an interquartile range increase in the instrument for local  $\text{PM}_{2.5}$  was associated with a 0.90% increase in daily deaths (95% CI: 0.25, 1.56). A similar result was found for BC, and a weaker association with  $\text{NO}_2$ . The Granger test found no evidence of omitted variable confounding for the instrument. A separate test confirmed the instrument was not associated with mortality independent of pollution. Furthermore, the association remained when all days with  $\text{PM}_{2.5}$  concentrations  $> 30 \mu\text{g}/\text{m}^3$  were excluded from the analysis (0.84% increase in daily deaths; 95% CI: 0.19, 1.50).

**CONCLUSIONS:** We conclude that there is a causal association of local air pollution with daily deaths at concentrations below U.S. EPA standards. The estimated attributable risk in Boston exceeded 1,800 deaths during the study period, indicating that important public health benefits can follow from further control efforts.

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## Introduction

Starting in the late 1980s, a large literature of time series studies have reported associations of daily air pollution concentrations with daily deaths (Analitis et al. 2006; Bell et al. 2004, 2013; Carbajal-Arroyo et al. 2011; Dominici et al. 2005; Fischer et al. 2003; Krall et al. 2013; Maynard et al. 2007; Peng et al. 2013; Samoli et al. 2006; Schwartz 2004a, 2004b; Stölzel et al. 2007; Zanobetti and Schwartz 2008, 2009). The most consistent results have been that particle concentrations are associated with daily mortality.

Fewer studies have examined the effects of source-specific particle contributions or individual particle species. Several large multicity studies have reported stronger associations for particle sulfate and nickel (Bell et al. 2014; Dai et al. 2014; Franklin et al. 2008). The U.S. Environmental Protection Agency's (EPA's) recent transport regulation has already produced substantial reductions in sulfate particles, and is scheduled to reduce remaining sulfur emissions further in the next few years (U.S. EPA 2016). As the sulfate contribution to particle mass declines and  $\text{NO}_x$  (nitrogen oxides) controls affect secondary organic particle formation, local

emissions of particulate and gaseous pollutants will become a more important part of the pollution mix; thus it is important to enhance our understanding of their health impact.

The observational epidemiology studies cited above have been associational studies, which do not assess causality. In general, when arguing for the causality of observed associations, authors have relied on Hill's Criteria (Hill 1965). For example, Brook et al. (2010) state "Many potential biological mechanisms exist whereby PM exposure could exacerbate existing CVDs [cardiovascular diseases] and trigger acute cardiovascular events (over the short term) and instigate or accelerate chronic CVDs (over the long run)." Besides biological plausibility, the  $\text{PM}_{2.5}$  (particulate matter  $\leq 2.5 \mu\text{m}$ ) epidemiological studies were relatively consistent, and exposure preceded effect.

The strength of the biological plausibility argument has grown over time (Brook et al. 2004), and includes studies indicating that particle exposure can induce lung and systemic inflammation (Adamkiewicz et al. 2004; Adar et al. 2007a; Araujo 2010; Brook 2008; Driscoll 2000; Dye et al. 2001; Folkmann et al. 2007), increase blood pressure (Baccarelli

et al. 2011; Bartoli et al. 2009; Brook et al. 2009; Hoffmann et al. 2012; Schwartz et al. 2012; Wilker et al. 2010; Zanobetti et al. 2014), impair microvascular function (Brauner et al. 2008), increase coagulation and thrombosis (Baccarelli et al. 2007, 2008; Bind et al. 2012; Bonzini et al. 2010; Carlsten et al. 2007; Chuang et al. 2007; Gilmour et al. 2005; Nemmar et al. 2002), produce autonomic changes (Adar et al. 2007b; Chahine et al. 2007; Chan et al. 2004; Ghelfi et al. 2008; Zhong et al. 2015), accelerate atherosclerosis (Adar et al. 2010; Allen et al. 2009; Araujo et al. 2008; Bauer et al. 2010; Bhatnagar 2006; Hansen et al. 2007; Hoffmann et al. 2007; Sun et al. 2005, 2008; Suwa et al. 2002; Tzeng et al. 2007), and destabilize atherosclerotic plaque (Suwa et al. 2002).

There are fewer and less consistent studies assessing the effects of particle components. For example, Krall et al. (2013) and Bell et al. (2014) reported a greater toxicity for elemental (or black) carbon, a large fraction of which is associated with local traffic and domestic heating, whereas Franklin et al. (2008), Beelen et al. (2015) and Dai et al. (2014) found greater effects for sulfur and not elemental carbon.

There is biological support for a role of local traffic particles. Diesel particles have been shown to increase oxidative stress in endothelial cells (Furuyama et al. 2006; Hirano et al. 2003), inducing the production of heme oxygenase-1, a rapid response part of

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the body's defense system against oxidative stress (Choi and Alam 1996). The viability of cell cultures of microvascular endothelial cells was also impaired by diesel particles with an accompanying large increase in induction of heme oxygenase-1 (Hirano et al. 2003).

A key gap in the analysis of the acute effects of local air pollution sources has been the lack of studies done in the framework of causal modeling, specifying potential outcomes, and basing their analysis on estimating the difference or ratio of potential outcomes under different exposures. In this paper, we use a causal modeling framework to estimate the causal acute effects of local pollution on daily deaths.

## Methods

### Causal Modeling

To establish causality specification of potential outcomes is required. We designate  $Y_i^A = a$  as the outcome that would occur given an exposure  $A = a$  for the unit  $i$ , and  $Y_i^{A'} = a'$  to be the outcome that would occur if the unit  $i$  were instead exposed to an alternative exposure,  $A = a'$ . Causal modeling seeks to estimate the ratio of the expected value of outcome in the population of subjects  $i$  under the exposure they received versus what it would have been had they received the alternative exposure:  $E(Y_i^A = a)/E(Y_i^{A'} = a')$ . Because only one potential outcome is observed, various methods seek legitimate surrogates for the unobserved potential outcome (Hernán et al. 2008). In this paper, we apply the approach of instrumental variables. An instrumental variable is a variable that is related to outcome only through the exposure of interest.

### Instrumental Variables

Let  $Y_t^A = a$  be the potential outcome (total deaths) in the population of a city exposed to  $A = a$  on day  $t$ , and let  $Y_t^{A'} = a'$  be the potential outcome under the alternative exposure  $a'$ . We would like to estimate  $E(Y_t^A = a)/E(Y_t^{A'} = a')$ , but only  $Y_t^A = a$  is observed. We assume the potential outcome depends on predictors as follows:

$$\text{Log}[E(Y_t^A = a)] = \theta_0 + a\theta_1 + \Phi_t \quad [1]$$

where  $Y_t^A = a$  represents the potential outcome at time  $t$  under exposure  $a$ ,  $\theta_0$  and  $\theta_1$  are the intercept and the slope of exposure, respectively, and  $\Phi_t$  represents all of the other predictors of outcome. Unless we have measured all of the confounders, standard methods, including standard approaches to causal modeling, will give biased estimates of  $\theta_1$ . However, air pollution has many sources of variation. If there is a variable  $Z$  that is one such source of variation in exposure, and  $Z$  is associated with  $Y$  only through  $A$ , then  $Z$  is called

an instrumental variable. Figure 1 shows the directed acyclic graph (DAG) for this scenario. Consequently,  $A_t$  can be expressed as follows:

$$A_t = Z_t\delta + \eta_t \quad [2]$$

where  $\eta_t$  represents the other sources of variation in exposure, and particularly all of the exposure variations that are associated with other measured or unmeasured predictors of outcome. This follows because of the instrument assumption, that  $Z$  is only related to  $Y$  through  $A$ . Formally,  $E(Z|\Phi_t) = 0$  because of the instrument assumption. Then let  $Z1$  and  $Z2$  be equal to  $Z$  such that:

$$E(A|Z1) = a, \text{ and } E(A|Z2) = a'.$$

Consequently,

$$\begin{aligned} \text{Log}[E(Y_t^Z = Z1)] \\ &= E(\theta_0 + \theta_1 a + \Phi_t | Z = Z1) \\ &= \theta_0 + \theta_1 a + E(\Phi_t) \end{aligned} \quad [3]$$

and

$$\begin{aligned} \text{Log}[E(Y_t^Z = Z2)] \\ &= E(\theta_0 + \theta_1 a' + \Phi_t | Z = Z2) \\ &= \theta_0 + \theta_1 a' + E(\Phi_t) \end{aligned} \quad [4]$$

therefore

$$\begin{aligned} \text{Log}[E(Y_t^Z = Z1)] - \text{Log}[E(Y_t^Z = Z2)] \\ &= \theta(a - a'). \end{aligned} \quad [5]$$

As a result, if we use  $Z$  as an instrument for  $A$ , we can recover a causal estimate for  $\theta$ , which is the log rate ratio. Importantly, this is true even if there are unmeasured confounders.

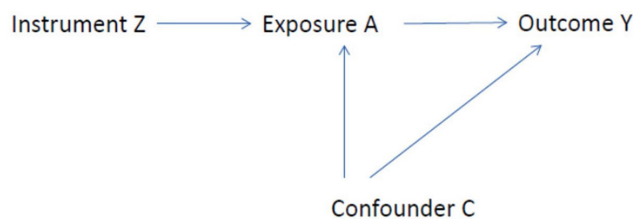
Put less formally, in an observational study the exposure is not randomly assigned, so it may be correlated with other predictors of the outcome. However, air pollution (and other exposures) varies for many reasons. Some of them may be correlated with other predictors of daily deaths. For example, worse-than-average traffic on 1 day will increase both air pollution and stress. However, some sources of variation in air pollution may not be correlated with other predictors of daily deaths. For example, wind speed is unlikely to be correlated with daily stress, smoking, and the like. Hence, if this is true, the fraction of air pollution variation that is produced by wind speed

is randomized with respect to confounders, including unmeasured ones; and if that fraction is associated with daily deaths, the estimated effect should be causal. We discuss this further below.

### Planetary Boundary Layer and Wind Speed as Instruments

The difficulty with instrumental variable analyses is finding a valid instrument that is associated only with outcome through the exposure of interest. Mendelian randomization is an example of an instrumental variable successfully applied in epidemiology, and is justified by knowledge that the biological pathway by which the genotype is associated with exposure is not associated with other predictors of outcome (Holmes et al. 2014). Hence external knowledge is critical to the technique.

The air pollution above a city is a mix of locally emitted pollutants and pollutants transported from elsewhere. The lowest part of the atmosphere, along with its behavior, is influenced by its contact with a planetary surface, which is called planetary boundary layer (PBL) and is characterized by strong vertical mixing (Finlayson-Pitts and Pitts 1986). Above the PBL lies the free atmosphere, which is mostly nonturbulent. The transport of pollutants from the boundary layer to the free atmosphere is slow relative to their vertical mixing within the boundary layer (Seinfeld and Pandis 1998). Therefore, the impact of local emissions on pollutant levels is directly related to the height of the PBL (e.g., for the same local emissions, concentrations of locally emitted pollutants are higher when the boundary layer is low and vice versa) (Seinfeld and Pandis 1998). As a result, the influence of the local emissions is modified by the atmospheric conditions. Over land, the PBL height exhibits a strong diurnal variability, with lower values at night. In addition, the mean PBL height varies substantially from day to day (Seinfeld and Pandis 1998). Besides the vertical transport (influenced by the PBL), locally emitted air pollutants are also transported horizontally, where the influence of local sources increases with decreasing wind speed and vice versa. It is hard to imagine how the PBL height can be directly related to health except through air



**Figure 1.** Directed acyclic graph illustrating an instrumental variable  $Z$ . The association between  $Z$  and  $Y$  is not confounded by  $C$ . By calibrating the instrument to  $A$ , estimates of causal effects of increases in  $A$  can be obtained.

pollution. Similarly, outside of extreme events, wind speed is an unlikely predictor of health other than through air pollution. As such, PBL height and wind speed represent attractive options as instruments for local pollution. However, PBL height and wind speed may vary seasonally and with temperature and other meteorological parameters. We believe that within strata of month and deciles of temperature, further association with predictors of health is unlikely. Hence we looked at local air pollution variation only within month-by-year strata and within deciles of temperature (for the full period), and calibrated that variation with our instruments—that is, we assume short-term predictors of mortality such as smoking, anger, and the like to be uncorrelated with PBL height on a day-to-day level, within month-by-year and decile of temperature. Our analysis took this into account.

A low PBL height and low wind speed are associated with increases in the concentrations of all locally emitted pollutants. Hence, when combined into an instrument, it can tell us that local pollution increases mortality rates (or not), but it will be difficult to identify which pollutants are responsible for the changed mortality rate.

If a single variable is used as an instrument, that variable can obtain the estimated causal effect of exposure on the outcome by regressing the outcome on the instrument, and the instrument on the exposure of interest. The product of those coefficients is the estimated causal effect per unit increase in exposure. Because we have four instrumental variables (PBL and wind speed at lag 0 and lag 1), we regressed the pollution against the four variables first, and used that result (the variation in pollution explained by the four instrumental variables) to generate a single instrumental variable for regression on the outcome. We have chosen to use these variables as instruments for PM<sub>2.5</sub> (particulate matter with aerodynamic diameter  $\leq 2.5$   $\mu\text{m}$ ) as the pollutant most strongly associated with daily deaths. However, this does not demonstrate that the results are attributable exclusively to particles. We evaluated two alternative air pollutant exposures as a sensitivity analysis: black carbon (BC), which represents traffic particles, a large fraction of them locally emitted, and nitrogen dioxide (NO<sub>2</sub>), which is mostly from local combustion.

## Data

### Mortality Data

We analyzed data from the Boston metropolitan area, which includes the following counties: Middlesex, Norfolk, and Suffolk. Mortality data were obtained from the Massachusetts Department of Public Health for the years 2000–2009. The mortality files provided information on the exact date of

death and the underlying cause of death. We chose all-cause non-accidental daily mortality [*International Classification of Diseases, 9th Revision* (ICD-9) codes 0–799] as our outcome to ensure sufficient statistical power.

### Air Quality Data

PM<sub>2.5</sub> and BC measurements were conducted at the Harvard Supersite located on the roof of the Countway Library of the Harvard Medical School near downtown Boston. Ambient BC was measured continuously using an aethalometer (Magee Scientific), and PM<sub>2.5</sub> was measured continuously using a tapered element oscillating microbalance (model 1400a; Rupprecht & Pataschnick Co). Daily averages were computed from the hourly values. We used publicly available daily data on the height of the PBL obtained from the NOAA (National Oceanic and Atmospheric Administration) Reanalysis Data (NOAA 2010). Ambient temperature and wind speed were obtained from the Logan Airport meteorological station.

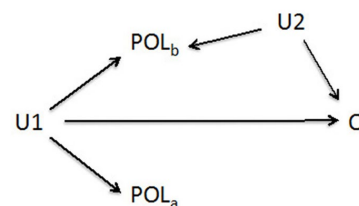
### Analysis

First we orthogonalized our local air pollution exposures to season and temperature by fitting them to a model with dummy variables for each month of each year, and for each decile of temperature. We used four individual variables to derive one single pollution-calibrated instrumental variable: PBL height and wind speed on the day of death (lag 0) and PBL height and wind speed on the day before death (lag 1). To do this, we used a support vector regression (SVM) (Cortes and Vapnik 1995) with a radial kernel to estimate the remaining variation in PM<sub>2.5</sub> (or in BC or NO<sub>2</sub>) that was explained by those four variables and their products including potential nonlinear dependencies on the predictors. This approach (support vector kernel regression with the radial basis kernel) combines our four instruments into one pollution calibrated instrument, and allows us to compare interquartile range (IQR) changes in the instruments for local pollution computed using each of the pollutants (PM<sub>2.5</sub>, BC, or NO<sub>2</sub>) as an indicator. The kernel regression also incorporates a ridge penalty to shrink the coefficients of the multiple terms to avoid overfitting and collinearity problems. We chose the parameters of the SVM to maximize 10-fold cross-validated  $R^2$ . We used the svm function in the R package *e1071* (version 3.2; R Project for Statistical Computing). We checked the  $R^2$  of the instrument predicting exposure to ensure our instrument was not too weakly associated with exposure to detect an effect. Because previous literature has most commonly used the mean of PM<sub>2.5</sub> on the day of death and the day preceding death as the exposure of interest, we used the mean of the instrumental variable on the day of death and the

day preceding the death as our exposure, and fit a quasi-Poisson regression (allowing for overdispersion) predicting all-cause mortality. We stratified by each month of each year and by deciles of temperature, using indicator variables, and estimated the rate ratio for the instrument.

Boston has lower than average pollution levels for a U.S. city, and there were no violations of the NO<sub>2</sub> annual National Ambient Air Quality (<https://www.epa.gov/criteria-air-pollutants/naaqs-table>) standard of 53 ppb during the study period. There were 19 days which exceeded the new U.S. EPA PM<sub>2.5</sub> daily standard of 35  $\mu\text{g}/\text{m}^3$ . To assure our results apply to low-dose exposures, we repeated the analyses with the instrument excluding days when PM<sub>2.5</sub> exceeded 30  $\mu\text{g}/\text{m}^3$  to ensure that even with measurement error the exposure was below the ambient standard. This excluded 39 days. There are currently no standards for BC.

Granger causality is not a true causal modeling approach, but a heuristic one that argues that omitted covariates that are correlated with time-varying exposure and outcome are as likely to be correlated with tomorrow's exposure as with yesterday's exposure. Hence, if no association is found between future values of exposure and outcome, that suggests there is no omitted confounder. Flanders et al. (2011) give a stronger causal framework using DAGs, and note that the Granger causality approach assumes that, conditional on exposure and all confounders, exposure after the outcome should be uncorrelated with the outcome. However, exposure after the outcome and exposure before the outcome are both associated with the confounders, as illustrated in the DAG in Figure 2. Therefore, in the presence of omitted confounders an association may be expected with the future exposure. Hence, if we fit a model with the past exposure and the future exposure and find an association only with the past exposure, that would argue against such omitted confounders, and vice versa. We tested this approach by rerunning our instrumental variable model with the mean of the instrument (lags 0 and 1) and the



**Figure 2.** Directed acyclic graph for the Granger causality model. Confounder  $U_2$  is measured and controlled, but confounder  $U_1$  is not.  $POL_b$  is pollution before the outcome ( $O$ ), and  $POL_a$  is pollution after the outcome. If  $U_1$  is not controlled, there is a backdoor path from  $O$  to  $POL_a$ , and an association would be expected. Hence, failure to find an association is evidence of a lack of confounding (i.e., no  $U_1$ ).



mean value of the instrument on the second and third days after death. We left 1 day between the exposure before the event and the exposure after the event to produce more stable estimates for each association, given the serial correlation in pollution.

We also conducted a sensitivity analysis to test our assumption that we had a valid instrument. Looking at Figure 1 again, we see that the instrumental variable ( $Z$ ) is associated only with the outcome through the exposure ( $A$ ) (the assumption for instrumental variables). That is, the exposure can be viewed as a mediator of the association of the instrumental variable with the outcome. Then if we control for  $A$ , there should be no association with the instrument any longer (no direct effect) by that assumption. If, in contrast, an association remains, then there is another path from  $Z$  to the outcome, through some confounder. We tested this by fitting a model with both our instrument and the original exposure variable ( $PM_{2.5}$ ).

To put our results in context, we performed a quantitative health impact assessment. Specifically, we estimated the reduction in deaths during the 10 years of study for an IQR reduction in our instrumental variable (after ensuring that such a reduction from the mean would result in an exposure above zero). This was estimated as

$$\text{change in deaths} = \frac{RR - 1}{RR} \text{Total Deaths}$$

where  $RR$  is the rate ratio for the change in exposure,  $\exp(b1 \times \text{IQR})$  where  $b1$  is the coefficient of the instrumental variable, and  $\text{IQR}$  is its interquartile range. This approach is standard in risk assessment (Fann et al. 2011; GBD 2013 Risk Factors Collaborators et al. 2015; U.S. EPA 1999). We computed the total deaths during follow-up (204,386) from our data.

## Results

Table 1 shows descriptive statistics for the variables in our study. Air pollution concentrations were low, and almost always well

**Table 1.** Descriptive statistics of the data: air pollution and daily deaths in Boston, 2000–2009.

Variable	Mean $\pm$ SD	Min	Max
Daily deaths <sup>a</sup>	55.8 $\pm$ 9.5	27	94
$PM_{2.5}$ ( $\mu\text{g}/\text{m}^3$ ) <sup>b</sup>	9.8 $\pm$ 5.8	0.2	67.2
BC ( $\mu\text{g}/\text{m}^3$ ) <sup>b</sup>	0.70 $\pm$ 0.41	0.10	4.70
$\text{NO}_2$ (ppb) <sup>c</sup>	18.4 $\pm$ 6.4	4.0	46.9
PBL (m) <sup>d</sup>	770 $\pm$ 356	110	2,392
Temperature ( $^{\circ}\text{C}$ ) <sup>e</sup>	10.8 $\pm$ 9.4	−16.9	31.5
Wind speed (knots) <sup>e</sup>	9.6 $\pm$ 3.2	2.5	26

Abbreviations: max, maximum; min, minimum.

<sup>a</sup>Data from MA Department of Public Health.

<sup>b</sup>Data measured at Harvard Supersite.

<sup>c</sup>Data from MA Department of Environmental Protection.

<sup>d</sup>Data from NOAA North America Reanalysis data set (NOAA 2010).

<sup>e</sup>Data from National Climatic Data Center.

below the current U.S. EPA standards (results not shown). Table 2 shows the correlations among the covariates. The correlation between  $PM_{2.5}$  and BC was 0.65, between  $PM_{2.5}$  and  $\text{NO}_2$  was 0.45, and between BC and  $\text{NO}_2$  was 0.57. The correlation between air pollution and the candidate instruments were modest. For example, for  $PM_{2.5}$ , the correlation with PBL height was  $-0.35$ , and with wind speed was  $-0.28$ .

## Instrumental Variable Model

If a model predicting a variable is over fit (e.g., uses too many degrees of freedom), then one would expect the predicted  $R^2$  on left-out monitors to be noticeably smaller than the model  $R^2$  in the training data set. The cross-validated  $R^2$  of the instrumental variable predicting  $PM_{2.5}$  was 0.180, little changed from the  $R^2$  in the training data (0.189). Although low, this is consistent with the fact that most of the  $PM_{2.5}$  in Boston is transported rather than locally emitted, and with PM having other important sources of variation besides PBL and wind speed (Masri et al. 2015). Overfitting was avoided because the tuning parameters of the model calibrating the instrument to  $PM_{2.5}$  were chosen by cross-validation, and because the SVM uses a ridge penalty, where a penalty term is added to the cost function proportional to the sum of the square of the regression coefficients. This penalty constrains the coefficients from varying wildly, or growing too large.

As expected, PBL height and wind speed were better predictors of BC (a large fraction of which is locally emitted) than of  $PM_{2.5}$ . The cross-validated  $R^2$  of the SVM model for BC was 0.36, versus 0.37 without cross-validation. Similarly, the SVM model for  $\text{NO}_2$  had a cross-validated  $R^2$  of 0.39, versus 0.40 without cross-validation.

## Mortality Model

An IQR change in the instrument for local  $PM_{2.5}$  was associated with a 0.90% increase in daily deaths [95% confidence interval (CI): 0.25, 1.56], whereas an IQR change in the instrument for BC was associated with a 0.90% increase in daily deaths (95% CI: 0.08, 1.73). For  $\text{NO}_2$ , an IQR increase in the instrument was associated with a 0.62% increase in daily deaths (95% CI:  $-0.12$ , 1.64). We compared IQR changes for the instrumental variables to

have some basis for comparing effects between the models for  $PM_{2.5}$ , BC, and  $\text{NO}_2$ . When the mortality analysis was restricted to days when  $PM_{2.5}$  was  $< 30 \mu\text{g}/\text{m}^3$  (which excluded 39 days), we found a 0.84% increase in daily deaths for the same increase in the instrument (95% CI: 0.19, 1.50).

When we used the Granger causality approach, the estimated effect of an IQR change in the instrument for  $PM_{2.5}$  remained the same (0.90%; 95% CI: 0.25, 1.96), whereas the forward lagged instrument was not associated with mortality (0.18%; 95% CI:  $-0.45$ , 0.81), suggesting no omitted confounders. Although the power for a Granger causality test may not be strong, the much smaller effect size as well as lack of significance both indicate a lack of confounding.

Finally, when we added the mean of  $PM_{2.5}$  on lags 0 and 1 to the model in addition to the instrumental variable, the instrumental variable was far from significant ( $p > 0.29$ ) while the  $PM_{2.5}$  variable was significant. This indicates that there was no path from instrument to the outcome except through  $PM_{2.5}$ , and hence that the instrumental variable assumption was valid.

## Discussion

Using a framework based on potential outcomes, we have estimated the causal effect of an IQR increase in local air pollution on daily deaths in Boston. The increase in deaths for an IQR increase in the instrument for exposure was about 0.90% using either particle measure to calibrate the instrument; for  $\text{NO}_2$  it was lower (0.62%) with confidence intervals that crossed zero. Using the approach of Granger causality, we saw no change in the estimated effect of our instrument when controlling for exposure on future days and the association with future exposure was close to zero and far from significant. Further, the association persisted when restricted to days well below the recently tightened U.S. EPA 24-hr standard for  $PM_{2.5}$  ( $35 \mu\text{g}/\text{m}^3$ ), and in a city that never violated the hourly  $\text{NO}_2$  National Ambient Air Quality standard during the study period. Hence, these effects are evident at levels below currently permissible limits.

A key advantage of the instrumental variable approach is that it provides protection against unmeasured confounders. We have approached this in three ways. First, we

**Table 2.** Correlation matrix of the exposures.

	$PM_{2.5}$ ( $\mu\text{g}/\text{m}^3$ )	BC ( $\mu\text{g}/\text{m}^3$ )	$\text{NO}_2$ (ppb)	PBL (m)	Temperature ( $^{\circ}\text{C}$ )	Wind speed (knots)
$PM_{2.5}$ ( $\mu\text{g}/\text{m}^3$ )	1					
BC ( $\mu\text{g}/\text{m}^3$ )	0.65	1				
$\text{NO}_2$ (ppb)	0.45	0.57	1			
PBL (m)	$-0.35$	$-0.52$	$-0.35$	1		
Temperature ( $^{\circ}\text{C}$ )	0.30	0.26	$-0.25$	$-0.23$	1	
Wind speed (knots)	$-0.28$	$-0.52$	$-0.37$	0.54	$-0.28$	1

have shown that if we have a valid instrument, then the association will be causal even in the presence of unmeasured confounders. We focused on the variation in local pollution within deciles of temperature and also stratified on each month of each year. We then chose as instruments variables (PBL height and wind speed) we believed, based on external knowledge, are unlikely to be associated with mortality except through air pollution. Second, we have confirmed that values of the instrument following the day of death are not significantly associated ( $p = 0.57$ ) with daily deaths, and that control for them did not change the estimated effect of the instrument. This assures that omitted confounders with the same broad temporal variability are not confounding our instrument. And third, we have tested the instrument assumption (that the association of the instrument is only through air pollution) by controlling for air pollution, and showing that no significant association with the instrument remained ( $p > 0.29$ ). We believe that this makes a strong case for a causal effect.

Support for this causal interpretation also comes from an extensive toxicological and human exposure literature on some of these local pollutants. For example, Furuyama et al. (2006) found increased oxidative stress in endothelial cells exposed to diesel exhaust, and in humans Rossner et al. (2007) reported increased levels of F-2 isoprostane and 8-OHdG (8-hydroxy-2'-deoxyguanosine) in bus drivers compared with controls. The human study contrasted urinary 8-OHdG in 50 bus drivers and 50 controls measured in three successive seasons in Prague. In logistic regression analysis,  $PM_{2.5}$ , but not volatile organic compound or polycyclic aromatic hydrocarbon exposure, was associated with 8-OHdG. Romieu et al. (2008) measured malondialdehyde in exhaled breath condensate at 480 visits in a panel of 108 children with asthma seen every 2 weeks, and found it was positively associated with  $PM_{2.5}$  at the nearest monitoring station within 5 km of their home and school.

Increased atherosclerosis has also been reported in animals with long-term exposure to particles, much of which was from traffic (Sun et al. 2005, 2008). Another study (Soares et al. 2009) placed hyperlipemic mice in two exposure chambers 20 m from a road. One chamber was filtered to remove particles and the other was not. After 120 days of exposure they documented increased oxidation of low-density lipoprotein, increased thickness of the arterial wall, and greater plaque growth and instability (Soares et al. 2009). Along with the increased oxidative stress, atherosclerosis, and plaque instability, increased thrombosis has also been associated with local pollution. Nemmar et al. (2002, 2003) found

that both diesel and ultrafine particles were associated with increased thrombosis in an animal model, and Carlsten et al. (2007, 2008) found that controlled exposure to diesel exhaust increased coagulation markers and thrombosis in human volunteers. Ischemia has likewise been produced experimentally by diesel exposure in a double-blind randomized crossover exposure of 20 people with previous myocardial infarction to 1 hr of dilute diesel exhaust or filtered air (Mills et al. 2007).

An intervention trial in Beijing had 15 young adults (median age, 28 years) walk the streets for 2 hr twice, once wearing a particle-filtering mask, and once without a mask. Blood pressure was measured continuously during the two 2-hr walks and was 7 mmHg lower when wearing the mask (Langrish et al. 2009). These results, combined with the instrumental variable approach and Granger causality model, support a causal interpretation.

The weaker association of the instrumental variables when calibrated to  $NO_2$  than to particles suggests that local particles may be more important in this relationship, but no definite conclusions can be drawn.

To put this result in context, the mean  $PM_{2.5}$ ,  $NO_2$ , and BC ( $9.8 \mu g/m^3$ , 18.4 ppb, and  $0.7 \mu g/m^3$ ) were all greater than their IQRs ( $6.32 \mu g/m^3$ , 8.4 ppb, and  $0.50 \mu g/m^3$ , respectively), indicating that IQR changes in the pollutant concentrations would result in levels above zero, and hence are plausible. Computing the attributable risk for an IQR change in exposure to the instrument, we estimated that local air pollution was responsible for 1,826 deaths in the Boston metropolitan area during the study period. This is a substantial public health burden.

Local air pollution in Boston has multiple sources, including traffic, combustion of fuel oil and residual oil for heating, and wood burning (Masri et al. 2015). Traffic pollution has fallen because of reduced U.S. EPA emission standards on vehicles, low-sulfur diesel oil requirements, the retrofit of particle filters onto buses, and the introduction of compressed natural gas buses for part of the fleet (Masri et al. 2015; U.S. EPA 2012). Continuing retirement of older vehicles will likely continue this trend. Wood burning, on the other hand has increased and now accounts for 19% of particles in Boston (Masri et al. 2015), and though the U.S. EPA has proposed new emission standards for future stoves and furnaces, there is no retrofit requirement. Heating oil, while similar to diesel oil, is still allowed much higher sulfur content. Hence, there are opportunities for local action to reduce this public health burden.

There are several limitations to our study. First, we have assumed we have a valid instrument. Although we have good evidence that

this is the case, one can never guarantee it. It is possible that behavior is modified on low-PBL or low-wind speed days in a way that affects mortality risk. A second limitation is that we have provided our proof that an instrumental variable protects against unmeasured confounding in the context of a log-linear model between mortality and air pollution, and assume that model is correct. This is the traditional approach for daily death counts, but we cannot be sure it is correct. In addition, all-cause mortality includes some causes of death unlikely to be associated with air pollution. This decreases power in our analysis, but still leaves us with a valid estimate of the impact on all deaths. The air pollutants, PBL, and wind speed were measured at only one location, which may introduce some error into the instrumental variable, which, if the instrument assumption is valid, should result in an underestimate of risk. Power is always an issue, and the power for a Poisson regression depends on the total number of events. In our case, there were 204,386 deaths during the study period, which indicates good power for our hypothesis tests.

In summary, we have used causal methods to estimate the acute effect of local air pollution on daily deaths, and found that concentrations below current limits are associated with important increases in daily deaths. If, when stratified by month and temperature, our instrument is independent of other causes of mortality, this association is causal, an interpretation supported by toxicological studies.

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# Association of Short-term Exposure to Air Pollution With Mortality in Older Adults

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**IMPORTANCE** The US Environmental Protection Agency is required to reexamine its National Ambient Air Quality Standards (NAAQS) every 5 years, but evidence of mortality risk is lacking at air pollution levels below the current daily NAAQS in unmonitored areas and for sensitive subgroups.

**OBJECTIVE** To estimate the association between short-term exposures to ambient fine particulate matter (PM<sub>2.5</sub>) and ozone, and at levels below the current daily NAAQS, and mortality in the continental United States.

**DESIGN, SETTING, AND PARTICIPANTS** Case-crossover design and conditional logistic regression to estimate the association between short-term exposures to PM<sub>2.5</sub> and ozone (mean of daily exposure on the same day of death and 1 day prior) and mortality in 2-pollutant models. The study included the entire Medicare population from January 1, 2000, to December 31, 2012, residing in 39 182 zip codes.

**EXPOSURES** Daily PM<sub>2.5</sub> and ozone levels in a 1-km × 1-km grid were estimated using published and validated air pollution prediction models based on land use, chemical transport modeling, and satellite remote sensing data. From these gridded exposures, daily exposures were calculated for every zip code in the United States. Warm-season ozone was defined as ozone levels for the months April to September of each year.

**MAIN OUTCOMES AND MEASURES** All-cause mortality in the entire Medicare population from 2000 to 2012.

**RESULTS** During the study period, there were 22 433 862 million case days and 76 143 209 control days. Of all case and control days, 93.6% had PM<sub>2.5</sub> levels below 25 µg/m<sup>3</sup>, during which 95.2% of deaths occurred (21 353 817 of 22 433 862), and 91.1% of days had ozone levels below 60 parts per billion, during which 93.4% of deaths occurred (20 955 387 of 22 433 862). The baseline daily mortality rates were 137.33 and 129.44 (per 1 million persons at risk per day) for the entire year and for the warm season, respectively. Each short-term increase of 10 µg/m<sup>3</sup> in PM<sub>2.5</sub> (adjusted by ozone) and 10 parts per billion (10<sup>-9</sup>) in warm-season ozone (adjusted by PM<sub>2.5</sub>) were statistically significantly associated with a relative increase of 1.05% (95% CI, 0.95%-1.15%) and 0.51% (95% CI, 0.41%-0.61%) in daily mortality rate, respectively. Absolute risk differences in daily mortality rate were 1.42 (95% CI, 1.29-1.56) and 0.66 (95% CI, 0.53-0.78) per 1 million persons at risk per day. There was no evidence of a threshold in the exposure-response relationship.

**CONCLUSIONS AND RELEVANCE** In the US Medicare population from 2000 to 2012, short-term exposures to PM<sub>2.5</sub> and warm-season ozone were significantly associated with increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated.

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In the United States, the Clean Air Act<sup>1</sup> requires a review of National Ambient Air Quality Standards (NAAQS) for fine particulate matter (PM<sub>2.5</sub>) and ozone every 5 years.<sup>2</sup> In 2012, the annual and 24-hour NAAQS for PM<sub>2.5</sub> were set to 12 µg/m<sup>3</sup> and 35 µg/m<sup>3</sup>, respectively. With no annual standard for ozone, the 8-hour NAAQS for ozone was set to 70 parts per billion (ppb). Currently, the review of these standards is ongoing, with public comments expected in the fall of 2017.<sup>3</sup>

Several studies have provided evidence that short-term exposures to PM<sub>2.5</sub> and ozone were associated with mortality,<sup>4-8</sup> but these studies primarily included large and well-monitored metropolitan areas. While the US Environmental Protection Agency (EPA) is considering more stringent NAAQS, evidence is needed to clarify the association between mortality risk and exposure levels below the daily NAAQS and in rural and unmonitored areas.

The Clean Air Act<sup>1</sup> also requires the US EPA to set standards to protect “sensitive subgroups.” To estimate the health risk of short-term exposure to air pollution for specific subgroups (eg, underrepresented minorities and those with low socioeconomic status, such as persons eligible for Medicaid), a large population is necessary to achieve maximum accuracy and adequate statistical power.

A case-crossover study was conducted to examine all deaths of Medicare participants in the continental United States from 2000 throughout 2012 and estimate the mortality risk associated with short-term exposures to PM<sub>2.5</sub> and ozone in the general population as well as in subgroups. The study was designed to estimate the association between daily mortality and air pollution at levels below current daily NAAQS to evaluate the adequacy of the current air quality standards for PM<sub>2.5</sub> and ozone.

## Methods

This study was approved by the institutional review board at the Harvard T.H. Chan School of Public Health. As a study of previously collected administrative data, it was exempt from informed consent requirements.

### Study Population

Using claims data from the Centers for Medicare & Medicaid Services, all deaths among all Medicare beneficiaries were identified during the period 2000 to 2012, providing enough power to analyze the risk of mortality associated with PM<sub>2.5</sub> and ozone concentrations much lower than the current standards (Table 1). For each beneficiary, information was extracted on the date of death, age, sex, race, ethnicity, zip code of residence, and eligibility for Medicaid (a proxy for low income) to assess the associations of mortality with PM<sub>2.5</sub> and ozone concentrations in potentially vulnerable subgroups. Self-reported information on race and ethnicity was obtained from Medicare beneficiary files.

### Outcome

The study outcome was all-cause mortality. Individuals with a verified date of death between January 1, 2000, and

## Key Points

**Question** What is the association between short-term exposure to air pollution below current air quality standards and all-cause mortality?

**Finding** In a case-crossover study of more than 22 million deaths, each 10-µg/m<sup>3</sup> daily increase in fine particulate matter and 10-parts-per-billion daily increase in warm-season ozone exposures were associated with a statistically significant increase of 1.42 and 0.66 deaths per 1 million persons at risk per day, respectively.

**Meaning** Day-to-day changes in fine particulate matter and ozone exposures were significantly associated with higher risk of all-cause mortality at levels below current air quality standards, suggesting that those standards may need to be reevaluated.

December 31, 2012, were included. Individuals with an unverified date of death, or still living after December 31, 2012, were excluded.

### Study Design

We estimated the association between short-term exposure to PM<sub>2.5</sub> (adjusted by ozone) and short-term exposure to ozone (adjusted by PM<sub>2.5</sub>) and all-cause mortality using a case-crossover design.<sup>9</sup> Specifically, “case day” was defined as the date of death. For the same person, we compared daily air pollution exposure on the case day vs daily air pollution exposure on “control days.” Control days were chosen (1) on the same day of the week as the case day to control for potential confounding effect by day of week; (2) before and after the case day (bidirectional sampling) to control for time trend<sup>10,11</sup>; and (3) only in the same month as the case day to control for seasonal and subseasonal patterns.<sup>10,12</sup> Individual-level covariates and zip code-level covariates that did not vary day to day (eg, age, sex, race/ethnicity, socioeconomic status, smoking, and other behavioral risk factors) were not considered to be confounders as they remain constant when comparing case days vs control days.

### Environmental Data

Daily ambient levels of PM<sub>2.5</sub> and ozone were estimated from published and validated air pollution prediction models.<sup>13,14</sup> Combining monitoring data from the EPA, satellite-based measurements, and other data sets, neural networks were used to predict 24-hour PM<sub>2.5</sub> and 8-hour maximum ozone concentrations at each 1-km × 1-km grid in the continental United States, including locations with no monitoring sites. Cross-validation indicated good agreement between predicted values and monitoring values ( $R^2 = 0.84$  for PM<sub>2.5</sub> and  $R^2 = 0.76$  for ozone) and at low concentrations ( $R^2 = 0.85$  when constraining to 24-hour PM<sub>2.5</sub> <25 µg/m<sup>3</sup> and  $R^2 = 0.75$  when constraining to daily 8-hour maximum ozone <60 ppb). Details have been published elsewhere.<sup>13,14</sup> Warm season was defined to be from April 1 to September 30, which is the specific time window to examine the association between ozone and mortality.

Meteorological variables, including air and dew point temperatures, were retrieved from North American Regional Reanalysis data and estimated daily mean values were determined for each 32-km × 32-km grid in the continental United States.<sup>15</sup>

For each case day (date of death) and its control days, the daily 24-hour PM<sub>2.5</sub>, 8-hour maximum ozone, and daily air and dew point temperatures were assigned based on zip code of residence of the individual (eAppendix 1 in the [Supplement](#)). Because we estimated air pollution levels everywhere in the

continental United States, the number of zip codes included in this study was 39 182, resulting in a 33% increase compared with the number of zip codes with a centroid less than 50 km from a monitor (n = 26 115).

### Statistical Analysis

The relative risk (RR) of all-cause mortality associated with short-term exposures to PM<sub>2.5</sub> (adjusted by ozone) and warm-season ozone (adjusted by PM<sub>2.5</sub>) was estimated by fitting a conditional logistic regression to all pairs of case days and matched control days (eAppendix 2 in the [Supplement](#)).<sup>9</sup> The regression model included both pollutants as main effects and natural splines of air and dew point temperatures with 3 *df* to control for potential residual confounding by weather. For each case day, daily exposure to air pollution was defined as the mean of the same day of death (lag 0-day) and 1 day prior (lag 1-day), denoted as lag 01-day.<sup>5,16,17</sup> Relative risk increase (RRI) was defined as  $RR - 1$ . The absolute risk difference (ARD) of all-cause mortality associated with air pollution was defined as  $ARD = \alpha \times (RR - 1)/RR$ , where  $\alpha$  denotes the baseline daily mortality rate (eAppendix 3 in the [Supplement](#)).

The robustness of the analysis results was assessed with respect to (1) choosing the *df* used for the confounding adjustment for temperature, (2) using lag 01-day exposure as the exposure metric, (3) the definition of warm season, and (4) using only air pollution measurements from the nearest EPA monitoring sites. Splines on meteorological variables with 6 and 9 *df* yielded results with a difference of less than 5% of the standard error (eFigure 1 in the [Supplement](#)). The main analysis, which used the lag 01-day exposure, yielded the lowest values of the Akaike Information Criteria values, indicating better fit to the data (eTable in the [Supplement](#)). Different definitions of warm season yielded similar risk estimates (eAppendix 4 in the [Supplement](#)), and using exposure mea-

Table 1. Baseline Characteristics of Study Population (2000-2012)

Baseline Characteristic	Value
Case days, No.	22 433 862
Control days, No.	76 143 209
Among All Cases (n = 22 433 862), %	
Age at death, y	
≤69	10.38
70-74	13.37
75-84	38.48
≥85	37.78
Sex	
Male	44.73
Female	55.27
Race/ethnicity	
White	87.34
Black	8.87
Asian	1.03
Hispanic	1.51
Native American	0.31
Medicaid Eligibility (n = 22 433 862), %	
Ineligible	77.36
Eligible	22.64

Table 2. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With Each 10-μg/m<sup>3</sup> Increase in PM<sub>2.5</sub> and Each 10-ppb Increase in Ozone

Air Pollutant Analysis	Relative Risk Increase, % (95% CI)		Absolute Risk Difference in Daily Mortality Rates, No. per 1 Million Persons at Risk per Day (95% CI) <sup>a</sup>	
	PM <sub>2.5</sub>	Ozone <sup>b</sup>	PM <sub>2.5</sub>	Ozone <sup>b</sup>
Main analysis <sup>c</sup>	1.05 (0.95-1.15)	0.51 (0.41-0.61)	1.42 (1.29-1.56)	0.66 (0.53-0.78)
Low-exposure analysis <sup>d</sup>	1.61 (1.48-1.74)	0.58 (0.46-0.70)	2.17 (2.00-2.34)	0.74 (0.59-0.90)
Single-pollutant analysis <sup>e</sup>	1.18 (1.09-1.28)	0.55 (0.48-0.62)	1.61 (1.48-1.73)	0.71 (0.62-0.79)
Nearest monitors analysis <sup>f</sup>	0.83 (0.73-0.93)	0.35 (0.28-0.41)	1.13 (0.99-1.26)	0.45 (0.37-0.53)

Abbreviations: PM<sub>2.5</sub>, fine particulate matter; ppb, parts per billion.

<sup>a</sup> The daily baseline mortality rate was 137.33 per 1 million persons at risk per day; the warm-season daily baseline mortality rate was 129.44 per 1 million persons at risk per day.

<sup>b</sup> Ozone analyses included days from the warm season only (April 1 to September 30).

<sup>c</sup> The main analysis used the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for both PM<sub>2.5</sub> and ozone, and controlled for natural splines of air and dew point temperatures with 3 *df*. The main analysis considered the 2 pollutants jointly included into the regression model and estimated the percentage increase in the daily mortality rate associated with a 10-μg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure adjusted for ozone and the percentage increase in daily mortality rate associated with a 10-ppb increase in warm-season ozone exposure adjusted for PM<sub>2.5</sub>.

<sup>d</sup> The low-exposure analysis had the same model specifications as the 2-pollutant analysis and was constrained for days when PM<sub>2.5</sub> was below 25 μg/m<sup>3</sup> or ozone below 60 ppb.

<sup>e</sup> The single-pollutant analysis estimated the percentage increase in the daily mortality rate associated with a 10-μg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure without adjusting for ozone and the percentage increase in the daily mortality rate associated with a 10-ppb increase in ozone exposure without adjusting for PM<sub>2.5</sub>.

<sup>f</sup> PM<sub>2.5</sub> and ozone monitoring data were retrieved from the US Environmental Protection Agency Air Quality System, which provides the daily mean of PM<sub>2.5</sub> and daily 8-hour maximum ozone levels at each monitoring site. Daily ozone concentrations were averaged from April 1 to September 30. Individuals were assigned to the PM<sub>2.5</sub> and ozone levels from the nearest monitor site within 50 km. Those living 50 km from any monitoring site were excluded.

surements from the nearest monitors resulted in attenuated, but still significant, risk estimates (Table 2).

The subgroup analyses were conducted by sex (male and female), race/ethnicity (white, nonwhite, and others), age ( $\leq 69$ , 70-74, 75-84, and  $\geq 85$  years), eligibility for Medicaid, and population density (quartiles). We fitted separate conditional logistic regressions to the data for each subgroup and obtained subgroup-specific estimates of RR and ARD. We implemented a 2-sample test for assessing statistically significant differences in the estimated RR and ARD between categories within each subgroup (eg, female vs male), based on the point estimate and standard error (se) (Appendix 5 in the Supplement):

$$Z = \frac{RR_{\text{male}} - RR_{\text{female}}}{\sqrt{se(RR_{\text{male}})^2 + se(RR_{\text{female}})^2}}$$

The goal was to estimate mortality rate increases (both RRI and ARD) at air pollution levels well below the current daily NAAQS. The analysis was restricted to days with daily air pollution concentrations below 25  $\mu\text{g}/\text{m}^3$  for  $\text{PM}_{2.5}$  and 60 ppb for ozone. We chose 25  $\mu\text{g}/\text{m}^3$  and 60 ppb instead of the current daily NAAQS (35  $\mu\text{g}/\text{m}^3$  for daily  $\text{PM}_{2.5}$  and 70 ppb for 8-hour maximum ozone) because levels of  $\text{PM}_{2.5}$  and ozone on most of the days included in the analysis were already below the current safety standards.

Exposure-response curves were estimated between  $\text{PM}_{2.5}$  or ozone and mortality by replacing linear terms for the 2 pollutants with penalized splines for both  $\text{PM}_{2.5}$  and ozone.

All analyses were performed in R software version 3.3.2 (R Foundation). Computations were run on (1) the Odyssey cluster supported by the Faculty of Arts and Sciences Division of Science, Research Computing Group at Harvard University and (2) the Research Computing Environment supported by the Institute for Quantitative Social Science in the Faculty of Arts and Sciences at Harvard University.

## Results

During the study period, there were more than 22 million case days (deaths) and more than 76 million control days (Table 1). Of all case and control days, 93.6% had  $\text{PM}_{2.5}$  levels below 25  $\mu\text{g}/\text{m}^3$ , during which 95.2% of deaths occurred (21 353 817 of 22 433 862), and 91.1% of days had ozone levels below 60 ppb, during which 93.4% of deaths occurred (20 955 387 of 22 433 862). The baseline daily mortality rates were 137.33 and 129.44 (per 1 million persons at risk per day [per 1M per day]) for the entire year and for the warm season, respectively. The mean time between case and control days was 12.55 days (range 7-28 days), with minimal differences in air and dew point temperatures between case and control days (0.003°C and 0.01°C, respectively). During the study period, the mean concentrations of  $\text{PM}_{2.5}$  and ozone were 11.6  $\mu\text{g}/\text{m}^3$  and 37.8 ppb, respectively. Figure 1 and Figure 2 show the daily  $\text{PM}_{2.5}$  and ozone time series by state, respectively.

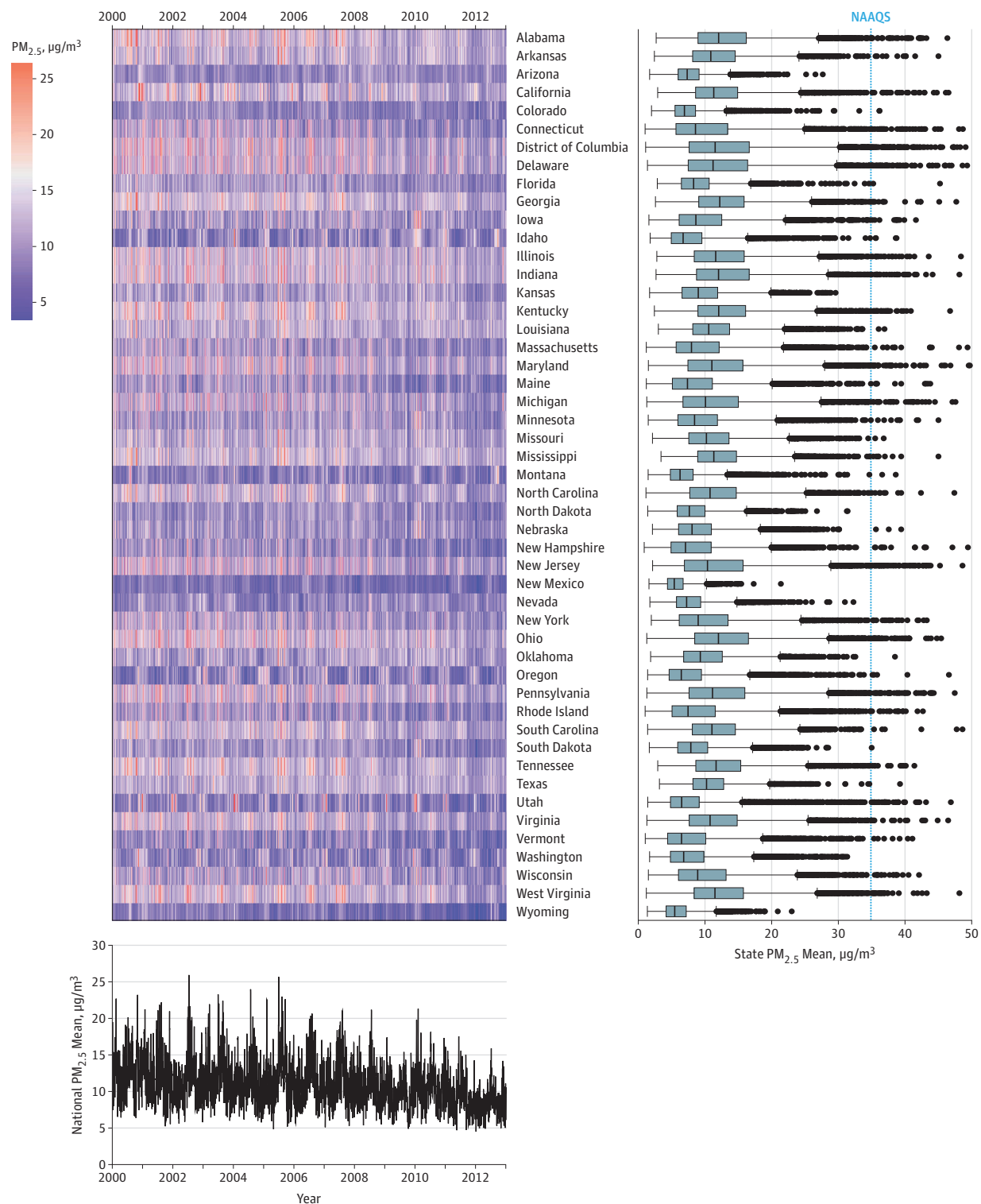
Each 10- $\mu\text{g}/\text{m}^3$  and 10-ppb increase in the lag 01-day exposure for  $\text{PM}_{2.5}$  and warm-season ozone was associated with

an RRI of 1.05% (95% CI, 0.95%-1.15%) and 0.51% (95% CI, 0.41%-0.61%) in the daily mortality rate. The ARDs were 1.42 (95% CI, 1.29-1.56) and 0.66 (95% CI, 0.53-0.78) per 1M per day. These associations remained significant when examining days below 25  $\mu\text{g}/\text{m}^3$  for  $\text{PM}_{2.5}$  and below 60 ppb for ozone, with larger effect size estimates for both  $\text{PM}_{2.5}$  and ozone (RRI: 1.61% [95% CI, 1.48%-1.74%] and 0.58% [95% CI, 0.46%-0.70%]; ARD: 2.17 [95% CI, 2.00-2.34] and 0.74 [95% CI, 0.59-0.90] per 1M per day, respectively) (Table 2).  $\text{PM}_{2.5}$  was associated with higher mortality rate in some subgroups, including Medicaid-eligible individuals (RRI: 1.49% [95% CI, 1.29%-1.70%]; ARD: 3.59 [95% CI, 3.11-4.08] per 1M per day; interaction:  $P < .001$ ), individuals older than 70 years (eg, for  $\geq 85$  years, RRI: 1.38% [95% CI, 1.23%-1.54%]; ARD: 5.35 [95% CI, 4.75-5.95] per 1M per day; interaction:  $P < .001$ ), and females (RRI: 1.20% [95% CI, 1.07%-1.33%]; ARD: 1.56 [95% CI, 1.39-1.72] per 1M per day; interaction:  $P = .02$ ) (Figure 3 and Figure 4). The effect estimates for  $\text{PM}_{2.5}$  increased with age. The effect estimate for black individuals was higher than that for white individuals ( $P = .001$ ; eFigure 2 in the Supplement). For ozone, similar patterns were observed, but with less contrast between groups. No significant differences were found in the short-term associations between air pollution exposure ( $\text{PM}_{2.5}$  and ozone) and mortality across areas with different population density levels (Figure 3 and Figure 4). Effect estimates using different lags of exposure are shown in eFigure 3 in the Supplement.

Figure 5 shows the estimated exposure-response curves for  $\text{PM}_{2.5}$  and ozone. The slope was steeper at  $\text{PM}_{2.5}$  levels below 25  $\mu\text{g}/\text{m}^3$  ( $P < .001$ ), consistent with the low-exposure analysis (Table 2). Both  $\text{PM}_{2.5}$  and ozone exposure-responses were almost linear, with no indication of a mortality risk threshold at very low concentrations. eFigure 4 in the Supplement shows the exposure-response curves for  $\text{PM}_{2.5}$  when restricted to just the warm season and for ozone when not restricted to the warm season; results were similar.

## Discussion

In this large case-crossover study of all Medicare deaths in the continental United States from 2000 to 2012, a 10- $\mu\text{g}/\text{m}^3$  daily increase in  $\text{PM}_{2.5}$  and a 10-ppb daily increase in warm-season ozone exposures were associated with a statistically significant increase of 1.42 and 0.66 deaths per 1M per day, respectively. The risk of mortality remained statistically significant when restricting the analysis to days with  $\text{PM}_{2.5}$  and ozone levels much lower than the current daily NAAQS.<sup>18</sup> This study included individuals living in smaller cities, towns, and rural areas that were unmonitored and thus excluded from previous time series studies. There were no significant differences in the mortality risk associated with air pollution among individuals living in urban vs rural areas. Taken together, these results provide evidence that short-term exposures to  $\text{PM}_{2.5}$  and ozone, even at levels much lower than the current daily standards, are associated with increased mortality, particularly for susceptible populations.

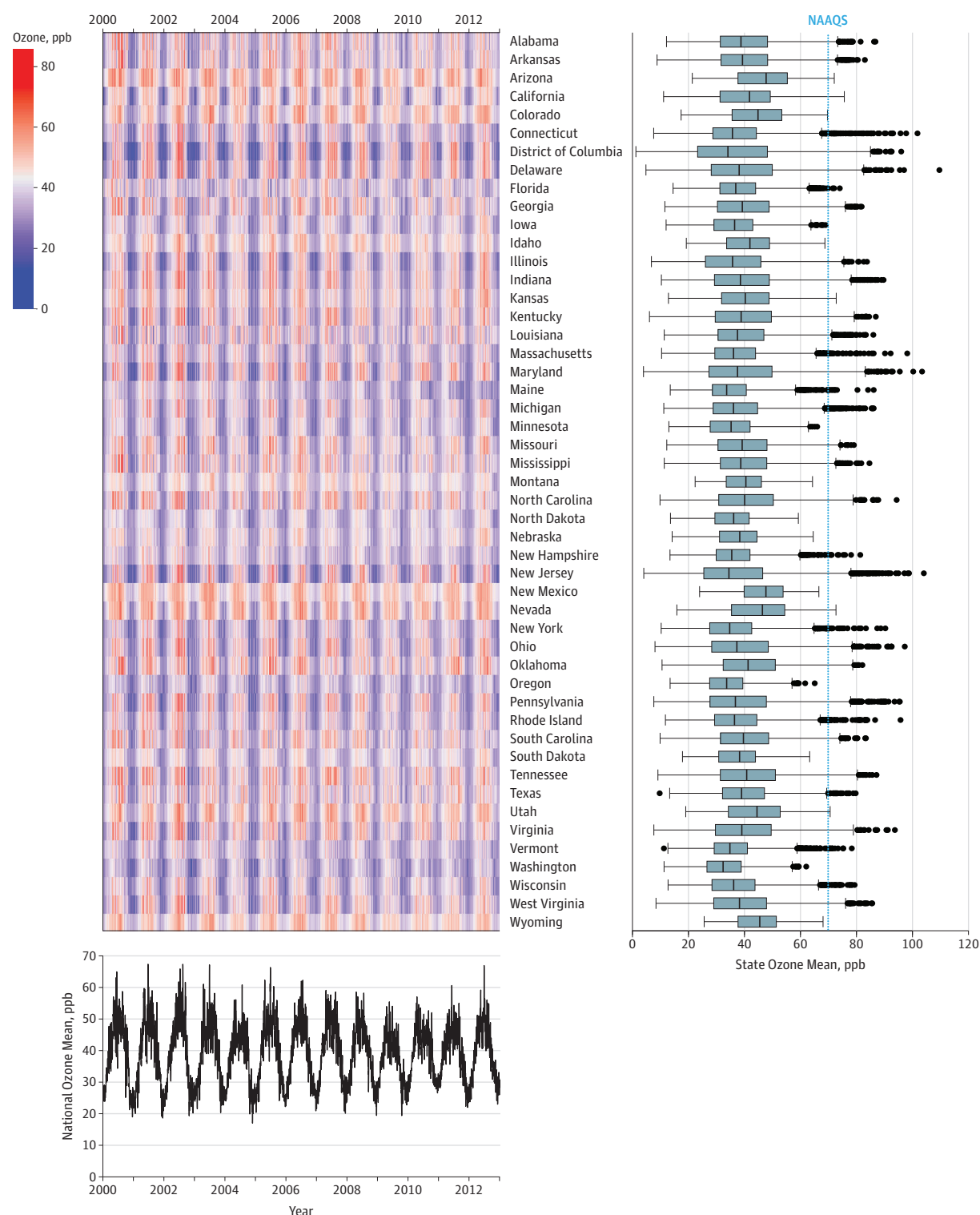
Figure 1. Daily Mean  $PM_{2.5}$  Concentrations in the Continental United States, 2000-2012

Daily mean fine particulate matter ( $PM_{2.5}$ ) concentrations were calculated and plotted by state. The time-series plot at the bottom indicates the national daily mean values across all locations. Boxplots show the distribution of daily  $PM_{2.5}$  levels for each state. The blue dashed line indicates the daily National Ambient Air Quality Standards (NAAQS) for  $PM_{2.5}$  (35  $\mu g/m^3$ ). The line across the box,

upper hinge, and lower hinge represent the median value, 75th percentile (Q3), and 25th percentile (Q1), respectively. The upper whisker is located at the smaller of the maximal value and  $Q3 + 1.5 \times$  interquartile range; the lower whisker is located at the larger of the minimal value and  $Q1 - 1.5 \times$  interquartile range. Any values that lie beyond the upper and lower whiskers are outliers.

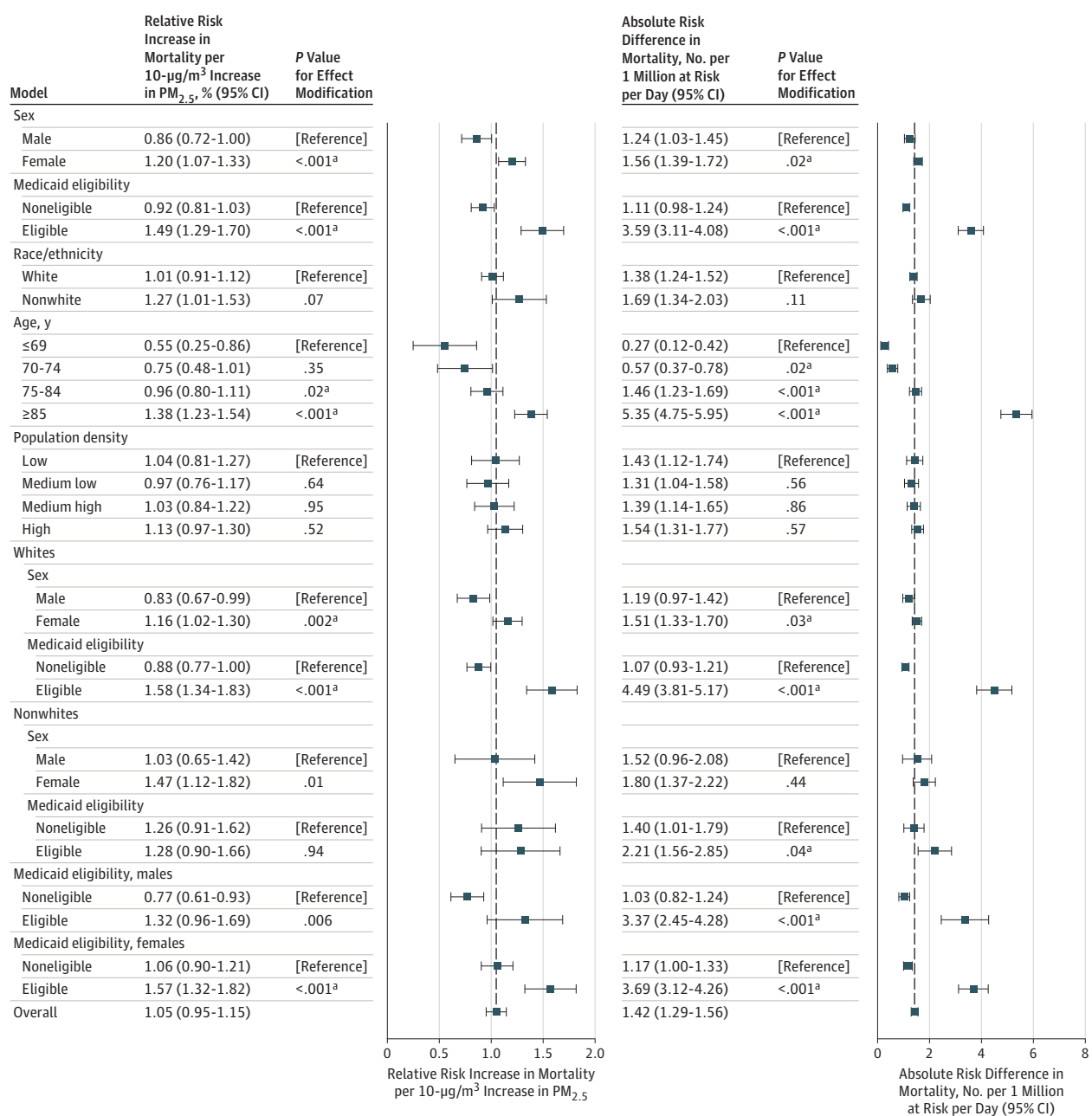


Figure 2. Daily 8-Hour Maximum Ozone Concentrations in the Continental United States, 2000-2012



Daily mean 8-hour maximum ozone concentrations were calculated and plotted by state. The time-series plot at the bottom indicates the national daily mean values across all locations. Boxplots show the distribution of daily ozone levels for each state. The blue dashed line indicates the daily National Ambient Air Quality Standards (NAAQS) for ozone (70 parts per billion [ppb]). The line across the box, upper hinge, and lower hinge represent the median value,

75th percentile (Q3), and 25th percentile (Q1), respectively. The upper whisker is located at the smaller of the maximal value and  $Q3 + 1.5 \times$  interquartile range; the lower whisker is located at the larger of the minimal value and  $Q1 - 1.5 \times$  interquartile range. Any values that lie beyond the upper and lower whiskers are outliers.

Figure 3. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With 10- $\mu\text{g}/\text{m}^3$  Increase in Fine Particulate Matter ( $\text{PM}_{2.5}$ )

For the main analysis, subgroup analyses used a 2-pollutant analysis (with both  $\text{PM}_{2.5}$  and ozone), based on the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for  $\text{PM}_{2.5}$ , and controlled for natural splines of air and dew point temperatures (each with 3 df). Vertical lines indicate effects for the entire study population. Subgroup analyses were conducted for each subgroup (eg, male or female, white or nonwhite, Medicare eligible or Medicare ineligible, age groups, and quartiles of population density). For the main analysis and each subgroup, conditional logistic

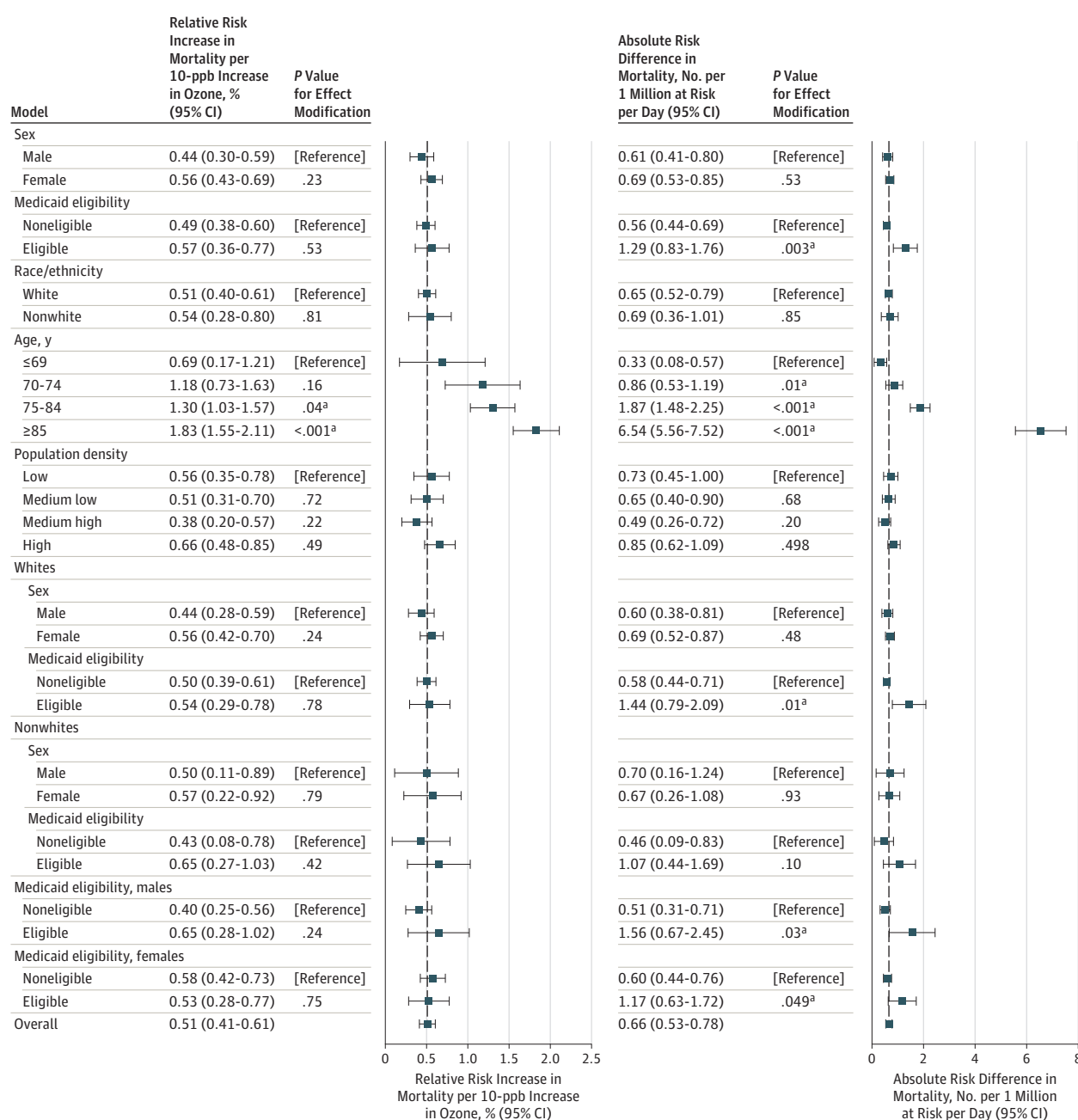
regressions were run to obtain relative risk increases and calculated absolute risk difference based on baseline mortality rates (eAppendix 2 in the Supplement). Numbers in the figure represent point estimates, 95% CIs, and P values for effect modifications. The reference groups were used when assessing effect modification.

<sup>a</sup> Statistically significant effect estimate (at 5% level) compared with the reference group.

The Clean Air Act<sup>1</sup> requires the administrator of the US EPA to set NAAQS at levels that provide “protection for at-risk populations, with an adequate margin of safety.”<sup>19</sup> In this study, Medicaid-eligible individuals, females, and elderly individuals had higher mortality rate increases associated with  $\text{PM}_{2.5}$

than other groups. Previous studies have found similar results in some subgroups.<sup>20,21</sup> Poverty, unhealthy lifestyle, poor access to health care, and other factors may make some subgroups more vulnerable to air pollution. The exact mechanism is worth exploring in future studies.

Figure 4. Relative Risk Increase and Absolute Risk Difference of Daily Mortality Associated With 10-Parts-per-Billion (ppb) Increase in Ozone



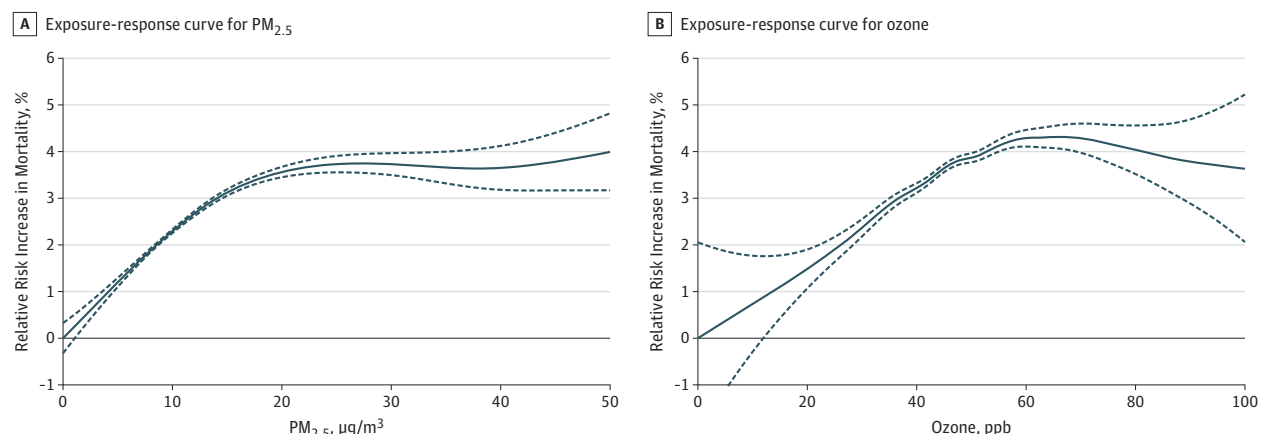
For the main analysis, subgroup analyses used a 2-pollutant analysis (with both  $PM_{2.5}$  and ozone), based on the mean of daily exposure on the same day of death and 1 day prior (lag 01-day) as the exposure metric for ozone, and controlled for natural splines of air and dew point temperatures (each with 3 *df*). Vertical lines indicate effects for the entire study population. Subgroup analyses were conducted for each subgroup (eg, male or female, white or nonwhite, Medicare eligible or Medicare ineligible, age groups, and quartiles of population density). For the main analysis and each subgroup, conditional logistic regressions were run to obtain relative risk increases, and calculated absolute

risk difference based on baseline mortality rates (eAppendix 2 in the Supplement). For ozone, analyses were restricted to the warm season (April to September). Numbers in the figure represent point estimates, 95% CIs, and P values for effect modifications. The reference groups were used when assessing effect modification.

<sup>a</sup> Statistically significant effect estimate (at 5% level) compared with the reference group.

The current NAAQS for daily  $PM_{2.5}$  is 35  $\mu g/m^3$ . When restricting the analysis to daily  $PM_{2.5}$  levels below 25  $\mu g/m^3$ , the association between short-term  $PM_{2.5}$  exposure and mortality remained but was elevated. The current daily

NAAQS for ozone is 70 ppb; when restricting the analysis to daily warm-season ozone concentrations below 60 ppb, the effect size also increased slightly. The exposure-response curves revealed a similar pattern. These results indicate

Figure 5. Estimated Exposure-Response Curves for Short-term Exposures to Fine Particulate Matter (PM<sub>2.5</sub>) and Ozone

A 2-pollutant analysis with separate penalized splines on PM<sub>2.5</sub> (A) and ozone (B) was conducted to assess the percentage increase in daily mortality at various pollution levels. Dashed lines indicate 95% CIs. The mean of daily

exposure on the same day of death and 1 day prior (lag 01-day) was used as metrics of exposure to PM<sub>2.5</sub> and ozone. Analysis for ozone was restricted to the warm season (April to September). Ppb indicates parts per billion.

that air pollution is associated with an increase in daily mortality rates, even at levels well below the current standards.

The exposure-response relationship between PM<sub>2.5</sub> exposure and mortality was consistent with findings of previous studies. One study combined exposure-response curves from 22 European cities and reported an almost linear relationship between PM<sub>2.5</sub> and mortality.<sup>22</sup> Another multicity study reported a linear relationship down to 2-µg/m<sup>3</sup> PM<sub>2.5</sub>.<sup>23</sup> The present study found a similarly linear exposure-response relationship below 15-µg/m<sup>3</sup> PM<sub>2.5</sub> and a less steep slope above this level.

For ozone, the linear exposure-response curve with no threshold described in this study is consistent with earlier research. An almost linear exposure-response curve for ozone was previously reported with no threshold or a threshold at very low concentrations.<sup>24</sup> A study from the Netherlands also concluded that if an ozone threshold exists, it does so at very low levels.<sup>25</sup>

Findings from this study are also consistent with the literature regarding the observed effect sizes of both PM<sub>2.5</sub><sup>5,8,16,26-28</sup> and ozone.<sup>7,20,29,30</sup> This study further demonstrates that in more recent years, during which air pollution concentrations have fallen, statistically significant associations between mortality and exposures to PM<sub>2.5</sub> and ozone persisted.

The association of mortality and PM<sub>2.5</sub> exposure is supported by a large number of published experimental studies in animals<sup>31-33</sup> and in humans exposed to traffic air pollution,<sup>34,35</sup> diesel particles,<sup>36</sup> and unfiltered urban air.<sup>37</sup> Similarly, a review of toxicological studies and a recent panel study found that ozone exposure was associated with multiple adverse health outcomes.<sup>38,39</sup>

### Strengths

This study has several strengths. First, to our knowledge, this is the largest analysis of daily air pollution exposure

and mortality to date, with approximately 4 times the number of deaths included in a previous large study.<sup>5</sup> Second, this study assessed daily exposures using air pollution prediction models that provide accurate estimates of daily levels of PM<sub>2.5</sub> and ozone for most of the United States, including previously unmonitored areas. An analysis that relied only on exposure data from monitoring stations was found to result in a downward bias in estimates (Table 2). Third, the inclusion of more than 22 million deaths from 2000 to 2012 from the entire Medicare population provided large statistical power to detect differences in mortality rates in potentially vulnerable populations and to estimate mortality rates at very low PM<sub>2.5</sub> and ozone concentrations. Fourth, this study estimated the air pollution-mortality association well below the current daily NAAQS and in unmonitored areas, and it did not identify significant differences in the mortality rate increase between urban and rural areas. Fifth, this study used a case-crossover design that individually matched potential confounding factors by month, year, and other time-invariant variables and controlled for time-varying patterns, as demonstrated by the minimal differences in meteorological variables between case and control days.

### Limitations

This study also has several limitations. First, the case-crossover design does not allow estimation of mortality rate increase associated with long-term exposure to air pollution. Long-term risks in the same study population have been estimated elsewhere.<sup>40</sup> Second, because this study used residential zip code to ascertain exposure level rather than exact home address or place of death, some measurement error is expected. Third, the Medicare population primarily consists of individuals older than 65 years, which limits the generalizability of findings to younger populations. However, because more than two-thirds of deaths in



the United States occur in people older than 65 years of age, and air pollution-related health risk rises with age, the Medicare population in this study includes most cases of air pollution-induced mortality. Fourth, Medicare files do not report cause-specific mortality. Fifth, the most recent data used in this study are nearly 5 years old, and it is uncertain whether exposures and outcomes would be the same with more current data.

## Conclusions

In the US Medicare population from 2000 to 2012, short-term exposures to PM<sub>2.5</sub> and warm-season ozone were significantly associated with increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated.

### ARTICLE INFORMATION

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**Author Contributions:** Mr Di had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Mr Di and Dr Dai contributed equally to this study.

**Concept and design:** Di, Dai, Zanobetti, Schwartz, Dominici.

**Acquisition, analysis, or interpretation of data:** All authors.

**Drafting of the manuscript:** Di, Dai, Choirat, Dominici.

**Critical revision of the manuscript for important intellectual content:** All authors.

**Statistical analysis:** Di, Dai, Choirat, Schwartz, Dominici.

**Obtained funding:** Zanobetti, Schwartz, Dominici.

**Administrative, technical, or material support:** Wang, Choirat.

**Supervision:** Zanobetti, Schwartz, Dominici.

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# Ambient Particulate Matter Air Pollution Exposure and Mortality in the NIH-AARP Diet and Health Cohort

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**BACKGROUND:** Outdoor fine particulate matter ( $\leq 2.5 \mu\text{m}$ ;  $\text{PM}_{2.5}$ ) has been identified as a global health threat, but the number of large U.S. prospective cohort studies with individual participant data remains limited, especially at lower recent exposures.

**OBJECTIVES:** We aimed to test the relationship between long-term exposure  $\text{PM}_{2.5}$  and death risk from all nonaccidental causes, cardiovascular (CVD), and respiratory diseases in 517,041 men and women enrolled in the National Institutes of Health-AARP cohort.

**METHODS:** Individual participant data were linked with residence  $\text{PM}_{2.5}$  exposure estimates across the continental United States for a 2000–2009 follow-up period when matching census tract-level  $\text{PM}_{2.5}$  exposure data were available. Participants enrolled ranged from 50 to 71 years of age, residing in six U.S. states and two cities. Cox proportional hazard models yielded hazard ratio (HR) estimates per  $10 \mu\text{g}/\text{m}^3$  of  $\text{PM}_{2.5}$  exposure.

**RESULTS:**  $\text{PM}_{2.5}$  exposure was significantly associated with total mortality (HR = 1.03; 95% CI: 1.00, 1.05) and CVD mortality (HR = 1.10; 95% CI: 1.05, 1.15), but the association with respiratory mortality was not statistically significant (HR = 1.05; 95% CI: 0.98, 1.13). A significant association was found with respiratory mortality only among never smokers (HR = 1.27; 95% CI: 1.03, 1.56). Associations with  $10\text{-}\mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$  exposures in yearly participant residential annual mean, or in metropolitan area-wide mean, were consistent with baseline exposure model results. Associations with  $\text{PM}_{2.5}$  were similar when adjusted for ozone exposures. Analyses of California residents alone also yielded statistically significant  $\text{PM}_{2.5}$  mortality HRs for total and CVD mortality.

**CONCLUSIONS:** Long-term exposure to  $\text{PM}_{2.5}$  air pollution was associated with an increased risk of total and CVD mortality, providing an independent test of the  $\text{PM}_{2.5}$ –mortality relationship in a new large U.S. prospective cohort experiencing lower post-2000  $\text{PM}_{2.5}$  exposure levels.

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## Introduction

Over the past several decades, numerous published epidemiologic studies have documented a consistent association between long-term exposure to fine particulate matter mass ( $\leq 2.5 \mu\text{m}$ ;  $\text{PM}_{2.5}$ ) air pollution and an increase in the risk of mortality around the globe (e.g., Beelen et al. 2014; Brook et al. 2010; Crouse et al. 2012; Dockery et al. 1993; Eftim et al. 2008; Ostro et al. 2010; Ozkaynak and Thurston 1987; Pope et al. 1995, 2002, 2004). Pope and collaborators notably found elevated relative risks of cardiovascular (CVD) mortality in association with long-term  $\text{PM}_{2.5}$  exposure [hazard ratio (HR) per  $10 \mu\text{g}/\text{m}^3 = 1.12$ ; 95% confidence interval (CI): 1.08, 1.15] in the largest and most definitive U.S. nationwide cohort considered to date (Pope et al. 2002, 2004), providing a cardiovascular mortality HR of 1.12 per  $10 \mu\text{g}/\text{m}^3$  (95% CI: 1.08, 1.15). However, existing U.S. cohort studies of  $\text{PM}_{2.5}$  health effects are still being questioned (e.g., Reis 2013). In addition, particulate matter air

pollution levels have been declining in recent years in the United States, so there is a need to confirm whether studies conducted in the past at higher levels are replicable today. Thus, it is important to test these associations in another large U.S. cohort with detailed individual-level risk factor information on participants, especially one for which pollution exposures can be estimated at the individual participant residence level, and in more recent lower  $\text{PM}_{2.5}$  exposure years, as we report here. This research addresses these needs using the newly available U.S. National Institutes of Health–AARP Diet & Health cohort (NIH-AARP Study) (Schatzkin et al. 2001).

## Methods

**Study population.** The NIH-AARP Study was initiated when members of the AARP, 50–71 years of age from six U.S. states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and two metropolitan areas (Atlanta, Georgia, and Detroit, Michigan), responded to a mailed

questionnaire in 1995 and 1996. Details of the NIH-AARP Study have been described previously (Schatzkin et al. 2001). Among 566,398 participants enrolled in the NIH-AARP cohort and available for analysis in 2014, we first excluded for this analysis those individuals for whom the forms were filled out by a proxy ( $n = 15,760$ , or 2.8%); who moved out of their study region before January 2000 ( $n = 13,863$ , or 2.4%); who died before 1 January 2000 ( $n = 21,415$ , or 3.8%); and those for whom census-level outdoor  $\text{PM}_{2.5}$  exposure was not estimable using the methods discussed below ( $n = 737$ , or 0.1%). After accounting for overlapping exclusions, the analytic cohort includes 517,041 (91.3%) participants for whom matching  $\text{PM}_{2.5}$  air pollution data were available. The NIH-AARP cohort questionnaires elicited information on demographic and anthropometric characteristics, dietary intake, and numerous health-related variables (e.g., marital status, body mass index, education, race, smoking status, physical activity, and alcohol consumption) at enrollment only. Contextual environment characteristics (e.g., median income) for the census tract of each of this cohort's participants have also been compiled for this population by the NIH-AARP Study (NIH-AARP 2006), allowing us to also incorporate contextual socioeconomic variables at the census-tract level. All participants provided informed consent before completing the study

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G.D.T. has provided expert witness testimony on the human health effects of air pollution before the U.S. Congress, in the U.S. EPA public hearings, and in legal cases. The other authors declare they have no actual or potential competing financial interests.

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questionnaire. The study was approved by the institutional review boards of the National Cancer Institute and New York University School of Medicine.

**Cohort follow-up and mortality ascertainment.** Vital status was ascertained through a periodic linkage of the cohort to the Social Security Administration Death Master File and follow-up searches of the National Death Index Plus for participants who matched to the Social Security Administration Death Master File (unpublished data, available on request from <https://www.ssa.gov/dataexchange/>), cancer registry linkage, questionnaire responses, and responses to other mailings. Participants were followed for address changes using the U.S. Postal Service's National Change of Address database, responses to other study-related mailings such as newsletters, and directly from cohort members' notifications (Michaud et al. 2005). We used the *International Classification of Diseases, 9th Revision* (ICD-9) and the *International Statistical Classification of Diseases, 10th Revision* to define death due to CVD (ICD-10: I00–I99), nonmalignant respiratory disease (ICD-10: J00–J99), and deaths from nonexternal and nonaccidental deaths (ICD-10 A00–R99). During the follow-up period considered here (2000 through 2009), 86,864 (16.8%) participants died, of whom 84,404 (97.2% of deaths) participants died of nonexternal and nonaccidental causes.

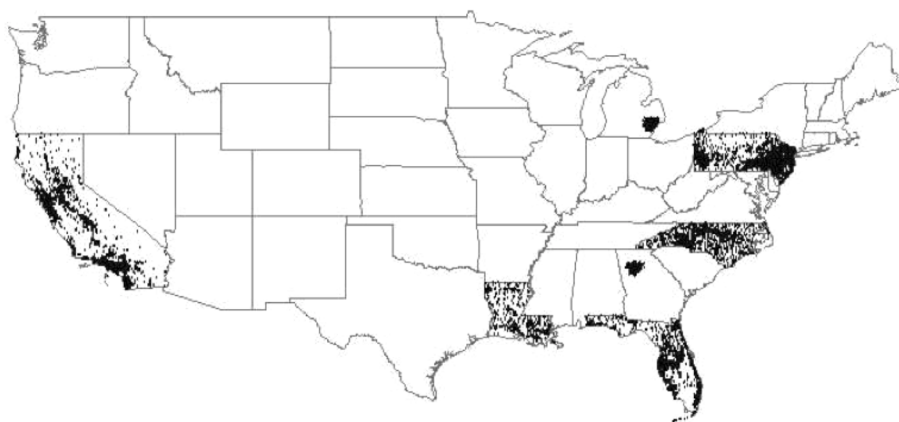
**Air pollution exposure assessment.** Outdoor annual PM<sub>2.5</sub>-related exposures at the census-tract level for residences at NIH-AARP cohort entry were estimated using data from the U.S. Environmental Protection Agency (EPA) nationwide Air Quality System (AQS, formerly AIRS) (<http://www.epa.gov/airdata/>). The nationwide AQS Network includes nearly 3,000 sites, has operated since the 1970s, and has included measurement of PM<sub>2.5</sub> mass since mid-1999. The year 2000 was selected as the start of follow-up in this study because that is the first full year that outdoor PM<sub>2.5</sub> exposure data were available nationwide. The contiguous U.S. map in Figure 1 displays the census tracts in which the members of this cohort resided at the start of the study. Private residence locations were not included in the original NIH-AARP Cohort data set in order to protect participant privacy. As a result, we employed census tract centroid estimates of monthly average PM<sub>2.5</sub> mass exposures available through the year 2008, as obtained from a published hybrid land-use regression (LUR) geostatistical model (Beckerman et al. 2013), and as matched with individuals by NIH to further protect participant anonymity. Exposure was considered only through 2008 because the time-dependent model matched deaths with exposure in each prior year, and follow-up ended in 2009 for these analyses.

These estimates used ambient AQS PM<sub>2.5</sub> as the dependent variable and traffic and land use information as predictors (Beckerman et al. 2013). Residuals from this model were interpolated with a Bayesian maximum entropy (BME) model, and the estimates from the LUR and BME were combined post hoc to derive monthly estimates of PM<sub>2.5</sub>. To allow investigation of possible confounding by O<sub>3</sub> exposure, annual primary metropolitan statistical area (PMSA) mean ozone (O<sub>3</sub>) exposures were also estimated for the year 2000 by averaging annual O<sub>3</sub> means from all ambient monitoring sites with > 75% of possible days of data in each PMSA (including 391 sites among 93 PMSAs) (U.S. EPA 2014). The PMSA mean PM<sub>2.5</sub> mass concentrations in 2008, at the end of the exposure period, were lower than but highly correlated with their paired PMSA mean concentration in 2000 ( $R^2 = 0.77$ ), suggesting that the spatial rank ordering of PM<sub>2.5</sub> concentrations remained consistent over the follow-up period. However, the number of cohort participants living below the U.S. annual PM<sub>2.5</sub> standard (12 µg/m<sup>3</sup>) increased over time, rising steadily from only 33% of cohort participants in 2000 (mean ± SD = 13.6 ± 3.6 µg/m<sup>3</sup>) up to 78% of cohort participants living below the 12 µg/m<sup>3</sup> annual PM<sub>2.5</sub> standard in 2008 (mean ± SD = 10.2 ± 2.3 µg/m<sup>3</sup>). Therefore, to incorporate these exposure level changes over the follow-up time, we also developed annual mean exposures at the census tract centroid of each participant's residence at baseline to incorporate into a time-dependent sensitivity analysis of the PM<sub>2.5</sub>–mortality association, with censoring for those known to have moved.

**Statistical methods.** Person-years of follow-up were included for each participant from 1 January 2000 to the date of death, the end of follow-up (31 December 2009), or the date the participant moved out of the state or city where s/he lived at enrollment, whichever occurred first. This period was selected because that is the time period

for which outdoor PM<sub>2.5</sub> exposure estimates were available nationwide at the census-tract level for matching with the cohort mortality data (Beckerman et al. 2013). For the time-independent exposure model, the exposure metric was each participant's annual mean enrollment census tract–centroid PM<sub>2.5</sub> exposure in the first year of this mortality analysis, 2000, which was the first complete year of PM<sub>2.5</sub> data availability across the United States. In addition, we also considered a time-dependent (annual mean) model, for which annual mean census tract–level exposure to PM<sub>2.5</sub> was treated as time-varying, with a 1-year lag. For example, mortality risk during 2000 was related to each participant's enrollment residence census tract–specific average PM<sub>2.5</sub> for 1999.

We used the Cox proportional hazards models (Cox and Oakes 1984; Fleming and Harrington 1991) to estimate relative risks (RRs) of mortality and 95% confidence intervals (CIs) in relation to ambient PM<sub>2.5</sub> (per 10 µg/m<sup>3</sup>). In multivariate models including individual-level variables, we treated age (in 3-year groupings), sex and region (six U.S. States and two municipalities of residence at study entry) as strata and adjusted for the following individual covariates and potential risk factors at enrollment: race (non-Hispanic white, non-Hispanic black, other), education (< 8 years, 8–11 years, high school, some college, college graduate), marital status (married, never married, or other, including widowed/divorced/separated and unknown), body mass index (BMI; < 18.5, 18.5 to < 25.0, 25.0 to < 30.0, 30 to < 35, and ≥ 35 kg/m<sup>2</sup>), alcohol consumption (none, < 1, 1–2, 2–5, and ≥ 5 drinks per day), and smoking history (never smoker, former smoker who quit at least 1 year ago of ≤ 1 pack/day, former smoker who quit at least 1 years ago of > 1 pack/day, quit less than 1 year ago or current smoker of ≤ 1 pack/day, quit less than 1 year ago or current smoker of > 1 pack/day). We also included two contextual characteristics of the



**Figure 1.** Continental U.S. map of NIH-AARP study participants' census tracts.



participants' residential census tracts found to modify the PM<sub>2.5</sub>–mortality HR estimates and have statistical significance in our analyses (data not shown): *a*) median census tract household income; and *b*) percent of census tract population with less than a high school education, based on the 2000 decennial census for the residence at study entry, as included in the cohort data set (NIH-AARP 2006). Potential effect modification was assessed by including multiplicative interaction terms between PM<sub>2.5</sub> concentrations and each covariate [e.g., sex, age < 65 or ≥ 65 years, age and sex combined, education (< high school, high school, > high school), and smoking (never, former, current) at baseline] in the proportional hazards models. Likelihood ratio statistic *p*-values (two-sided) comparing model fit with and without interaction terms were used to test the statistical significance of each interaction, with *p*-values of < 0.05 defined as statistically significant. Statistical analyses were carried out in SAS (version 9.3; SAS Institute Inc.) and R (version 3.0.1), using the “survival” package (R Core Team 2013).

Additional sensitivity analyses were conducted, including models without adjusting for contextual variables; limiting the analysis to California residents; without censoring data after people moved; adjusting for O<sub>3</sub>, and using PM<sub>2.5</sub> exposures estimated at the metropolitan area average level (rather than at the census tract level). In addition, other contextual characteristics were also considered: *a*) Gini coefficient, a metric of income inequality; *b*) percent of census tract population who are black; *c*) percent of census tract population who are unemployed; and *d*) percent of census tract population living below the poverty level, but were not included in the final model, as addition of these variables did not significantly affect results. To allow more direct comparisons with past work applying random effects methods (e.g., Krewski et al. 2009), we also evaluated HRs in relation to baseline (2000) PM<sub>2.5</sub> exposure levels while incorporating random effects for state of residence using the “coxme” package in R.

To show how the shape of the PM<sub>2.5</sub>–mortality relationship response varies with concentration in this cohort, PM<sub>2.5</sub> natural spline (ns) plots with 4 degrees of freedom (df) were prepared for both total (all cause) and cardiovascular mortality using standard Cox models for the baseline case, stratified by age and sex, and adjusted for all individual-level covariates and contextual variables, as described above.

## Results

The cohort was exposed to a wide range of PM<sub>2.5</sub> concentrations (Table 1), with a concentration range similar to the nation as a whole (U.S. EPA 2009). Except for race

(for which Table 1 indicates a rising exposure with increasing percentage of black participants), cohort characteristics were generally similar across PM<sub>2.5</sub> exposure level, limiting the potential for confounding in our PM<sub>2.5</sub> mortality relationship analyses.

In our time-independent baseline exposure Cox model analyses of the selected cohort (using the study entry tract of residence PM<sub>2.5</sub> mean as the exposure reference for each participant), higher levels of ambient PM<sub>2.5</sub> exposure were significantly associated with increased mortality due to all causes of (nonaccidental) death (HR = 1.03 per 10 µg/m<sup>3</sup> PM<sub>2.5</sub>; 95% CI: 1.00, 1.05) and cardiovascular disease (HR = 1.10; 95% CI: 1.05, 1.15), as presented in Table 2. Stratified analyses by sex, age, and education for this cohort did not indicate significant differences in PM<sub>2.5</sub> effect estimates across categories (Table 2). However, although PM<sub>2.5</sub> exposure was not significantly associated overall with increased risk of respiratory mortality (HR = 1.05; 95% CI: 0.98, 1.13), an association was found for never smokers (HR = 1.27; 95% CI: 1.03, 1.56). Figure 2 graphically demonstrates, for the time-independent model, the monotonically rising nature of the concentration–response curve for both all-cause and CVD mortality (vs. a referent HR = 1.0 at the mean level of exposure).

A number of sensitivity analyses for alternative models were also conducted (Table 3). In general, associations were stronger and *p*-values were smaller when we did not adjust for census tract–level contextual environmental variables, including the association with respiratory mortality (HR = 1.09; 95% CI: 1.02, 1.18). Adding random-effects terms to the time-independent model yielded very similar results to those without random-effects terms. Time-dependent yearly exposure models gave comparable results to the year 2000 time-independent baseline exposure model for total mortality (HR = 1.03; 95% CI: 0.99, 1.05), CVD mortality (HR = 1.11; 95% CI: 1.06, 1.16), and respiratory mortality (HR = 1.05; 95% CI: 0.97, 1.15). Limiting the analysis to only California (the state with the largest number of cohort participants) gave similar results to the entire cohort. To assess the extent to which our censoring of those who moved out of the study state/city might have affected the results, we also present overall results for participants without that censoring, retaining those who moved after 2000, finding that it gave similar results to our base model case with censoring (as shown in Table 2). In addition, in a model that simultaneously also included exposure to the gaseous pollutant O<sub>3</sub> along with PM<sub>2.5</sub>, the PM<sub>2.5</sub> effect estimate was found to be still significant and its CVD mortality effect

estimate not statistically different from the model without the addition of O<sub>3</sub>, indicating the PM<sub>2.5</sub>–CVD mortality association to be robust to the addition of O<sub>3</sub>.

## Discussion

In this large prospective cohort study with detailed baseline individual-level risk factor information on study participants (e.g., smoking, BMI, alcohol use), we confirmed a monotonically increasing, and statistically significant, relationship between long-term exposure to PM<sub>2.5</sub> air pollution and both all-cause and CVD mortality, even at the decreased PM<sub>2.5</sub> levels experienced in the United States since 2000. Comparisons by sex, age, and education for this cohort did not indicate statistically significant differences in the mortality–PM<sub>2.5</sub> association across categories.

With significant overall associations with all-cause and cardiovascular mortality, the results presented here are consistent with many, but not all, of the prior published results examining PM<sub>2.5</sub> and mortality. We estimated a 3% increase (95% CI: 0, 5%) in all-cause mortality for a 10-µg/m<sup>3</sup> annual increase in PM<sub>2.5</sub> that, though statistically significant in this large cohort, is lower than many other past estimates. For example, a recent literature review reported a pooled effect estimate of 6% per 10 µg/m<sup>3</sup> PM<sub>2.5</sub> (95% CI: 4, 8%) for all-cause mortality (Hoek et al. 2013). Our overall estimate for CVD mortality (10% effect per 10 µg/m<sup>3</sup>; 95% CI: 5, 15%), agrees more closely with the pooled estimate for CVD mortality reported by Hoek et al. (2013) (11% per 10 µg/m<sup>3</sup>; 95% CI: 6, 16%).

Comparisons with the American Cancer Society (ACS) cohort, a similarly large nationwide cohort, provides an opportunity to evaluate the issue of association consistency over time in the United States. Although participants in the ACS cohort (Pope et al. 2002) were somewhat younger (mean 56 years at recruitment, vs. mean 65 years in the NIH-AARP cohort in 2000), and were exposed during that study's follow-up to pollution at an earlier period of time (when the mix of air pollution sources was likely different), it has a similar racial (> 90% white) and educational (> 50% post-high school education) composition, is of similar size (> 500,000 participants), and also spans the United States, making it probably the most similar U.S. cohort for comparison here. The ACS cohort reported that a 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub> was associated with a 4% increase in all-cause mortality (95% CI: 1, 8%) (Pope et al. 2002), which is consistent with the corresponding estimate from the present analysis (3% per 10 µg/m<sup>3</sup>; 95% CI: 0, 5%), as shown in Figure 3. Moreover,

the PM<sub>2.5</sub>–CVD mortality effect estimate reported for the ACS cohort (12% per 10 µg/m<sup>3</sup>; 95% CI: 8, 15%) (Pope et al. 2004) is very similar to the corresponding association in the NIH-AARP cohort (10% per 10 µg/m<sup>3</sup>; 95% CI: 5, 15%) (Figure 3). This new prospective cohort study's follow-up begins at approximately the time that most of the published ACS cohort's follow-up analyses ended, providing an independent test as to whether the effects continue to the lower PM<sub>2.5</sub> levels in the 21st century. The ACS cohort study started in 1982 with follow-up through 1998, with an annual PM<sub>2.5</sub> study period mean ± SD = 17.7 ± 3.7 µg/m<sup>3</sup>

(Pope et al. 2002); in contrast, this new NIH-AARP analysis started in 2000 with much lower study follow-up mean PM<sub>2.5</sub> of 12.2 ± 3.4 µg/m<sup>3</sup> through 2008. Our study therefore documents for the first time that the PM<sub>2.5</sub>–mortality effects still occur at the much lower post-2000 levels of exposures across the United States. In California, the ACS follow-up ended with a mean 1998–2002 PM<sub>2.5</sub> concentration of 14.1 µg/m<sup>3</sup> (Jerrett et al. 2013), versus a much lower end of follow-up mean 2008 PM<sub>2.5</sub> concentration of 10.4 µg/m<sup>3</sup> in the present study. Figure 3 provides comparative plots of these two cohort's PM<sub>2.5</sub> mortality estimates across

mortality outcomes, for both the United States and the State of California (Jerrett et al. 2013; Krewski et al. 2009; Pope et al. 2002, 2004), indicating consistency in their effect estimates, despite the notable decline in pollution levels after 2000.

We have also considered and compared effect estimates per 10 µg/m<sup>3</sup> PM<sub>2.5</sub> as a function of alternative PM<sub>2.5</sub> exposure metrics. In addition to the year 2000 base PM<sub>2.5</sub> exposure index, we also considered time-dependent annual mean exposure models for each mortality outcome that directly addressed the declining concentration levels of PM<sub>2.5</sub> exposures during follow-up.

**Table 1.** Selected participant characteristics according to quintile of PM<sub>2.5</sub> exposure in 2000 [mean ± SD or n (%)].

Characteristic	PM <sub>2.5</sub> concentration (µg/m <sup>3</sup> )				
	2.9–10.7	10.7–12.6	12.6–14.2	14.2–15.9	15.9–28.0
n <sup>a</sup>	103,576	103,330	103,345	103,410	103,380
Age in 2000 (years)	66.1 ± 5.3	65.8 ± (5.4)	65.6 ± (5.4)	65.6 ± (5.4)	65.6 ± (5.4)
Sex					
Male	60,996 (58.9)	61,716 (59.7)	61,541 (59.5)	61,076 (59.1)	58,053 (56.2)
Female	42,580 (41.1)	41,614 (40.3)	41,804 (40.5)	42,334 (40.9)	45,327 (43.8)
BMI (kg/m <sup>2</sup> )					
≤ 18.5	845 (0.8)	817 (0.8)	842 (0.8)	809 (0.8)	860 (0.8)
18.5–25	37,390 (36.1)	34,657 (33.5)	33,316 (32.2)	32,861 (31.8)	35,545 (34.4)
> 25 and ≤ 30	42,709 (41.2)	43,141 (41.8)	43,329 (41.9)	43,327 (41.9)	41,781 (40.4)
> 30 and ≤ 35	14,714 (14.2)	15,959 (15.4)	16,546 (16.0)	16,794 (16.2)	15,823 (15.3)
> 35	5,329 (5.1)	6,041 (5.8)	6,510 (6.3)	6,816 (6.6)	6,531 (6.3)
Unknown	2,589 (2.5)	2,715 (2.6)	2,802 (2.7)	2,803 (2.7)	2,840 (2.7)
Smoking status					
Never smoking	34,685 (33.5)	35,363 (34.2)	37,100 (35.9)	37,413 (36.2)	38,377 (37.1)
Former, ≤ 1 pack/day	28,700 (27.7)	27,572 (26.7)	27,307 (26.4)	27,219 (26.3)	27,442 (26.5)
Former, > 1 pack/day	23,163 (22.4)	22,575 (21.8)	21,285 (20.6)	20,414 (19.7)	19,696 (19.1)
Currently, ≤ 1 pack/day	8,555 (8.3)	8,709 (8.4)	8,855 (8.6)	9,541 (9.2)	9,368 (9.1)
Currently, > 1 pack/day	4,657 (4.5)	5,232 (5.1)	4,895 (4.7)	4,812 (4.7)	4,543 (4.4)
Unknown	3,816 (3.7)	3,879 (3.8)	3,903 (3.8)	4,011 (3.9)	3,954 (3.8)
Race/ethnicity					
White	95,786 (92.5)	95,942 (92.9)	96,283 (93.2)	94,670 (91.5)	88,741 (85.8)
Black	1,807 (1.7)	2,501 (2.4)	3,532 (3.4)	5,421 (5.2)	7,067 (6.8)
Hispanic	2,691 (2.6)	1,974 (1.9)	1,180 (1.1)	920 (0.9)	3,011 (2.9)
Asian	1,957 (1.9)	1,573 (1.5)	1,004 (1.0)	1,043 (1.0)	2,863 (2.8)
Unknown	1,335 (1.3)	1,340 (1.3)	1,346 (1.3)	1,356 (1.3)	1,698 (1.6)
Marital status					
Married	71,327 (68.9)	72,457 (70.1)	72,094 (69.8)	70,980 (68.6)	65,450 (63.3)
Widowed/divorced/separated	26,664 (25.7)	25,923 (25.1)	25,816 (25.0)	26,592 (25.7)	30,330 (29.3)
Never married	4,743 (4.6)	4,135 (4.0)	4,563 (4.4)	5,019 (4.9)	6,646 (6.4)
Unknown	842 (0.8)	815 (0.8)	872 (0.8)	819 (0.8)	954 (0.9)
Education					
Less than 11 years	5,081 (4.9)	6,011 (5.8)	6,829 (6.6)	7,198 (7.0)	5,672 (5.5)
High school completed	17,019 (16.4)	19,880 (19.2)	22,604 (21.9)	24,055 (23.3)	17,750 (17.2)
Post-high school	9,560 (9.2)	10,590 (10.2)	10,652 (10.3)	10,933 (10.6)	8,890 (8.6)
Some college	25,852 (25.0)	24,470 (23.7)	21,809 (21.1)	21,616 (20.9)	25,854 (25.0)
College and post graduate	43,103 (41.6)	39,343 (38.1)	38,347 (37.1)	36,498 (35.3)	42,001 (40.6)
Unknown	2,961 (2.9)	3,036 (2.9)	3,104 (3.0)	3,110 (3.0)	3,213 (3.1)
State of residence					
California	49,086 (47.4)	26,087 (25.2)	12,303 (11.9)	13,238 (12.8)	59,495 (57.5)
Florida	47,001 (45.4)	42,769 (41.4)	14,647 (14.2)	5,851 (5.7)	82 (0.1)
Georgia	0 (0.0)	0 (0.0)	0 (0.0)	156 (0.2)	14,331 (13.9)
Louisiana	265 (0.3)	3,717 (3.6)	12,150 (11.8)	3,295 (3.2)	145 (0.1)
Michigan	78 (0.1)	1,157 (1.1)	3,051 (3.0)	15,546 (15.0)	6,307 (6.1)
North Carolina	156 (0.2)	8,022 (7.8)	11,596 (11.2)	18,402 (17.8)	4,583 (4.4)
New Jersey	4,585 (4.4)	14,568 (14.1)	29,238 (28.3)	14,657 (14.2)	2,149 (2.1)
Pennsylvania	2,405 (2.3)	7,010 (6.8)	20,360 (19.7)	32,265 (31.2)	16,288 (15.8)
Contextual variables					
Median income (\$)	57,399 ± 27,037	52,980 ± 23,695	53,453 ± 22,793	51,280 ± 20,502	53,746 ± 22,979
Percent high school or less	13.6 ± 9.6	15.5 ± 10.0	15.6 ± 9.7	16.2 ± 9.8	18.0 ± 13.7

<sup>a</sup>Number of participants in PM<sub>2.5</sub> quintile, after accounting for missing covariate data.

The fixed exposure model has the advantage that it provides results using methods directly comparable to those used in many past such analyses (e.g., the ACS CP-II cohort). We

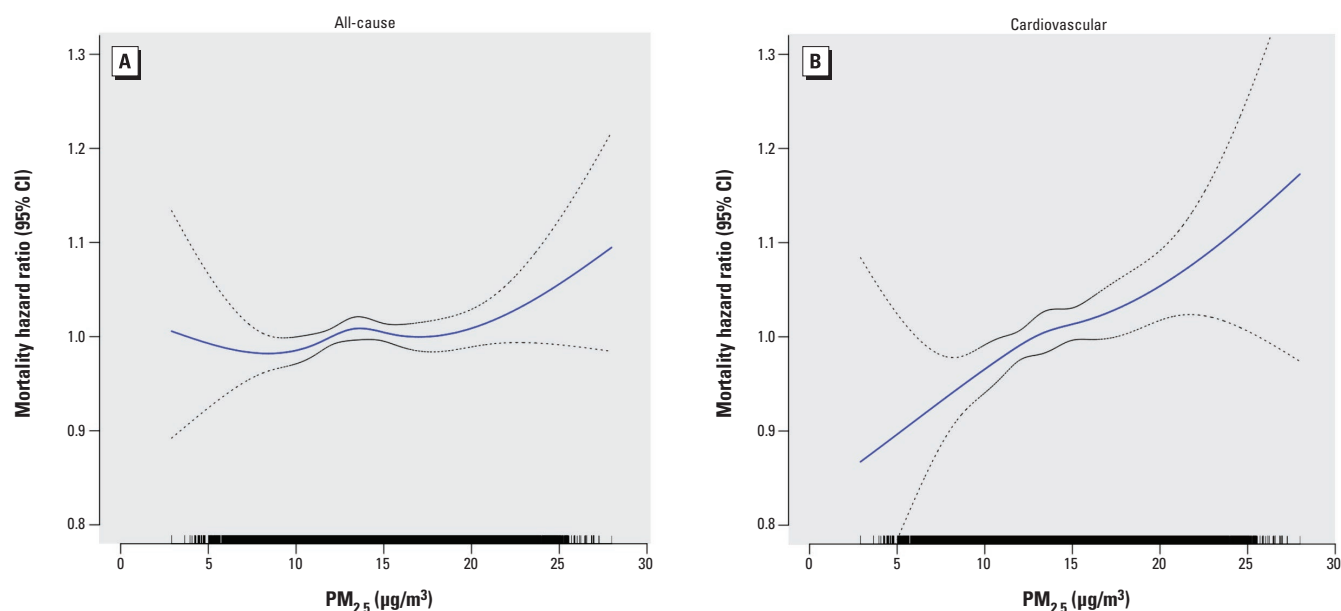
found that the annual mean model yielded results consistent with the baseline (year 2000) exposure time-independent model. Lepeule et al. (2012) also found that varying

the exposure metric choice had little effect on  $PM_{2.5}$  effect estimates in their analysis of the Harvard Six Cities Study cohort. Not censoring those participants who moved out

**Table 2.** NIH-AARP cohort time independent Cox model  $PM_{2.5}$  mortality hazard ratios (and 95% CIs) per 10  $\mu g/m^3$ , by cause and cohort subgroup.

Cohort subset	All-cause mortality			Cardiovascular mortality			Respiratory mortality		
	HR (95% CI)	<i>n</i> deaths	<i>p</i> -int	HR (95% CI)	<i>n</i> deaths	<i>p</i> -int	HR (95% CI)	<i>n</i> deaths	<i>p</i> -int
All	1.03 (1.00, 1.05)	84,404		1.10 (1.05, 1.15)	26,009		1.05 (0.98, 1.13)	8,397	
Age (years)									
< 65	1.00 (0.95, 1.05)	20,422		1.09 (0.99, 1.19)	5,614		1.00 (0.85, 1.19)	1,592	
≥ 65	1.03 (1.00, 1.06)	63,982	0.67	1.10 (1.05, 1.15)	20,395	0.97	1.06 (0.98, 1.15)	6,805	0.24
Sex									
Male	1.03 (1.00, 1.06)	55,685		1.09 (1.04, 1.15)	18,200		1.02 (0.93, 1.12)	5,193	
Female	1.02 (0.98, 1.06)	28,719	0.77	1.10 (1.02, 1.19)	7,809	0.33	1.10 (0.98, 1.23)	3,204	0.73
Sex and age (years)									
Male: < 65	0.99 (0.94, 1.06)	13,117		1.08 (0.97, 1.21)	3,975		0.99 (0.80, 1.23)	923	
Male: ≥ 65	1.04 (1.01, 1.08)	42,568		1.10 (1.03, 1.16)	14,225		1.03 (0.92, 1.14)	4,270	
Female: < 65	1.01 (0.94, 1.10)	7,305		1.11 (0.94, 1.30)	1,639		1.01 (0.78, 1.31)	669	
Female: ≥ 65	1.02 (0.97, 1.06)	21,414	0.88	1.10 (1.01, 1.19)	6,170	0.82	1.12 (0.99, 1.28)	2,535	0.56
Education									
< High school education	1.02 (0.97, 1.07)	25,886		1.05 (0.97, 1.15)	8,176		1.04 (0.91, 1.19)	2,900	
High school education	1.06 (0.98, 1.15)	8,668		1.21 (1.05, 1.40)	2,708		1.00 (0.79, 1.26)	883	
> High school education	1.02 (0.99, 1.05)	46,577	0.65	1.10 (1.04, 1.16)	14,057	0.86	1.07 (0.97, 1.18)	4,275	0.38
Smoking									
Never smoked	1.04 (0.99, 1.08)	19,785		1.11 (1.02, 1.20)	6,384		1.27 (1.03, 1.56)	1,004	
Former smoker	1.02 (0.99, 1.06)	44,590		1.07 (1.01, 1.14)	13,934		1.04 (0.94, 1.14)	4,677	
Current smoker	1.01 (0.95, 1.06)	16,354	0.58	1.14 (1.02, 1.25)	4,451	0.46	1.01 (0.88, 1.16)	2,372	0.70

*p*-int, *p*-value for interaction.



**Figure 2.** Concentration–response curves (solid lines) and 95% CIs (dashed lines) based on natural spline models with 4 df, standard Cox models stratified by age and sex, adjusted for all individual-level covariates (race, education, marital status, BMI, alcohol consumption, and smoking history) and contextual covariates [median income (\$), and percent high school or less] for (A) all nonaccidental causes and (B) cardiovascular disease. The tick marks on the x-axis identify the distribution of observations according to  $PM_{2.5}$  concentrations.

**Table 3.** NIH-AARP cohort  $PM_{2.5}$  mortality hazard ratios and 95% CIs per 10  $\mu g/m^3$   $PM_{2.5}$  for alternative model specifications.

Model	<i>n</i>	All	Cardiovascular	Respiratory
Full baseline model, time-independent 2000 census tract mean $PM_{2.5}$ exposures	517,041	1.03 (1.00, 1.05)	1.10 (1.05, 1.15)	1.05 (0.98, 1.13)
Full model, time-dependent annual census tract mean $PM_{2.5}$ exposures	517,041	1.03 (0.99, 1.05)	1.11 (1.06, 1.16)	1.05 (0.97, 1.15)
Full baseline model, 2000 PMSA mean $PM_{2.5}$ exposures	474,565	1.01 (0.98, 1.04)	1.10 (1.04, 1.16)	1.06 (0.97, 1.16)
Full baseline model without contextual variations	517,041	1.06 (1.03, 1.08)	1.15 (1.10, 1.20)	1.09 (1.02, 1.18)
Full baseline model with random effects	517,041	1.03 (1.00, 1.05)	1.10 (1.05, 1.14)	1.06 (0.99, 1.14)
Full baseline model with $O_3$	466,121	1.02 (0.99, 1.05)	1.07 (1.02, 1.12)	1.02 (0.94, 1.11)
Full baseline model retaining all who moved from study area after 2000	517,041	1.02 (1.00, 1.05)	1.10 (1.06, 1.15)	1.04 (0.97, 1.12)
Full baseline model for California only	160,209	1.02 (0.99, 1.04)	1.10 (1.05, 1.16)	1.01 (0.93, 1.10)

of the study areas between 2000 and 2006 ( $n = 28,923$ ) had little effect on these results. We also compared the results using both PMSA and census tract-level mean exposure metrics, finding similar and confirmatory results with either approach. This may suggest that the fact that people are mobile, and often do not stay at their home residence all day, may limit the exposure assessment accuracy gain derived from knowing home residence locale versus an area-wide average. Overall, we found that the PM<sub>2.5</sub>–mortality associations in this work are robust to various PM<sub>2.5</sub> exposure modeling choices.

Numerous past long-term PM<sub>2.5</sub>–mortality analyses have found higher relative risks among those with less education. For example, Krewski et al. (2000), in their reanalysis of the Six Cities and ACS cohorts, found that the relative risk of mortality associated with fine particles was greater among individuals with high school education or less, compared to those with more than high school education in the Six Cities Study, and that the fine particle air pollution mortality risk decreased significantly ( $p < 0.05$ ) with increasing educational attainment in the ACS cohort. They concluded that “it is possible that educational attainment is a marker for socioeconomic status, which in turn may be correlated with exposure to fine particle air pollution.” Similarly, Brunekreef et al. (2009) found in their NLCS (Netherlands Cohort Study on Diet and Cancer)–AIR cohort examination of long-term exposure to traffic air pollution that associations with mortality tended to be stronger in case-cohort participants with lower levels of education, but that differences between strata were not statistically significant. Ostro et al. (2008) also estimated stronger

associations with components of PM<sub>2.5</sub> among individuals with lower educational attainment, attributing this trend to the effects of lower socioeconomic status. However, no such trend was found in this NIH-AARP cohort, perhaps because the reported annual incomes of this cohort did not vary with PM<sub>2.5</sub> concentration (Table 1). Indeed, although the association of education with median income in this cohort was strong ( $r = 0.49$ ), the correlation between PM<sub>2.5</sub> and median income was much lower ( $p = 0.03$ ). Thus, it may be that the lack of a strong socioeconomic–PM<sub>2.5</sub> covariation in this cohort is the reason we did not see the mortality effect modification by education status found in past studies.

This study has both strengths and limitations relative to past such studies. One strength is that we have employed estimates of PM<sub>2.5</sub> exposure at the participant residence census tract level, rather than applying the overall county or metropolitan area average exposure that has been used in some major prior studies (e.g., the Medicare and ACS cohorts, respectively) (Eftim et al. 2008; Krewski et al. 2009). In addition, most previous studies have assigned only a single fixed exposure level for each study participant (e.g., at the start of the follow-up), whereas we also considered a sensitivity model applying time-varying exposure estimates to address the declining PM<sub>2.5</sub> exposure levels over time. Another strength of this study is that covariate risk factors were collected at the individual level, but a limitation is that this was ascertained only at enrollment, and we could not account for temporal changes in risk factors (e.g., smoking and BMI) during follow up. Another limitation is that, other than knowing if and when participants leave the NIH-AARP

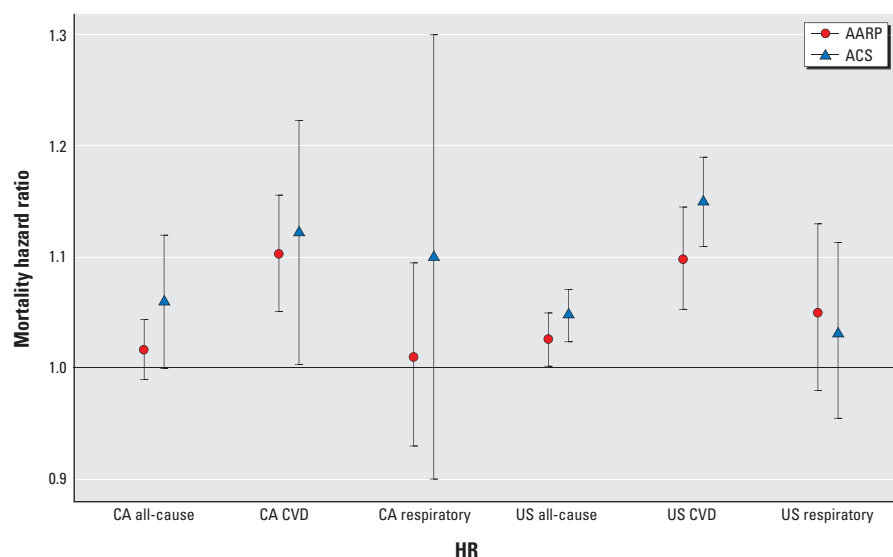
cohort study areas, we presently lack information on residence location after those participants moved out of the study region. Despite these limitations, as discussed above, our derived effect estimates were found to be largely consistent with other PM<sub>2.5</sub> mortality results, notably the ACS cohort study (Pope et al. 2002, 2004), the only prior prospective U.S. cohort study of such size with detailed individual-level risk factor information.

## Conclusions

Long-term exposure to PM<sub>2.5</sub> air pollution was associated with a significant increase in CVD and total nonaccidental mortality in the cohort as a whole, as well as with a significant increase in respiratory mortality among never smokers, in a new, large, U.S. cohort having detailed individual level participant data and census tract-level PM<sub>2.5</sub> exposure information. This independent evaluation of the PM<sub>2.5</sub>–mortality association, in this new large cohort, was robust to various model specification and PM<sub>2.5</sub> exposure assessment sensitivity analyses, and has found effect estimates (per 10  $\mu\text{g}/\text{m}^3$  of PM<sub>2.5</sub> exposure) that are consistent with past estimates, even at the much lower PM<sub>2.5</sub> air pollution levels experienced in the United States since 2000.

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**Figure 3.** Comparison of NIH-AARP cohort vs. published ACS cohort all-cause and by-cause mortality hazard ratios per 10  $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub>, with 95% CIs, for the state of California (CA) and nationwide (US) (Jerrett et al. 2013; Krewski et al. 2009).



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# Chronic Exposure to Fine Particles and Mortality: An Extended Follow-up of the Harvard Six Cities Study from 1974 to 2009

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**BACKGROUND:** Epidemiologic studies have reported associations between fine particles (aerodynamic diameter  $\leq 2.5 \mu\text{m}$ ;  $\text{PM}_{2.5}$ ) and mortality. However, concerns have been raised regarding the sensitivity of the results to model specifications, lower exposures, and averaging time.

**OBJECTIVE:** We addressed these issues using 11 additional years of follow-up of the Harvard Six Cities study, incorporating recent lower exposures.

**METHODS:** We replicated the previously applied Cox regression, and examined different time lags, the shape of the concentration–response relationship using penalized splines, and changes in the slope of the relation over time. We then conducted Poisson survival analysis with time-varying effects for smoking, sex, and education.

**RESULTS:** Since 2001, average  $\text{PM}_{2.5}$  levels, for all six cities, were  $< 18 \mu\text{g}/\text{m}^3$ . Each increase in  $\text{PM}_{2.5}$  ( $10 \mu\text{g}/\text{m}^3$ ) was associated with an adjusted increased risk of all-cause mortality ( $\text{PM}_{2.5}$  average on previous year) of 14% [95% confidence interval (CI): 7, 22], and with 26% (95% CI: 14, 40) and 37% (95% CI: 7, 75) increases in cardiovascular and lung-cancer mortality ( $\text{PM}_{2.5}$  average of three previous years), respectively. The concentration–response relationship was linear down to  $\text{PM}_{2.5}$  concentrations of  $8 \mu\text{g}/\text{m}^3$ . Mortality rate ratios for  $\text{PM}_{2.5}$  fluctuated over time, but without clear trends despite a substantial drop in the sulfate fraction. Poisson models produced similar results.

**CONCLUSIONS:** These results suggest that further public policy efforts that reduce fine particulate matter air pollution are likely to have continuing public health benefits.

**KEY WORDS:** air pollution, cohort studies, concentration–response, follow-up studies, lag, lung cancer, mortality, particles,  $\text{PM}_{2.5}$ , threshold. *Environ Health Perspect* 120:965–970 (2012). <http://dx.doi.org/10.1289/ehp.1104660> [Online 28 March 2012]

All-cause, cardiopulmonary, cardiovascular, and lung-cancer mortality have been associated with chronic air pollution exposure in prospective studies that controlled for individual covariates (Abbey et al. 1999; Beelen et al. 2008b; Beeson et al. 1998; Cao et al. 2011; Dockery et al. 1993; Eftim et al. 2008; Filleul et al. 2005; Gehring et al. 2006; Katanoda et al. 2011; Laden et al. 2006; Miller et al. 2007; Naftstad et al. 2004; Ostro et al. 2010; Pope et al. 2002; Puett et al. 2009; Yorifuji et al. 2011). The studies that specifically considered lung-cancer mortality associations with fine particles (aerodynamic diameter  $< 2.5 \mu\text{m}$ ;  $\text{PM}_{2.5}$ ), all found positive associations (Beelen et al. 2008b; Dockery et al. 1993; Laden et al. 2006; McDonnell et al. 2000), although this association was only statistically significant ( $p < 0.05$ ) in the American Cancer Society study (ACS) (Pope et al. 2002; Turner et al. 2011).

Although compelling evidence supports the harmful effects of  $\text{PM}_{2.5}$  on longevity, concerns have been raised regarding the sensitivity of the results to model specifications. In particular, Moolgavkar (2005, 2007) suggested that covariates may not be proportional and hence were not controlled for properly in proportional hazards models; that the concentration–response relation may not be linear; and that there are few observations at levels as low as or below the

current World Health Organization and U.S. Environmental Protection Agency (EPA) air quality standards. In addition, the relative toxicity of particle elements is still controversial, and most of the recent reduction in  $\text{PM}_{2.5}$  concentrations in the United States has come from sulfate control. Hence it is of interest whether the concentration–response curve has changed over time as particle composition has changed. Health impact assessments in the United States assume that health benefits of reducing particles are only fully realized after 20 years (U.S. EPA 2010), so examination of the lag between exposure and mortality is also relevant for consideration of changes in the standard.

Our goal was to test the robustness of the association between chronic exposure to  $\text{PM}_{2.5}$  and mortality observed in the original study (Dockery et al. 1993), and the first extended follow-up of the Harvard Six Cities study (Laden et al. 2006) by replicating the analyses using 11 additional years of follow-up with exposures well below the U.S. annual standard ( $15 \mu\text{g}/\text{m}^3$ ) (U.S. EPA 1997). We examined different lags of exposure, tested the shape of the  $\text{PM}_{2.5}$  concentration–mortality relationship, tested for changes in this slope over time, and relaxed the proportion assumption by allowing the effects of covariates to vary each year. We reexamined the association of  $\text{PM}_{2.5}$  with specific causes of death such as

lung cancer and examined the effects of  $\text{PM}_{2.5}$  depending on participants' chronic conditions and smoking status.

## Methods

**Study population.** The Harvard Six Cities study population has been previously described (Dockery et al. 1993). Briefly, adults were randomly sampled from six cities in the eastern and midwestern United States between 1974 and 1977: in 1974, Watertown, Massachusetts; in 1975, Kingston and Harriman, Tennessee, and specific census tracts of St. Louis, Missouri; in 1976, Steubenville, Ohio, and Portage, Wyocena, and Pardeeville, Wisconsin; and in 1977, Topeka, Kansas. Information on age, sex, weight, height, educational level, smoking history, hypertension, and diabetes was collected by questionnaire at enrollment. All participants underwent spirometry tests at enrollment (Dockery et al. 1985) and chronic obstructive pulmonary disease (COPD) was defined as having

$$(\text{FEV}_1 \div \text{FVC}) < 70\%,$$

where  $\text{FEV}_1$  is forced expiratory volume in 1 sec, and FVC is forced vital capacity. This analysis, as in the previous analyses, was restricted to 8,096 white participants with acceptable pulmonary function measurements. The study was approved by the Harvard School of Public Health Human Subjects Committee and all participants signed an informed consent before participation.

**Mortality follow-up.** Vital status and cause of death were determined by searching the National Death Index (NDI) for calendar years 1979–2009. Deaths before the NDI started in 1979 were identified by next of kin and Social Security records, and the cause of death was determined by a certified nosologist

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who reviewed death certificates (Dockery et al. 1993).

**Survival time.** Survival times were calculated from enrollment until death or the end of follow-up (31 December 2009). For the 6 participants who were lost to follow-up before 1979, the censored survival times were calculated from enrollment to date of the last follow-up contact plus 6 months or the first day of the NDI (1 January 1979), whichever came first. For each cause of death category, participants who died from another cause were censored at time of death.

**Air pollution estimates.** Annual PM<sub>2.5</sub> concentration was assigned for each participant until death or censoring. PM<sub>2.5</sub> concentration was measured in the participant's city by a centrally located monitor from 1979 to 1986–1988, depending on the city (Dockery et al. 1993). Therefore, the study has no spatial contrast on the within-city scale. PM<sub>2.5</sub> concentrations for the years before monitoring started were assumed to be equal to the earliest monitored year. From the end of monitoring until 1998, PM<sub>2.5</sub> concentration was estimated from PM<sub>10</sub> (aerodynamic diameter < 10 µm) data from U.S. EPA monitors and visibility (extinction) data from the National Weather Service (Laden et al. 2006). From 1999 through 2009, direct measurements of PM<sub>2.5</sub> were available from U.S. EPA monitors. For sensitivity analyses, we also predicted PM<sub>2.5</sub> for 1999–2009 (correlation between predicted and measured was 0.97) using the formula applied to derive exposure estimates during the earlier period when PM<sub>2.5</sub> was not measured.

**Statistical analysis.** We first replicated the original analysis separately for all-cause mortality, cardiovascular mortality as coded by the *International Classification of Diseases, 9th Revision* [ICD-9; World Health Organization (WHO) 1977] or the *10th Revision* [ICD-10; WHO 1992], 400.0–440.9, I10.0–I70.9, respectively, lung-cancer mortality (ICD-9 162, ICD-10 C33.0–C34.9), and COPD mortality (ICD-9 490.0–496.0, ICD-10 J40.0–J47.0) for the 36-year follow-up from 1974 to 2009 using a Cox proportional hazards model with follow-up time as the time scale (Dockery et al. 1993; Laden et al. 2006). PM<sub>2.5</sub> was included in each model as an annual time-dependent variable. The model was stratified by sex, age (1-year intervals) and time in the study (1-year intervals), so that each age/sex group had its own baseline hazard for each year of follow-up. The analysis was adjusted for potential confounders collected at baseline: smoking status (never, former, current), cumulative smoking (pack-years included separately for current and former smokers), educational level (< high-school, ≥ high school), and a linear and quadratic term for body mass index (BMI; kilograms per meter squared), using

the Cox proportional hazards model formulated as follows:

$$h_{is}(t) = h_{0s}(t) \exp[\beta_1 X_i + \beta_2 Z_i(t)], \quad [1]$$

where  $h_i$  is the instantaneous hazard probability of death for subject  $i$  in stratum  $s$  (defined by sex, age, and time in the study),  $h_{0s}(t)$  is the baseline hazard function,  $X_i$  is the vector of time-independent variables, and  $Z_i(t)$  is the vector of time-dependent variables. We evaluated models with 1-year (i.e., exposure during the year before death or censor) to 5-year lagged moving averages and chose the best fit model using Akaike's information criterion (AIC) (Akaike 1973). The best fit moving average was determined from participants who survived at least 5 years from enrollment, so that AIC criteria were evaluated among populations with comparable sizes. We then estimated mortality rate ratios (RR) associated with PM<sub>2.5</sub> exposure during the best fit moving average on the whole sample size. Once the best exposure window was determined, we fit a penalized spline model using a cubic regression spline with 12 knots to estimate the shape of the concentration–response relation, and chose the optimal degree of freedom by minimizing AIC and evaluated nonlinearity with a Wald test. We investigated whether PM<sub>2.5</sub> advanced date of death for participants with chronic conditions at enrollment. We also investigated the potential for effect modification of PM<sub>2.5</sub> on mortality by smoking status at enrollment using interaction terms between such variables and PM<sub>2.5</sub>. Finally, we tested the hypothesis that the effect of PM<sub>2.5</sub> changed over time by dividing the follow-up into four equally spaced time periods and testing interactions between period and PM<sub>2.5</sub>.

**Sensitivity analyses.** We performed sensitivity analyses using a second-degree polynomial distributed lag model to allow the effects of PM<sub>2.5</sub> exposure to be distributed from 1 to 5 years before death or censor (Lepeule et al. 2006; Schwartz 2000); using predicted PM<sub>2.5</sub> concentrations after 1999 instead of the measured PM<sub>2.5</sub>; considering only deaths from natural causes, with external causes of deaths (ICD-9 E800–E999, ICD-10 S00–T88 and V00–Y99) being censored at time of death; and considering only deaths that occurred in the state where the participants lived at enrollment. We next investigated the robustness of the results to alternative modeling assumptions by using a Poisson model with dummy variables for each year of follow-up, which is equivalent to a piecewise exponential proportionate hazard model with the baseline hazard changing each year (Laird and Oliver 1981):

$$\log \mu_{it} = \log E_{it} + \gamma_t T_t + \beta_1 X_i + \beta_2 Z_i(t), \quad [2]$$

where  $\mu_{it}$  is the expected value of the death indicator for subject  $i$  at time  $t$ ,  $E_{it}$  is the

exposure duration of subject  $i$  at time  $t$  ( $\log E_{it}$  being the offset),  $T_t$  is the vector of dummy variables for time by 1 year (piecewise baseline hazard),  $X_i$  is the vector of the time-independent covariates, and  $Z_i(t)$  is the vector of time-dependent variables. Using this Poisson survival analysis, we first compared the results to the Cox model and then relaxed the proportionate hazard assumption for sex, education, and cumulative smoking by including interaction terms of these variables with each year of follow-up. As an alternative to the previous analyses (Dockery et al. 1993; Laden et al. 2006), we used age in 5-year groups as the time scale, and adjusted the model for time trends (linear term). For specific causes of death, convergence issues led us to group age by 10 years. We then fit penalized spline models. Because RRs may vary over time and period-specific RRs may be biased, we used the Poisson model to calculate adjusted survival curves (Hernan 2010). We included product terms between PM<sub>2.5</sub> and time in model 2 [Equation 2], thereby allowing the effect of PM<sub>2.5</sub> to flexibly vary from year to year. We then predicted the survival probability for each year of follow-up for each participant under three scenarios using concentrations of PM<sub>2.5</sub> throughout the entire follow-up period equal to 10, 15, or 20 µg/m<sup>3</sup>.

$p$ -Values < 0.05 were considered statistically significant. All analyses were repeated separately for all- and specific-causes of deaths. Analyses were conducted with SAS software, version 9.2 (SAS Institute Inc., Cary, NC) and R statistical software, version 2.12.2 (R Foundation for Statistical Computing, Vienna, Austria).

## Results

**Study population.** The 8,096 participants were 25–74 years of age at enrollment (mean ± SD, 49.6 ± 13.4) and 54.7% were female. More than half of the participants had a high school degree or higher, 35.8% were current smokers, and 23.9% were former smokers and the average BMI was 25.8 ± 4.5. As for chronic conditions, 17.8% reported hypertension, 11.6% COPD, and 6.9% diabetes.

**Mortality rates and PM<sub>2.5</sub> levels.** At the end of 2009, there were 212,067 person-years of follow-up and 55.5% of the participants had died, among whom 40.8% died from cardiovascular diseases, 7.8% from lung cancer, and 5.5% from COPD (Table 1). Overall, PM<sub>2.5</sub> concentration decreased during the study period (Figure 1). After 1998, annual average levels declined by 1.8 µg/m<sup>3</sup> in St. Louis and by 10.5 µg/m<sup>3</sup> in Steubenville, whereas levels increased by 1.5 µg/m<sup>3</sup> in the Portage–Wyocena–Pardeeville area. Since 2000, all the cities experienced average PM<sub>2.5</sub> levels < 15 µg/m<sup>3</sup> except Kingston–Harriman and Steubenville, which had average concentrations of ≤ 18 µg/m<sup>3</sup>.

**Association between PM<sub>2.5</sub> and mortality.** Using the Cox proportional hazards model, statistically significant associations between PM<sub>2.5</sub> exposure and all-cause, cardiovascular, and lung-cancer mortality were observed (Table 2). The AIC indicated lag 1 (i.e., exposure during the previous year) to be the best fit exposure window for all-cause mortality [see Supplemental Material, Table 1 (<http://dx.doi.org/10.1289/ehp.1104660>)]. For cause-specific mortality, the best fit moving average differed between the Cox and the Poisson regressions. Because the differences in AIC were very small between the 1- and 5-year moving averages for both the Cox and Poisson regressions, we chose the longer of the two moving averages to produce more stable results, specifically, a 1- to 3-year moving average for cardiovascular and lung-cancer mortality, and a 1- to 5-year moving average for COPD mortality. Each 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub> was associated with a 14% increased risk of all-cause death [95% confidence interval (CI): 7%, 22%], a 26% increase in cardiovascular death (95% CI: 14%, 40%), and a 37% increase in lung-cancer death (95% CI: 7%, 75%). For both all-cause mortality and

specific causes of death, the model fit was better without the spline ( $p$ -values between 0.24 and 0.43), indicating a linear relationship with PM<sub>2.5</sub>. Results restricted to participants with chronic conditions at enrollment (i.e., hypertension, COPD, or diabetes) were consistent with those estimated for all participants (Table 2). Although, the interaction between smoking status and PM<sub>2.5</sub> was not statistically significant, there was a trend for a stronger estimated effect of PM<sub>2.5</sub> on mortality in current and former smokers. However, positive associations between PM<sub>2.5</sub> and all-cause and cardiovascular mortality were still evident in never smokers. RR for PM<sub>2.5</sub> fluctuated over time for all-cause mortality and specific causes of death, without clear trends (Table 2).

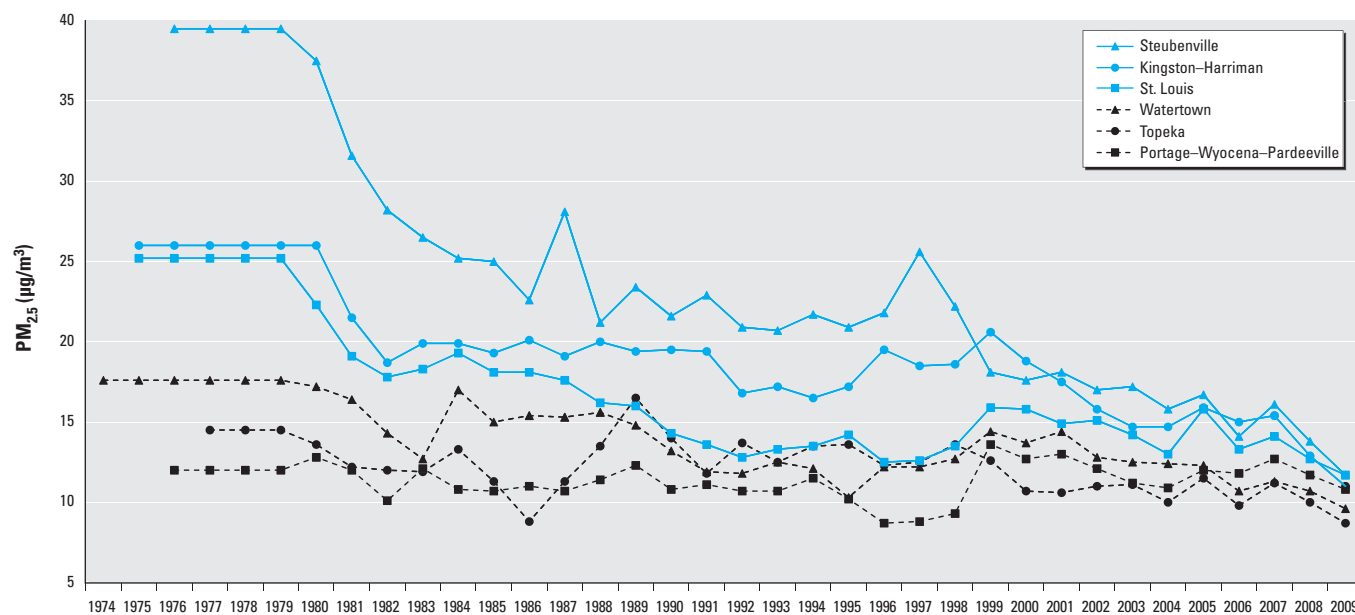
**Sensitivity analysis.** For both all causes and specific causes of death, the cumulative effects estimated from the polynomial distributed lag model were similar to the effect estimates obtained with the selected moving averages (Table 2). However, the five lags were too correlated (between 0.90 and 0.96) to disentangle the relative importance of each one. Using predicted PM<sub>2.5</sub> instead of measured PM<sub>2.5</sub> for exposures after 1999, excluding the

138 deaths from external causes and excluding the 702 participants who died in a state other than the state where they lived at enrollment, did not change the results (data not shown) except for the lung-cancer mortality association with PM<sub>2.5</sub>, which was slightly attenuated (increased risk of 28%; 95% CI: -2%, 67% compared with 37%; 95% CI: 7%, 75%) when the 702 participants were excluded.

With the Poisson framework, using basic assumptions, relaxed proportionate hazard assumption for covariates, or age as the time scale, the effect estimates and  $p$ -values fluctuated slightly but without any notable change in the results compared with estimates from the Cox models for all-cause mortality and for specific-causes of death (data not shown). The penalized spline models also indicated linear concentration-response relationships without a threshold for PM<sub>2.5</sub> and mortality from all-causes and specific-causes [see Supplemental Material, Figure 1 (<http://dx.doi.org/10.1289/ehp.1104660>)]. With the Poisson survival analysis, we predicted survival assuming every participant was exposed to a constant concentration of PM<sub>2.5</sub> (10, 15, or 20  $\mu\text{g}/\text{m}^3$ ) during the entire follow-up period. Adjusted

**Table 1.** Number of participants, mortality, and average PM<sub>2.5</sub> levels in the Harvard Six Cities study, 1974–2009.

Characteristic	Six cities (combined)	Steubenville	Kingston–Harriman	St. Louis	Watertown	Topeka	Portage–Wyocena– Pardeeville
Participants ( $n$ )	8,096	1,346	1,258	1,292	1,332	1,238	1,630
Person-years ( $n$ )	212,067	33,276	33,067	32,225	36,818	32,877	43,804
Cause of death							
All causes ( $n$ [%])	4,495 (55.5)	822 (61.1)	733 (58.3)	827 (64.0)	700 (52.6)	617 (49.8)	796 (48.8)
Cardiovascular (%)	40.8	45.3	41.1	42.2	39.3	37.4	38.6
Lung cancer (%)	7.8	9.0	8.0	8.7	6.6	7.3	6.8
COPD (%)	5.5	4.9	7.0	5.1	4.9	7.3	4.6
1974–2009 average of individual PM <sub>2.5</sub> concentrations	15.9	23.6	19.1	16.7	14.0	12.2	11.4



**Figure 1.** Annual mean PM<sub>2.5</sub> levels during 1974–2009 in the Harvard Six Cities study.



for individual covariates, the lowest PM<sub>2.5</sub> concentration was associated with the highest survival (Figure 2). The three adjusted survival curves showed that the proportionate hazard was a reasonable assumption for PM<sub>2.5</sub> and that PM<sub>2.5</sub> effects were quite stable over time.

## Discussion

Including more recent observations with PM<sub>2.5</sub> exposures down to 8 µg/m<sup>3</sup>, we continued to find a statistically significant association between chronic exposure to PM<sub>2.5</sub> and all-cause and cardiovascular mortality. Furthermore, in the present extended follow-up, PM<sub>2.5</sub> exposure was also statistically significantly associated with lung-cancer mortality. Our study indicated no sensitivity of

the results for all-cause mortality and specific causes of death when we allowed the effects of smoking, education, and sex to vary over time, or when we used age as the time scale instead of follow-up time. Using very flexible modeling assumptions, our results did not show any rationale for change of PM<sub>2.5</sub> effect size over the whole study period, as indicated by the adjusted survival curves and the lack of a clear interaction of PM<sub>2.5</sub> with the four study periods. The concentration–response relationship was linear without any threshold, even at exposure levels below the U.S. annual 15-µg/m<sup>3</sup> standard (U.S. EPA 1997). Taken together with the results of a previous reanalysis of the Harvard Six Cities study (Krewski et al. 2005b), there is evidence for a robust

association between chronic PM<sub>2.5</sub> exposure and early mortality.

**Consistency of the results.** Our results indicated a statistically significant 14% increase in all-cause mortality for a 10-µg/m<sup>3</sup> annual increase in PM<sub>2.5</sub>, which is similar to the results of the previous follow-ups (Dockery et al. 1993; Laden et al. 2006). The Netherlands Cohort Study on Diet (NLCS–Air) in Europe (Beelen et al. 2008b), the Adventist Study (McDonnell et al. 2000), and the male Health Professionals Follow-up Study in the United States (Puett et al. 2011) did not show statistically significant associations between PM<sub>2.5</sub> and all-cause mortality. However, our current results are consistent with those from the ACS cohort (Pope et al. 2002), the Nurses' Health Study (Puett et al. 2009), and the Medicare cohort (Eftim et al. 2008), which indicated mortality increases ranging from 3–26% per 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub>.

The 26% increase in cardiovascular mortality for each 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub> exposure during the previous 3 years estimated in this extended follow-up is similar to the previous estimate (Laden et al. 2006). Although the NLCS–Air study (Beelen et al. 2008b) found no statistically significant association, the magnitude of the estimated effect reported here is between the 12% increase estimated for the ACS cohort (Pope et al. 2004) and the 76% increase estimated for the Women's Health Initiative study (Miller et al. 2007). Puett et al. (2009) also estimated a 100% increase in fatal coronary heart diseases for a 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub> during the prior year. Underlying mechanisms for the effects of PM<sub>2.5</sub> on cardiovascular mortality are still poorly understood, but changes in vasoconstriction might explain the associations (Anderson et al. 2011).

The previous extended follow-up of the Harvard Six Cities study showed an elevated, but not statistically significant, risk of lung-cancer mortality (Laden et al. 2006), whereas the present extended follow-up estimated a statistically significant 37% increase in lung-cancer mortality (for each 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub>), which is greater than that estimated for both the ACS cohort (14%) (Pope et al. 2002) and a Japanese cohort (27%) (Katanoda et al. 2011). Lungs are one of the organs that are most directly affected by particulate air pollution. Fine particles, which may carry toxic chemicals of carcinogenic potential (Laden et al. 2000), can reach lung alveoli where the clearance is slow (Pinkerton et al. 1995) and induce durable pulmonary and systemic inflammation (Riva et al. 2011). Recent findings in the ACS cohort indicated that a 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub> concentration was associated with a statistically significant 15% to 27% increase in lung-cancer mortality in never smokers (Turner et al. 2011).

**Table 2.** Adjusted<sup>a</sup> association between PM<sub>2.5</sub><sup>b</sup> and mortality, for the 8,096 participants and certain subpopulations of the Harvard Six Cities study, 1974–2009.

Cause of death/stratum-specific estimates according to characteristics at enrollment	n participants (n person-years)	RR (95% CI) for 10-µg/m <sup>3</sup> increase in PM <sub>2.5</sub>
All-cause	8,096 (212,067)	1.14 (1.07, 1.22)
Chronic conditions <sup>c</sup>		
Hypertension	1,439 (30,540)	1.17 (1.03, 1.32)
COPD	942 (17,723)	1.09 (0.95, 1.26)
Diabetes	563 (11,473)	1.04 (0.85, 1.27)
Smoking status ( <i>p</i> -interaction = 0.58)		
Never smoker	3,265 (90,372)	1.09 (0.98, 1.21)
Former smoker	1,934 (48,049)	1.17 (1.04, 1.30)
Current smoker	2,897 (73,646)	1.17 (1.06, 1.28)
Follow-up period ( <i>p</i> -interaction = 0.06)		
1974–1982	8,096 (58,798)	1.06 (0.96, 1.17)
1983–1991	7,478 (63,129)	1.32 (1.16, 1.50)
1992–2000	6,391 (51,800)	1.11 (0.98, 1.27)
2001–2009	4,910 (38,340)	1.19 (0.91, 1.55)
Cardiovascular	7,961 (195,941)	1.26 (1.14, 1.40)
Smoking status ( <i>p</i> -interaction = 0.45)		
Never smoker	3,232 (83,861)	1.21 (1.04, 1.41)
Former smoker	1,891 (44,205)	1.21 (1.02, 1.44)
Current smoker	2,838 (67,875)	1.36 (1.17, 1.58)
Follow-up period ( <i>p</i> -interaction = 0.07)		
1974–1982	7,961 (42,672)	1.08 (0.92, 1.27)
1983–1991	7,478 (63,129)	1.46 (1.21, 1.76)
1992–2000	6,391 (51,800)	1.30 (1.06, 1.59)
2001–2009	4,910 (38,340)	1.57 (1.01, 2.43)
Lung cancer	7,961 (195,941)	1.37 (1.07, 1.75)
Smoking status ( <i>p</i> -interaction = 0.15)		
Never smoker	3,232 (83,861)	1.25 (0.54, 2.89)
Former smoker	1,891 (44,205)	1.96 (1.29, 2.99)
Current smoker	2,838 (67,875)	1.25 (0.95, 1.64)
Follow-up period ( <i>p</i> -interaction = 0.19)		
1974–1982	7,961 (42,672)	1.45 (0.98, 2.15)
1983–1991	7,478 (63,129)	0.94 (0.58, 1.52)
1992–2000	6,391 (51,800)	1.54 (0.98, 2.41)
2001–2009	4,910 (38,340)	2.84 (1.06, 7.59)
COPD	7,805 (180,106)	1.17 (0.85, 1.62)
Smoking status ( <i>p</i> -interaction = 0.35)		
Never smoker	3,191 (77,422)	0.85 (0.36, 2.02)
Former smoker	1,847 (40,453)	1.64 (0.92, 2.93)
Current smoker	2,767 (62,231)	1.10 (0.74, 1.62)
Follow-up period ( <i>p</i> -interaction = 0.35)		
1974–1982	7,805 (26,837)	0.79 (0.36, 1.72)
1983–1991	7,478 (63,129)	1.52 (0.90, 2.56)
1992–2000	6,391 (51,800)	1.31 (0.74, 2.31)
2001–2009	4,910 (38,340)	0.68 (0.25, 1.83)

<sup>a</sup>Cox proportional hazards model stratified by sex, age, and time in the study and adjusted for BMI, education, and smoking history. <sup>b</sup>PM<sub>2.5</sub> moving average was 1 year before death or censor for all-cause deaths, 1–3 years for cardiovascular and lung-cancer deaths, and 1–5 years for COPD deaths. <sup>c</sup>Estimates restricted to participants with the specified chronic condition.

We did not find such an association in our study, which might be due to a lack of statistical power (350 lung-cancer deaths, 26 among never smokers). However, estimated effects of PM<sub>2.5</sub> on all-cause and cardiovascular mortality were also statistically significant (or borderline significant) in never smokers, and higher in current smokers compared to never or former smokers (Table 2).

Regarding COPD mortality, we found a positive but not statistically significant risk of COPD death associated with PM<sub>2.5</sub> exposure. In the ACS cohort, Pope et al. (2004) estimated an unexpected inverse association between PM<sub>2.5</sub> exposure and COPD mortality, whereas Katanoda et al. (2011) estimated an inverse but not statistically significant association between PM<sub>2.5</sub> and COPD in a Japanese cohort.

**Chronic conditions at enrollment and mortality.** The central deposition of particles in lungs has been shown to be enhanced in COPD patients (Bennett et al. 1997). Although PM<sub>2.5</sub> has been associated with early mortality in COPD patients (Zanobetti et al. 2008), and ozone has been associated with early mortality in susceptible subjects (i.e., with COPD, diabetes, heart failure, or myocardial infarction) (Zanobetti and Schwartz 2011), our results did not indicate stronger associations in participants with such chronic conditions at enrollment compared with the population as a whole. This might have been due to a lack of statistical power as few participants had COPD ( $n = 942$ ) or diabetes ( $n = 563$ ) at enrollment.

**Exposure assessment.** Use of outdoor measurements from central monitoring stations as a proxy measure of mean personal exposure to PM<sub>2.5</sub> is prone to measurement error because the measures do not capture fine spatial contrasts that may occur within a city, which may bias the results. Recent reanalyses of the ACS cohort using land use regression models showed that the impact on the PM<sub>2.5</sub>–mortality association was heterogeneous depending on the city (Krewski et al. 2009). However, other recent studies have suggested that considering a more precise exposure model focused on the home address might not improve health effects estimates in terms of bias and variance (Kim et al. 2009; Lepeule et al. 2010; Szpiro et al. 2011). In the Harvard Six Cities study, there were not enough monitors in the cities to implement a land use regression model.

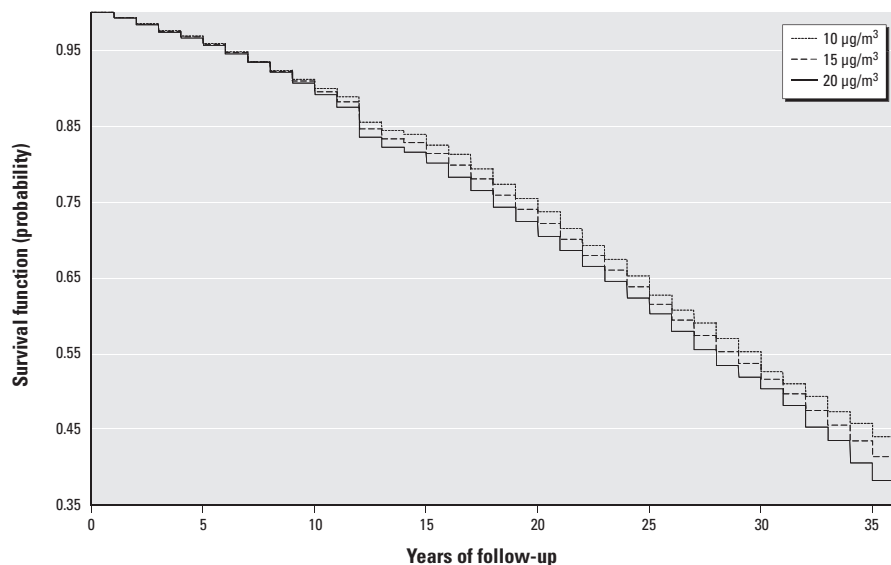
**Strengths and limitations.** Our results were adjusted for baseline factors, but there is potential for residual confounding for risk factors after enrollment and for unmeasured factors such as occupational exposures or medication use if those factors co-vary with PM<sub>2.5</sub>. Some other limitations are that we did not measure PM<sub>2.5</sub> in the same locations throughout the

study period, that death certificates might have listed misclassified specific causes of death, and that hypertension and diabetes were assessed by questionnaire only. An extensive body of methodological work has been performed regarding the sensitivity of estimated associations between long-term exposure to air pollution and mortality, especially for the ACS and Harvard Six Cities study cohorts. More specifically, it has been shown that results were robust to alternative model specifications, alternative metrics of PM<sub>2.5</sub>, and adjustment for individual and ecological risk factors such as occupational exposures and socioeconomic variables (Krewski et al. 2005a, 2005b). It was also shown that using a spatial covariance structure did not change the results (Pope et al. 2002), but with only six locations, that methodology is not applicable in our study. Whereas the primary analysis from the Harvard Six Cities study (Dockery et al. 1993) estimated associations were based on between-city contrasts in exposure, in the current study, with age used as time scale, the exposure relied on both between- and within-city contrasts, limiting the potential for residual cross-sectional confounding. The strengths of the present study are the randomly sampled participants and its extended follow-up through 2009, which included more observations of participants with lower exposures during recent years and provided more statistical power.

**Critical periods of PM<sub>2.5</sub> exposure.** Our results indicated that the best fit moving average for PM<sub>2.5</sub> was 1 year for all-cause mortality. For cardiovascular and lung-cancer mortality, no clear pattern was identified because of the high correlation between PM<sub>2.5</sub> concentrations in the 5 lagged years tested. These results suggest that PM<sub>2.5</sub> exposure can act to promote cardiovascular diseases

and lung-cancer growth, although the design of this study precludes us from determining whether PM<sub>2.5</sub> initiates these diseases as suggested by other studies (Beelen et al. 2008a; Beeson et al. 1998). These results agree with the literature (Gehring et al. 2006; Krewski et al. 2009; Puett et al. 2009; Schwartz et al. 2008) and suggest that health improvements can be expected almost immediately after a reduction in air pollution. This conclusion should be taken into account for cost–benefit analyses related to air pollution standards.

**Role of sulfates and public health implications.** Although RRs for PM<sub>2.5</sub> fluctuated over time, our extended follow-up did not indicate any clear pattern over time during the study period. Between 1979–1988 (Laden et al. 2000) and 2009 (Nehls and Akland 1973), the sulfates/PM<sub>2.5</sub> ratio for exposures measured for the Harvard Six Cities study dropped between 13% and 54%, depending on the city. If sulfates are unrelated to mortality, as some have argued (Grahame and Schlesinger 2005), the elimination of a substantial fraction of nontoxic material from PM<sub>2.5</sub> mass should result in a substantial increase in the PM<sub>2.5</sub> coefficient, which would otherwise have been suppressed by the large fraction of mass that was nontoxic. This was not the case, and hence our results indicate that sulfate particles are about as toxic as the average fine particle. This is consistent with the results of Pope et al. (2007), who found that the 2.5- $\mu\text{g}/\text{m}^3$  decrease in sulfate particle concentrations observed during an 8-month smelters strike were associated with a 2.5% decrease in the number of deaths in the region. In comparison, a 2.5- $\mu\text{g}/\text{m}^3$  decrease in PM<sub>2.5</sub> in our follow-up of the Harvard Six Cities study was associated with a 3.5% reduction in all-cause deaths, but that was for reductions in PM<sub>2.5</sub> lasting at least a year, not



**Figure 2.** Survival probabilities under three hypothetical scenarios: participants of the Harvard Six Cities study are exposed to 10, 15, or 20  $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub> during the entire follow-up period of 1974–2009.

8 months. Given that there were 2,423,712 deaths in the United States in 2007 (Xu et al. 2010) and that the average  $PM_{2.5}$  level was  $11.9 \mu\text{g}/\text{m}^3$  (U.S. EPA 2011), our estimated association between  $PM_{2.5}$  and all-cause mortality implies that a decrease of  $1 \mu\text{g}/\text{m}^3$  in population-average  $PM_{2.5}$  would result in approximately 34,000 fewer deaths per year.

## Conclusion

Including recent observations with  $PM_{2.5}$  exposures well below the U.S. annual standard of  $15 \mu\text{g}/\text{m}^3$  and down to  $8 \mu\text{g}/\text{m}^3$ , the relationship between chronic exposure to  $PM_{2.5}$  and all-cause, cardiovascular, and lung-cancer mortality was found to be linear without a threshold. Our results were not sensitive to various model specifications. Furthermore, estimated effects of  $PM_{2.5}$  did not change over time, suggesting a stable toxicity of  $PM_{2.5}$ , even at lower exposure levels and with a lower sulfates proportion. These results suggest that further public policy efforts that reduce fine particulate matter air pollution are likely to have continuing public health benefits.

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**UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
WASHINGTON D.C. 20460**

March 26, 2007

**OFFICE OF THE ADMINISTRATOR  
SCIENCE ADVISORY BOARD**

**Memorandum**

**SUBJECT:** Formation of Clean Air Scientific Advisory Committee (CASAC)  
NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel

**FROM:** Fred Butterfield */Signed/*  
Designated Federal Officer (DFO)  
Clean Air Scientific Advisory Committee  
EPA Science Advisory Board Staff Office (1400F)

**TO:** Vanessa Vu, Ph.D.  
Staff Director  
EPA Science Advisory Board (1400F)

**VIA:** Daniel Fort */Signed/*  
Ethics & FACA Policy Officer  
EPA Science Advisory Board Staff Office (1400F)

The Clean Air Scientific Advisory Committee (CASAC), which is comprised of seven members appointed by the EPA Administrator, was established under section 109(d)(2) of the Clean Air Act (CAA or Act) (42 U.S.C. 7409) as an independent scientific advisory committee. The CASAC provides advice, information and recommendations on the scientific and technical aspects of air quality criteria and National Ambient Air Quality Standards (NAAQS) under sections 108 and 109 of the Act. The CASAC is a Federal advisory committee chartered under the Federal Advisory Committee Act (FACA), as amended, 5 U.S.C., App. Section 109(d)(1) of the CAA requires that the Agency carry out a periodic review and revision, where appropriate, of the air quality criteria and the NAAQS for "criteria" air pollutants including oxides of nitrogen (NO<sub>x</sub>) and sulfur oxides (SO<sub>x</sub>).

On August 7, 2006 the SAB Staff Office announced in the *Federal Register* (71 FR 44695) the formation of the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel and solicited nominations for experts to supplement the statutory CASAC. This memorandum addresses the set of determinations that were necessary for forming the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel (Panel). Over the next two to three years, the Panel will provide advice and recommendations to the EPA Administrator on the scientific and technical aspects of the primary (health-based) policy-relevant science and the NAAQS for both NO<sub>x</sub> and SO<sub>x</sub>. Specifically, this will involve the Panel's review of EPA's updated draft Integrative Science Assessment (ISA) for NO<sub>x</sub> and SO<sub>x</sub> health effects; and subsequently, as the basis for possible revisions to the primary NAAQS for NO<sub>x</sub> and SO<sub>x</sub>, the Primary NO<sub>x</sub> and SO<sub>x</sub> Risk/Exposure Assessment (RA), and the

Primary NO<sub>x</sub> and SO<sub>x</sub> Policy Assessment (PA) to be published as an advance notice of proposed rulemaking (ANPR). This memo provides background information on this Panel, and addresses the following determinations:

- (A) The type of review body that will be used to conduct the review, the name of the Panel, and identification of the Panel Chair;
- (B) The types of expertise needed to address the general charge;
- (C) Financial conflict of interest considerations, including identification of parties who are potentially interested in or may be affected by the topic to be reviewed;
- (D) How regulations concerning “appearance of a lack of impartiality,” pursuant to 5 C.F.R. § 2635.502 apply to members of the Panel; and
- (E) How individuals were selected for the Panel.

#### **DETERMINATIONS:**

- (A) The type of review body that will be used to conduct the review, the name of the Panel, and identification of the Panel Chair.

The CASAC, augmented by additional subject-matter experts, known collectively as the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel, will conduct the ongoing review of, and offer advice and recommendations to the EPA Administrator concerning, the primary (health-based NAAQS for NO<sub>x</sub> and SO<sub>x</sub>. Dr. Rogene Henderson, the CASAC Chair, will chair this Panel. The CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel will comply with the provisions of FACA and all appropriate SAB Staff Office procedural policies.

- (B) The types of expertise needed to address the general charge.

Per the solicitation for nominees to form the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel that was published in the *Federal Register* on August 7, 2006 (see citation above), recognized, national-level experts were sought in one or more of the following six (6) disciplines to augment the expertise of the statutory CASAC:

(a) Atmospheric Science. Expertise in physical/chemical properties of nitrogen oxides and sulfur oxides and atmospheric processes involved in the formation, transport on urban to global scales, transformation of these pollutants in the atmosphere, and movement of the pollutants between media through deposition and other such mechanisms. Also, expertise in the evaluation of natural and anthropogenic sources and emissions of nitrogen oxides and sulfur oxides and resulting ambient levels due to natural sources, pertinent monitoring or measurement methods for these pollutants, and spatial and temporal trends in their atmospheric concentrations.

(b) Exposure and Risk Assessment/Modeling. Expertise in measuring human population exposure to nitrogen oxides and sulfur oxides and/or in modeling human population exposure to pollutants from ambient and indoor sources. Expertise in human health risk analysis modeling for nitrogen oxides and sulfur oxides related to respiratory and other non-cancer health effects.

(c) Dosimetry. Expertise in evaluation of the dosimetry of animal and human subjects, including identification of factors determining differential patterns of inhalation and/or deposition/uptake in respiratory tract regions that may contribute to differential susceptibility of human population subgroups and animal-to-human dosimetry extrapolations.

(d) Toxicology. Expertise in evaluation of experimental laboratory animal studies and *in vitro* studies of the effects of sulfur oxides and/or oxides of nitrogen on pulmonary and extra-pulmonary (*e.g.*, cardiovascular, immunological) endpoints.

(e) Controlled Human Exposure. Expertise in evaluations of controlled human exposure studies of the effects of nitrogen oxides and sulfur oxides on health and compromised (*e.g.*, having pertinent preexisting disease such as asthma) human adults and children, including physicians with experience in the clinical treatment of asthma and chronic lung diseases.

(f) Epidemiology and Biostatistics. Expertise in epidemiologic evaluation of the effects of exposures to ambient nitrogen oxides and sulfur oxides and/or other major air pollutants (*e.g.*, particulate matter, ozone, carbon monoxide) on human population groups, including mortality and morbidity effects (*e.g.*, respiratory symptoms, lung function decrements, asthma medication use, emergency department visits, respiratory-related hospital admissions). Also, expertise in associated biostatistics and/or health risk analysis.

(C) Financial conflict of interest considerations, including identification of parties who are potentially interested in or may be affected by the topic to be reviewed.

(a) Identification of parties who are potentially interested in or may be affected by the topic to be reviewed: The principal interested and affected parties for this topic are: (1) EPA; (2) State, regional and local air program (or air pollution control) agencies, and State regulatory officials; (3) State and local health officials; (4) research universities; (5) environmental interest groups/non-Governmental organizations (NGOs); (6) potentially responsible parties (PRP) and their contractors; and (7) various industry sectors interested in, or affected by, the current or any revised NAAQS for NO<sub>x</sub> and SO<sub>x</sub>, including the power-generating and automotive industries.

(b) Conflict of interest considerations: For Financial Conflict of Interest (COI) issues, the basic 18 U.S.C. § 208 provision states that: “An employee is prohibited from participating *personally and substantially* in an official capacity in any *particular matter* in which he, to his knowledge, or any person whose interests are imputed to him under this statute has a *financial interest*, if the particular matter will have a direct and predictable effect on that interest [emphasis added].” For a conflict of interest to be present, all elements in the above provision must be present. If an element is missing the issue does not involve a formal conflict of interest; however, the general provisions in the appearance of impartiality guidelines must still apply and need to be considered.

(i) Does the general charge to the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel involve a particular matter? A “particular matter” refers to matters that “...will involve deliberation, decision, or action that is focused upon the interests of specific people, or a discrete and identifiable class of people.” It does not refer to “...consideration or adoption of broad policy options directed to the interests of a large and diverse group of people.” [5 C.F.R. § 2640.103 (a)(1)]. A particular matter of general applicability means a particular matter that is

focused on the interests of a discrete and identifiable class of persons, but does not involve specific parties. [5 C.F.R. § 2640.102 (m)].

The CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel's activity in addressing the charge for the peer review of the draft NO<sub>x</sub> and SO<sub>x</sub> Primary ISA, RA and PA and related technical support documents will qualify as a *particular matter of general applicability* because the resulting advice will be part of a deliberation, and under certain circumstances the advice could involve the interests of a discrete and identifiable class of people but does not involve specific parties. That group of people constitutes those who are associated or involved with the potentially interested or affected parties, as identified in Section (3)(a) above.

(ii) Will there be personal and substantial participation on the part of Panel members? Participating personally means direct participation in this review. Participating substantially refers to involvement that is of significance to the matter under consideration. [5 C.F.R. § 2640.103(a)(2)]. For this review, the EPA Science Advisory Board (SAB) Staff Office has determined that *CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel members will be participating personally in the matter*. Panel members will be providing the Agency with advice and recommendations that is expected to include an assessment as to whether the proposed air quality criteria (by means of the ISA) accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of these pollutants (that is, NO<sub>x</sub> or SO<sub>x</sub>) in the ambient air. *Therefore, participation in this review will also be substantial.*

(iii) Will there be a direct and predictable effect on CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel members' financial interest? A direct effect on a participant's financial interest exists if "...a close causal link exists between any decision or action to be taken in the matter and any expected effect of the matter on the financial interest. ...A particular matter does not have a direct effect ...if the chain of causation is attenuated or is contingent upon the occurrence of events that are speculative or that are independent of, and unrelated to, the matter. A particular matter that has an effect on a financial interest only as a consequence of its effects on the general economy is not considered to have a direct effect." [5 C.F.R. § 2640.103(a)(i)] A predictable effect exists if, "...there is an actual, as opposed to a speculative, possibility that the matter will affect the financial interest." [5 C.F.R. § 2640.103(a)(ii)]

(D) How regulations concerning "appearance of a lack of impartiality," pursuant to 5 C.F.R. § 2635.502, apply to members of the Panel.

The Code of Federal Regulations at 5 C.F.R. § 2635.502(a) states that: "Where an employee knows that a particular matter involving specific parties is likely to have a direct and predictable effect on the financial interest of a member of his household, or knows that a person with whom he has a covered relationship is or represents a party to such matter, and where the person determines that the circumstances would cause a reasonable party to such matter, and where the person determines that the circumstances would cause a reasonable person with knowledge of the relevant facts to question his impartiality in the matter, the employee should not participate in the matter unless he has informed the agency designee of the appearance problem and received authorization from the agency designee." Further, § 2635.502(a)(2) states that, "An employee who is concerned that circumstances other than those specifically described

in this section would raise a question regarding his impartiality should use the process described in this section to determine whether he should or should not participate in a particular matter.”

To ascertain whether there is any appearance of a lack of impartiality, the following five questions will be posed to each member of CASAC and prospective members of the NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel with respect to the forthcoming charge for the Panel:

(a) Do you know of any reason that you might be unable to provide impartial advice on the matter to come before the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel or any reason that your impartiality in the matter might be questioned?

(b) Have you had any previous involvement with the review document(s) under consideration, *i.e.*, EPA’s 1<sup>st</sup> Draft NO<sub>x</sub> and SO<sub>x</sub> Primary Integrated Science assessment — including authorship, collaboration with the authors, or previous peer review functions? If so, please identify and describe that involvement.

(c) Have you served on previous advisory panels, committees or subcommittees (Federal or otherwise) that have addressed the topic under consideration? If so please identify those activities.

(d) Have you made any public statements (written or oral) on the issue? If so, please identify those statements.

(e) Have you made any public statements that would indicate to an observer that you have taken a position on the issue under consideration? If so, please identify those statements.

(E) How individuals were selected for the Panel.

As described in Section (B) above, the SAB Staff Office announced the formation of the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel in the *Federal Register* (71 FR 44695) on August 7, 2006, and requested nominations for recognized, national-level experts in one or more of the six scientific/technical expertise areas delineated in Section (B) above to augment the expertise of the statutory CASAC. In response to that solicitation, as well as from other sources, the SAB Staff Office identified 27 experts for the Panel’s “Short List.”

In December 2006, the SAB Staff Office published the “Short List” in the form of an “Invitation for Comments” memorandum and posted this on the Panel’s page on the SAB Web site at: [http://www.epa.gov/sab/panels/casac\\_nox\\_and\\_sox\\_primary\\_panel.htm](http://www.epa.gov/sab/panels/casac_nox_and_sox_primary_panel.htm). The purpose of the “Invitation for Comments” memo is to solicit comments from members of the public or the Agency with respect to any relevant information or other documentation that the SAB Staff Office should consider in the final selection of this Panel.

The SAB Staff Office received two (2) public comments on this Short List from individuals representing the following organizations (date):

- Clean Air Watch (January 12, 2007)
- Center for Science in the Public Interest (CSPI) (January 17, 2007)

The SAB Staff Office Director makes the final decision about who serves on the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel, based on all relevant information. This includes a review

of the member's confidential financial disclosure form (EPA Form 3110-48) and an evaluation of a lack of impartiality. For the EPA SAB Staff Office, a balanced committee or panel is characterized by inclusion of candidates who possess the necessary domains of knowledge, the relevant scientific perspectives (which, among other factors, can be influenced by work history and affiliation), and the collective breadth of experience to adequately address the general charge. Specific criteria to be used in evaluating an individual Panel member include: (a) scientific and/or technical expertise, knowledge, and experience (primary factors); (b) availability and willingness to serve; (c) absence of financial conflicts of interest; (d) absence of an appearance of a lack of impartiality; and (e) skills working in committees, subcommittees and advisory panels; and, for the Panel as a whole, (f) diversity of, and balance among, scientific expertise, viewpoints, *etc.*

*On the basis of the above-specified criteria, the CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Review Panel is as follows:*

*Members of the statutory (chartered) Clean Air Scientific Advisory Committee:*

1. **Dr. Rogene Henderson**, Lovelace Respiratory Research Institute (NM) – CASAC Chair
2. **Dr. Ellis Cowling**, North Carolina State University (NC)
3. **Dr. James D. Crapo**, National Jewish Medical and Research Center (CO)
4. **Dr. Douglas Crawford-Brown**, University of North Carolina at Chapel Hill (NC)
5. **Mr. Richard L. Poirot**, Vermont Agency of Natural Resources (VT)
6. **Dr. Armistead (Ted) Russell**, Georgia Institute of Technology (GA)
7. **Dr. Frank Speizer**, Harvard Medical School (MA)

*Additional CASAC NO<sub>x</sub> and SO<sub>x</sub> Primary Panel members:*

1. **Dr. Ed Avol**, University of Southern California (CA)
2. **Dr. John Balmes**, University of California, San Francisco (CA)
3. **Dr. Henry Gong**, University of Southern California (CA)
4. **Dr. Terry Gordon**, New York University School of Medicine (NY)
5. **Dr. Dale Hattis**, Clark University (MA)
6. **Dr. Patrick Kinney**, Columbia University, Mailman School of Public Health (NY)
7. **Dr. Steven Kleeberger**, National Institute of Environmental Health Sciences (NC)
8. **Dr. Timothy Larson**, University of Washington (WA)
9. **Dr. Kent E. Pinkerton**, University of California, Davis (CA)
10. **Dr. Edward M. Postlethwait**, University of Alabama at Birmingham (AL)
11. **Dr. Richard B. Schlesinger**, Pace University (NY)
12. **Dr. Christian Seigneur**, Atmospheric & Environmental Research, Inc. (CA)
13. **Dr. Elizabeth A. (Lianne) Sheppard**, University of Washington (WA)
14. **Dr. George Thurston**, New York University School of Medicine (NY)
15. **Dr. James Ultman**, Pennsylvania State University (Emeritus) (PA)
16. **Dr. Ronald Wyzga**, Electric Power Research Institute (CA)

Concurred:

*/Signed/*

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Vanessa T. Vu, Ph.D.  
Staff Director  
EPA Science Advisory Board (1400F)

*April 2, 2007*

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Date



# Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter





EPA-452/R-20-002  
January 2020

Policy Assessment for the Review of the National Ambient Air Quality Standards for  
Particulate Matter

U.S. Environmental Protection Agency  
Office of Air Quality Planning and Standards  
Health and Environmental Impacts Division  
Research Triangle Park, NC

## **DISCLAIMER**

This Policy Assessment has been prepared by staff in the U.S. Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards. Any findings and conclusions are those of the authors and do not necessarily reflect the views of the EPA. Questions or comments related to this document should be addressed to Dr. Scott Jenkins, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C539-06, Research Triangle Park, North Carolina 27711 (email: [jenkins.scott@epa.gov](mailto:jenkins.scott@epa.gov)).

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## **LIST OF ACRONYMS AND ABBREVIATIONS**

AAMS	Ambient Air Monitoring Subcommittee
ACS	American Cancer Society
AMTIC	Ambient Monitoring Technology Information Center
APEX	Air Pollutants Exposure model
AQCD	Air Quality Criteria Document
AQI	Air Quality Index
AQS	Air Quality System
ATUS	American Time Use Survey
BC	Black carbon
BenMAP-CE	Environmental Benefits Mapping and Analysis Program – Community Edition
CAA	Clean Air Act
CASAC	Clean Air Scientific Advisory Committee
CBSA	Core-based statistical area
CHAD	Consolidated Human Activity Database
CPL	Candidate protection level
C-R	Concentration-response
CSN	Chemical Speciation Network
dv	Deciview
EC	Elemental carbon
U.S. EPA	United States Environmental Protection Agency
FEM	Federal Equivalent Method
FR	Federal Register
FRM	Federal Reference Method
HERO	Health and Environmental Research Online
HREA	Health Risk and Exposure Assessment
IARC	International Agency for Research on Cancer
IHD	Ischemic heart disease
IMPROVE	Interagency Monitoring of Protected Visual Environments
IPCC	Intergovernmental Panel on Climate Change
IRP	Integrated Review Plan
ISA	Integrated Science Assessment
LML	Lowest measured level
Mm-1	Megameters
N	Nitrogen
NAAQS	National Ambient Air Quality Standards

NATTS	National Air Toxics 1 Trends Stations
NCEA	National Center for Environmental Assessment
NCore	National Core
NO <sub>2</sub>	Nitrogen dioxide
NO <sub>x</sub>	Oxides of nitrogen
O <sub>3</sub>	Ozone
OAR	Office of Air and Radiation
OAQPS	Office of Air Quality Planning and Standards
OC	Organic carbon
OMB	Office of Management and Budget
ORD	Office of Research and Development
PA	Policy Assessment
PM	Particulate matter
PM <sub>2.5</sub>	<p>In general terms, particulate matter with an aerodynamic diameter less than or equal to a nominal 2.5 µm; a measurement of fine particles</p> <p>In regulatory terms, particles with an upper 50% cut-point of 2.5 µm aerodynamic diameter (the 50% cut point diameter is the diameter at which the sampler collects 50% of the particles and rejects 50% of the particles) and a penetration curve as measured by a reference method based on Appendix L of 40 CFR Part 50 and designated in accordance with 40 CFR Part 53, by an equivalent method designated in accordance with 40 CFR Part 53, or by an approved regional method designated in accordance with Appendix C of 40 CFR Part 58</p>
PM <sub>10</sub>	<p>In general terms, particulate matter with an aerodynamic diameter less than or equal to a nominal 10 µm; a measurement of thoracic particles (i.e., that subset of inhalable particles thought small enough to penetrate beyond the larynx into the thoracic region of the respiratory tract.</p> <p>In regulatory terms, particles with an upper 50% cut-point of 10 ± 0.5 µm aerodynamic diameter (the 50% cut point diameter is the diameter at which the sampler collects 50% of the particles and rejects 50% of the particles) and a penetration curve as measured by a reference method based on Appendix J of 40 CFR Part 50 and designated in accordance with 40 CFR Part 53, or by an equivalent method designated in accordance with 40 CFR Part 53</p>
PM <sub>10-2.5</sub>	<p>In general terms, particulate matter with an aerodynamic diameter less than or equal to a nominal 10 µm and greater than a nominal 2.5 µm; a measurement of thoracic particles or the coarse fraction of PM<sub>10</sub></p> <p>In regulatory terms, particles with an upper 50% cut-point of 10 µm aerodynamic diameter and a lower 50% cut-point of 2.5 µm aerodynamic diameter (the 50% cut point diameter is the diameter at which the sampler collects 50% of the particles and rejects 50% of the particles) as measured by a reference method based on Appendix O of 40 CFR Part 50 and designated in accordance with 40 CFR Part 53, or by an equivalent method designated in accordance with 40 CFR Part 53</p>

PRB	Policy relevant background
QA	Quality assurance
QMP	Quality Management Plan
REA	Risk and Exposure Assessment
RIA	Regulatory impact analysis
S	Sulfur
SES	Socioeconomic status
SIP	State Implementation Plan
SLAMS	State and Local Air Monitoring Stations
SO <sub>2</sub>	Sulfur dioxide
SO <sub>x</sub>	Sulfur oxides
SOPM	Secondary Organic Particulate Matter
STN	Speciation Trends Network
TAD	Technical Assistance Document
TRIM	Total Risk Integrated Methodology
TSP	Total Suspended Particles
UFP	Ultrafine Particles: Generally considered as particulates with a diameter less than or equal to 0.1 µm, typically based on physical size, thermal diffusivity or electrical mobility
UFVA	Urban-Focused Visibility Assessment
VAQ	Visual air quality
VOC	Volatile organic compound
WHO	World Health Organization
WREA	Welfare Risk and Exposure Assessment

# 1 INTRODUCTION

This document, *Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter* (hereafter referred to as the PA), presents the policy assessment for the U.S. Environmental Protection Agency's (EPA's) current review of the national ambient air quality standards (NAAQS) for particulate matter (PM). The overall plan for this review was presented in the *Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter* (IRP; U.S. EPA, 2016). The IRP also identified key policy-relevant issues to be addressed in this review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA) and a PA.

This document is organized into five chapters. Chapter 1 presents introductory information on the purpose of the PA, legislative requirements for reviews of the NAAQS, an overview of the history of the PM NAAQS, including background information on prior reviews, and a summary of the progress to date for the current review. Chapter 2 provides an overview of the available information on PM-related emissions, atmospheric chemistry, monitoring and air quality. Chapters 3 and 4 focus on policy-relevant aspects of the currently available health effects evidence and exposure/risk information, identifying and summarizing key considerations related to this review of the primary standards for PM<sub>2.5</sub> and PM<sub>10</sub>, respectively. Chapter 5 focuses on policy-relevant aspects of the currently available welfare evidence and associated quantitative analyses, identifying and summarizing key considerations related to this review of the PM secondary standards.<sup>1</sup>

## 1.1 PURPOSE

The PA evaluates the potential policy implications of the available scientific evidence, as assessed in the ISA, and the potential implications of the available air quality, exposure or risk analyses. The role of the PA is to help “bridge the gap” between the Agency's scientific assessments and quantitative technical analyses, and the judgments required of the Administrator in determining whether it is appropriate to retain or revise the NAAQS.

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<sup>1</sup> The welfare effects considered in this review include visibility impairment, climate effects, and materials effects (i.e., damage and soiling). Ecological effects associated with PM, and the adequacy of protection provided by the secondary PM standards for them, are being addressed in the separate review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM in recognition of the linkages between oxides of nitrogen, oxides of sulfur, and PM with respect to atmospheric chemistry and deposition, and with respect to ecological effects. Information on the current review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM can be found at <https://www.epa.gov/naaqs/nitrogen-dioxide-no2-and-sulfur-dioxide-so2-secondary-air-quality-standards>.

In evaluating the question of adequacy of the current standards, and whether it may be appropriate to consider alternative standards, the PA focuses on information that is most pertinent to evaluating the standards and their basic elements: indicator, averaging time, form, and level.<sup>2</sup> These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection the standards afford.

The PA is also intended to facilitate advice to the Agency and recommendations to the Administrator from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act (CAA). As discussed below in section 1.2, the CASAC is to advise on subjects including the Agency's assessment of the relevant scientific information and on the adequacy of the current standards, and to make recommendations as to any revisions of the standards that may be appropriate. The EPA generally makes available to the CASAC and the public one or more drafts of the PA for CASAC review and public comment.

In this PA, we<sup>3</sup> take into account the available scientific evidence, as assessed in the *Integrated Science Assessment for Particulate Matter (Final Report)* (ISA [U.S. EPA, 2019]), and additional policy-relevant analyses of air quality and risks. Our approach to considering the available evidence and analyses in this PA has been informed by the advice received from the CASAC, based on its review of the draft IRP and the draft ISA, and also by public comment received thus far in the review. This final PA is also informed by the advice and recommendations received from the CASAC during its review of the draft PA, and also by public comments received. The final PA is intended to help the Administrator in considering the currently available scientific and technical information, and in formulating judgments regarding the adequacy of the current standards and regarding alternative standards, as appropriate.

Beyond informing the Administrator and facilitating the advice and recommendations of the CASAC, the PA is also intended to be a useful reference to all parties interested in the review of the PM NAAQS. In these roles, it is intended to serve as a source of policy-relevant information that informs the Agency's review of the NAAQS for PM, and it is written to be understandable to a broad audience.

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<sup>2</sup> The indicator defines the chemical species or mixture to be measured in the ambient air for the purpose of determining whether an area attains the standard. The averaging time defines the period over which air quality measurements are to be averaged or otherwise analyzed. The form of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. For example, the form of the annual NAAQS for fine particulate matter is the average of annual mean concentrations for three consecutive years, while the form of the 8-hour NAAQS for carbon monoxide is the second-highest 8-hour average in a year. The level of the standard defines the air quality concentration used for that purpose.

<sup>3</sup> The terms "we," "our," and "staff" throughout this document refer to the staff in the EPA's Office of Air Quality Planning and Standards (OAQPS).



## 1.2 LEGISLATIVE REQUIREMENTS

Two sections of the Clean Air Act (CAA) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those pollutants “emissions of which, in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare”; “the presence of which in the ambient air results from numerous or diverse mobile or stationary sources”; and for which he “plans to issue air quality criteria...” (42 U.S.C. § 7408(a)(1)). Air quality criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air...” 42 U.S.C. § 7408(a)(2).

Section 109 [42 U.S.C. 7409] directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued [42 U.S.C. § 7409(a)]. Section 109(b)(1) defines primary standards as ones “the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”<sup>4</sup> Under section 109(b)(2), a secondary standard must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”<sup>5</sup>

In setting primary and secondary standards that are “requisite” to protect public health and welfare, respectively, as provided in section 109(b), the EPA’s task is to establish standards that are neither more nor less stringent than necessary. In so doing, the EPA may not consider the costs of implementing the standards. See generally, *Whitman v. American Trucking Associations*, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, “[a]ttainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards.” *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1185 (D.C. Cir. 1981). At the same time, courts have clarified the EPA may consider “relative proximity to peak background ... concentrations” as a factor in deciding how to revise the NAAQS in the context of considering

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<sup>4</sup> The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

<sup>5</sup> Under CAA section 302(h) (42 U.S.C. § 7602(h)), effects on welfare include, but are not limited to, “effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”

standard levels within the range of reasonable values supported by the air quality criteria and judgments of the Administrator. *American Trucking Associations, Inc. v. EPA*, 283 F.3d 355, 379 (D.C. Cir. 2002).

The requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. See *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d at 1186 (D.C. Cir. 1981), *cert. denied*, 455 U.S. 1034 (1982); *Coalition of Battery Recyclers Ass'n v. EPA*, 604 F.3d 613, 617-18 (D.C. Cir. 2010); *Mississippi v. EPA*, 744 F.3d 1334, 1353 (D.C. Cir. 2013). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51, *Mississippi v. EPA*, 744 F.3d at 1351, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s), and the kind and degree of uncertainties. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62; *Mississippi v. EPA*, 744 F.3d at 1353.

Section 109(d)(1) of the Act requires a review be completed every five years and, if appropriate, revision of existing air quality criteria to reflect advances in scientific knowledge on the effects of the pollutant on public health and welfare. Under the same provision, the EPA is also to review every five years and, if appropriate, revise the NAAQS, based on the revised air quality criteria.<sup>6</sup>

Section 109(d)(2) addresses the appointment and advisory functions of an independent scientific review committee. Section 109(d)(2)(A) requires the Administrator to appoint this

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<sup>6</sup> This section of the Act requires the Administrator to complete these reviews and make any revisions that may be appropriate "at five-year intervals."

committee, which is to be composed of “seven members including at least one member of the National Academy of Sciences, one physician, and one person representing State air pollution control agencies.” Section 109(d)(2)(B) provides that the independent scientific review committee “shall complete a review of the criteria...and the national primary and secondary ambient air quality standards...and shall recommend to the Administrator any new...standards and revisions of existing criteria and standards as may be appropriate....” Since the early 1980s, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of the EPA’s Science Advisory Board. A number of other advisory functions are also identified for the committee by section 109(d)(2)(C), which reads:

Such committee shall also (i) advise the Administrator of areas in which additional knowledge is required to appraise the adequacy and basis of existing, new, or revised national ambient air quality standards, (ii) describe the research efforts necessary to provide the required information, (iii) advise the Administrator on the relative contribution to air pollution concentrations of natural as well as anthropogenic activity, and (iv) advise the Administrator of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards.

As previously noted, the Supreme Court has held that section 109(b) “unambiguously bars cost considerations from the NAAQS-setting process” (*Whitman v. Am. Trucking Associations*, 531 U.S. 457, 471 [2001]). Accordingly, while some of these issues regarding which Congress has directed the CASAC to advise the Administrator are ones that are relevant to the standard setting process, others are not. Issues that are not relevant to standard setting may be relevant to implementation of the NAAQS once they are established.<sup>7</sup>

### **1.3 HISTORY OF REVIEWS OF THE PM NAAQS**

This section summarizes the PM NAAQS that have been promulgated in past reviews (Table 1-1). Each of these reviews is discussed briefly below.

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<sup>7</sup> Some aspects of CASAC advice may not be relevant to EPA’s process of setting primary and secondary standards that are requisite to protect public health and welfare. Indeed, were EPA to consider costs of implementation when reviewing and revising the standards “it would be grounds for vacating the NAAQS.” *Whitman*, 531 U.S. at 471 n.4. At the same time, the Clean Air Act directs CASAC to provide advice on “any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance” of the NAAQS to the Administrator under section 109(d)(2)(C)(iv). In *Whitman*, the Court clarified that most of that advice would be relevant to implementation but not standard setting, as it “enable[s] the Administrator to assist the States in carrying out their statutory role as primary *implementers* of the NAAQS.” *Id.* at 470 (emphasis in original). However, the Court also noted that CASAC’s “advice concerning certain aspects of ‘adverse public health ... effects’ from various attainment strategies is unquestionably pertinent” to the NAAQS rulemaking record and relevant to the standard setting process. *Id.* at 470 n.2.

**Table 1-1. Summary of NAAQS promulgated for particulate matter 1971-2012.**

Review Completed	Indicator	Averaging Time	Level	Form
1971	Total Suspended Particles (TSP)	24-hour	260 µg/m <sup>3</sup> (primary) 150 µg/m <sup>3</sup> (secondary)	Not to be exceeded more than once per year
		Annual	75 µg/m <sup>3</sup> (primary) 60 µg/m <sup>3</sup> (secondary)	Annual geometric mean
1987	PM <sub>10</sub>	24-hour	150 µg/m <sup>3</sup>	Not to be exceeded more than once per year on average over a 3-year period
		Annual	50 µg/m <sup>3</sup>	Annual arithmetic mean, averaged over 3 years
1997	PM <sub>2.5</sub>	24-hour	65 µg/m <sup>3</sup>	98 <sup>th</sup> percentile, averaged over 3 years
		Annual	15.0 µg/m <sup>3</sup>	Annual arithmetic mean, averaged over 3 years <sup>a</sup>
	PM <sub>10</sub>	24-hour	150 µg/m <sup>3</sup>	99 <sup>th</sup> percentile, averaged over 3 years <sup>b</sup>
		Annual	50 µg/m <sup>3</sup>	Annual arithmetic mean, averaged over 3 years
2006	PM <sub>2.5</sub>	24-hour	35 µg/m <sup>3</sup>	98 <sup>th</sup> percentile, averaged over 3 years
		Annual	15.0 µg/m <sup>3</sup>	Annual arithmetic mean, averaged over 3 years <sup>c</sup>
	PM <sub>10</sub>	24-hour <sup>d</sup>	150 µg/m <sup>3</sup>	Not to be exceed more than once per year on average over a 3-year period
2012	PM <sub>2.5</sub>	24-hour	35 µg/m <sup>3</sup>	98 <sup>th</sup> percentile, averaged over 3 years
		Annual	12.0 µg/m <sup>3</sup> (primary) 15.0 µg/m <sup>3</sup> (secondary)	Annual mean, averaged over 3 years <sup>e</sup>
	PM <sub>10</sub>	24-hour	150 µg/m <sup>3</sup>	Not to be exceeded more than once per year on average over 3 years
<p>Note: When not specified, primary and secondary standards are identical.</p> <p><sup>a</sup> The level of the 1997 annual PM<sub>2.5</sub> standard was to be compared to measurements made at the community-oriented monitoring site recording the highest concentration or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged (i.e., “spatial averaging”) (62 FR 38652, July 18, 1997).</p> <p><sup>b</sup> When the 1997 standards were vacated (see below), the form of the 1987 standards remained in place (i.e., not to be exceeded more than once per year on average over a 3-year period).</p> <p><sup>c</sup> The EPA tightened the constraints on the spatial averaging criteria by further limiting the conditions under which some areas may average measurements from multiple community-oriented monitors to determine compliance (71 FR 61144, October 17, 2006).</p> <p><sup>d</sup> The EPA revoked the annual PM<sub>10</sub> NAAQS in 2006 (71 FR 61144, October 17, 2006).</p> <p><sup>e</sup> In the 2012 decision, the EPA eliminated the option for spatial averaging (78 FR 3086, January 15, 2013).</p>				

### 1.3.1 Reviews Completed in 1971 and 1987

The EPA first established NAAQS for PM in 1971 (36 FR 8186, April 30, 1971), based on the original Air Quality Criteria Document (AQCD) (DHEW, 1969).<sup>8</sup> The federal reference method (FRM) specified for determining attainment of the original standards was the high-volume sampler, which collects PM up to a nominal size of 25 to 45 micrometers ( $\mu\text{m}$ ) (referred to as total suspended particulates or TSP). The primary standards were set at  $260 \mu\text{g}/\text{m}^3$ , 24-hour average, not to be exceeded more than once per year, and  $75 \mu\text{g}/\text{m}^3$ , annual geometric mean. The secondary standards were set at  $150 \mu\text{g}/\text{m}^3$ , 24-hour average, not to be exceeded more than once per year, and  $60 \mu\text{g}/\text{m}^3$ , annual geometric mean.

In October 1979 (44 FR 56730, October 2, 1979), the EPA announced the first periodic review of the air quality criteria and NAAQS for PM. Revised primary and secondary standards were promulgated in 1987 (52 FR 24634, July 1, 1987). In the 1987 decision, the EPA changed the indicator for particles from TSP to  $\text{PM}_{10}$ , in order to focus on the subset of inhalable particles small enough to penetrate to the thoracic region of the respiratory tract (including the tracheobronchial and alveolar regions), referred to as thoracic particles.<sup>9</sup> The level of the 24-hour standards (primary and secondary) was set at  $150 \mu\text{g}/\text{m}^3$ , and the form was one expected exceedance per year, on average over three years. The level of the annual standards (primary and secondary) was set at  $50 \mu\text{g}/\text{m}^3$ , and the form was annual arithmetic mean, averaged over three years.

### 1.3.2 Review Completed in 1997

In April 1994, the EPA announced its plans for the second periodic review of the air quality criteria and NAAQS for PM, and in 1997 the EPA promulgated revisions to the NAAQS (62 FR 38652, July 18, 1997). In the 1997 decision, the EPA determined that the fine and coarse fractions of  $\text{PM}_{10}$  should be considered separately. This determination was based on evidence that serious health effects were associated with short- and long-term exposures to fine particles in areas that met the existing  $\text{PM}_{10}$  standards. The EPA added new standards, using  $\text{PM}_{2.5}$  as the indicator for fine particles (with  $\text{PM}_{2.5}$  referring to particles with a nominal mean aerodynamic diameter less than or equal to  $2.5 \mu\text{m}$ ). The new primary standards were as follows: (1) an annual standard with a level of  $15.0 \mu\text{g}/\text{m}^3$ , based on the 3-year average of annual arithmetic mean

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<sup>8</sup> Prior to the review initiated in 2007 (see below), the AQCD provided the scientific foundation (i.e., the air quality criteria) for the NAAQS. Beginning in that review, the Integrated Science Assessment (ISA) has replaced the AQCD.

<sup>9</sup>  $\text{PM}_{10}$  refers to particles with a nominal mean aerodynamic diameter less than or equal to  $10 \mu\text{m}$ . More specifically,  $10 \mu\text{m}$  is the aerodynamic diameter for which the efficiency of particle collection is 50 percent.

PM<sub>2.5</sub> concentrations from single or multiple community-oriented monitors;<sup>10</sup> and (2) a 24-hour standard with a level of 65 µg/m<sup>3</sup>, based on the 3-year average of the 98<sup>th</sup> percentile of 24-hour PM<sub>2.5</sub> concentrations at each monitor within an area. Also, the EPA established a new reference method for the measurement of PM<sub>2.5</sub> in the ambient air and adopted rules for determining attainment of the new standards. To continue to address the health effects of the coarse fraction of PM<sub>10</sub> (referred to as thoracic coarse particles or PM<sub>10-2.5</sub>; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 µm and less than or equal to 10 µm), the EPA retained the annual primary PM<sub>10</sub> standard and revised the form of the 24-hour primary PM<sub>10</sub> standard to be based on the 99<sup>th</sup> percentile of 24-hour PM<sub>10</sub> concentrations at each monitor in an area. The EPA revised the secondary standards by setting them equal in all respects to the newly established primary standards.

Following promulgation of the 1997 PM NAAQS, petitions for review were filed by several parties, addressing a broad range of issues. In May 1999, the U.S. Court of Appeals for the District of Columbia Circuit (D.C. Circuit) upheld the EPA's decision to establish fine particle standards, holding that "the growing empirical evidence demonstrating a relationship between fine particle pollution and adverse health effects amply justifies establishment of new fine particle standards." *American Trucking Associations v. EPA*, 175 F. 3d at 1027, 1055-56 (D.C. Cir. 1999). The D.C. Circuit also found "ample support" for the EPA's decision to regulate coarse particle pollution, but vacated the 1997 PM<sub>10</sub> standards, concluding that the EPA had not provided a reasonable explanation justifying use of PM<sub>10</sub> as an indicator for coarse particles. *American Trucking Associations v. EPA*, 175 F. 3d at 1054-55. Pursuant to the D.C. Circuit's decision, the EPA removed the vacated 1997 PM<sub>10</sub> standards, and the pre-existing 1987 PM<sub>10</sub> standards remained in place (65 FR 80776, December 22, 2000). The D.C. Circuit also upheld the EPA's determination not to establish more stringent secondary standards for fine particles to address effects on visibility. *American Trucking Associations v. EPA*, 175 F. 3d at 1027.

The D.C. Circuit also addressed more general issues related to the NAAQS, including issues related to the consideration of costs in setting NAAQS and the EPA's approach to establishing the levels of NAAQS. Regarding the cost issue, the court reaffirmed prior rulings holding that in setting NAAQS the EPA is "not permitted to consider the cost of implementing those standards." *American Trucking Associations v. EPA*, 175 F. 3d at 1040-41. Regarding the

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<sup>10</sup> The 1997 annual PM<sub>2.5</sub> standard was to be compared with measurements made at the community-oriented monitoring site recording the highest concentration or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged (i.e., "spatial averaging"). In the last review (completed in 2012) the EPA replaced the term "community-oriented" monitor with the term "area-wide" monitor. Area-wide monitors are those sited at the neighborhood scale or larger, as well as those monitors sited at micro- or middle-scales that are representative of many such locations in the same CBSA (78 FR 3236, January 15, 2013).

levels of NAAQS, the court held that the EPA's approach to establishing the level of the standards in 1997 (i.e., both for PM and for the ozone NAAQS promulgated on the same day) effected "an unconstitutional delegation of legislative authority." *American Trucking Associations v. EPA*, 175 F. 3d at 1034-40. Although the court stated that "the factors EPA uses in determining the degree of public health concern associated with different levels of ozone and PM are reasonable," it remanded the rule to the EPA, stating that when the EPA considers these factors for potential non-threshold pollutants "what EPA lacks is any determinate criterion for drawing lines" to determine where the standards should be set.

The D.C. Circuit's holding on the cost and constitutional issues were appealed to the United States Supreme Court. In February 2001, the Supreme Court issued a unanimous decision upholding the EPA's position on both the cost and constitutional issues. *Whitman v. American Trucking Associations*, 531 U.S. 457, 464, 475-76. On the constitutional issue, the Court held that the statutory requirement that NAAQS be "requisite" to protect public health with an adequate margin of safety sufficiently guided the EPA's discretion, affirming the EPA's approach of setting standards that are neither more nor less stringent than necessary.

The Supreme Court remanded the case to the Court of Appeals for resolution of any remaining issues that had not been addressed in that court's earlier rulings. *Id.* at 475-76. In a March 2002 decision, the Court of Appeals rejected all remaining challenges to the standards, holding that the EPA's PM<sub>2.5</sub> standards were reasonably supported by the administrative record and were not "arbitrary and capricious" *American Trucking Associations v. EPA*, 283 F. 3d 355, 369-72 (D.C. Cir. 2002).

### **1.3.3 Review Completed in 2006**

In October 1997, the EPA published its plans for the third periodic review of the air quality criteria and NAAQS for PM (62 FR 55201, October 23, 1997). After the CASAC and public review of several drafts, the EPA's NCEA finalized the AQCD in October 2004 (U.S. EPA, 2004a, U.S. EPA, 2004b). The EPA's OAQPS finalized a Risk Assessment and Staff Paper in December 2005 (Abt Associates, 2005, U.S. EPA, 2005).<sup>11</sup> On December 20, 2005, the EPA announced its proposed decision to revise the NAAQS for PM and solicited public comment on a broad range of options (71 FR 2620, January 17, 2006). On September 21, 2006, the EPA announced its final decisions to revise the primary and secondary NAAQS for PM to provide increased protection of public health and welfare, respectively (71 FR 61144, October 17, 2006).

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<sup>11</sup> Prior to the review initiated in 2007, the Staff Paper presented the EPA staff's considerations and conclusions regarding the adequacy of existing NAAQS and, when appropriate, the potential alternative standards that could be supported by the evidence and information. More recent reviews present this information in the Policy Assessment.

With regard to the primary and secondary standards for fine particles, the EPA revised the level of the 24-hour  $PM_{2.5}$  standards to  $35 \mu g/m^3$ , retained the level of the annual  $PM_{2.5}$  standards at  $15.0 \mu g/m^3$ , and revised the form of the annual  $PM_{2.5}$  standards by narrowing the constraints on the optional use of spatial averaging. With regard to the primary and secondary standards for  $PM_{10}$ , the EPA retained the 24-hour standards, with levels at  $150 \mu g/m^3$ , and revoked the annual standards.<sup>12</sup> The Administrator judged that the available evidence generally did not suggest a link between long-term exposure to existing ambient levels of coarse particles and health or welfare effects. In addition, a new reference method was added for the measurement of  $PM_{10-2.5}$  in the ambient air in order to provide a basis for approving federal equivalent methods (FEMs) and to promote the gathering of scientific data to support future reviews of the PM NAAQS.

Several parties filed petitions for review following promulgation of the revised PM NAAQS in 2006. These petitions addressed the following issues: (1) selecting the level of the primary annual  $PM_{2.5}$  standard; (2) retaining  $PM_{10}$  as the indicator of a standard for thoracic coarse particles, retaining the level and form of the 24-hour  $PM_{10}$  standard, and revoking the  $PM_{10}$  annual standard; and (3) setting the secondary  $PM_{2.5}$  standards identical to the primary standards. On February 24, 2009, the U.S. Court of Appeals for the District of Columbia Circuit issued its opinion in the case *American Farm Bureau Federation v. EPA*, 559 F. 3d 512 (D.C. Cir. 2009). The court remanded the primary annual  $PM_{2.5}$  NAAQS to the EPA because the Agency failed to adequately explain why the standards provided the requisite protection from both short- and long-term exposures to fine particles, including protection for at-risk populations. *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 520-27 (D.C. Cir. 2009). With regard to the standards for  $PM_{10}$ , the court upheld the EPA's decisions to retain the 24-hour  $PM_{10}$  standard to provide protection from thoracic coarse particle exposures and to revoke the annual  $PM_{10}$  standard. *American Farm Bureau Federation*, 559 F. 2d at 533-38. With regard to the secondary  $PM_{2.5}$  standards, the court remanded the standards to the EPA because the Agency failed to adequately explain why setting the secondary PM standards identical to the primary standards provided the required protection for public welfare, including protection from visibility impairment. *American Farm Bureau Federation*, 559 F. 2d at 528-32. The EPA responded to the

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<sup>12</sup> In the 2006 proposal, the EPA proposed to revise the 24-hour  $PM_{10}$  standard in part by establishing a new  $PM_{10-2.5}$  indicator for thoracic coarse particles (i.e., particles generally between 2.5 and 10  $\mu m$  in diameter). The EPA proposed to include any ambient mix of  $PM_{10-2.5}$  that was dominated by resuspended dust from high density traffic on paved roads and by PM from industrial sources and construction sources. The EPA proposed to exclude any ambient mix of  $PM_{10-2.5}$  that was dominated by rural windblown dust and soils and by PM generated from agricultural and mining sources. In the final decision, the existing  $PM_{10}$  standard was retained, in part due to an "inability...to effectively and precisely identify which ambient mixes are included in the [ $PM_{10-2.5}$ ] indicator and which are not" (71 FR 61197, October 17, 2006).



court's remands as part of the next review of the PM NAAQS, which was initiated in 2007 (discussed below).

### **1.3.4 Review Completed in 2012**

In June 2007, the EPA initiated the fourth periodic review of the air quality criteria and the PM NAAQS by issuing a call for information in the *Federal Register* (72 FR 35462, June 28, 2007). Based on the NAAQS review process, as revised in 2008 and again in 2009,<sup>13</sup> the EPA held science/policy issue workshops on the primary and secondary PM NAAQS (72 FR 34003, June 20, 2007; 72 FR 34005, June 20, 2007), and prepared and released the planning and assessment documents that comprise the review process (i.e., IRP (U.S. EPA, 2008), ISA (U.S. EPA, 2009a), REA planning documents for health and welfare (U.S. EPA, 2009b, U.S. EPA, 2009c), a quantitative health risk assessment (U.S. EPA, 2010a) and an urban-focused visibility assessment (U.S. EPA, 2010b), and PA (U.S. EPA, 2011)). In June 2012, the EPA announced its proposed decision to revise the NAAQS for PM (77 FR 38890, June 29, 2012).

In December 2012, the EPA announced its final decisions to revise the primary NAAQS for PM to provide increased protection of public health (78 FR 3086, January 15, 2013). With regard to primary standards for PM<sub>2.5</sub>, the EPA revised the level of the annual PM<sub>2.5</sub> standard<sup>14</sup> to 12.0 µg/m<sup>3</sup> and retained the 24-hour PM<sub>2.5</sub> standard, with its level of 35 µg/m<sup>3</sup>. For the primary PM<sub>10</sub> standard, the EPA retained the 24-hour standard to continue to provide protection against effects associated with short-term exposure to thoracic coarse particles (i.e., PM<sub>10-2.5</sub>). With regard to the secondary PM standards, the EPA generally retained the 24-hour and annual PM<sub>2.5</sub> standards<sup>15</sup> and the 24-hour PM<sub>10</sub> standard to address visibility and non-visibility welfare effects.

As with previous reviews, petitioners challenged the EPA's final rule. Petitioners argued that the EPA acted unreasonably in revising the level and form of the annual standard and in amending the monitoring network provisions. On judicial review, the revised standards and monitoring requirements were upheld in all respects. *NAM v EPA*, 750 F.3d 921 (D.C. Cir. 2014).

## **1.4 CURRENT REVIEW OF THE PM NAAQS**

In December 2014, the EPA announced the initiation of the current periodic review of the air quality criteria for PM and of the PM<sub>2.5</sub> and PM<sub>10</sub> NAAQS and issued a call for information

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<sup>13</sup> The history of the NAAQS review process, including revisions to the process, is discussed at <http://www3.epa.gov/ttn/naaqs/review2.html>.

<sup>14</sup> The EPA also eliminated the option for spatial averaging.

<sup>15</sup> Consistent with the primary standard, the EPA eliminated the option for spatial averaging with the annual standard.

in the *Federal Register* (79 FR 71764, December 3, 2014). On February 9 to 11, 2015, the EPA's NCEA and OAQPS held a public workshop to inform the planning for the current review of the PM NAAQS (announced in 79 FR 71764, December 3, 2014). Workshop participants, including a wide range of external experts as well as EPA staff representing a variety of areas of expertise (e.g., epidemiology, human and animal toxicology, risk/exposure analysis, atmospheric science, visibility impairment, climate effects), were asked to highlight significant new and emerging PM research, and to make recommendations to the Agency regarding the design and scope of this review. This workshop provided for a public discussion of the key science and policy-relevant issues around which the EPA has structured the current review of the PM NAAQS and of the most meaningful new scientific information that would be available in this review to inform our understanding of these issues.

The input received at the workshop guided the EPA staff in developing a draft IRP, which was reviewed by the CASAC Particulate Matter Panel and discussed on public teleconferences held in May 2016 (81 FR 13362, March 14, 2016) and August 2016 (81 FR 39043, June 15, 2016). Advice from the CASAC, supplemented by the Particulate Matter Panel, and input from the public were considered in developing the final IRP for this review (U.S. EPA, 2016). The final IRP discusses the approaches to be taken in developing key scientific, technical, and policy documents in this review and the key policy-relevant issues that will frame the EPA's consideration of whether the current primary and/or secondary NAAQS for PM should be retained or revised.

In May 2018, the Administrator issued a memorandum describing a "back-to-basics" process for reviewing the NAAQS (Pruitt, 2018). This memo announced the Agency's intention to conduct the current review of the PM NAAQS in such a manner as to ensure that any necessary revisions are finalized by December 2020. Following this memo, on October 10, 2018 the Administrator additionally announced that the role of reviewing the key science assessments developed as part of the ongoing review of the PM NAAQS (i.e., drafts of the ISA and PA) would be performed by the seven-member chartered CASAC (i.e., rather than the CASAC Particulate Matter Panel that reviewed the draft IRP).<sup>16</sup>

The EPA released the draft ISA in October 2018 (83 FR 53471, October 23, 2018). The draft ISA was reviewed by the chartered CASAC at a public meeting held in Arlington, VA in December 2018 (83 FR 55529, November 6, 2018) and was discussed on a public teleconference in March 2019 (84 FR 8523, March 8, 2019). The CASAC provided its advice on the draft ISA in a letter to the EPA Administrator dated April 11, 2019 (Cox, 2019a). In that letter, the

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<sup>16</sup> Announcement available at: <https://www.epa.gov/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee>

CASAC's recommendations address both the draft ISA's assessment of the science for PM-related effects and the process under which this review of the PM NAAQS is being conducted.

Regarding the assessment of the evidence, the CASAC letter states that "the Draft ISA does not provide a sufficiently comprehensive, systematic assessment of the available science relevant to understanding the health impacts of exposure to particulate matter (PM)" (Cox, 2019, p. 1 of letter). The CASAC recommends that this and other limitations (i.e., "[i]nadequate evidence for altered causal determinations" and the need for a "[c]learer discussion of causality and causal biological mechanisms and pathways") be remedied in a revised ISA (Cox, 2019, p. 1 of letter). The EPA has taken steps to address these comments in the Final PM ISA (U.S. EPA, 2019). In particular, the final ISA includes additional text and a new appendix to clarify the comprehensive and systematic process employed by the EPA to develop the PM ISA. In addition, several causality determinations were re-examined and the final ISA reflects a revised causality determination for long-term ultrafine particle exposures and nervous system effects (i.e., from "likely to be causal" to "suggestive of, but not sufficient to infer, a causal relationship"). The final ISA also contains additional text to clarify the evidence for biological pathways of particular PM-related effects and the role of that evidence in causality determinations.

Among its comments on the process, the chartered CASAC recommended "that the EPA reappoint the previous CASAC PM panel (or appoint a panel with similar expertise)" (Cox, 2019a). The Agency's response to this advice was provided in a letter from the Administrator to the CASAC chair dated July 25, 2019.<sup>17</sup> As indicated in that letter, on September 13, 2019 the Administrator announced the selection of a pool of non-member subject matter experts. These experts were intended to "provide technical expertise to help CASAC ensure a rigorous and timely review of the National Ambient Air Quality Standards for particulate matter and ozone."<sup>18</sup> Input from members of this pool of experts informed the CASAC's review of the draft PA.

The EPA released the draft PA in September 2019 (84 FR 47944, September 11, 2019). The draft PA was reviewed by the chartered CASAC and discussed in October 2019 at a public meeting held in Cary, NC. Public comments were received via a separate public teleconference (84 FR 51555, September 30, 2019). A public meeting to discuss the chartered CASAC letter and response to charge questions on the draft PA was held in Cary, NC in December 2019 (84 FR 58713, November 1, 2019), and the CASAC provided its advice on the draft PA, including its

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<sup>17</sup> Available at:

[https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002\\_Response.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002_Response.pdf)

<sup>18</sup> Available at: <https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>

advice on the current primary and secondary PM standards, in a letter to the EPA Administrator dated December 16, 2019 (Cox, 2019b).

With regard to the primary standards, the CASAC recommends retaining the current 24-hour PM<sub>2.5</sub> and PM<sub>10</sub> standards, but does not reach consensus on the adequacy of the current annual PM<sub>2.5</sub> standard. With regard to the secondary standards, the CASAC recommends retaining the current standards. The CASAC's advice on the primary and secondary PM standards is discussed in detail in chapters 3 (primary PM<sub>2.5</sub> standards), 4 (primary PM<sub>10</sub> standards), and 5 (secondary standards) of this final PA.

The CASAC additionally makes a number of recommendations regarding the information and analyses presented in the draft PA. Specifically, the CASAC recommends that a revised PA include (1) additional discussion of the current CASAC and NAAQS review process; (2) additional characterization of PM-related emissions, monitoring and air quality information, including uncertainties in that information; (3) additional discussion and examination of uncertainties in the PM<sub>2.5</sub> health evidence and the risk assessment; (4) updates to reflect changes in the ISA's causality determinations; and (5) additional discussion of the evidence for PM-related welfare effects, including uncertainties (Cox, 2019b, pp. 2-3 in letter). In response to the CASAC's comments, we have incorporated a number of changes into this final PA, including the following:

- (1) We have added text to Chapter 1 (see above) to clarify the process followed for this review of the PM NAAQS, including how the process has evolved since the initiation of the review.
- (2) We have added text and figures to Chapter 2 on emissions of PM and PM precursors, and we have added a section discussing uncertainty in emissions estimates. We have also added new discussion of measurement uncertainty for FRM, FEM, CSN, and IMPROVE monitors.
- (3) In Chapter 3 and Appendices B and C, we have made a number of changes:
  - a. We have reduced the emphasis on evidence for long-term ultrafine particle exposures and nervous system effects to reflect the change in the final ISA's causality determination from "likely to be causal" to "suggestive of, but not sufficient to infer, a causal relationship."
  - b. We have expanded the characterization and discussion of the evidence related to exposure measurement error, the potential confounders examined by key studies, the shapes of concentration-response functions, and the results of causal inference and quasi-experimental studies.

- c. We have expanded and clarified the discussion of uncertainties in the risk assessment,<sup>19</sup> and we have added additional air quality model performance evaluation for each of the urban study areas included in the risk assessment.
  - d. We have provided additional detail on the procedure used to derive concentration-response functions used in the risk assessment.
- (4) Throughout the document (Chapters 3, 4 and 5), we have added summaries of the CASAC advice on the PM standards, and we have expanded the discussion of data gaps and areas for future research in the health and welfare effects evidence.

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<sup>19</sup> The CASAC's comments on the risk assessment include recommending additional analyses to quantify uncertainty in estimates of how PM<sub>2.5</sub>-related risks may change with changing ambient PM<sub>2.5</sub> concentrations (Cox, 2019b, p. 7 of consensus responses). While this final PA includes additional discussion of sources of uncertainty in the risk assessment, and additional qualitative consideration of the potential impacts of those uncertainties on risk estimates, we have not conducted additional analyses to further quantify uncertainty. This approach to addressing the CASAC's comments on the risk assessment reflects our consideration of the timeline for this review as well as the likely impact of such additional analyses on decision making.

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## 2 PM AIR QUALITY

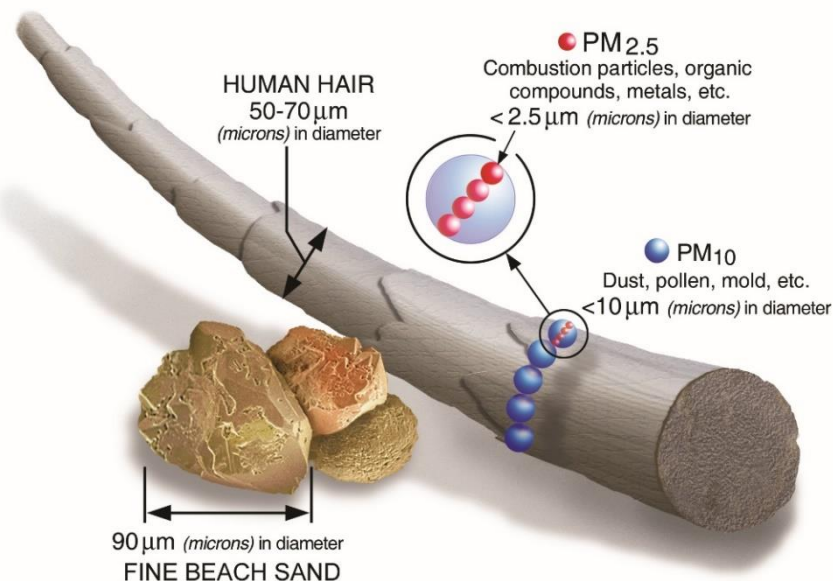
This chapter provides an overview of recent ambient air quality with respect to PM. It summarizes information on the distribution of particle size in ambient air, including discussions about size fractions and components (section 2.1), ambient monitoring of PM in the U.S. (section 2.2), ambient concentrations of PM in the U.S. (section 2.3), and background PM (section 2.4).

### 2.1 DISTRIBUTION OF PARTICLE SIZE IN AMBIENT AIR

In ambient air, PM is a mixture of substances suspended as small liquid and/or solid particles. Particle size is an important consideration for PM, as distinct health and welfare effects have been linked with exposures to particles of different sizes. Particles in the atmosphere range in size from less than 0.01 to more than 10 micrometers ( $\mu\text{m}$ ) in diameter (U.S. EPA, 2019a, section 2.2). When describing PM, subscripts are used to denote the aerodynamic diameter<sup>1</sup> of the particle size range in micrometers ( $\mu\text{m}$ ) of 50% cut points of sampling devices. The EPA defines  $\text{PM}_{2.5}$ , also referred to as fine particles, as particles with aerodynamic diameters generally less than or equal to 2.5  $\mu\text{m}$ . The size range for  $\text{PM}_{10-2.5}$ , also called coarse or thoracic coarse particles, includes those particles with aerodynamic diameters generally greater than 2.5  $\mu\text{m}$  and less than or equal to 10  $\mu\text{m}$ .  $\text{PM}_{10}$ , which is comprised of both fine and coarse fractions, includes those particles with aerodynamic diameters generally less than or equal to 10  $\mu\text{m}$ . Figure 2-1 provides perspective on these particle size fractions. In addition, ultrafine particles (UFP) are often defined as particles with a diameter of less than 0.1  $\mu\text{m}$  based on physical size, thermal diffusivity or electrical mobility (U.S. EPA, 2019a, section 2.2).

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<sup>1</sup> Aerodynamic diameter is the size of a sphere of unit density (i.e., 1  $\text{g}/\text{cm}^3$ ) that has the same terminal settling velocity as the particle of interest (U.S. EPA, 2018, U.S. EPA, 2019a, section 4.1.1).



**Figure 2-1. Comparisons of PM<sub>2.5</sub> and PM<sub>10</sub> diameters to human hair and beach sand.**  
 (Adapted from: <https://www.epa.gov/pm-pollution/particulate-matter-pm-basics>)

Atmospheric distributions of particle size generally exhibit distinct modes that roughly align with the PM size fractions defined above. The nucleation mode is made up of freshly generated particles, formed either during combustion or by atmospheric reactions of precursor gases. The nucleation mode is especially prominent near sources like heavy traffic, industrial emissions, biomass burning, or cooking (Vu et al., 2015). While nucleation mode particles are only a minor contributor to overall ambient PM mass and surface area, they are the main contributors to ambient particle number (U.S. EPA, 2019a, section 2.2). By number, most nucleation mode particles fall into the UFP size range, though some fraction of the nucleation mode number distribution can extend above 0.1  $\mu\text{m}$  in diameter. Nucleation mode particles can grow rapidly through coagulation or uptake of gases by particle surfaces, giving rise to the accumulation mode. The accumulation mode is typically the predominant contributor to PM<sub>2.5</sub> mass and surface area, though only a minor contributor to particle number (U.S. EPA, 2019a, section 2.2). PM<sub>2.5</sub> sampling methods measure most of the accumulation mode mass, although a small fraction of particles that make up the accumulation mode are greater than 2.5  $\mu\text{m}$  in diameter. Coarse mode particles are formed by mechanical generation, and through processes like dust resuspension and sea spray formation (Whitby et al., 1972). Most coarse mode mass is captured by PM<sub>10-2.5</sub> sampling, but small fractions of coarse mode mass can be smaller than 2.5  $\mu\text{m}$  or greater than 10  $\mu\text{m}$  in diameter (U.S. EPA, 2019a, section 2.2).

Most particles are found in the lower troposphere, where they can have residence times ranging from a few hours to weeks. Particles are removed from the atmosphere by wet

deposition, such as when they are carried by rain or snow, or by dry deposition, when particles settle out of suspension due to gravity. Atmospheric lifetimes are generally longest for PM<sub>2.5</sub>, which often remains in the atmosphere for days to weeks (U.S. EPA, 2019a, Table 2-1) before being removed by wet or dry deposition. In contrast, atmospheric lifetimes for UFP and PM<sub>10-2.5</sub> are shorter. Within hours, UFP can undergo coagulation and condensation that lead to formation of larger particles in the accumulation mode, or can be removed from the atmosphere by evaporation, deposition, or reactions with other atmospheric components. PM<sub>10-2.5</sub> are also generally removed from the atmosphere within hours, through wet or dry deposition (U.S. EPA, 2019a, Table 2-1).

### **2.1.1 Sources of PM Emissions**

PM is composed of both primary (directly emitted particles) and secondary chemical components. Primary PM is derived from direct particle emissions from specific PM sources while secondary PM originates from gas-phase chemical compounds present in the atmosphere that have participated in new particle formation or condensed onto existing particles (U.S. EPA, 2019a, section 2.3). Primary particles, and gas-phase compounds contributing to secondary formation PM, are emitted from both anthropogenic and natural sources.

Anthropogenic sources of PM include both stationary and mobile sources. Stationary sources include fuel combustion for electricity production and other purposes, industrial processes, agricultural activities, and road and building construction and demolition. Mobile sources of PM include diesel- and gasoline-powered highway vehicles and other engine-driven sources (e.g., ships, aircraft, and construction and agricultural equipment). Both stationary and mobile sources directly emit primary PM to ambient air, along with secondary PM precursors (e.g., SO<sub>2</sub>) that contribute to the secondary formation of PM in the atmosphere (U.S. EPA, 2019a, section 2.3, Table 2-2).

Natural sources of PM include dust from the wind erosion of natural surfaces, sea salt, wildland fires, primary biological aerosol particles (PBAP) such as bacteria and pollen, oxidation of biogenic hydrocarbons such as isoprene and terpenes to produce secondary organic aerosol (SOA), and geogenic sources such as sulfate formed from volcanic production of SO<sub>2</sub> (U.S. EPA, 2009, section 3.3, Table 3-2). While most of the above sources release or contribute predominantly to fine aerosol, some sources including windblown dust, and sea salt also produce particles in the coarse size range (U.S. EPA, 2019a, section 2.3.3).

Generally, the sources of PM for different size fractions vary. While PM<sub>2.5</sub> in ambient air is largely emitted directly by sources such as those described above or through secondary PM formation in the atmosphere, PM<sub>10-2.5</sub> is almost entirely from primary sources (i.e., directly emitted) and is produced by surface abrasion or by suspension of sea spray or biological

materials such as microorganisms, pollen, and plant and insect debris (U.S. EPA, 2019a, section 2.3.2.1).

In sections 2.1.1.1 and 2.1.1.2 below, we describe the most recently available information on sources contributing to PM<sub>2.5</sub> and PM<sub>10-2.5</sub> emissions into ambient air, respectively, based on the U.S. EPA 2014 National Emissions Inventory (NEI).<sup>2</sup> In section 2.1.1.3, we describe information on sources contributing to emissions of PM components and precursor gases.

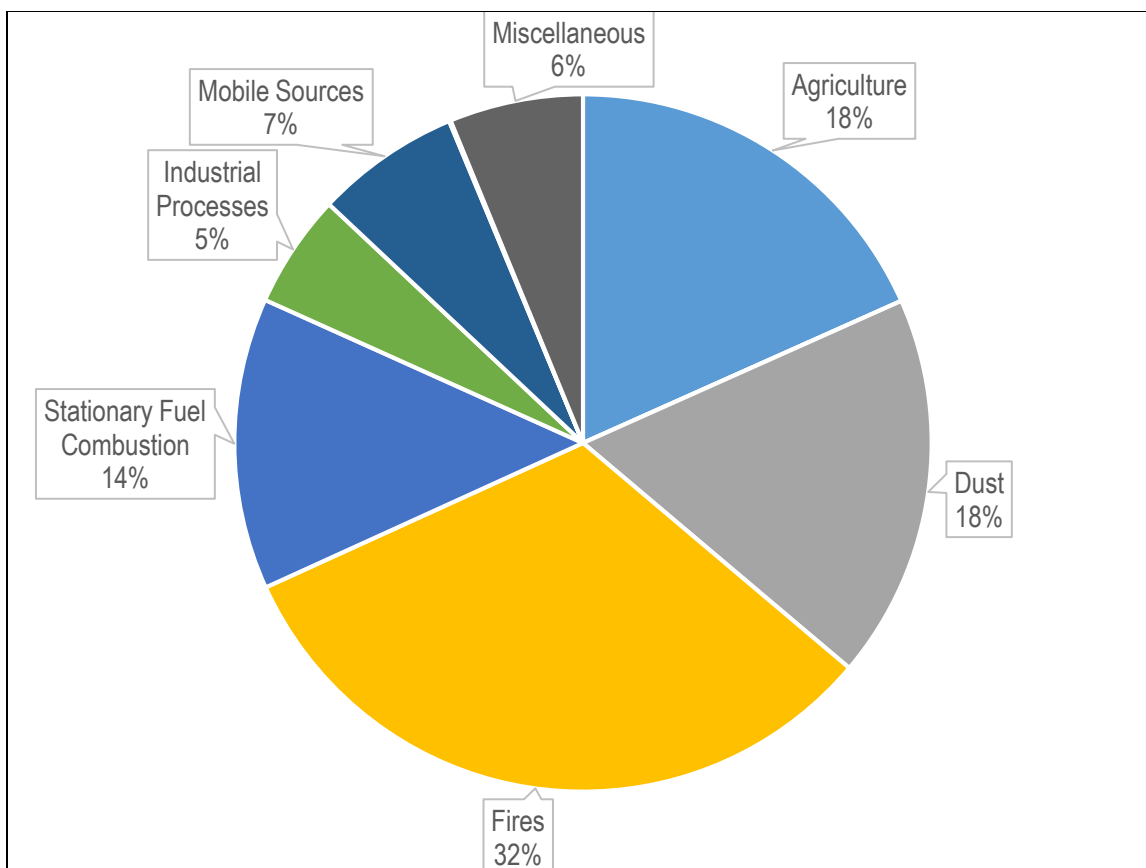
#### **2.1.1.1 Sources Contributing to Primary PM<sub>2.5</sub> Emissions**

The National Emissions Inventory (NEI) is a comprehensive and detailed estimate of air emissions of criteria pollutants, criteria precursors, and hazardous air pollutants from a comprehensive set of air emissions sources, including point sources (electric generating units, boilers, etc.), nonpoint (or area) sources (oil & gas, residential wood combustion, and many other dispersed sources), mobiles sources, and events (large fires). There are over 3,000 sources for which the NEI is developed. The NEI is released every three years based primarily upon data provided by State, Local, and Tribal air agencies for sources in their jurisdictions and supplemented by data developed by the U.S. EPA. The NEI is built using the Emissions Inventory System (EIS) first to collect the data from State, Local, and Tribal air agencies and then to blend that data with other data sources.

Based on the 2014 NEI, approximately 5.4 million tons/year of PM<sub>2.5</sub> were estimated to be directly emitted to the atmosphere from a number of source sectors in the U.S. This total excludes sources that are not a part of the NEI (e.g., windblown dust, geogenic sources). As shown in Figure 2-2, nearly half of the total primary PM<sub>2.5</sub> emissions nationally are contributed by the dust and fire sectors together. Dust includes agricultural, construction, and road dust. Of these, agricultural dust and road dust in sum make the greatest contributions to PM<sub>2.5</sub> emissions nationally. Fires include wildfires, prescribed fires, and agricultural fires, with wildfires and prescribed fires accounting for most of the fire-related primary PM<sub>2.5</sub> emissions nationally (U.S. EPA, 2019a, section 2.3.1.1). Other lesser-contributing anthropogenic sources of PM<sub>2.5</sub> emissions nationally include stationary fuel combustion and agriculture sources (e.g., agricultural tilling).

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<sup>2</sup> These sections do not provide a comprehensive list of all sources, nor does it provide estimates of emission rates or emission factors for all source categories. Individual subsectors of source types were aggregated up to a sector level as used in Figure 2-2 and Figure 2-4. More information about the sectors and subsectors can be found as a part of the 2014 NEI available from [https://www.epa.gov/sites/production/files/2018-07/documents/nei2014v2\\_tsd\\_05jul2018.pdf](https://www.epa.gov/sites/production/files/2018-07/documents/nei2014v2_tsd_05jul2018.pdf).



**Figure 2-2. Percent contribution of PM<sub>2.5</sub> emissions by national source sectors.** (Source: 2014 NEI)

The relative contributions of specific sources to annual emissions of primary PM<sub>2.5</sub> can vary from location to location, with a notable difference in contributions of sources of PM<sub>2.5</sub> emissions in urban areas compared to national emissions. For example, the ISA illustrates this variation of primary PM<sub>2.5</sub> emissions with data from five urban counties in the U.S. (U.S. EPA, 2019a, Figure 2-3).<sup>3</sup> Across the majority of these urban areas, the largest PM<sub>2.5</sub>-emitting sectors are mobile sources and fuel combustion. This is in contrast to fires, which account for the largest fraction of primary emissions nationally but make much smaller contributions in many urban counties (U.S. EPA, 2019a, section 2.3.1.2, Figure 2-3). While primary PM<sub>2.5</sub> from mobile sources are a dominant contributor in some urban areas, accounting for an estimated 13 to 30% of the total primary PM<sub>2.5</sub> emissions, mobile sources contribute only about 7% to total primary PM<sub>2.5</sub> emissions nationally as shown in Figure 2-2.

Another way to look at the emissions data shown in Figure 2-2 is by county. Figure 2-4 presents county-based total PM<sub>2.5</sub> emissions divided by the area of the county to normalize for

<sup>3</sup> The five counties included in the ISA analysis include Queens County, NY, Philadelphia County, PA, Los Angeles County, CA, Sacramento County, CA, and Maricopa County (Phoenix), AZ (U.S. EPA, 2019a, section 2.3.1.2).

differences in county size. This “emissions density” map highlights regions of the country with the strongest emitting sectors for PM<sub>2.5</sub>.



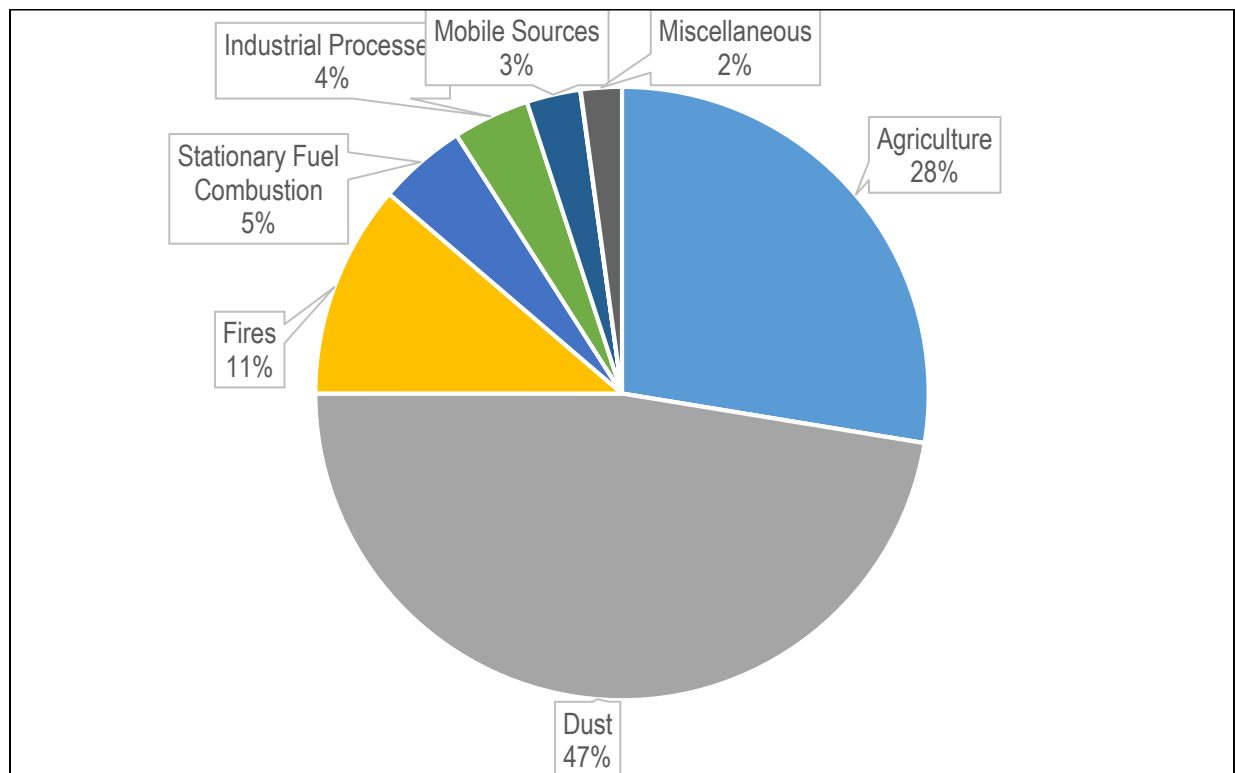
**Figure 2-3. 2014 NEI PM<sub>2.5</sub> Emissions Density Map, tons per square mile**

#### **2.1.1.2 Sources Contributing to Primary PM<sub>10</sub> Emissions**

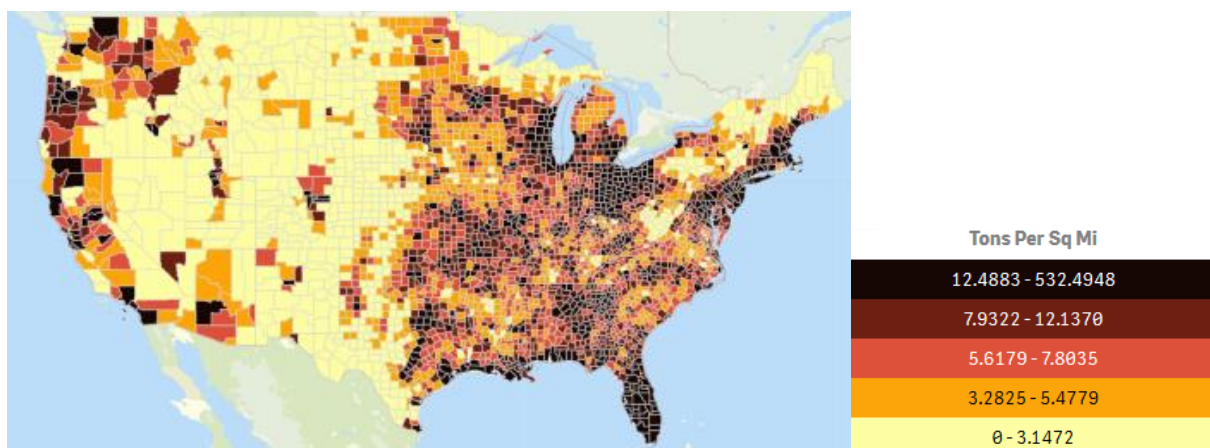
Although the NEI does not estimate emissions of PM<sub>10-2.5</sub> specifically, estimates of PM<sub>10</sub> emissions can provide insight into sources of coarse particles. Thus, the discussion below focuses on PM<sub>10</sub> emissions. The relative contributions of key sources to national PM<sub>10</sub> emissions, based on the 2014 NEI, are shown in Figure 2-4. Total PM<sub>10</sub> emissions are estimated to be about 13 million tons. National emissions of PM<sub>10</sub> are dominated by dust and agriculture, contributing a combined 75% of the total emissions. Current NEI estimates of dust emissions across the U.S. are based on limited emissions profile and activity information. For a number of reasons, quantification of dust emissions is highly uncertain. Much like wildfires, dust emissions are common but intermittent emissions sources. Additionally, the suspension and resuspension of dust is difficult to quantify. Moreover, some dust particles in the PM<sub>10-2.5</sub> size range are also transported internationally and considered as a part of the background component of PM as opposed to a primary emission of coarse PM (U.S. EPA, 2019a, section 2.3.3).

As with PM<sub>2.5</sub>, the relative contributions of particular sources to total PM<sub>10</sub> emissions varies from location to location (e.g., depending on local climate, geography, degree of urbanization, etc.). However, unlike with PM<sub>2.5</sub>, the sectors included in Figure 2-4 and found to be the largest contributors to coarse PM emissions are expected to be among the most important contributors at both the national and more regional levels, particularly given the sources of the particles in these source categories (e.g., mineral dust, primary biological aerosols (including pollen), sea spray). As noted previously, the NEI does not include sources such as pollen, sea spray, windblown dust, or geogenic sources, though those sources also likely contribute to PM<sub>10</sub>

emissions. Figure 2-4 shows the national contributions to PM<sub>10</sub> emissions from particular source sectors and Figure 2-5 shows the emissions density map for PM<sub>10</sub>.



**Figure 2-4. Percent contribution of PM<sub>10</sub> emissions by national source sectors.** (Source: 2014 NEI)



**Figure 2-5. PM<sub>10</sub> Emissions Density Map, tons per square mile**

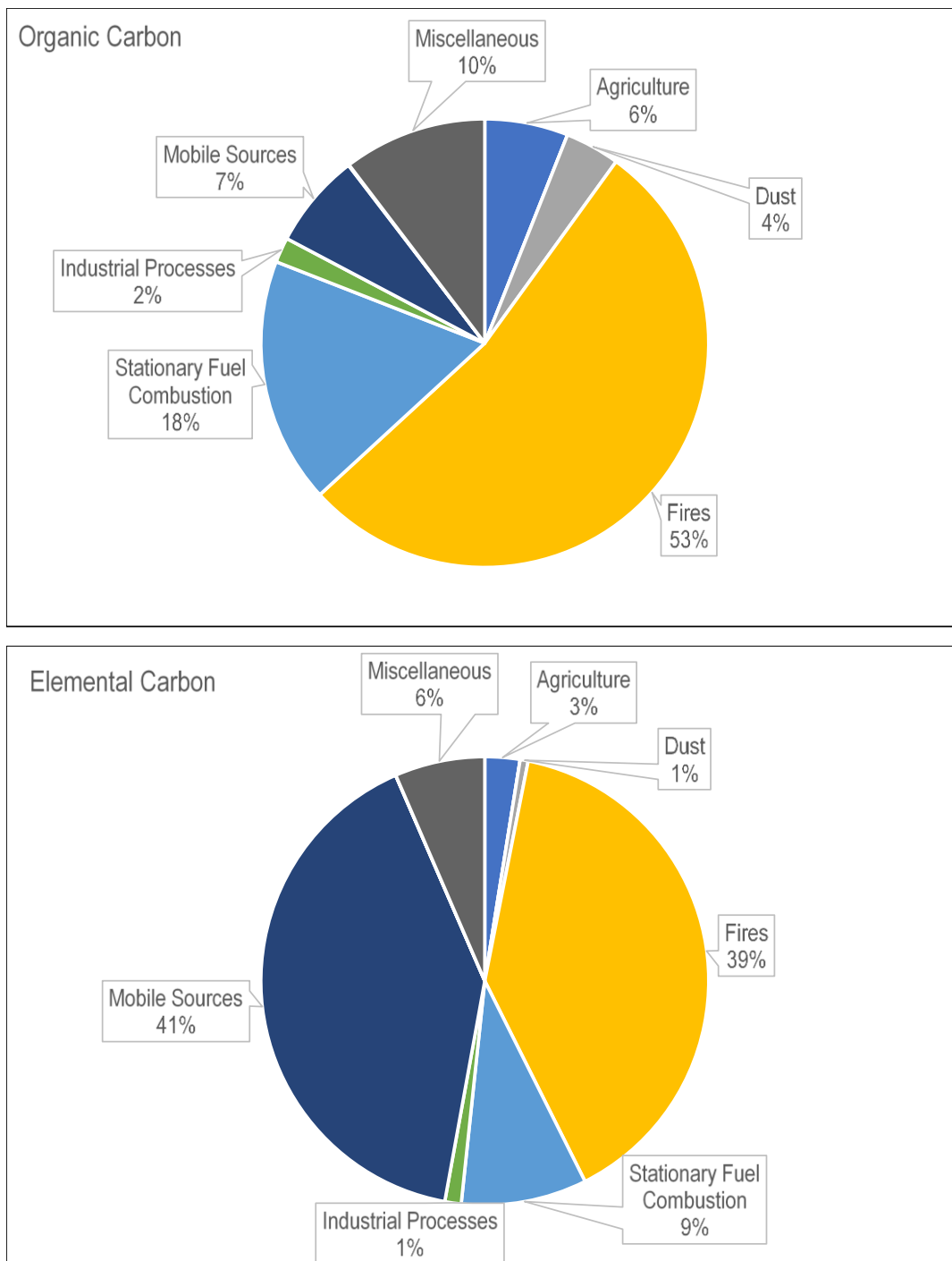
### **2.1.1.3 Sources Contributing to Emissions of PM Components and Precursor Gases**

Understanding the components of PM is particularly important for providing insight into which sources contribute to PM mass, as well as for better understanding the health and welfare effects of particles. Major components of PM<sub>2.5</sub> mass include sulfate (SO<sub>4</sub><sup>2-</sup>), nitrate (NO<sub>3</sub><sup>-</sup>), elemental or black carbon (EC or BC), organic carbon (OC), and crustal materials. Some of these PM components are emitted directly to the air (e.g., EC, BC) while others are formed secondarily through reactions by gaseous precursors (e.g., sulfate, nitrate). The following sections specifically discuss the sources that contribute to the specific PM<sub>2.5</sub> components, including particulate carbon (section 2.1.1.3.1) and precursor gases (section 2.1.1.3.2).

#### **2.1.1.3.1 Sources Contributing to Emissions of Particulate Carbon**

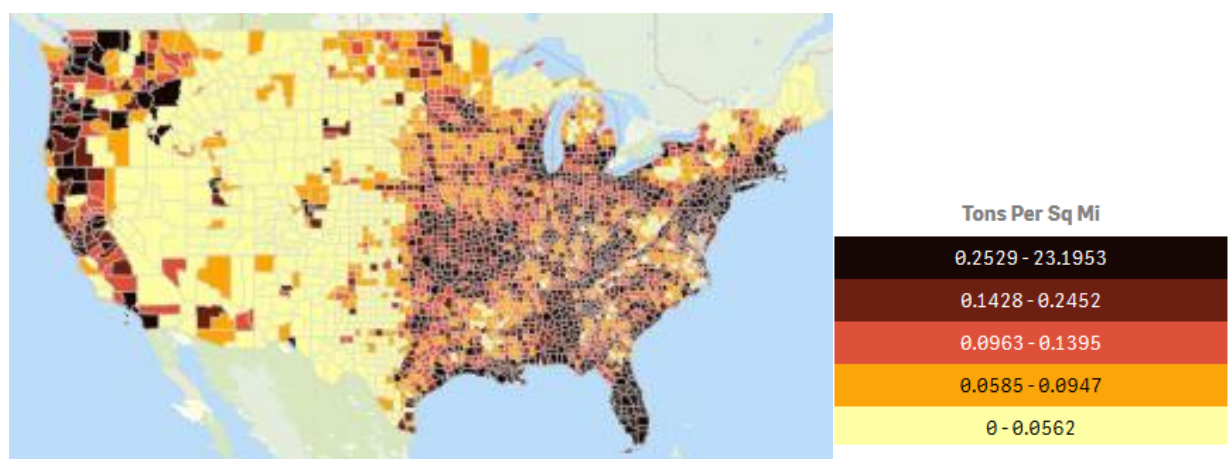
Of the directly emitted components of PM<sub>2.5</sub>, emissions of elemental (or black) carbon and organic carbon often make up the largest percentage of directly emitted PM<sub>2.5</sub> mass. Figure 2-6 illustrates the sources that contribute to national emissions of elemental and organic carbon based on the 2014 NEI. The top panel of Figure 2-6 shows that fires account for most (i.e., 53%) of the 1.5 million tons of particulate OC emissions estimated in the 2014 NEI, while the bottom panel of Figure 2-6 shows that fires and mobile sources (mostly diesel sources) contribute 80% of the estimated 431,000 tons of particulate EC in the 2014 NEI.





**Figure 2-6. Percent contribution to organic carbon (top panel) and elemental carbon (bottom panel) national emissions by source sectors. (Source: 2014 NEI)**

Figure 2-7 shows the emissions density map for elemental carbon. This map illustrates that the elemental carbon emissions signals are strong in the Southeast U.S. and parts of the West and Northwest U.S., where fires make substantial contributions to PM<sub>2.5</sub>. In addition, areas where diesel off-road and on-road sources are a large part of the emissions mix also stand out (urban and highway corridors). The OC density map (not shown) shows the highest emissions density in locations with substantial biomass burning activity, consistent with most of the OC emissions coming from fires (Figure 2-6).



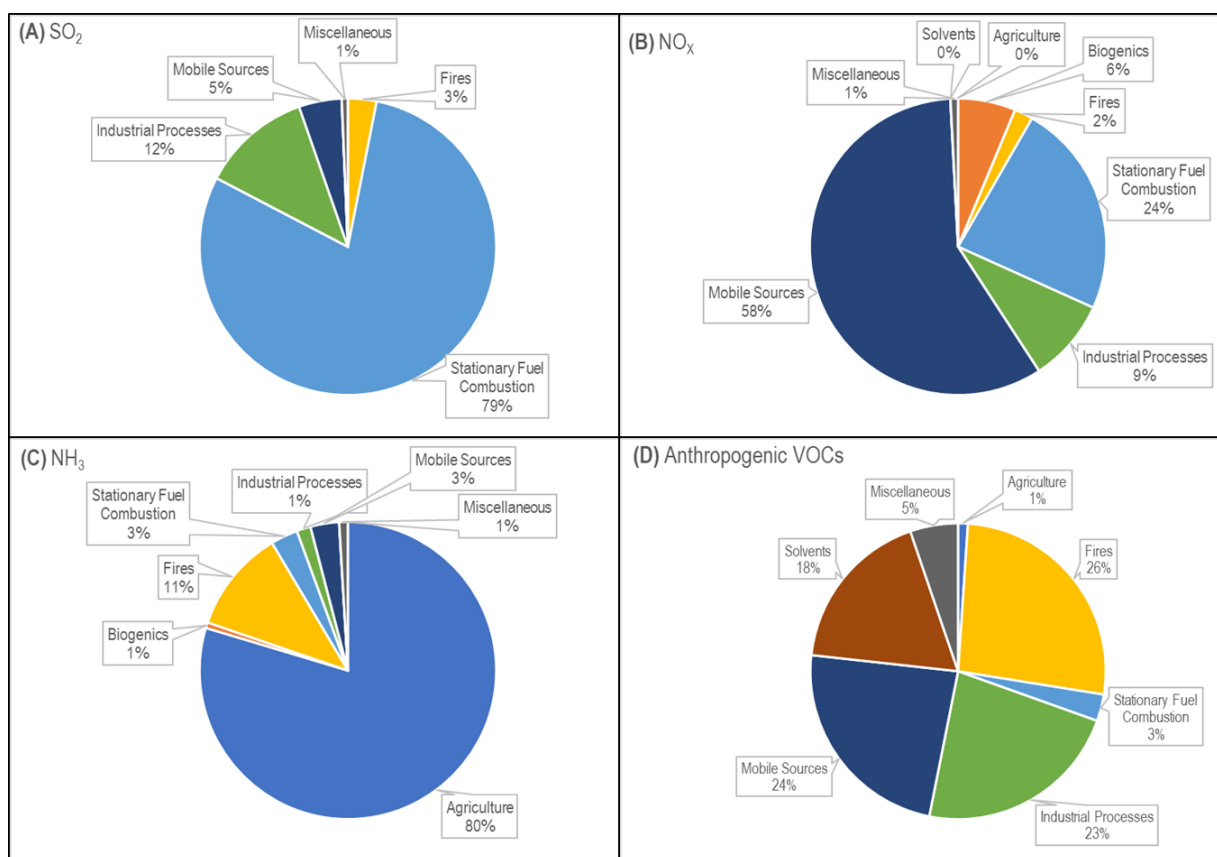
**Figure 2-7. Elemental Carbon Emissions Density Map, tons per square mile**

#### **2.1.1.3.2 Sources Contributing to Emissions of Precursor Gases**

As discussed further in the ISA (U.S. EPA, 2019a, section 2.3.2.1), secondary PM is formed in the atmosphere by photochemical oxidation reactions of both inorganic and organic gas-phase precursors. Precursor gases include SO<sub>2</sub>, NO<sub>x</sub>, and volatile organic compound (VOC) gases of anthropogenic or natural origin (U.S. EPA, 2019a, section 2.3.2.1). Anthropogenic SO<sub>2</sub> and NO<sub>x</sub> are the predominant precursor gases in the formation of secondary PM<sub>2.5</sub>, and ammonia also plays an important role in the formation of nitrate PM by neutralizing sulfuric acid and nitric acid. In addition, atmospheric oxidation of VOCs, both anthropogenic and biogenic, is an important source of organic aerosols, particularly in summer. The semi-volatile and non-volatile products of VOC oxidation reactions can condense onto existing particles or can form new particles (U.S. EPA, 2009, section 3.3.2; U.S. EPA, 2019a, section 2.3.2).

Emissions of each of the precursor gases noted above are estimated in the NEI and have unique source signatures at the national level. Figure 2-8 illustrates the source contributions at the national level for these PM<sub>2.5</sub> precursor gases. As shown in Panel A in Figure 2-8, stationary fuel combustion sources contribute nearly 80% of the estimated total of 4.8 million tons of national SO<sub>2</sub> national emissions. Within this source category, nearly all of the SO<sub>2</sub> emitted to the

atmosphere comes from electricity generating units, or EGUs. NO<sub>x</sub> emissions, shown in panel B, are emitted by a range of combustion sources, including mobile sources (58%) and stationary fuel combustion sources (24%). In the 2014 NEI, there is an estimated total of 14.4 million tons of NO<sub>x</sub> emitted. Of the total estimate of 3.6 million tons of ammonia (NH<sub>3</sub>) emissions shown in panel C of Figure 2-8, NH<sub>3</sub> emissions are dominated by the agriculture source categories. In these categories, NH<sub>3</sub> is predominantly emitted by livestock waste from animal husbandry operations (55%) and fertilizer application (25%). In urban areas, on-road mobile sources may also contribute significantly to NH<sub>3</sub> emissions (U.S. EPA, 2019a, Figure 2-3; Sun et al., 2014). Of the estimated 17 million tons of VOC emissions from anthropogenic sources, fires (26%) and mobile sources (24%) are the largest contributors to national VOC emissions, along with industrial processes (23%), as shown in panel D.



**Figure 2-8. Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B), ammonia (panel C), and anthropogenic volatile organic compounds (panel D) national emissions by source sectors. (Source: 2014 NEI)**

Figure 2-9 to Figure 2-12 below show the emissions density maps corresponding to each of the PM<sub>2.5</sub> precursors included in Figure 2-8.



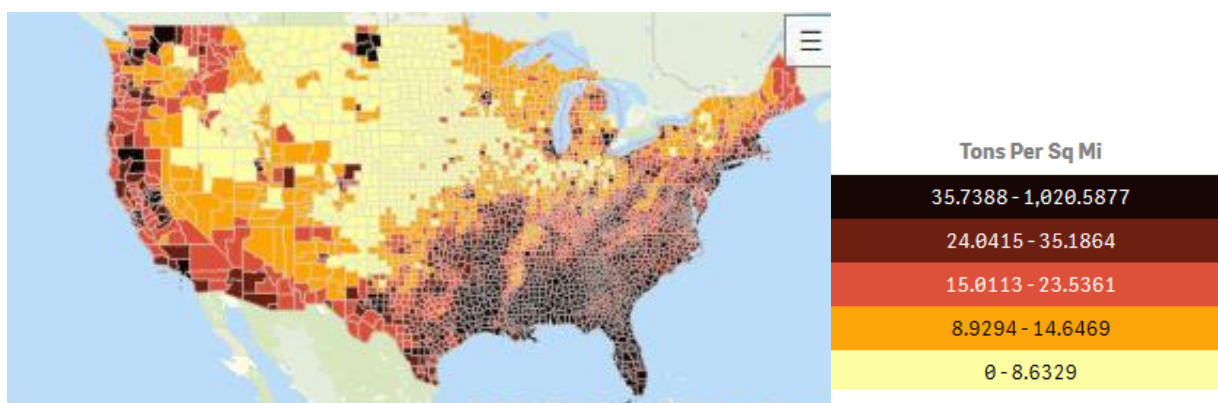
**Figure 2-9. SO<sub>2</sub> Emissions Density Map, tons per square mile**



**Figure 2-10. NO<sub>x</sub> Emissions Density Map, tons per square mile**



**Figure 2-11. NH<sub>3</sub> Emissions Density Map, tons per square mile**



**Figure 2-12. Anthropogenic (including wildfires) VOC Emissions Density Map, tons per square mile**

### 2.1.1.3.3 Uncertainty in Emission Estimates

Accuracy in an emissions inventory reflects the extent to which the inventory represents the actual emissions that occurred. Anthropogenic emissions of air pollutants result from a variety of sources such as power plants, industrial sources, motor vehicles and agriculture. The emissions from any individual source typically vary in both time and space. It is not practically possible to monitor each of the emission sources individually and, therefore, emission inventories necessarily contain assumptions, interpolation and extrapolation from a limited set of sample data.

The NEI process is based on a “bottom up” approach to developing emission estimates. This means that a combination of activity and an appropriate emissions factor is used to estimate emissions for all processes. For the thousands of sources that make up the NEI, there is uncertainty in one or both of these factors. For some sources, such as EGUs, direct emission measurements enable the emission factors to be more certain than for sources without such direct measurements. For example, emission factors for residential wood combustion are taken from information available in the literature, regardless of its pedigree and direct applicability to the



source in question. Many of these issues related to the analysis of uncertainty in the NEI are discussed by Day et al., 2019).

It is not clear how uncertainties in emission estimates affect air quality modeling, as there are no numerical empirical uncertainty estimates available for the NEI. However, by comparing modeled concentrations to ambient measurements, overall uncertainty in model outputs can be characterized. Some of this uncertainty in model outputs is likely due to uncertainty in emission estimates.

## **2.2 AMBIENT PM MONITORING METHODS AND NETWORKS**

To promote uniform enforcement of the air quality standards set forth under the CAA and to achieve the degree of public health and welfare protection intended for the NAAQS, the EPA established PM Federal Reference Methods (FRMs)<sup>4</sup> for both PM<sub>10</sub> and PM<sub>2.5</sub> (40 CFR Appendix J and L to Part 50) and performance requirements for approval of Federal Equivalent Methods (FEMs) (40 CFR Part 53). Amended following the 2006 and 2012 PM NAAQS reviews, the current PM monitoring network relies on FRMs and automated continuous FEMs, in part to support changes necessary for implementation of the revised PM standards. The requirements for measuring ambient air quality and reporting ambient air quality data and related information are the basis for 40 CFR Appendices A through E to Part 58.

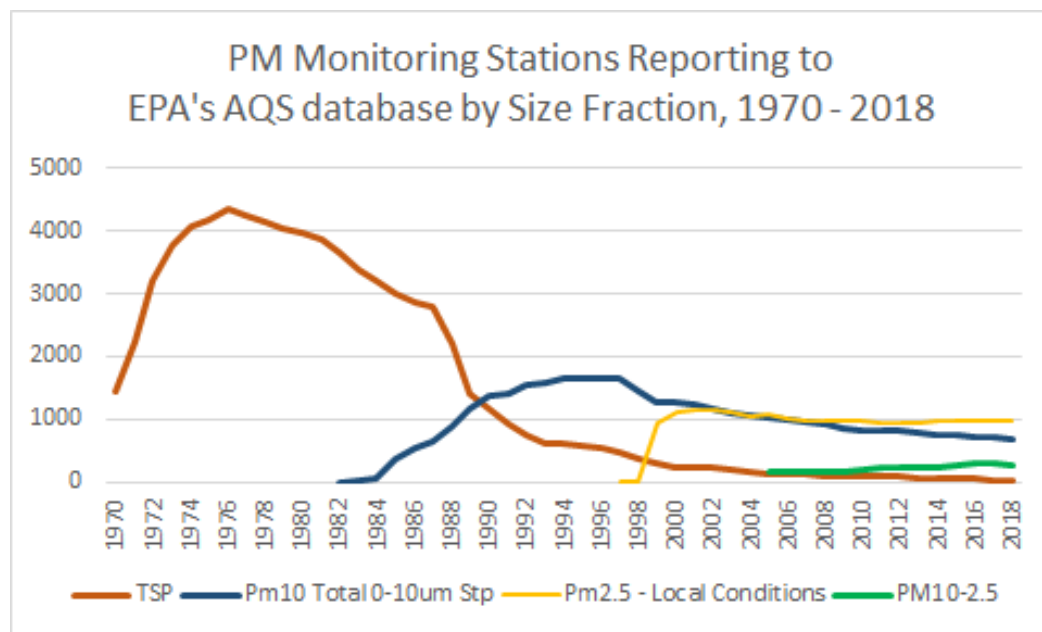
The EPA and its partners at state, local, and tribal monitoring agencies manage and operate the nation's ambient air monitoring networks. The EPA provides minimum monitoring requirements for criteria pollutants and related monitoring (e.g., the Chemical Speciation Network (CSN)), including identification of an FRM for criteria pollutants and guidance documents to support implementation and operation of the networks. Monitoring agencies carry out and perform ambient air monitoring in accordance with the EPA's requirements and guidance as well as often meeting their own state monitoring needs that may go beyond the minimum federal requirements. Data from the ambient air monitoring networks are available from two national databases: 1) the Air Quality System (AQS) database, which is the EPA's long-term repository of ambient air monitoring data and 2) the AirNow database, which provides near real-time data used in public reporting and forecasting of the Air Quality Index (AQI).<sup>5</sup>

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<sup>4</sup> FRMs provide the methodological basis for comparison to the NAAQS and also serve as the "gold-standard" for the comparison of other methods being reviewed for potential approval as equivalent methods. The EPA keeps a complete list of designated reference and equivalent methods available on its Ambient Monitoring Technology Information Center (AMTIC) website (<https://www.epa.gov/amtic/air-monitoring-methods-criteria-pollutants>).

<sup>5</sup> The AQI translates air quality data into numbers and colors to help people understand when to take action to protect their health against ambient air concentrations of criteria pollutants.

The EPA and monitoring agencies manage and operate robust national networks for both PM<sub>10</sub> and PM<sub>2.5</sub>, as these are the two measurement programs directly supporting the PM NAAQS. PM<sub>10</sub> measurements are based on gravimetric mass, while PM<sub>2.5</sub> measurements include gravimetric mass and chemical speciation. A smaller network of stations is operating and reporting data for PM<sub>10-2.5</sub> gravimetric mass and a few monitors are operated to support special projects, including pilot studies, for continuous speciation and particle count data. Monitoring networks and additional monitoring efforts for each of the various PM size fractions and for PM composition are discussed below.<sup>6</sup> Section 2.2.1 provides information on monitoring for total suspended particulates (TSP), section 2.2.2 provides information on monitoring for PM<sub>10</sub>, section 2.2.3 provides information on monitoring PM<sub>2.5</sub>, section 2.2.4 provides information on monitoring for PM<sub>10-2.5</sub>, and section 2.2.5 provides information on additional PM metrics. All sampler and monitor counts provided in these sections are based on data submitted to the EPA for calendar year 2018, unless otherwise noted. Figure 2-13 below illustrates the changes in PM monitoring stations reporting to the EPA's AQS database by size fraction since 1970.



**Figure 2-13. PM Monitoring stations reporting to EPA's AQS database by PM size fraction, 1970-2018.**

### 2.2.1 Total Suspended Particulates (TSP) Sampling

The EPA first established NAAQS for PM in 1971, based on the original air quality criteria document (DHEW, 1969). The reference method specified for determining attainment of

<sup>6</sup> More information on ambient monitoring networks can be found at <https://www.epa.gov/amtic>

the original standards was the high-volume sampler, which collects PM up to a nominal size of 25 to 45  $\mu\text{m}$  (referred to as total suspended particles or TSP). TSP was replaced by  $\text{PM}_{10}$  as the indicator for the PM NAAQS in the 1987 final rule (52 FR 24854, July 1, 1987). TSP sampling remains in operation at a limited number of locations primarily to provide aerosol collection for TSP lead (Pb) analysis as well as for instances where a state may continue to have state standards for TSP. The size of the TSP network peaked in the mid-1970s when over 4,300 TSP samplers were in operation. As of 2018, there were 164 TSP samplers still in operation as part of the Pb monitoring program; of these, 41 also report TSP mass.

### 2.2.2 $\text{PM}_{10}$ Monitoring

To support the 1987  $\text{PM}_{10}$  NAAQS, the EPA and its state and local partners implemented the first size-selective PM monitoring network in 1990 with the establishment of a  $\text{PM}_{10}$  network consisting of mainly high-volume samplers. The network design criteria emphasize monitoring at middle<sup>7</sup> and neighborhood<sup>8</sup> scales to effectively characterize the emissions from both mobile and

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<sup>7</sup> For  $\text{PM}_{10}$ , middle-scale is defined as follows: Much of the short-term public exposure to  $\text{PM}_{10}$  is on this scale and on the neighborhood scale. People moving through downtown areas or living near major roadways or stationary sources, may encounter particulate pollution that would be adequately characterized by measurements of this spatial scale. Middle scale  $\text{PM}_{10}$  measurements can be appropriate for the evaluation of possible short-term exposure public health effects. In many situations, monitoring sites that are representative of micro-scale or middle-scale impacts are not unique and are representative of many similar situations. This can occur along traffic corridors or other locations in a residential district. In this case, one location is representative of a neighborhood of small scale sites and is appropriate for evaluation of long-term or chronic effects. This scale also includes the characteristic concentrations for other areas with dimensions of a few hundred meters such as the parking lot and feeder streets associated with shopping centers, stadia, and office buildings. In the case of  $\text{PM}_{10}$ , unpaved or seldomly swept parking lots associated with these sources could be an important source in addition to the vehicular emissions themselves.

<sup>8</sup> For  $\text{PM}_{10}$ , neighborhood scale is defined as follows: Measurements in this category represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. In some cases, a location carefully chosen to provide neighborhood scale data would represent not only the immediate neighborhood but also neighborhoods of the same type in other parts of the city. Neighborhood scale  $\text{PM}_{10}$  sites provide information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for extended periods. Neighborhood scale data could provide valuable information for developing, testing, and revising models that describe the larger-scale concentration patterns, especially those models relying on spatially smoothed emission fields for inputs. The neighborhood scale measurements could also be used for neighborhood comparisons within or between cities.



stationary sources, although not ruling out microscale<sup>9</sup> monitoring in some instances (40 CFR Part 58 Appendix D, 4.6 (b)). The PM<sub>10</sub> monitoring network peaked in size in 1995 with 1,665 stations reporting data.

In 2018, there were 714 PM<sub>10</sub> stations in operation to support comparison of the PM<sub>10</sub> data to the NAAQS, trends, and reporting and forecasting of the AQI. Though the PM<sub>10</sub> network is relatively stable, monitoring agencies may continue divesting of some of the PM<sub>10</sub> monitoring stations where concentration levels are low relative to the NAAQS.

While the PM<sub>10</sub> network is national in scope, there are areas of the west, such as California and Arizona, with substantially higher PM<sub>10</sub> station density than the rest of the country. In the PM<sub>10</sub> mass network, 365 of the stations operate automated continuous mass monitors approved as FEMs and 391 operate FRMs. About 40 of the PM<sub>10</sub> stations have collocation with both continuous FEMs and FRMs. About two thirds of the PM<sub>10</sub> stations with FRMs operate on a sample frequency of one in every sixth day, with about 70 operating every third day and 60 operating every day.

### **2.2.3 PM<sub>2.5</sub> Monitoring**

To support the 1997 PM<sub>2.5</sub> NAAQS, the first PM standard with PM<sub>2.5</sub> as an indicator, the EPA and states implemented a PM<sub>2.5</sub> network consisting of ambient air monitoring sites with mass and/or chemical speciation measurements. Network operation began in 1999 with nearly 1,000 monitoring stations operating FRMs to measure fine particle mass. The PM<sub>2.5</sub> monitoring program remains one of the major ambient air monitoring programs operated across the country.

For most urban locations PM<sub>2.5</sub> monitors are sited at the neighborhood scale,<sup>10</sup> where PM<sub>2.5</sub> concentrations are reasonably homogeneous throughout an entire urban sub-region. In each

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<sup>9</sup> For PM<sub>10</sub>, microscale is defined as follows: This scale would typify areas such as downtown street canyons, traffic corridors, and fence line stationary source monitoring locations where the general public could be exposed to maximum PM<sub>10</sub> concentrations. Microscale particulate matter sites should be located near inhabited buildings or locations where the general public can be expected to be exposed to the concentration measured. Emissions from stationary sources such as primary and secondary smelters, power plants, and other large industrial processes may, under certain plume conditions, likewise result in high ground level concentrations at the microscale. In the latter case, the microscale would represent an area impacted by the plume with dimensions extending up to approximately 100 meters. Data collected at microscale sites provide information for evaluating and developing hot spot control measures.

<sup>10</sup> For PM<sub>2.5</sub>, neighborhood scale is defined as follows: Measurements in this category would represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. Much of the PM<sub>2.5</sub> exposures are expected to be associated with this scale of measurement. In some cases, a location carefully chosen to provide neighborhood scale data would represent the immediate neighborhood as well as neighborhoods of the same type in other parts of the city. PM<sub>2.5</sub> sites of this kind provide good information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for periods comparable to those specified in the NAAQS. In general, most PM<sub>2.5</sub> monitoring in urban areas should have this scale.

CBSA with a monitoring requirement, at least one PM<sub>2.5</sub> monitoring station representing area-wide air quality is to be sited in an area of expected maximum concentration. Sites that represent relatively unique microscale, localized hot-spot, or unique middle scale impact sites are only eligible for comparison to the 24-hour PM<sub>2.5</sub> NAAQS.

There are three main components of the current PM<sub>2.5</sub> monitoring program: FRMs, PM<sub>2.5</sub> continuous mass monitors, and CSN samplers. The FRMs are primarily used for comparison to the NAAQS, but also serve other important purposes such as developing trends and evaluating the performance of PM<sub>2.5</sub> continuous mass monitors. PM<sub>2.5</sub> continuous mass monitors are automated methods primarily used to support forecasting and reporting of the AQI, but are also used for comparison to the NAAQS where approved as FEMs. The CSN and related Interagency Monitoring of Protected Visual Environments (IMPROVE) network are used to provide chemical composition of the aerosol which serve a variety of objectives. This section provides an overview of each of these components of the PM<sub>2.5</sub> monitoring program and of recent changes to PM<sub>2.5</sub> monitoring requirements.

#### **2.2.3.1 Federal Reference Method and Continuous Monitors**

As noted above, the PM<sub>2.5</sub> monitoring network began operation in 1999 with nearly 1,000 monitoring stations operating FRMs. The PM<sub>2.5</sub> FRM network peaked in operation in 2001 with over 1,150 monitoring stations. In the PM<sub>2.5</sub> network, in 2018 there were 624 FRM filter-based samplers that provide 24-hour PM<sub>2.5</sub> mass concentration data. Of these operating FRMs, 70 are providing daily PM<sub>2.5</sub> data, 422 every third day, and 132 every sixth day.

As of 2018, there are 940 continuous PM<sub>2.5</sub> mass monitors that provide hourly data on a near real-time basis reporting across the country. A total of 579 of the PM<sub>2.5</sub> continuous monitors are FEMs and therefore used both for comparison with the NAAQS and to report the AQI. Another 361 monitors not approved as FEMs are operated primarily to report the AQI. These legacy PM<sub>2.5</sub> continuous monitors were largely purchased prior to the availability of PM<sub>2.5</sub> continuous FEMs.

The first method approved as a continuous PM<sub>2.5</sub> FEM was the Met One BAM 1020. This method, approved in 2008, accounts for just over 50% of the operating PM<sub>2.5</sub> continuous FEMs in the country. The EPA has approved a total of 11 PM<sub>2.5</sub> continuous methods as FEMs. Other methods approved as continuous PM<sub>2.5</sub> FEMs include beta attenuation from multiple instrument manufacturers; optical methods such as the GRIMM and Teledyne T640; and methods employing the Tapered Element Oscillating Microbalance (TEOM) with a Filter Dynamic Measurement System (FDMS) manufactured by Thermo Fisher Scientific.

The quality of the data generated by PM<sub>2.5</sub> FRMs and automated FEMs were analyzed for years 2016-2018. Data quality terms for measurement uncertainty regularly assessed in the

PM<sub>2.5</sub> monitoring program include precision and bias. Precision is calculated by comparing data from collocated methods of the same make and model operated by the same monitoring organization. Bias is calculated by comparing data from routinely operated FRMs or automated FEMs by the monitoring organization and comparing that to data from reference method audit samplers temporarily collocated and operated independently from the staff in the monitoring organization. Goals for measurement uncertainty are defined in Appendix A to 40 CFR Part 58. They state “Measurement Uncertainty for Automated and Manual PM<sub>2.5</sub> Methods. The goal for acceptable measurement uncertainty is defined for precision as an upper 90 percent confidence limit for the coefficient of variation (CV) of 10 percent and  $\pm 10$  percent for total bias.” The most recent three-year average estimate of national aggregate PM<sub>2.5</sub> FRM precision is 8.2% and bias is -4.7%.

Automated PM<sub>2.5</sub> FEMs include a wide variety of approved methods which can have different measurement principles. Data aggregated across all automated FEMs result in a collocated precision of 18.6% and a bias as compared to the reference method audit program of +7.6%. When evaluating automated FEMs as individual methods, only two of the seven methods with available collocated precision data meet the measurement uncertainty goal; however, as explained in the Notice of Proposed Rulemaking, January 17, 2006<sup>11</sup> when considering a requirement for approval of candidate FEMs: “Statistical analyses based on the DQO model show that the precision of a candidate method is not, statistically, very important to annual concentration averages used for NAAQS attainment decisions, but would be important for a daily standard.” When evaluating automated FEMs as individual methods for bias, eight of ten methods with data available to calculate a performance evaluation bias meet this goal. In summary, PM<sub>2.5</sub> automated FEMs tend to have higher collocated precision than FRMs and tend to have a positive bias relative to both State and local operated FRMs as well as performance evaluation audit FRMs.

### **2.2.3.2 Chemical Speciation and IMPROVE Networks**

Due to the complex nature of fine particles, the EPA and states implemented the CSN to better understand the components of fine particle mass at selected locations across the country. The CSN was first piloted at 13 sites in 2000, and after the pilot phase, the program continued with deployment of the Speciation Trends Network (STN) later that year. The CSN ultimately grew to 54 trends sites and peaked in operation in 2005 with 252 stations: the 54 trends stations and nearly 200 supplemental stations. The original CSN program had multiple sampler configurations including the Thermo Andersen RAAS, Met One SASS/SuperSASS, and URG

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<sup>11</sup> <https://www.govinfo.gov/content/pkg/FR-2006-01-17/pdf/06-177.pdf>

MASS. During the 2000s, the EPA and states worked to align the network to one common sampler for elements and ions, which was the Met One SASS/SuperSASS. In 2005, the CASAC provided recommendations to the EPA for making changes to the CSN. These changes were intended to improve data comparability with the rural IMPROVE carbon concentration data. To accomplish this, the EPA replaced the existing carbon channel sampling and analysis methods with a new modified IMPROVE version III module C sampler, the URG 3000N. Implementation of the new carbon sampler and analysis was broken into three phases starting in May 2007 through October 2009.

In the 2018 PM<sub>2.5</sub> CSN, long-term measurements are made at about 76 largely urban locations comprised of either the STN or the National Core (NCore) network.<sup>12</sup> NCore is a multipollutant network measuring particles, gases, and basic meteorology that has been in formal operation since January 1, 2011. Particle measurements made at NCore include PM<sub>2.5</sub> filter-based mass, which is largely the FRM, except in some rural locations that utilize the IMPROVE program PM<sub>2.5</sub> mass filter-based measurement; PM<sub>2.5</sub> speciation using either the CSN program or IMPROVE program; and PM<sub>10-2.5</sub> mass utilizing an FRM, FEM or IMPROVE for some of the rural locations. As of 2018, the NCore network includes a total of 78 stations of which 63 are in urban or suburban stations designed to provide representative population exposure and another 15 rural stations designed to provide background and transport information. The NCore network is deployed in all 50 States, DC, and Puerto Rico with at least one station in each state and two or more stations in larger population states (California, Florida, Illinois, Michigan, New York, North Carolina, Ohio, Pennsylvania, and Texas).

Both the STN and NCore networks are intended to remain in operation indefinitely. The CSN measurements at NCore and STN stations operate every third day. Another approximately 72 CSN stations, known as supplemental sites, are intended to be potentially less permanent locations used to support State Implementation Plan (SIP) development and other monitoring objectives.<sup>13</sup> Supplemental CSN stations typically operate every sixth day. In January 2015, 38 supplemental CSN stations that are largely located in the eastern half of the country stopped operations to ensure a sustainable CSN network moving forward.<sup>14</sup>

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<sup>12</sup> In most cases where a city has an STN station, it is located at the same site as the NCore station. In a few cases, a city may have an STN station located at a different location than the NCore station.

<sup>13</sup> See <https://www3.epa.gov/ttn/amtic/speciepg.html> for more information on the PM<sub>2.5</sub> speciation monitoring program.

<sup>14</sup> Based on assessments of the CSN network and IMPROVE protocol sites, monitoring resources were redistributed to focus on new or high priorities. More information on the CSN and IMPROVE protocol assessments is available at <https://www.sdas.battelle.org/CSNAssessment/html/Default.html>.

Specific components of fine particles are also measured through the IMPROVE monitoring program<sup>15</sup> which supports regional haze characterization and tracks changes in visibility in Class I areas as well as many other rural and some urban areas. As of 2018, the IMPROVE network includes 110 monitoring locations that are part of the base network supporting regional haze and another 46 locations operated as IMPROVE protocol sites where a monitoring agency has requested participation in the program. These IMPROVE protocol sites operate the same way as the IMPROVE program, but they may serve several monitoring objectives (i.e., the same objectives as the CSN) and are not explicitly tied to the Regional Haze Program. Samplers at IMPROVE stations operate every third day. In January 2016, eight IMPROVE protocol stations stopped operating to ensure a sustainable IMPROVE program moving forward. Details on the process and outcomes of the CSN supplemental and IMPROVE protocol assessments used to identify sites that would no longer be funded are available on an interactive website.<sup>16</sup> Together, the CSN and IMPROVE data provide chemical species information for fine particles that are critical for use in health and epidemiologic studies to help inform reviews of the primary PM NAAQS and can be used to better understand visibility through calculation of light extinction using the IMPROVE algorithm<sup>17</sup> to support reviews of the secondary PM NAAQS.

The quality of the data generated by the PM<sub>2.5</sub> speciation networks (CSN and IMPROVE) is assessed regularly, using a variety of metrics. Overall network precision, including uncertainties associated with both field operations and laboratory analyses, is assessed using the subset of sites with collocated samplers. Fractional uncertainty is one metric that both speciation networks regularly calculates using collocated data pairs above the MDL and reflects the overall percent uncertainty for the measurements. For CSN data collected between November 2015 and December 2016, the fractional uncertainties range from 6.6% for sulfate to 31.4% for chlorine.<sup>18</sup>

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<sup>15</sup> Recognizing the importance of visual air quality, Congress included legislation in the 1977 Clean Air Act to prevent future and remedy existing visibility impairment in Class I areas. To aid the implementation of this legislation, the IMPROVE program was initiated in 1985 and substantially expanded in 2000-2003. This program implemented an extensive long-term monitoring program to establish the current visibility conditions, track changes in visibility and determine causal mechanism for the visibility impairment in the National Parks and Wilderness Areas. For more information, see <https://www3.epa.gov/ttn/amtic/visdata.html>.

<sup>16</sup> See the Chemical Speciation Network Assessment Interactive Website at: <https://www.sdas.battelle.org/CSNAssessment/html/Default.html>.

<sup>17</sup> The IMPROVE algorithm is an equation to estimate light extinction based on the measured concentration of several PM components and is used to track visibility progress in the Regional Haze Rule. More information about the IMPROVE algorithm is at available at: <http://vista.cira.colostate.edu/Improve/the-improve-algorithm>.

<sup>18</sup> [https://airquality.ucdavis.edu/sites/g/files/dgvnsk1671/files/inline-files/CSN\\_AnnualReport\\_2016Data\\_03.06.2019\\_FINAL\\_APPROVED.pdf](https://airquality.ucdavis.edu/sites/g/files/dgvnsk1671/files/inline-files/CSN_AnnualReport_2016Data_03.06.2019_FINAL_APPROVED.pdf)

For IMPROVE data collected in 2016 and 2017, the fractional uncertainties range from 2% for sulfur and sulfate to 27% for phosphorous.<sup>19</sup> In general, uncertainties are higher for species with concentrations near the detection limit. Bias for the speciation networks can be assessed using reports from interlaboratory comparisons.<sup>20</sup>

### **2.2.3.3 Recent Changes to PM<sub>2.5</sub> Monitoring Requirements**

Key changes made to the EPA's monitoring requirements as a result of the 2012 PM NAAQS review included the addition of PM<sub>2.5</sub> monitoring at near-road locations in core-based statistical areas (CBSAs) over 1 million in population; the clarification of terms used in siting of PM<sub>2.5</sub> monitors and their applicability to the NAAQS; and the provision of flexibility on data uses to monitoring agencies where their PM<sub>2.5</sub> continuous monitors are not providing data that meets the performance criteria used to approve the continuous method as an FEM. The addition of PM<sub>2.5</sub> monitoring at near-road locations was phased in from 2015 to 2017. On January 1, 2015, 22 CBSAs with a population of 2.5 million or more were required to have a PM<sub>2.5</sub> FRM or FEM operating at a near-road monitoring station. On January 1, 2017, 30 CBSAs with a population between 1 million and 2.5 million were required to have a PM<sub>2.5</sub> FRM or FEM operating at a near-road monitoring station.

The terms clarified as a part of the 2012 rulemaking ensure consistency with all other NAAQS and long-standing definitions used by the EPA (78 FR 3234, January 15, 2013). The flexibility provided to monitoring agencies ensures that the incentives of utilizing PM<sub>2.5</sub> continuous monitors (e.g., efficiencies in operation and availability of hourly data in near-real time) are realized without having potentially poor performing data be used in situations where the data is not applicable to the NAAQS (78 FR 3241, January 15, 2013).

### **2.2.4 PM<sub>10-2.5</sub> Monitoring**

In the 2006 PM NAAQS review, the EPA promulgated a new FRM for the measurement of PM<sub>10-2.5</sub> mass in ambient air. Although the standard for coarse particles uses a PM<sub>10</sub> indicator, a new FRM for PM<sub>10-2.5</sub> mass was developed to provide a basis for approving FEMs and to promote the gathering of scientific data to support future reviews of the PM NAAQS. The PM<sub>10-2.5</sub> FRM (or approved FEMs, where available) was implemented at required NCore stations by January 1, 2011. In addition to NCore, there are other collocated PM<sub>10</sub> and PM<sub>2.5</sub> low-volume FRMs operating across the country that are essentially providing the PM<sub>10-2.5</sub> FRM measurement by the difference method.

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19 [http://vista.cira.colostate.edu/improve/wp-content/uploads/2019/11/IMPROVE\\_QAReport\\_11.15.2019.pdf](http://vista.cira.colostate.edu/improve/wp-content/uploads/2019/11/IMPROVE_QAReport_11.15.2019.pdf)

20 <https://www3.epa.gov/ttn/amtic/pmspec.html>

PM<sub>10-2.5</sub> measurements are currently performed across the country at NCore stations, IMPROVE monitoring stations, and at a few additional locations where state or local agencies choose to operate a PM<sub>10-2.5</sub> method. For urban NCore stations and other State and Local Air Monitoring Stations (SLAMS) the method employed is either a PM<sub>10-2.5</sub> FRM, which is performed using a low-volume PM<sub>10</sub> FRM collocated with a low volume PM<sub>2.5</sub> FRM of the same make and model, or FEMs for PM<sub>10-2.5</sub>, including filter-based dichotomous methods and continuous methods of which several makes and models are approved. Filter-based PM<sub>10-2.5</sub> measurements at NCore (i.e., the FRM or dichotomous filter-based FEM) operate every third day, while continuous methods have data available every hour of every day. PM<sub>10-2.5</sub> filter-based methods at other SLAMS typically operate every third or sixth day. For IMPROVE, which is largely a rural network, PM<sub>10-2.5</sub> measurements are made with two sample channels; one each for PM<sub>10</sub> and PM<sub>2.5</sub>. All IMPROVE program samplers operate every third day. All together there were 279 stations in 2018 where PM<sub>10-2.5</sub> data were being reported to the AQS database.

There is no operating chemical speciation network for characterizing the specific components of coarse particles. In 2015, Washington University at St. Louis, under contract to the U.S. EPA, reported on a coarse particle speciation pilot study with several objectives aimed at addressing this issue, such as evaluating a coarse particle species analyte list and evaluating sampling and analytical methods (U.S. EPA, 2015). The coarse particle speciation pilot study provides useful information for any organization wishing to pursue coarse particle speciation.

### **2.2.5 Additional PM Measurements and Metrics**

There are additional PM measurements and metrics made at a much smaller number of stations. These measurements may be associated with special projects or are complementary measurements to other networks where the monitoring agency has prioritized having the measurements. None of these measurements are required by regulation. They include PM measurements such as particle counts, continuous carbon, and continuous sulfate.

The EPA and state and local agencies have also been working together to pilot additional PM methods at near-road monitoring stations that may be of interest to data users. These methods include such techniques as particle counters, particle size distribution, and black carbon by aethalometer. These methods and their rationale for use at near-road monitoring stations are described in a Technical Assistance Document (TAD) on NO<sub>2</sub> near-road monitoring (U.S. EPA, 2012, section 16).

Aethalometer measurements of the concentration of optically absorbing particles have been submitted to AQS for many years. Data uses include characterizing black carbon and wood smoke. Ambient air monitoring stations that may have aethalometers include some of the near-road monitoring stations and National Air Toxics Trends Stations (NATTS). Data from about 72

monitoring sites across the county are being reported from aethalometers. While aethalometer data is available at high time resolutions (e.g., 5-minute data), it is typically reported to the AQS database in 1-hour periods.

Continuous elemental and organic carbon data were monitored at select locations participating in a pilot of the Sunset EC/OC analyzer as well as a few additional sites that were already operating before the EPA initiated the pilot study.<sup>21</sup> The Sunset EC/OC analyzer provides high time resolution carbon data, typically every hour, but in some remote locations the instrument is programmed to run every two hours to ensure collection of enough aerosol. The data from the Sunset EC/OC analyzer was compared to filter-based carbon methods from the carbon channel of the CSN program. The Sunset EC/OC analyzer was operated at each of the study sites for at least three years. Results from this pilot study are available in an EPA report (U.S. EPA, 2019b). A key finding from the study suggests that when the Sunset instrument was working well, OC and optical EC were comparable to CSN OC and EC; however, the time and resources needed to keep a Sunset analyzer operational did not merit replacement of CSN OC and EC measurements.

As of 2018, continuous sulfate is measured at four remaining monitoring sites, one in Maine and three in New York State. Several other stations have historical data but are no longer monitoring continuous sulfate. Discontinuing monitoring efforts for continuous sulfate is likely an outcome of the significantly lower sulfate concentrations throughout the east where these methods were operated. The continuous sulfate analyzer provides hourly data and these data can be readily compared to 24-hour sulfate data which are collected from the ion channel in both the CSN and IMPROVE programs.

In addition, over the last few years, the EPA has investigated the use of several PM sensor technologies as one of several areas of research intended to address the next generation of air measurements. The investigation into air sensors is envisioned to work towards near real-time or continuous measurement options that are smaller, cheaper, and more portable than traditional FRM or FEM methods. These sensor devices have the potential to be used in several applications such as identifying hotspots, informing network design, providing personal exposure monitoring, supporting risk assessments, and providing background concentration data for permitting. The EPA has hosted workshops and published several documents and peer-reviewed articles on this work.<sup>22</sup>

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<sup>21</sup> The six sites that participated in the study were Washington, DC; Chicago, IL; St. Louis, MO; Houston, TX; Las Vegas, NV; and Los Angeles, CA.

<sup>22</sup> For more information, see <https://www.epa.gov/sciencematters/epas-next-generation-air-measuring-research> and <https://www.epa.gov/air-sensor-toolbox/air-sensor-toolbox-what-epa-doing#pane-1>.



## 2.3 AMBIENT AIR CONCENTRATIONS

This section summarizes available information on recent ambient PM concentrations. Section 2.3.1 presents trends in emissions of PM and precursor gases, while section 2.3.2 presents trends in monitored ambient concentrations of PM in the U.S. Section 2.3.3 discusses approaches for predicting ambient PM<sub>2.5</sub> by hybrid modeling approaches.

### 2.3.1 Trends in Emissions of PM and Precursor Gases

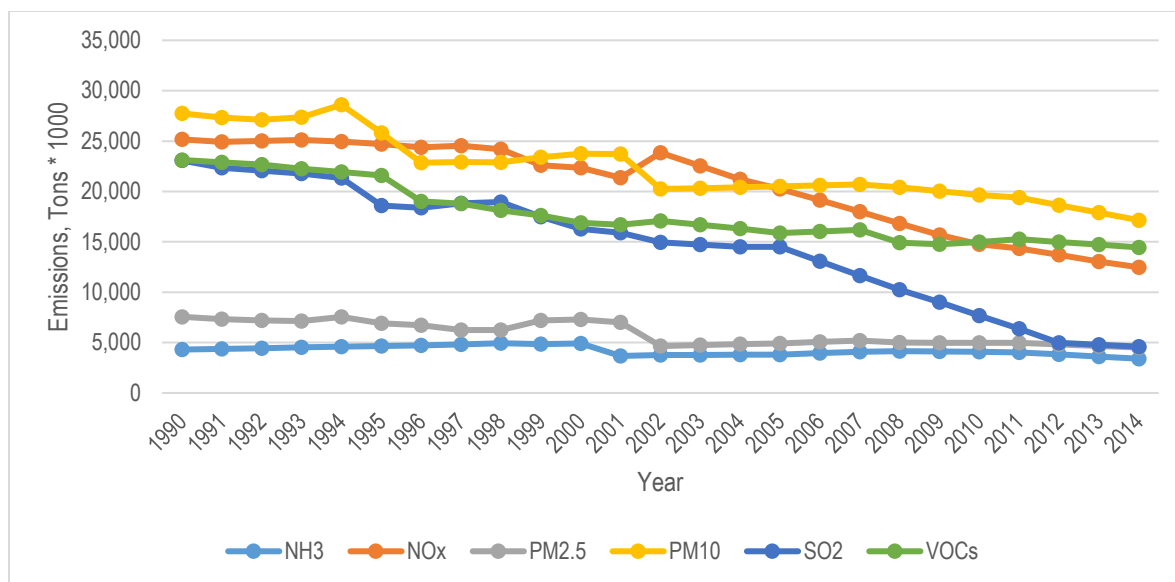
Direct emissions of PM have remained relatively unchanged in recent years, while emissions of some precursor gases have declined substantially.<sup>23</sup> As illustrated in Figure 2-14,<sup>24</sup> from 1990 to 2014, SO<sub>2</sub> emissions have undergone the largest declines while NH<sub>3</sub> emissions have undergone the smallest change. Declining SO<sub>2</sub> emissions during this time period are primarily a result of reductions at stationary sources such as EGUs, with substantial reductions also from mobile sources (U.S. EPA, 2019a, section 2.3.2.1). In more recent years (i.e., 2002 to 2014), emissions of SO<sub>2</sub> and NO<sub>x</sub> have undergone the largest declines, while direct PM<sub>2.5</sub> and NH<sub>3</sub> emissions have undergone the smallest changes, as shown in Table 2-1. Regional trends in emissions can differ from the national trends illustrated in Figure 2-14 and Table 2-1.<sup>25</sup> For example, Hand et al. (2012) studied reductions in EGU-related annual SO<sub>2</sub> emissions during the 2001–2010 period and found that while SO<sub>2</sub> emissions decreased throughout the U.S. by an average of 6.2% per year, the amount of change varied across the U.S. with the largest percent reductions in the western U.S. at 20.1% per year.

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<sup>23</sup> More information on these trends, including details on methods and explanations on the noted changes over time is available at <https://gispub.epa.gov/neireport/2014/>.

<sup>24</sup> Emission trends in Figure 2-14 do not include wildfire emissions.

<sup>25</sup> State-specific emission trends data for 1990 to 2014 can be found at: <https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data>.



**Figure 2-14. National emission trends of PM<sub>2.5</sub>, PM<sub>10</sub>, and precursor gases from 1990 to 2014.**

**Table 2-1. Percent Changes in PM and PM precursor emissions in the NEI for the time periods 1990-2014 and 2002-2014.**

Pollutant	Percent Change in Emissions: 1990 to 2014	Percent Change in Emissions: 2002 to 2014	Major Sources
NH <sub>3</sub>	-21%	-10%	Agricultural Sources (Fertilizer and Livestock Waste), Fires
NO <sub>x</sub>	-50%	-48%	EGUs, Mobile Sources
SO <sub>2</sub>	-80%	-69%	EGUs, other Stationary Sources
VOCs	-38%	-15%	Solvents, Fires, Mobile Sources
PM <sub>2.5</sub>	-40%	-4%	Dust, Fires
PM <sub>10</sub>	-38%	-15%	Dust, Fires

## 2.3.2 Trends in Monitored Ambient Concentrations

### 2.3.2.1 National Characterization of PM<sub>2.5</sub> Mass

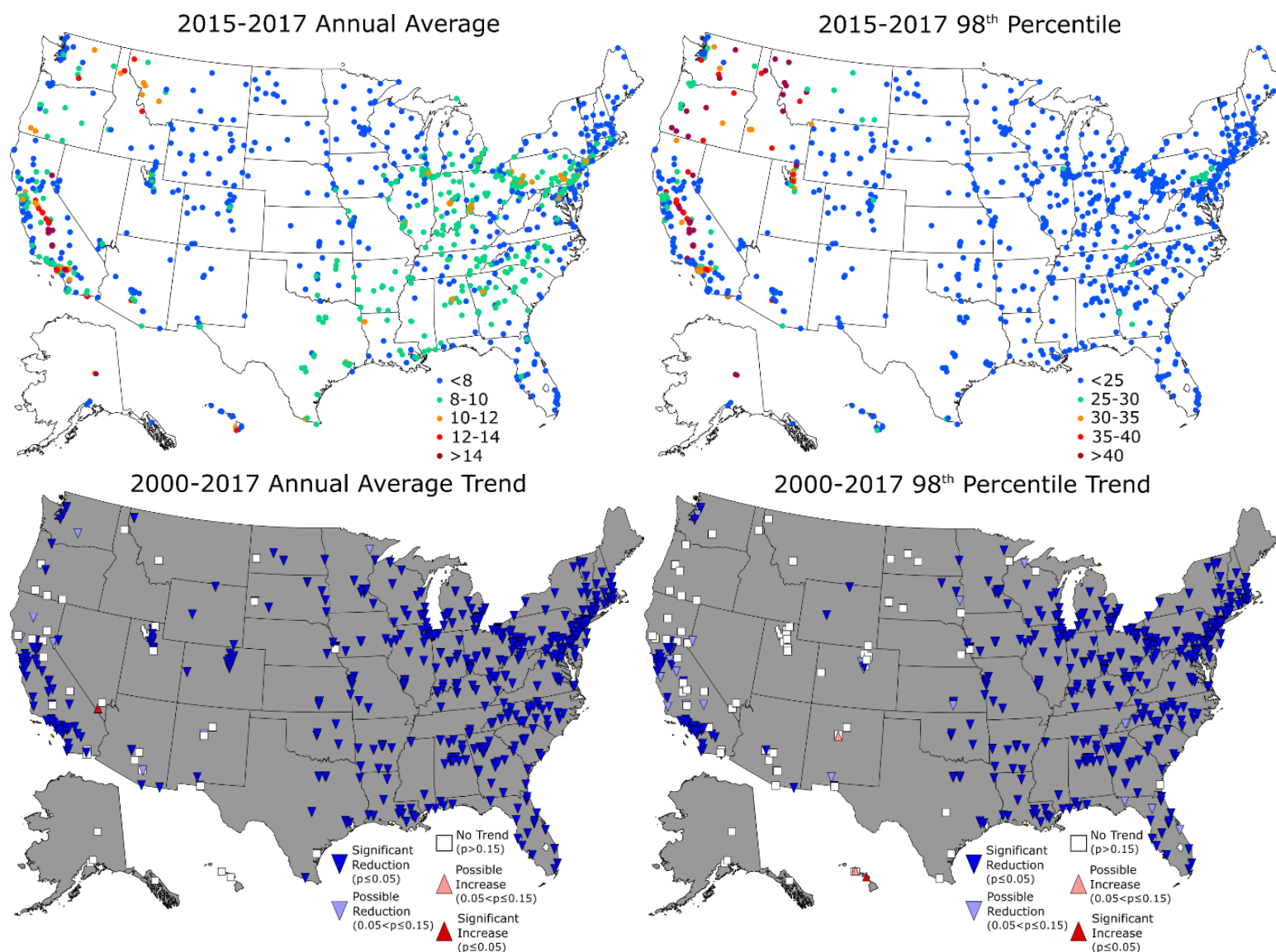
At long-term monitoring sites in the U.S., annual PM<sub>2.5</sub> concentrations from 2015 to 2017 averaged 8.0 µg/m<sup>3</sup> (ranging from 3.0 to 18.2 µg/m<sup>3</sup>) and the 98<sup>th</sup> percentiles of 24-hour concentrations averaged 20.9 µg/m<sup>3</sup> (ranging from 9.2 to 111 µg/m<sup>3</sup>). Figure 2-15 (top panels) shows that the highest ambient PM<sub>2.5</sub> concentrations occur in the west, particularly in California and the Pacific northwest. Much of the eastern U.S. has lower ambient concentrations, with annual average concentrations generally at or below 12.0 µg/m<sup>3</sup> and 98<sup>th</sup> percentiles of 24-hour concentrations generally at or below 30 µg/m<sup>3</sup>.

These concentrations are distinct from design values in part because they include days with episodic events like wildfires and dust storms which can have very high PM<sub>2.5</sub> and/or PM<sub>10</sub> concentrations. The EPA's Exceptional Events Rule,<sup>26</sup> most recently updated in 2016, describes the process by which these events can be excluded from the design values used for comparison to the NAAQS. For the remainder of Chapter 2, episodic events are included in the calculations of PM concentrations. When design values are discussed in Chapter 2, regionally-concurred exceptional events (as of July 2019) have been excluded from the analysis.<sup>27</sup>

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<sup>26</sup> The final version of the 2016 Exceptional Events Rule can be accessed at [https://www.epa.gov/sites/production/files/2018-10/documents/exceptional\\_events\\_rule\\_revisions\\_2060-as02\\_final.pdf](https://www.epa.gov/sites/production/files/2018-10/documents/exceptional_events_rule_revisions_2060-as02_final.pdf).

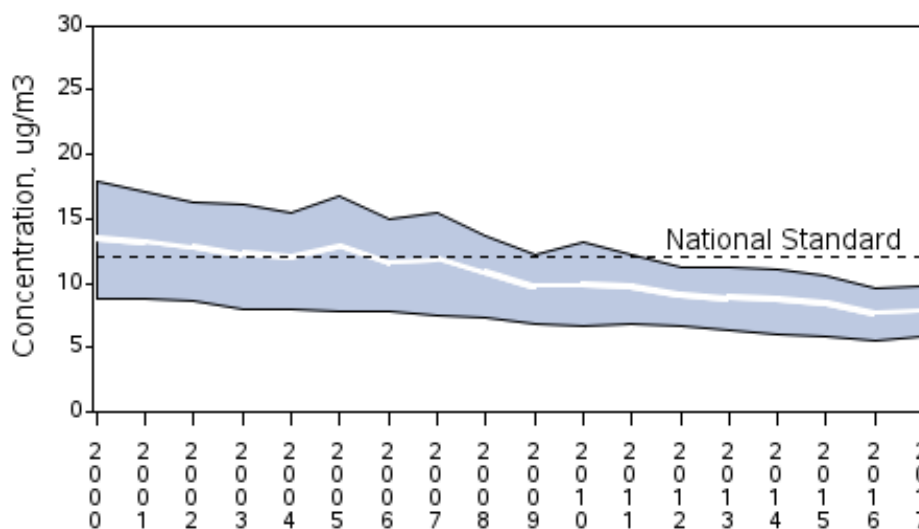
<sup>27</sup> Regionally-concurred exceptional events are unusual or naturally-occurring events such as wildfires or high wind dust events that have 1) resulted in PM<sub>2.5</sub> concentrations above the level of the NAAQS, 2) been submitted by tribal, state or local air agencies under the EPA's Exceptional Events Rule to their respective EPA Region, and 3) received concurrence.



**Figure 2-15. Annual average and 98<sup>th</sup> percentile PM<sub>2.5</sub> concentrations (in  $\mu\text{g}/\text{m}^3$ ) from 2015-2017 (top) and linear trends and their associated significance (based on p-values) in PM<sub>2.5</sub> concentrations from 2000-2017 (bottom).**

Analysis of monthly data indicate distinct peaks in national ambient PM<sub>2.5</sub> concentrations during the summer and the winter (U.S. EPA, 2019a, Figure 2-22). Through 2008, the summer peaks reflected the highest national average PM<sub>2.5</sub> concentrations. These summer peaks in ambient PM<sub>2.5</sub> concentrations were largely a consequence of summertime peaks in SO<sub>2</sub> emissions from power plants in the eastern U.S., and subsequent sulfate formation. However, substantial reductions in SO<sub>2</sub> emissions (see above and U.S. EPA, 2019a, sections 2.5.1.1.1 and 2.5.2.2.1) have changed this pattern. Starting in 2009, winter peaks in national average PM<sub>2.5</sub> concentrations have been higher than those in the summer (U.S. EPA, 2019a, section 2.5.2.2.1). This pattern is illustrated by data from 2013 to 2015, when average winter PM<sub>2.5</sub> concentrations were about 11 µg/m<sup>3</sup>, average summer concentrations were about 9 µg/m<sup>3</sup>, and average spring and fall concentrations were about 7 µg/m<sup>3</sup> (Chan et al., 2018).

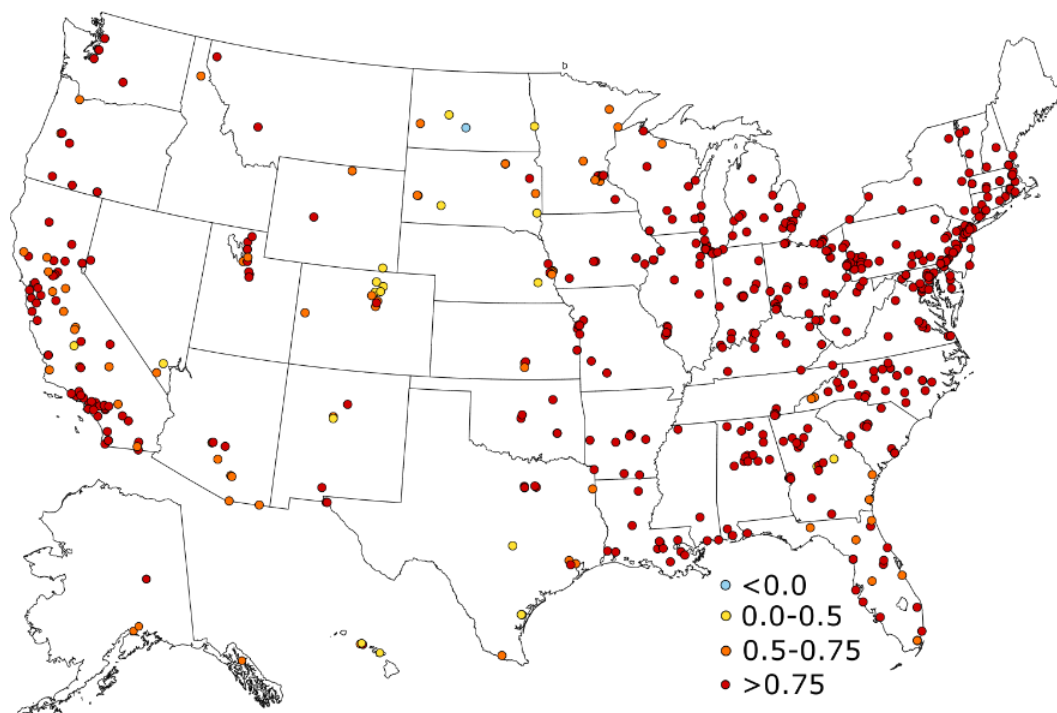
The ambient PM<sub>2.5</sub> concentrations in Figure 2-15 reflect the substantial reductions that have occurred across much of the U.S. over recent years (Figure 2-15, bottom panels and Figure 2-16). From 2000 to 2017, national annual average PM<sub>2.5</sub> concentrations have declined from 13.5 µg/m<sup>3</sup> to 8.0 µg/m<sup>3</sup>, a 41% decrease (Figure 2-16).<sup>28</sup> These declines have occurred at both urban and rural monitoring sites, although urban PM<sub>2.5</sub> concentrations remain consistently higher than those in rural areas (Chan et al., 2018) due to the so-called “urban increment” of PM<sub>2.5</sub> from local sources in an urban area that is additive to the regional and natural background PM<sub>2.5</sub> concentrations.



**Figure 2-16. Seasonally-weighted annual average PM<sub>2.5</sub> concentrations in the U.S. from 2000 to 2017 (429 sites).** (Note: The white line indicates the mean concentration while the gray shading denotes the 10<sup>th</sup> and 90<sup>th</sup> percentile concentrations.)

<sup>28</sup> See <https://www.epa.gov/air-trends/particulate-matter-pm25-trends> and <https://www.epa.gov/air-trends/particulate-matter-pm25-trends#pmnat> for more information.

Analyses at individual monitoring sites indicate that declines in ambient PM<sub>2.5</sub> concentrations have been most consistent across the eastern U.S. and in parts of coastal California, where both annual average and 98<sup>th</sup> percentiles of 24-hour concentrations have declined significantly (Figure 2-15, bottom panels). In contrast, trends in ambient PM<sub>2.5</sub> concentrations have been less consistent over much of the western U.S., with no significant changes since 2000 observed at some sites in the Pacific northwest, the northern Rockies and plains, and the southwest, particularly for 98<sup>th</sup> percentiles of 24-hour concentrations (Figure 2-15, bottom panels). Trends in annual average PM<sub>2.5</sub> concentrations have been highly correlated with trends in 98<sup>th</sup> percentiles of 24-hour concentrations at individual sites (Figure 2-17). Such correlations are highest across the eastern U.S. and in coastal California, and are somewhat lower, though still generally positive, at sites in the Central and Western U.S. (i.e., outside of coastal California).



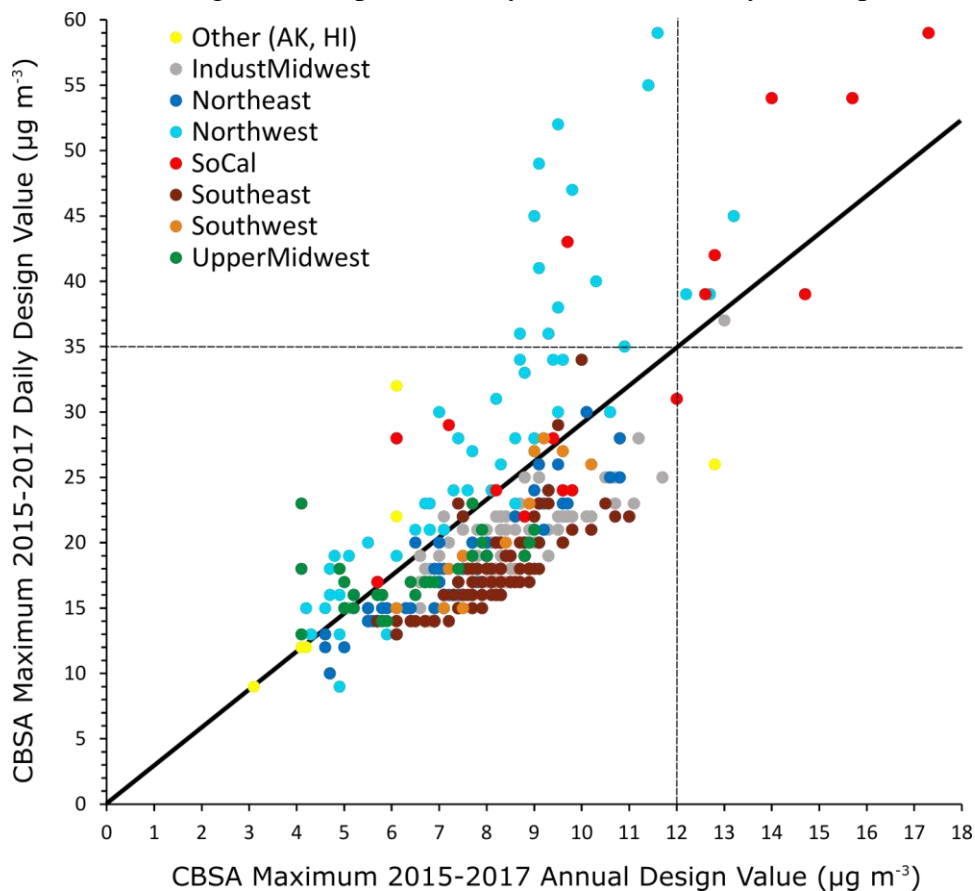
**Figure 2-17. Pearson's correlation coefficient between annual average and 98<sup>th</sup> percentile of 24-hour PM<sub>2.5</sub> concentrations from 2000-2017.**

### **2.3.2.2 Characterization of PM<sub>2.5</sub> Mass at Finer Spatial and Temporal Scales**

#### **2.3.2.2.1 CBSA Maximum Annual Versus Daily Design Values**

Analysis of recent air quality indicates that maximum annual and daily PM<sub>2.5</sub> design values within a CBSA are positively correlated with some noticeable regional variability (Figure 2-18). In the Southeast, Northeast, and Industrial Midwest regions, the annual design values are high relative to the daily design values due in part to the infrequent impacts of episodic events

like wildfire or dust storms. On the other hand, the Northwest region has very high daily design values relative to the annual design values. This is due to episodically high PM<sub>2.5</sub> concentrations that affect the region, both from wintertime stagnation events and summer/fall wildfire smoke events.<sup>29</sup> The relatively small population and low emissions in the region result in much lower PM<sub>2.5</sub> concentrations during the other parts of the year not affected by these episodes.



**Figure 2-18. Scatterplot of CBSA maximum annual versus daily design values (2015-2017) with the solid black line representing the ratio of daily and annual NAAQS values.**

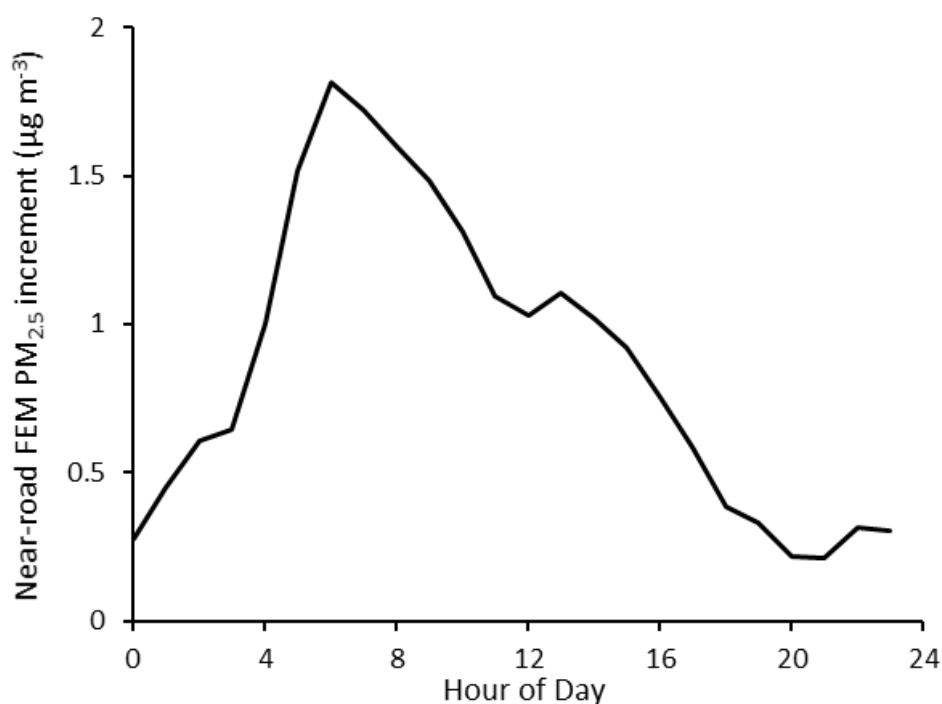
#### 2.3.2.2.2 PM<sub>2.5</sub> Near Major Roadways

Because of its longer atmospheric lifetime (U.S. EPA, 2019a, section 2.2), PM<sub>2.5</sub> is expected to exhibit less spatial variability on an urban scale than UFP or PM<sub>10-2.5</sub> (U.S. EPA, 2019a, section 2.5.1.2.1). Analyses in the 2009 ISA for PM indicated that correlations between PM<sub>2.5</sub> monitoring sites up to a distance of 100 km from each other were greater than 0.75 in most

<sup>29</sup> Due to the recent time period shown in Figure 2-18, it is likely that some of the annual and daily design values are affected by potential exceptional events associated with wildfire smoke that have yet to be regionally-concurred and removed from the design value calculations. The EPA defines exceptional events as unusual or natural-occurring events that affect air quality but are not reasonably controllable using techniques that tribal, state, or local air agencies may implement. This is especially likely for the daily design values in the Northwest region which experienced frequent wildfire smoke events during the 2015-2017 period.

urban areas. However, more substantial spatial variation has been reported for some urban areas, due in part to proximity between monitors and emissions sources (U.S. EPA, 2019a, section 2.5.1.2.1). The recent deployment of PM<sub>2.5</sub> monitors near major roads in large urban areas provides some insight into this spatial variation.

As discussed above, in the last review of the PM NAAQS the EPA required monitoring of PM<sub>2.5</sub>, along with NO<sub>2</sub> and CO, near major roads in CBSAs with populations greater than 1 million. PM<sub>2.5</sub> monitoring was required to start for the largest CBSAs at the beginning of 2015, and several years of data are now available for analysis at these sites. DeWinter et al. (2018) analyzed these data and found that the average near-road increment (difference between near-road PM<sub>2.5</sub> concentrations and the concentrations at other sites in the same CBSA) was 1.2 µg/m<sup>3</sup> for 2014 to 2015. The near-road increment has a diurnal cycle, with a peak during the morning rush hour (Figure 2-19). This near-road increment likely is additive to the urban increment of PM<sub>2.5</sub> from local sources in the CBSA including mobile sources on the numerous non-highway roads that are not monitored by the near-road network.



**Figure 2-19. Network-wide average of the hourly near-road PM<sub>2.5</sub> increment through 2017.**

Analyses of recent data indicate that, of the 25 CBSAs with valid design values<sup>30</sup> at the near-road site(s) from 2015 to 2017, 52% measured the highest annual design value at the near-

<sup>30</sup> A design value is considered valid if it meets the data handling requirements given in 40 CFR Appendix N to Part 50. Several large CBSAs such as Chicago-Naperville-Elgin, IL-IN-WI and Houston-The Woodlands-Sugar Land, TX had near-road sites that did not have valid PM<sub>2.5</sub> design values for the 2015-2017 period.



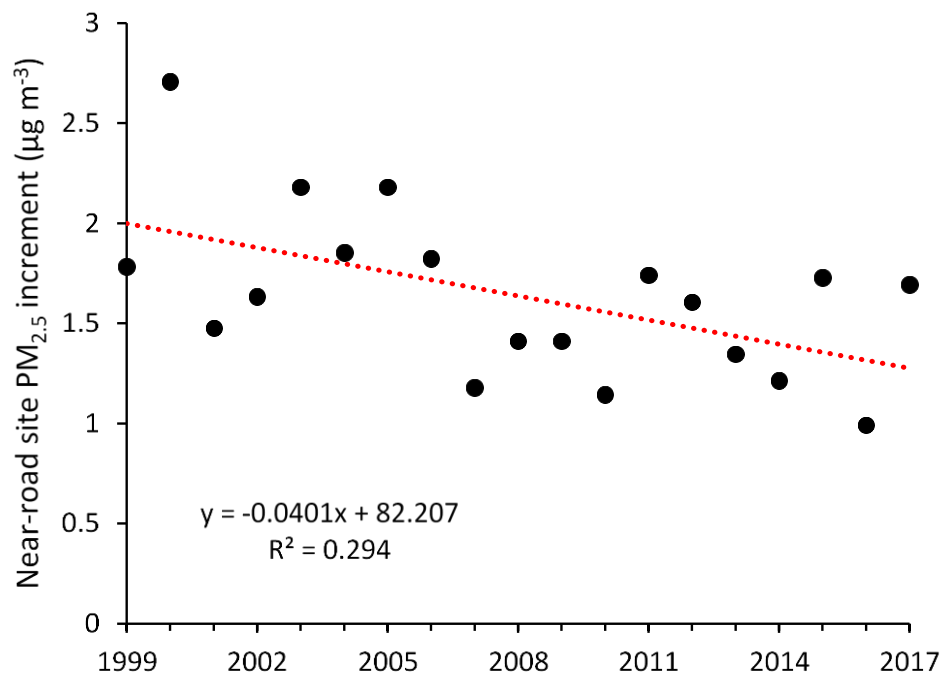
road site while 24% measured the highest 24-hour design value at the near-road site (Table 2-2). Of the CBSAs with highest annual design values at near-road sites, those design values were, on average, 0.7  $\mu\text{g}/\text{m}^3$  higher than at the highest measuring non-near-road sites (range is 0.1 to 2.0  $\mu\text{g}/\text{m}^3$  higher at near-road sites).

**Table 2-2. Daily and annual PM<sub>2.5</sub> design values for the near-road sites in major CBSAs (2015-2017).**

CBSA Name	Maximum Near-Road Daily Design Value	Maximum Non-Near-Road Daily Design Value	Maximum Near-Road Annual Design Value	Maximum Non-Near-Road Annual Design Value
New York-Newark-Jersey City, NY-NJ-PA	22	23	NA	9.7
Los Angeles-Long Beach-Anaheim, CA	33	39	12.6	12.1
Dallas-Fort Worth-Arlington, TX	18	18	8.7	8.9
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	24	25	9.5	10.6
Atlanta-Sandy Springs-Roswell, GA	23	20	10.5	9.9
Boston-Cambridge-Newton, MA-NH	16	16	7	7.2
San Francisco-Oakland-Hayward, CA	27	30	10.1	10.6
Phoenix-Mesa-Scottsdale, AZ	18	27	7.9	9.6
Riverside-San Bernardino-Ontario, CA	37	39	14.7	13.6
Detroit-Warren-Dearborn, MI	22	28	8.5	11.2
Seattle-Tacoma-Bellevue, WA	24	34	8.4	8.7
Minneapolis-St. Paul-Bloomington, MN-WI	18	19	8	7.5
St. Louis, MO-IL	19	21	8.7	9.8
Baltimore-Columbia-Towson, MD	20	23	9.1	8.9
Denver-Aurora-Lakewood, CO	23	20	8.5	7.1
Portland-Vancouver-Hillsboro, OR-WA	25	28	7.4	7.4
Kansas City, MO-KS	16	21	7.1	9.0
Indianapolis-Carmel-Anderson, IN	22	22	10.5	10.2
San Jose-Sunnyvale-Santa Clara, CA	28	27	9.4	9.3
Providence-Warwick, RI-MA	20	18	9.1	7.1
Louisville/Jefferson County, KY-IN	21	22	9.4	9.7
New Orleans-Metairie, LA	18	19	8.2	8.5
Hartford-West Hartford-East Hartford, CT	20	18	8.2	6.7
Birmingham-Hoover, AL	22	22	11	10.4
Buffalo-Cheektowaga-Niagara Falls, NY	17	18	7.8	7.6
Rochester, NY	17	16	7	6.5

Although most near-road monitoring sites do not have sufficient data to evaluate long-term trends in near-road PM<sub>2.5</sub> concentrations, analyses of the data at one near-road-like site in

Elizabeth, NJ,<sup>31</sup> show that the annual average increment has generally decreased between 1999 and 2017 from about 2.0  $\mu\text{g}/\text{m}^3$  to about 1.3  $\mu\text{g}/\text{m}^3$  (Figure 2-20). The trend in the near-road increment of elemental carbon at the Elizabeth, NJ site has shown a similar reduction, with values of  $\sim 1.0 \mu\text{g}/\text{m}^3$  in 2000 decreasing to  $\sim 0.5 \mu\text{g}/\text{m}^3$  in 2017. These data are consistent with the timing of EPA emission standards for motor vehicles.<sup>32</sup> Although long-term data are not available at other near-road sites, the national scope of the diesel vehicle controls suggests the near-road environment across the U.S. likely experienced similar decreasing trends in near-road  $\text{PM}_{2.5}$  increments.



**Figure 2-20. Annual average near-road increment for  $\text{PM}_{2.5}$  at the Elizabeth, NJ site.**

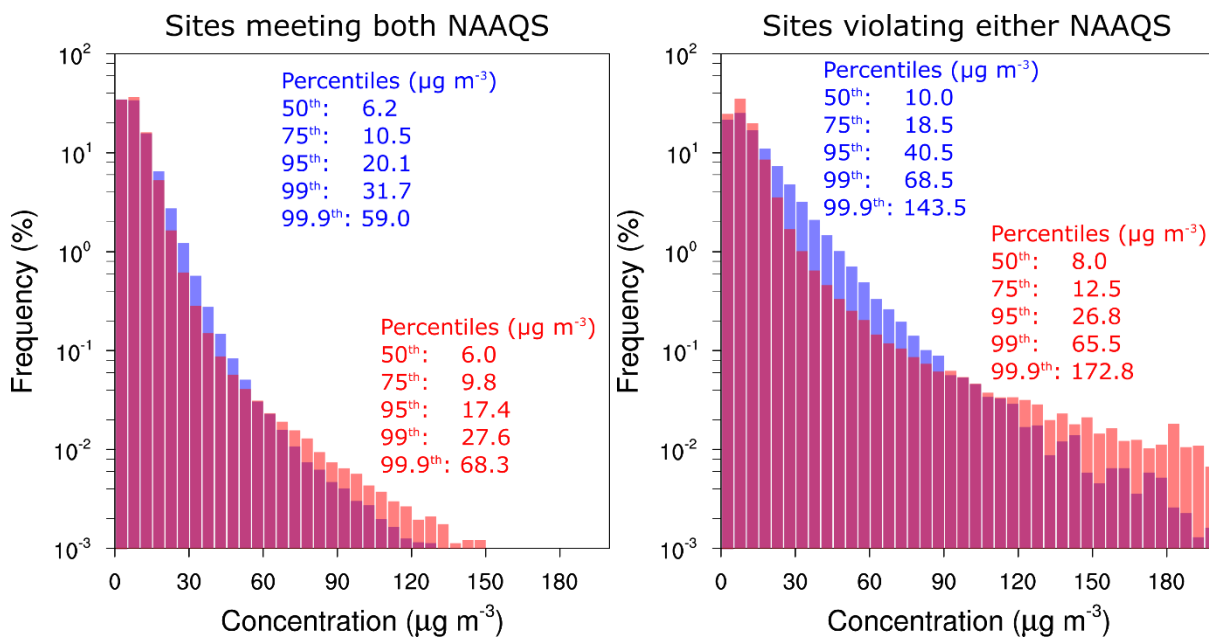
### 2.3.2.2.3 Sub-Daily Concentrations of $\text{PM}_{2.5}$

Ambient  $\text{PM}_{2.5}$  concentrations can exhibit a diurnal cycle that varies due to impacts from intermittent emission sources, meteorology, and atmospheric chemistry. The  $\text{PM}_{2.5}$  monitoring network in the U.S. has an increasing number of continuous FEM monitors reporting hourly  $\text{PM}_{2.5}$  mass concentrations that reflect this diurnal variation. The ISA describes a two-peaked diurnal pattern in urban areas, with morning peaks attributed to rush-hour traffic and afternoon peaks attributed to a combination of rush hour traffic, decreasing atmospheric dilution, and

<sup>31</sup> The Elizabeth Lab site in Elizabeth, NJ is situated approximately 30 meters from travel lanes of the Interchange 13 toll plaza of the New Jersey Turnpike and within 200 meters of travel lanes for Interstate 278 and the New Jersey Turnpike.

<sup>32</sup> See <https://www.epa.gov/diesel-fuel-standards/diesel-fuel-standards-and-rulemakings#nonroad-diesel>.

nucleation (U.S. EPA, 2019a, section 2.5.2.3, Figure 2-32). Because a focus on annual average and 24-hour average  $\text{PM}_{2.5}$  concentrations could mask sub-daily patterns, and because some health studies examine PM exposure durations shorter than 24-hours, it is useful to understand the broader distribution of sub-daily  $\text{PM}_{2.5}$  concentrations across the U.S. Figure 2-21 below presents the frequency distribution of 2-hour average  $\text{PM}_{2.5}$  mass concentrations from all FEM  $\text{PM}_{2.5}$  monitors in the U.S. for 2015-2017.<sup>33</sup> At sites meeting the current primary  $\text{PM}_{2.5}$  standards, these 2-hour concentrations generally remain below  $11 \mu\text{g}/\text{m}^3$ , and virtually never exceed  $32 \mu\text{g}/\text{m}^3$ . Two-hour concentrations are higher at sites violating the current standards, generally remaining below  $19 \mu\text{g}/\text{m}^3$  and virtually never exceeding  $69 \mu\text{g}/\text{m}^3$ .



**Figure 2-21. Frequency distribution of 2015-2017 2-hour averages for sites meeting both or violating either  $\text{PM}_{2.5}$  NAAQS for October to March (blue) and April to September (red).**

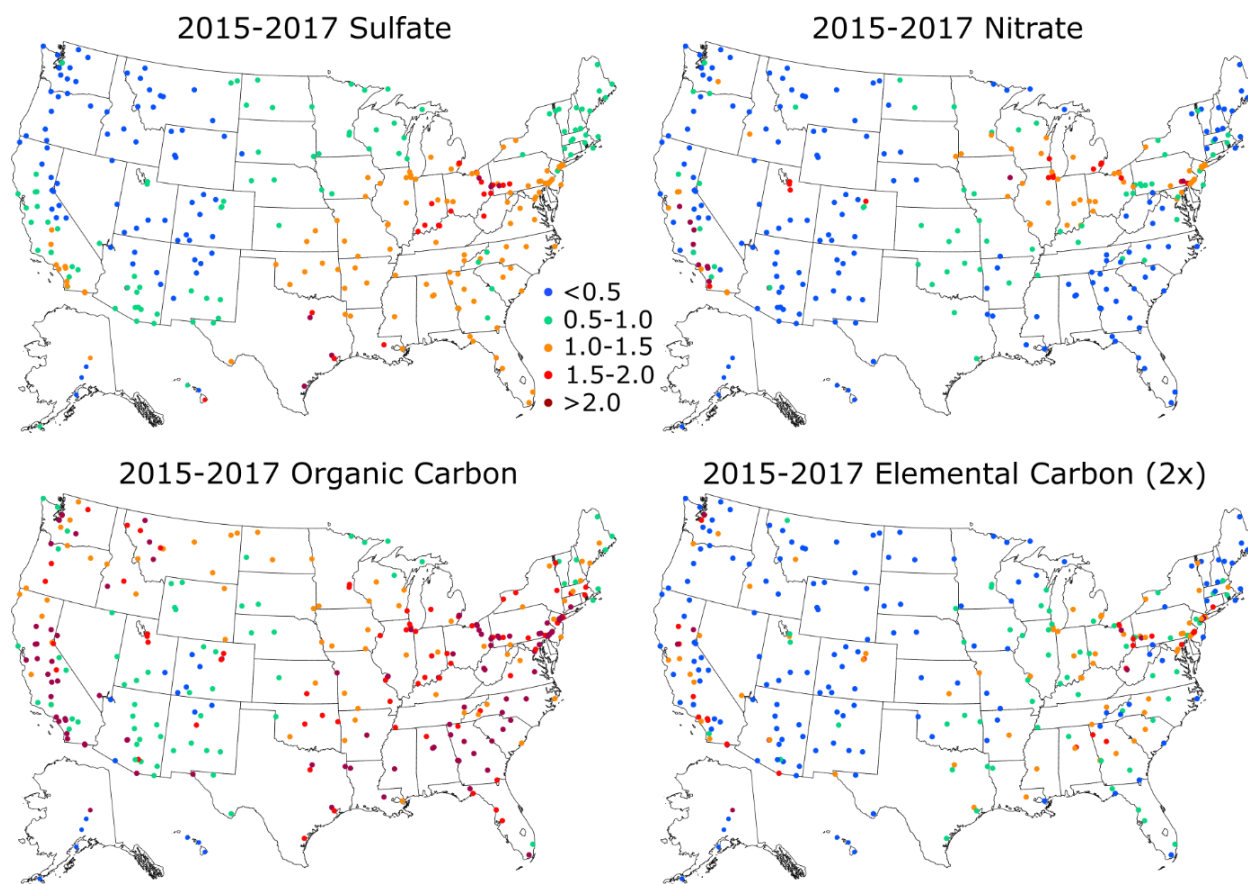
The extreme upper end of the distribution of 2-hour  $\text{PM}_{2.5}$  concentrations is shifted higher during the warmer months (red in Figure 2-21), generally corresponding to the period of peak wildfire frequency (April to September) in the U.S. At sites meeting the current primary standards, the highest 2-hour concentrations measured virtually never occur outside of the period of peak wildfire frequency. Most of the sites measuring these very high concentrations are in the northwestern U.S. and California, where wildfires have been relatively common in recent years

<sup>33</sup> As discussed further in section 3.2,  $\text{PM}_{2.5}$  controlled human exposure studies often examine 2-hour exposures. Thus, when evaluating those studies in the context of the current primary  $\text{PM}_{2.5}$  standards, it is useful to consider the distribution of 2-hour  $\text{PM}_{2.5}$  concentrations. Similar analyses of 5-hour  $\text{PM}_{2.5}$  concentrations are presented in Appendix A, Figure A-2.

(see Appendix A, Figure A-1). When the period of peak wildfire frequency is excluded from the analysis (blue in Figure 2-21), the extreme upper end of the distribution is reduced.

### 2.3.2.3 Chemical Composition of PM<sub>2.5</sub>

Based on recent air quality data, the major chemical components of PM<sub>2.5</sub> have distinct spatial distributions. Sulfate concentrations tend to be highest in the eastern U.S., while in the Ohio Valley, Salt Lake Valley, and California nitrate concentrations are highest and relatively high concentrations of organic carbon are widespread across most of the Continental U.S., as shown in Figure 2-22. Elemental carbon, crustal material, and sea-salt are found to have the highest concentrations in the northeast U.S., southwest U.S., and coastal areas, respectively.



**Figure 2-22. Annual average PM<sub>2.5</sub> sulfate, nitrate, organic carbon, and elemental carbon concentrations (in µg/m<sup>3</sup>) from 2015-2017.**

An examination of PM<sub>2.5</sub> composition trends can provide insight into the factors contributing to overall reductions in ambient PM<sub>2.5</sub> concentrations. The biggest change in PM<sub>2.5</sub> composition that has occurred in recent years is the reduction in sulfate concentrations due to reductions in SO<sub>2</sub> emissions. Between 2000 and 2015, the nationwide annual average sulfate concentration decreased by 17% at urban sites and 20% at rural sites. This change in sulfate

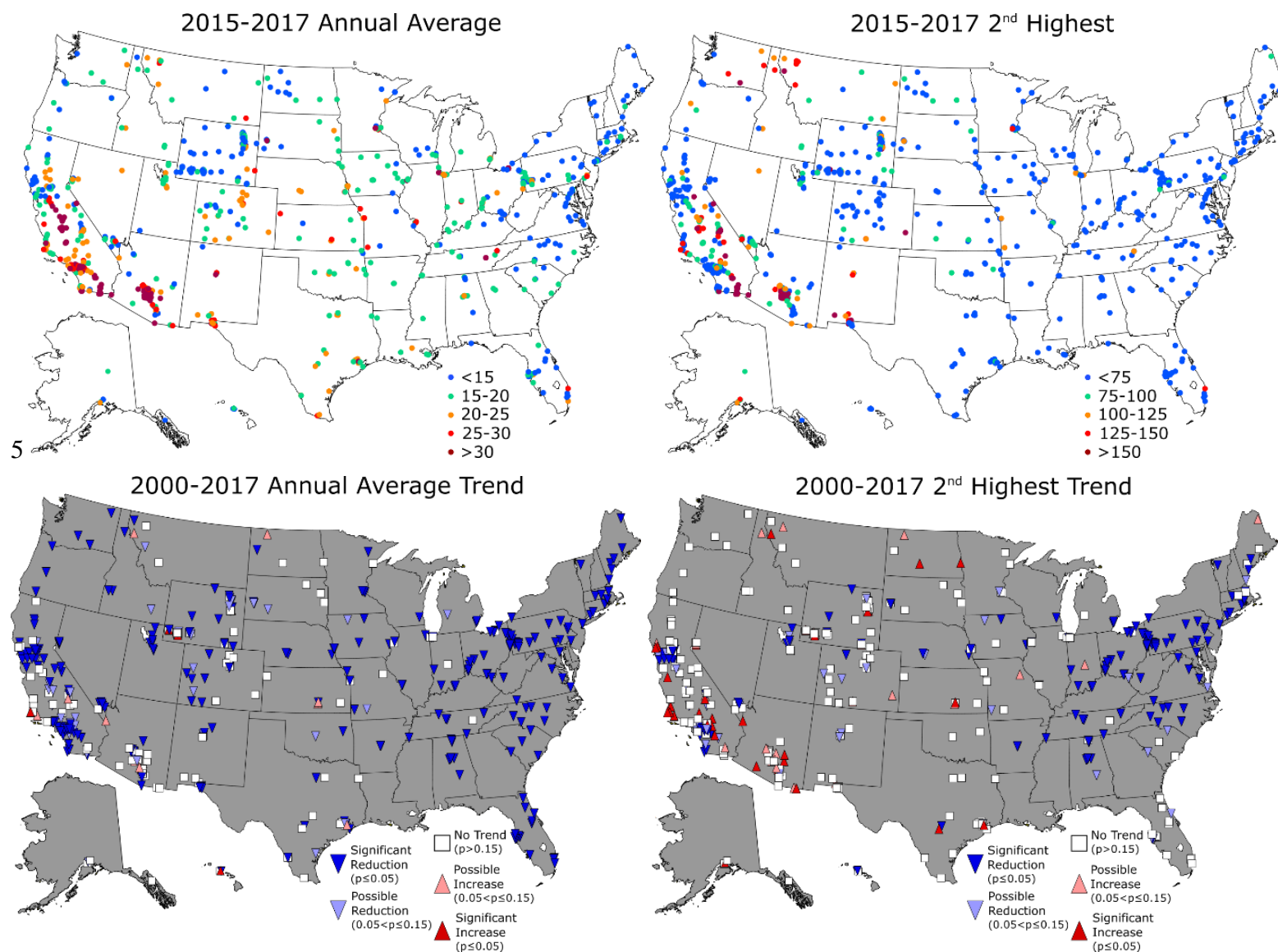
concentrations is most evident in the eastern U.S. and has resulted in organic matter or nitrate now being the greatest contributor to PM<sub>2.5</sub> mass in many locations (U.S. EPA, 2019a, Figure 2-19). The overall reduction in sulfate concentrations has contributed substantially to the decrease in national average PM<sub>2.5</sub> concentrations as well as the decline in the fraction of PM<sub>10</sub> mass accounted for by PM<sub>2.5</sub> (U.S. EPA, 2019a, section 2.5.1.1.6; section 2.3.1 above).

#### **2.3.2.4 National Characterization of PM<sub>10</sub> Mass**

At long-term monitoring sites in the U.S., the 2015-2017 average of 2<sup>nd</sup> highest 24-hour PM<sub>10</sub> concentration was 56 µg/m<sup>3</sup> (ranging from 18 to 173 µg/m<sup>3</sup>) (Figure 2-23, top panels).<sup>34</sup> The highest PM<sub>10</sub> concentrations tend to occur in the western U.S. Seasonal analyses indicate that ambient PM<sub>10</sub> concentrations are generally higher in the summer months than at other times of year, though the most extreme high concentration events are more likely in the spring (U.S. EPA, 2019a, Table 2-5). This is due to fact that the major PM<sub>10</sub> emission sources, dust and agriculture, are more active during the warmer and drier periods of the year.

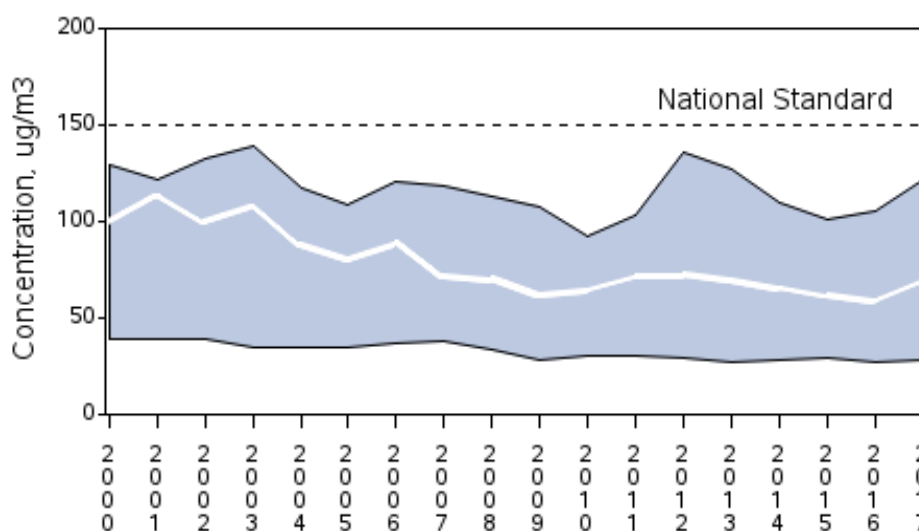
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<sup>34</sup> The form of the current 24-hour PM<sub>10</sub> standard is one-expected-exceedance, averaged over three years.



**Figure 2-23. Annual average and 2<sup>nd</sup> highest PM<sub>10</sub> concentrations (in  $\mu\text{g}/\text{m}^3$ ) from 2015-2017 (top) and linear trends and their associated significance in PM<sub>10</sub> concentrations from 2000-2017 (bottom).**

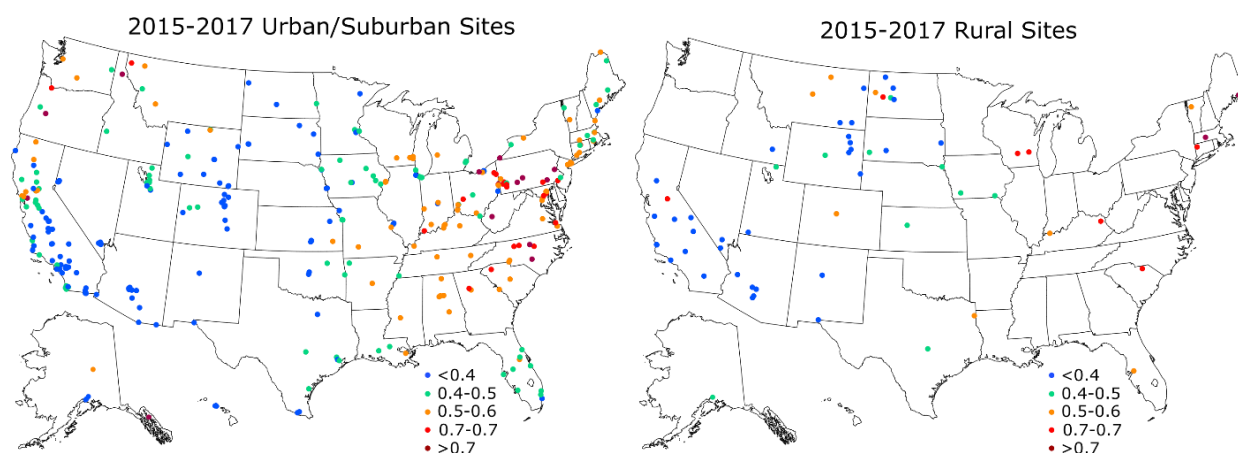
Recent ambient PM<sub>10</sub> concentrations reflect reductions that have occurred across much of the U.S. (Figure 2-23, bottom panels). From 2000 to 2017, 2<sup>nd</sup> highest 24-hour PM<sub>10</sub> concentrations have declined by about 30% (Figure 2-24).<sup>35</sup> Analyses at individual monitoring sites indicate that annual average PM<sub>10</sub> concentrations have declined at most sites across the U.S., with much of the decrease in the eastern U.S. associated with reductions in PM<sub>2.5</sub> concentrations. Annual second highest 24-hour PM<sub>10</sub> concentrations have generally declined in the eastern U.S., while concentrations in much of the midwest and western U.S. have remained unchanged or increased since 2000 (Figure 2-23, bottom panels).



**Figure 2-24. National trends in Annual 2<sup>nd</sup> Highest 24-Hour PM<sub>10</sub> concentrations from 2000 to 2017 (131 sites).** (Note: The white line indicates the mean concentration while the gray shading denotes the 10<sup>th</sup> and 90<sup>th</sup> percentile concentrations.)

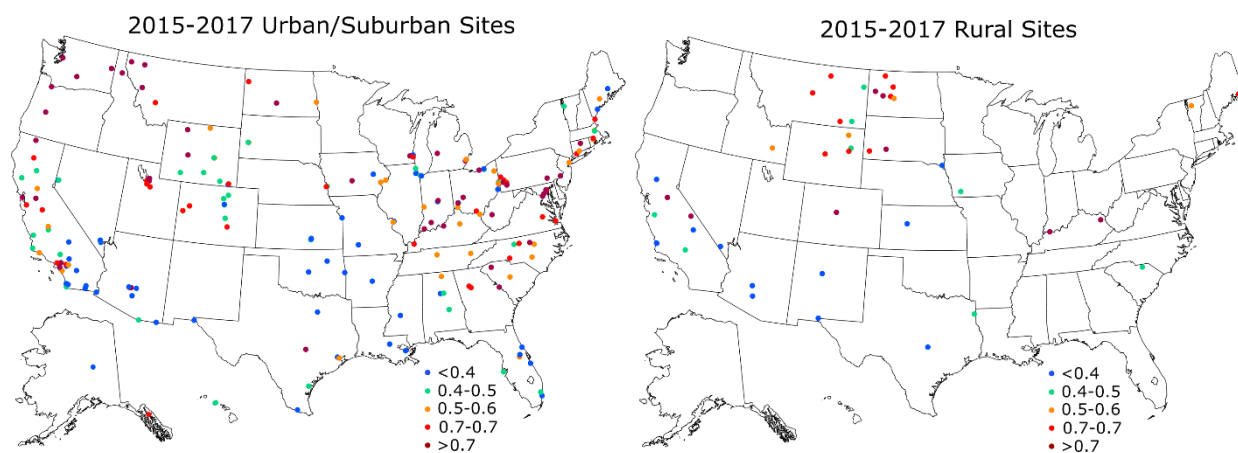
Compared to previous reviews, data available from the NCore monitoring network in the current review allows a more comprehensive analysis of the relative contributions of PM<sub>2.5</sub> and PM<sub>10-2.5</sub> to PM<sub>10</sub> mass. PM<sub>2.5</sub> generally contributes more to annual average PM<sub>10</sub> mass in the eastern U.S. than the western U.S. (Figure 2-25). At most sites in the eastern U.S., the majority of PM<sub>10</sub> mass is comprised of PM<sub>2.5</sub>. Similar east-west patterns are observed for both urban/suburban and rural sites. As ambient PM<sub>2.5</sub> concentrations have declined in the eastern U.S. (section 2.3.2.2, above), the ratios of PM<sub>2.5</sub> to PM<sub>10</sub> have also declined.

<sup>35</sup> For more information, see <https://www.epa.gov/air-trends/particulate-matter-pm10-trends#pmnat>.



**Figure 2-25. Annual average  $PM_{2.5}/PM_{10}$  ratio for 2015-2017.**

For days with very high  $PM_{10}$  concentrations (Figure 2-26), the  $PM_{2.5}/PM_{10}$  ratios are typically higher than the annual average ratios. This is particularly true in the northwestern U.S. where the high  $PM_{10}$  concentrations can occur during wildfires with high  $PM_{2.5}$ .



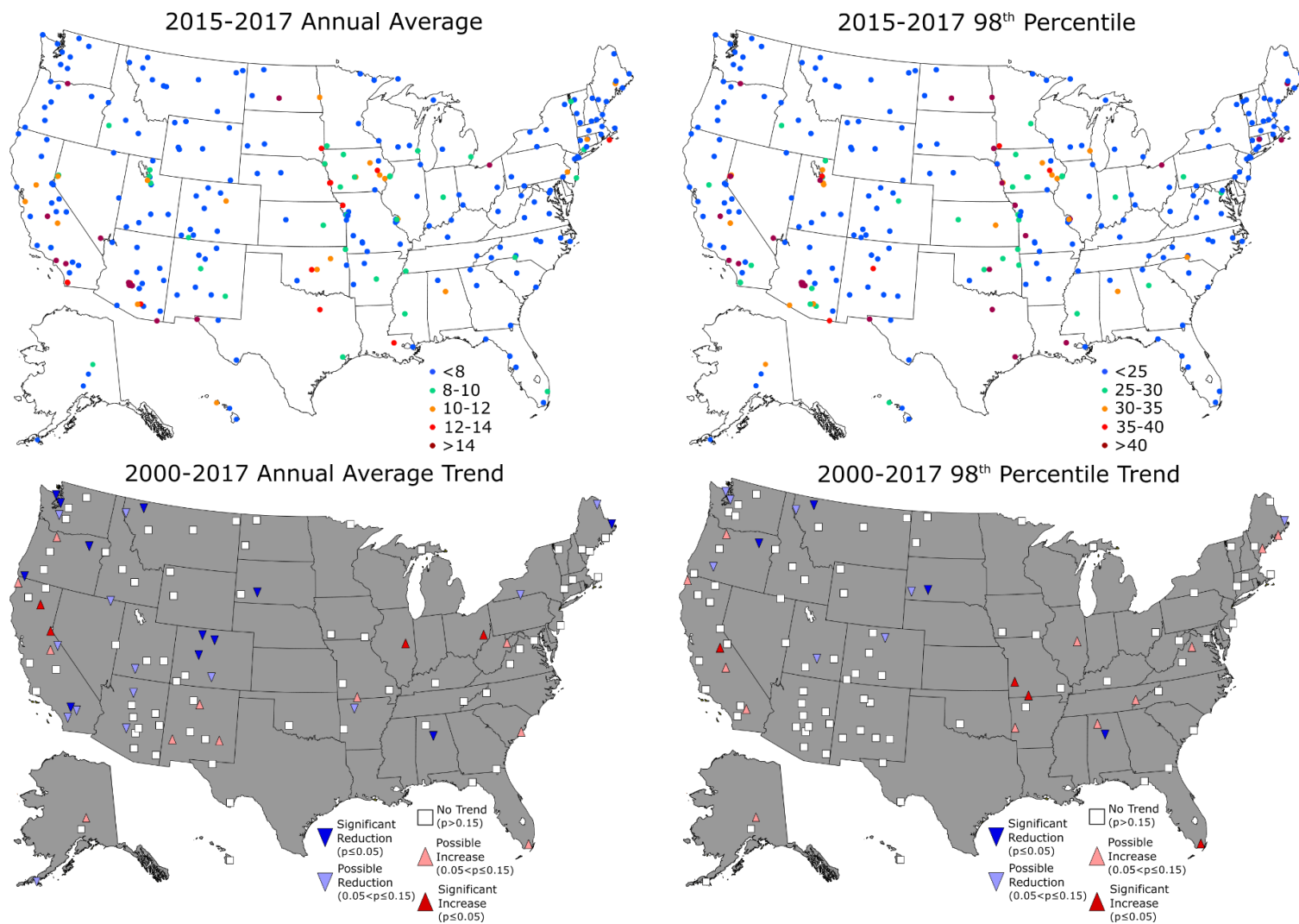
**Figure 2-26.  $PM_{2.5}/PM_{10}$  ratio for the second highest  $PM_{10}$  concentrations for 2015-2017.**

### 2.3.2.5 National Characterization of $PM_{10-2.5}$ Mass

Since the last review, the availability of  $PM_{10-2.5}$  ambient concentration data has greatly increased. As illustrated in Figure 2-27<sup>36</sup> (top panels), annual average and 98<sup>th</sup> percentile  $PM_{10-2.5}$  concentrations exhibit less distinct differences between the eastern and western U.S. than for either  $PM_{2.5}$  or  $PM_{10}$ . Additionally, compared to  $PM_{2.5}$  and  $PM_{10}$ , changes in  $PM_{10-2.5}$  concentrations have been small in magnitude and inconsistent in direction (Figure 2-27, lower panels).

<sup>36</sup> The sites shown in Figure 2-27 have a data completeness of either 75% or  $\geq 182$  valid days in each year.

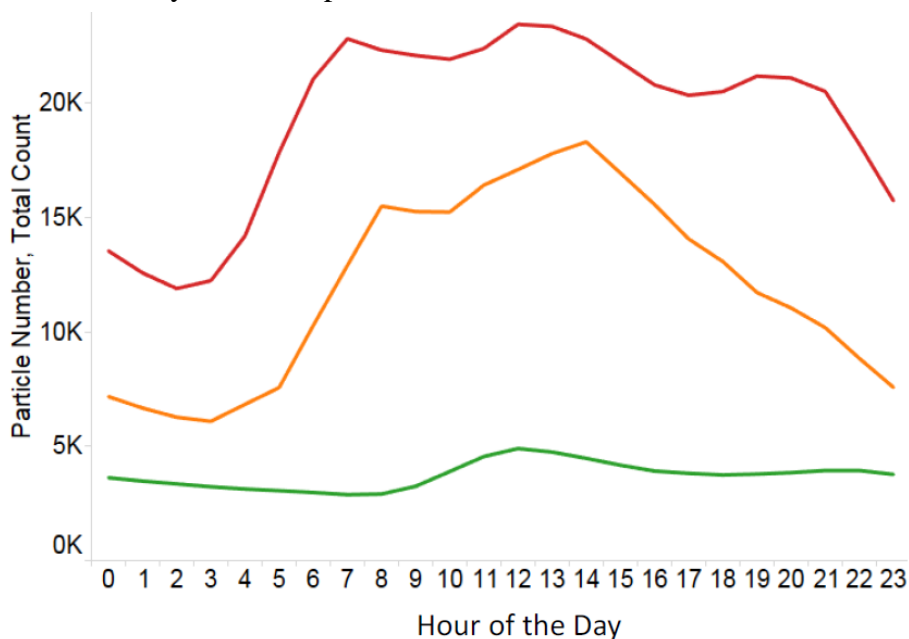




**Figure 2-27. Annual average and 98<sup>th</sup> percentile PM<sub>10-2.5</sub> concentrations ( $\mu\text{g}/\text{m}^3$ ) from 2015-2017 (top) and linear trends and their associated significance in PM<sub>10-2.5</sub> concentrations from 2000-2017 (bottom).**

### 2.3.2.6 Characterization of the Ultrafine Fraction of PM<sub>2.5</sub> Mass

Compared to PM<sub>2.5</sub> mass, there is relatively little data on U.S. particle number concentrations, which are dominated by UFP. In the published literature, annual average particle number concentrations reaching about 20,000 to 30,000 cm<sup>-3</sup> have been reported in U.S. cities (U.S. EPA, 2019a). In addition, based on UFP measurements in two urban areas (New York City, Buffalo) and at a background site (Steuben County) in New York, there is a pronounced difference in particle number concentration between different types of locations (Figure 2-28; U.S. EPA, 2019a, Figure 2-18). Urban particle number counts were several times higher than at the background site, and the highest particle number counts in an urban area with multiple sites (Buffalo) were observed at a near-road location. Hourly data indicate that particle numbers remain fairly constant throughout the day at the background site, that they peak around 8:00 a.m. in Buffalo and New York City (NYC), and that they remain high into the evening hours with distinct rush hour and early afternoon peaks.

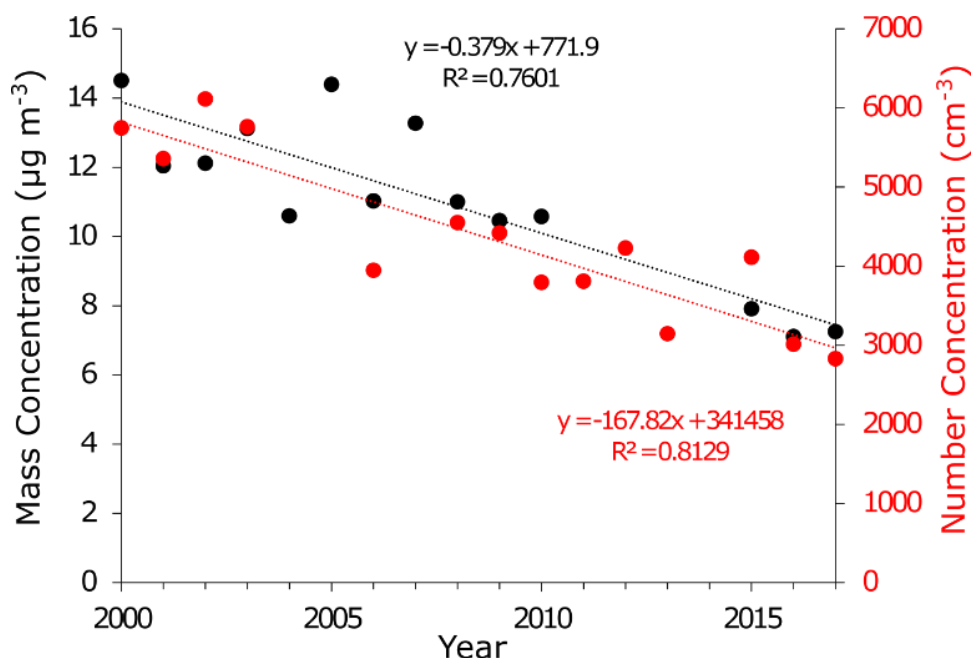


**Figure 2-28. Average hourly particle number concentrations from three locations in the State of New York for 2014 to 2015 (green is Steuben County, orange is Buffalo, red is New York City).** (Source: Figure 2-18 in U.S. EPA, 2019a).

Long-term trends in UFP are generally not available at U.S. monitoring sites. However, data on number size distribution have been reported for an 8-year period from 2002 to 2009 in Rochester, NY. Number concentrations averaged 4,730 cm<sup>-3</sup> for 0.01 to 0.05 µm particles and 1,838 cm<sup>-3</sup> for 0.05 to 0.1 µm particles (Wang et al., 2011). On average over the 8 years that UFP data were collected in Rochester, total particle number concentrations declined from the earlier period evaluated (i.e., 2001 to 2005) to the later period (2006 to 2009). This decline was

most evident for particles between 0.01 and 0.1  $\mu\text{m}$  and was attributed to changes in local sources resulting from the 2007 Heavy Duty Highway Rule, a reduction in local industrial activity, and the closure of a nearby coal-fired power plant (Wang et al., 2011; U.S. EPA, 2019a, section 2.5.2.1.4).

In addition, at a site in Illinois the annual average particle number concentration declined between 2000 and 2017, closely matching the reductions in annual  $\text{PM}_{2.5}$  mass over that same period (Figure 2-29, below). Particle number concentrations at this site are closer to those of the background site in Figure 2-28 than the urban sites. A recent study found that particle number concentrations in an urban area (Pittsburgh, PA) decreased between 2001-2002 and 2016-2017 along with decreases in  $\text{PM}_{2.5}$  associated with  $\text{SO}_2$  emission reductions (Saha et al., 2018). However, the relationship between changes in ambient  $\text{PM}_{2.5}$  and UFPs cannot be comprehensively characterized due to the high variability and limited monitoring of UFPs.



**Figure 2-29. Time series of annual average mass and number concentrations (left) and scatterplot of mass vs. number concentration (right) between 2000-2017 in Bondville, IL.**

### 2.3.3 Predicted Ambient $\text{PM}_{2.5}$ Based on Hybrid Modeling Approaches

Ambient concentrations of  $\text{PM}_{2.5}$  are often characterized using measurements from national monitoring networks due to the accuracy and precision of the measurements and the public availability of data. For applications requiring  $\text{PM}_{2.5}$  characterizations across urban areas, data averaging techniques such as area-wide and population-weighted averaging of monitors are sometimes used to provide complete coverage from the site measurements (U.S. EPA, 2019a, chapter 3). Yet data averaging methods may not adequately represent the spatial heterogeneity of

PM<sub>2.5</sub> within an area and are not practical for large unmonitored areas or time periods. As a result, additional methods have been developed to improve PM<sub>2.5</sub> characterizations in areas where monitoring is relatively sparse or unavailable. Methods include interpolation of monitored data, land-use regression models, chemical-transport models (CTMs), models based on satellite-derived aerosol optical depth (AOD), and hybrid spatiotemporal models that combine information from the individual approaches (U.S. EPA, 2019a, chapter 3). A number of recent studies have employed such methods to estimate PM<sub>2.5</sub> air quality concentrations across the U.S. and Canada, and to estimate population exposures for use in epidemiologic analyses (U.S. EPA, 2019a, sections 3.3 and 3.4). Given the increasing availability and application of these methods, in this section we provide an overview of recently developed hybrid modeling methods, their predictions and performance, and how predictions from various methods compare to each other.

#### **2.3.3.1.1 Overview of Hybrid Methods**

Hybrid methods are broadly classified into four categories: (1) methods based primarily on interpolation of monitor data, (2) Bayesian statistical downscalers, (3) methods based primarily on satellite-derived AOD, and (4) methods based on machine-learning algorithms. Each method is discussed briefly below.

Interpolation-based methods are the simplest approach for developing spatial fields of PM<sub>2.5</sub> concentrations and rely on the moderate degree of spatial autocorrelation in PM<sub>2.5</sub> in many areas of the U.S. Interpolation methods often use inverse-distance or inverse-distance-squared weighted averaging of monitoring data to predict PM<sub>2.5</sub> concentrations at unmonitored receptor points. Examples include the Voronoi neighbor averaging (VNA) approach and the enhanced VNA approach (eVNA). The VNA approach applies weighted averaging to the concentrations monitored in the Voronoi cells neighboring the cell containing the prediction point (Abt Associates, 2014). In the eVNA approach, monitored data are further weighted by the ratio of CTM predictions in the grid-cell containing the prediction point to the grid-cell containing the monitor (Abt Associates, 2014).

Bayesian statistical modeling has been used to calibrate CTM PM<sub>2.5</sub> predictions or satellite-derived AOD estimates to surface measurements (Berrocal et al., 2012; Wang et al., 2018b). This approach, commonly referred to as a Bayesian downscaler because it “downscales” grid-cell average values to points, first regresses the PM<sub>2.5</sub> predictions or AOD estimates on monitoring data. The resulting relationships are then used to develop a gridded PM<sub>2.5</sub> field from the CTM or AOD input field. Bayesian downscalers have been applied to develop gridded daily PM<sub>2.5</sub> fields at 12-km resolution for the conterminous U.S. (Wang et al., 2018b; U.S. EPA, 2017). An ensemble technique that optimally combines predictions of CTM and AOD

downscalers has also been developed to predict PM<sub>2.5</sub> at high resolution over Colorado during the fire season (Geng et al., 2018).

Surface PM<sub>2.5</sub> concentrations can also be predicted based on satellite retrievals of AOD and the relationship between surface PM<sub>2.5</sub> and AOD from CTM simulations (van Donkelaar et al., 2010). For example, in van Donkelaar et al. (2015a), satellite-based approaches (van Donkelaar et al., 2010; van Donkelaar et al., 2013) were used to estimate a gridded field of global mean PM<sub>2.5</sub> concentration for the 2001-2010 period that was combined with information from radiometrically stable satellite instruments (Boys et al., 2014) to develop global PM<sub>2.5</sub> fields over the 1998-2012 period (van Donkelaar et al., 2015a). Motivated by the limited use of surface measurements in this approach, van Donkelaar et al. (2015b) developed an updated method that incorporates additional information from PM<sub>2.5</sub> monitoring networks to improve performance. Specifically, geographically weighted regression (GWR) of residual PM<sub>2.5</sub> (i.e., the difference between monitored PM<sub>2.5</sub> and predictions based on satellite-derived AOD) with land-use and other variables is performed to improve PM<sub>2.5</sub> concentration estimates in areas such as North America where monitoring is relatively dense (van Donkelaar et al., 2019; van Donkelaar et al., 2015b). This approach has been used to create long-term PM<sub>2.5</sub> fields globally and for North America at about 1-km resolution. However, the developers caution that PM<sub>2.5</sub> gradients may not be fully resolved at 1-km resolution due to the influence of coarser-scale data used in the model<sup>37</sup> and report that mean error variance decreases when averaging the 1-km fields to coarser resolution (van Donkelaar et al., 2019).

Daily PM<sub>2.5</sub> fields based on non-parametric (i.e., machine learning) methods have also been developed to characterize PM<sub>2.5</sub> over the U.S. Non-parametric methods facilitate the use of large numbers of predictor variables that may have complex nonlinear relationships with PM<sub>2.5</sub> concentrations that would be challenging to specify with a parametric method. For example, a neural network algorithm was used to predict daily PM<sub>2.5</sub> fields at 1-km resolution over the conterminous U.S. during 2000-2012 using more than 50 predictor variables including satellite-derived AOD, CTM predictions, satellite-derived absorbing aerosol index, meteorological data, and land-use variables (Di et al., 2016). A random forest algorithm was also applied to develop daily PM<sub>2.5</sub> fields at 12-km resolution over the conterminous U.S. in 2011 and provide variable importance information for about 40 predictor variables including CTM results and satellite-derived AOD (Hu et al., 2017). Satellite-derived AOD and the convolution layer for nearby PM<sub>2.5</sub> measurements are ranked among the top five most important predictor variables for the importance metrics considered. A wide range of parametric and non-parametric hybrid PM<sub>2.5</sub> models have recently been reviewed in Chapter 3 of the ISA (U.S. EPA, 2019a).

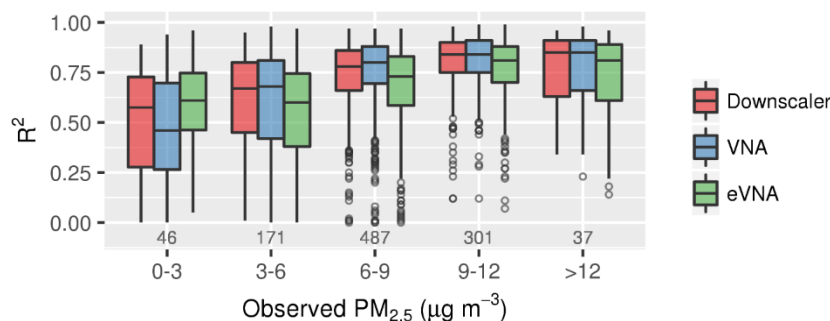
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<sup>37</sup> See [http://fizz.phys.dal.ca/~atmos/martin/?page\\_id=140](http://fizz.phys.dal.ca/~atmos/martin/?page_id=140)

### 2.3.3.1.2 Performance of the Methods

The performance of hybrid modeling methods is often evaluated against surface measurements using n-fold cross validation (i.e., 1/n of the data are reserved for validation with the rest used for model training, and the process is repeated n times). Although model evaluation methods are not consistent across studies, ten-fold cross-validation statistics are often reported and support use of the hybrid methods just described. For example, the neural network achieved total  $R^2$  of 0.84 and root-mean-square error (RMSE) of  $2.94 \mu\text{g m}^{-3}$  for daily  $\text{PM}_{2.5}$  predictions at sites in the conterminous U.S. during 2000-2012 (Di et al., 2016). The random forest achieved total  $R^2$  of 0.80 and RMSE of  $2.83 \mu\text{g m}^{-3}$  for daily  $\text{PM}_{2.5}$  predictions at U.S. sites in 2011 (Hu et al., 2017). The satellite-derived AOD approach with GWR yielded an  $R^2$  of 0.79 and RMSE of  $1.7 \mu\text{g m}^{-3}$  in cross validation for longer-term  $\text{PM}_{2.5}$  predictions at sites in North America (van Donkelaar et al., 2015b). The Bayesian downscalers had weaker performance in cross validation (e.g., national  $R^2$ : 0.66-0.70; Wang et al., 2018b; Kelly et al., 2019) than the other methods, possibly due to the relatively small number of predictor variables. However, the downscalers have advantages of simplicity, computational efficiency, and lower potential for overfitting compared with the machine learning methods.

Although model validation analyses often report favorable performance in terms of aggregate cross-validation statistics, studies have reported heterogeneity in performance by season, region, and concentration range. For example, several methods had relatively high cross-validation  $R^2$  in summer compared with other seasons (Kelly et al., 2019; Hu et al., 2017; Di et al., 2016; van Donkelaar et al., 2015b). Also, studies have noted relatively weak performance in parts of the western U.S., possibly due to the complex terrain, low concentrations (and therefore signal-to-noise ratio), less dense monitoring, prevalence of wildfire, and challenges in satellite retrievals and CTM modeling (Di et al., 2016; Wang et al., 2018b; Hu et al., 2017; Kelly et al., 2019). Predictive capability in terms of cross-validation  $R^2$  has also been reported to weaken with decreasing  $\text{PM}_{2.5}$  concentration in several studies (e.g., Kelly et al., 2019; Di et al., 2016; van Donkelaar et al., 2019). Trends in model performance associated with  $\text{PM}_{2.5}$  concentration (e.g., Figure 2-30) could be due in part to the relatively sparse monitoring in remote areas, where  $\text{PM}_{2.5}$  concentrations tend to be low. Consistent with this hypothesis, studies have reported degradation of model performance metrics with increasing distance to the nearest in-sample monitor, suggesting that predictions are most reliable in densely monitored urban areas (Jin et al., 2019; Huang et al., 2018; Kelly et al., 2019).



**Figure 2-30. R<sup>2</sup> for ten-fold cross-validation of daily PM<sub>2.5</sub> predictions in 2015 from three methods for individual sites as a function of observed concentration.** Text indicates the number of monitors in the PM<sub>2.5</sub> concentration range. Downscaler: Bayesian downscaler of CMAQ predictions; VNA: Voronoi Neighbor Averaging; eVNA: enhanced-VNA. From Kelly et al. (2019).

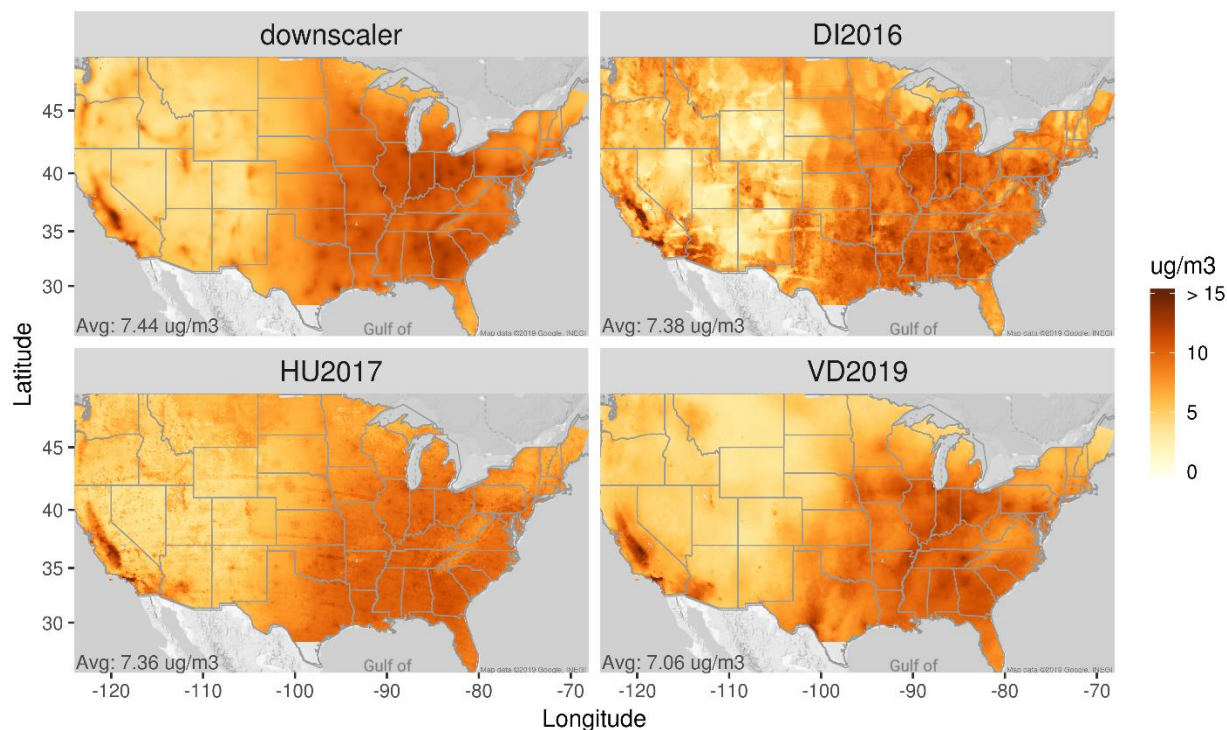
A limited number of studies have intercompared concentration predictions based on different PM<sub>2.5</sub> characterization methods. Huang et al. (2018) compared PM<sub>2.5</sub> concentrations from the method of Di et al., 2016 with concentrations from the CTM-based data fusion method of Friberg et al. (2016) and the satellite-derived AOD approach of Hu et al. (2014) for North Carolina. They reported general agreement in concentrations among methods, with some differences along the coast and in forested regions where monitoring is less dense. Yu et al. (2018) compared PM<sub>2.5</sub> concentrations from fourteen approaches of varying complexity for developing PM<sub>2.5</sub> spatial fields over the Atlanta, Georgia region. They reported that predictions of the methods can differ considerably, and the hybrid approaches that incorporate CTM predictions generally outperformed the simpler techniques (e.g., monitor interpolation). Also, model predictions appeared to be more reliable in the urban center based on relatively low cross validation R<sup>2</sup> for sites away from the urban core. Jin et al. (2019) reported increasing uncertainty in hybrid model predictions with distance to the nearest AQS monitor. Keller and Peng (2019) reported that a prediction model incorporating CTM output outperformed a monitor averaging approach and error reduction could be achieved by restricting the study to areas near monitors.

### 2.3.3.1.3 Comparison of PM<sub>2.5</sub> Fields Across Approaches

To illustrate features of the spatial fields reported in the literature, the annual mean PM<sub>2.5</sub> concentrations for 2011 from four methods is shown in Figure 2-31, where predictions from the methods were averaged to a common 12-km grid. The fields were developed using a Bayesian downscaler (downscaler, Berrocal et al., 2012), neural network (DI2016, Di et al., 2016), random forest (HU2017, Hu et al., 2017), and GWR of residuals from satellite-based PM<sub>2.5</sub> estimates (VD2019; van Donkelaar et al., 2019). Annual mean concentrations were developed from daily PM<sub>2.5</sub> predictions in the downscaler, DI2016, and HU2017 cases and from monthly PM<sub>2.5</sub> predictions in the VD2019 case. General features of the 2011 fields are in reasonable agreement



across methods, with elevated concentrations across broad areas of the eastern U.S. and in the San Joaquin Valley and South Coast Air Basin of California. The national mean PM<sub>2.5</sub> concentration for the VD2019 case (7.06  $\mu\text{g m}^{-3}$ ) is slightly lower than those of the other cases (7.36-7.44  $\mu\text{g m}^{-3}$ ), possibly because the VD2019 fields were developed using monthly (rather than daily) PM<sub>2.5</sub> measurements. Use of monthly averages provides greater influence on the annual mean of sites with less frequent monitoring that tend to be in rural areas with relatively low concentrations. Mean PM<sub>2.5</sub> concentrations predicted by the four methods in nine U.S. climate regions (Karl and Koss, 1984) are provided in Table 2-3.



**Figure 2-31. Comparison of 2011 annual average PM<sub>2.5</sub> concentrations from four methods.**  
(Note: These four methods include: downscaler (Berrocal et al., 2012), DI2016 (Di et al., 2016), HU2017 (Hu et al., 2017), and VD2019 (van Donkelaar et al., 2019). Predictions have been averaged to a common 12-km grid for this comparison.)

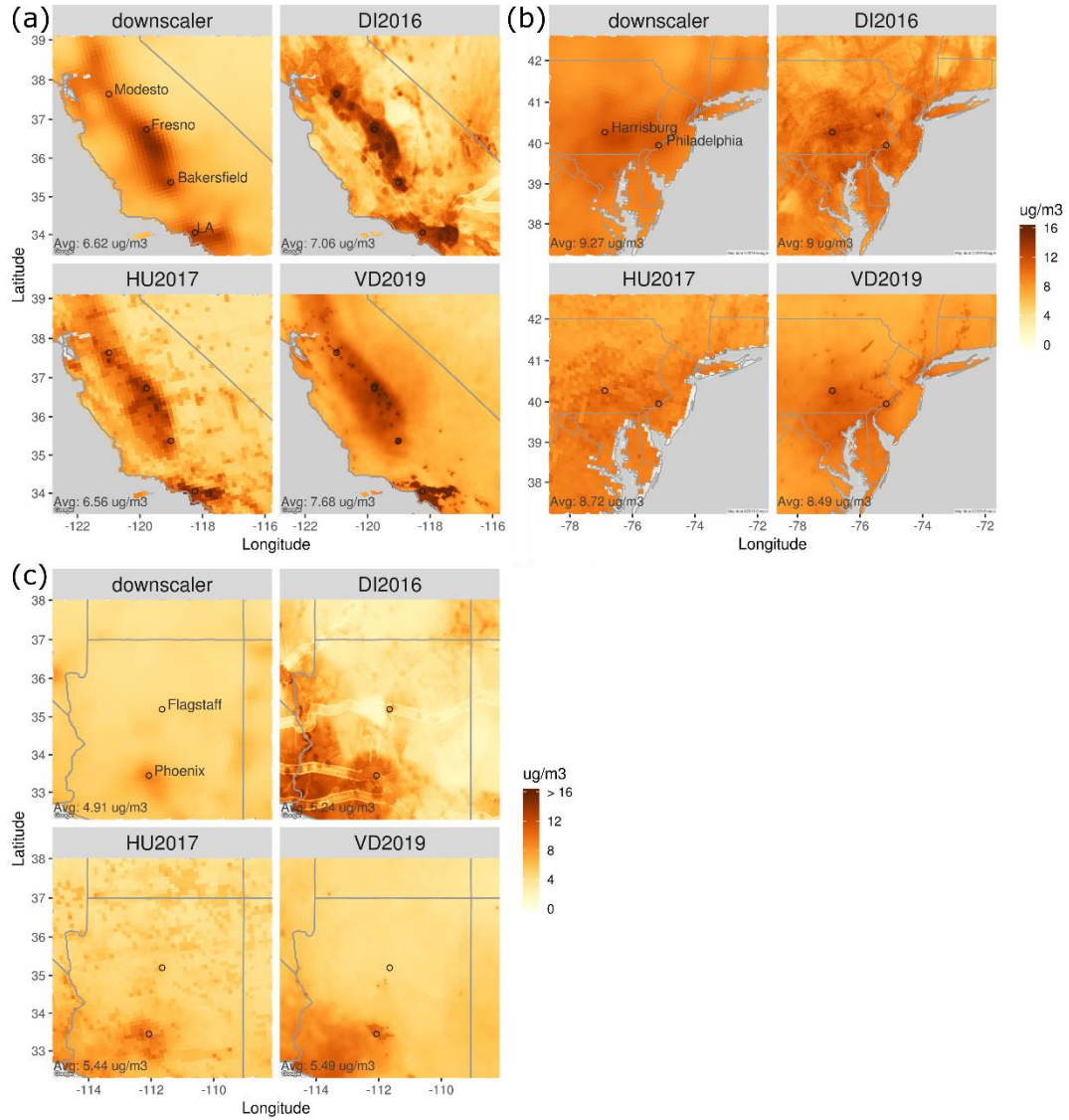
**Table 2-3. Mean 2011 PM<sub>2.5</sub> concentration by region for predictions in Figure 2-24**

Region <sup>1</sup>	downscaler	HU2017	DI2016	VD2019
Northeast	8.5	8.0	8.2	7.5
Southeast	9.9	10.0	9.4	9.8
Ohio Valley	10.7	9.6	9.8	10.0
Upper Midwest	8.8	7.9	7.9	7.1



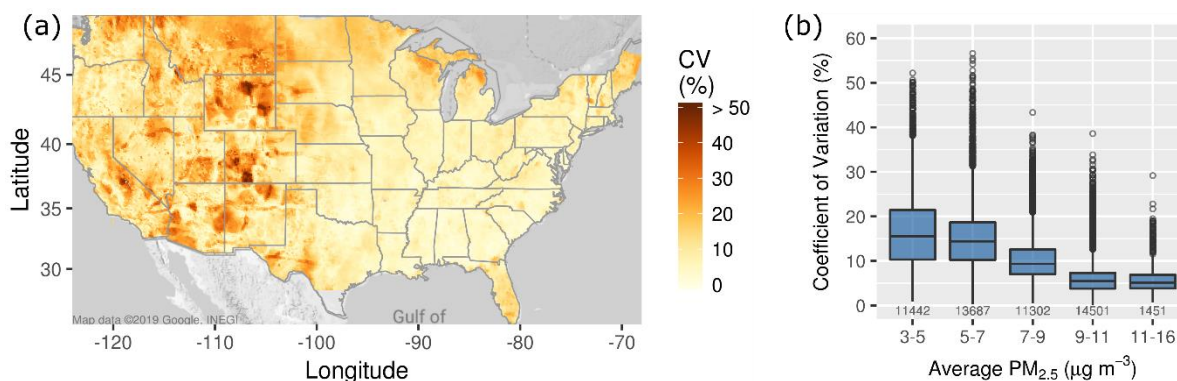
South	8.8	8.9	9.0	8.7
Southwest	5.0	5.3	5.2	5.1
N. Rockies & Plains	5.6	5.9	5.6	4.5
Northwest	5.0	5.3	6.1	4.9
West	5.5	5.7	6.0	6.5
<sup>1</sup> U.S. climate region: <a href="https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php">https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php</a> .				

In Figure 2-32, PM<sub>2.5</sub> concentrations predicted by the four methods are shown at their native resolution for regions centered on California, New Jersey, and Arizona. Predictions span a wider range of concentrations for the western regions centered on California and Arizona (Figure 2-32, panels a and c) than the eastern region centered on New Jersey (Figure 2-32, panel b). Despite general agreement among predictions for the California and the eastern U.S. areas, the spatial texture of the concentration fields differs among methods. For instance, the 12-km Bayesian downscaler produces the smoothest PM<sub>2.5</sub> concentration field, and the 1-km neural network (DI2016) produces the field with the greatest variance. Some of the largest differences in PM<sub>2.5</sub> concentration among methods occurred over southwest Arizona. The DI2016 and VD2019 methods predict higher concentrations in this area than the downscaler and HU2017 methods, and the DI2016 approach predicts distinct spatial features associated with Interstate 40, 10, and 8 that are not apparent in the other fields (Figure 2-32, panel c).

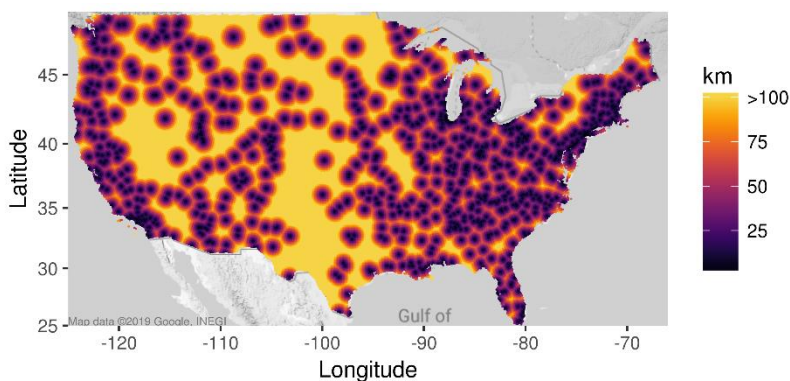


**Figure 2-32. Comparison of 2011 annual average PM<sub>2.5</sub> concentrations from four methods for regions centered on the (a) California (b) New Jersey, and (c) Arizona.** Predictions are shown at their native resolution (i.e., about 1-km for DI2016 and VD2019 and 12-km for downscaler and HU2017).

In Figure 2-33, the coefficient of variation (CV; i.e., the standard deviation divided by the mean) among methods is shown in percentage units based on predictions that were averaged to a common 12-km grid. The largest values occur in the western U.S. (Figure 2-33, panel a), where terrain is complex, wildfire is prevalent, monitoring is relatively sparse, and PM<sub>2.5</sub> concentrations tend to be low. The distance from the grid-cell center to the nearest monitor is greater than 100 km for broad areas of the west (Figure 2-34).



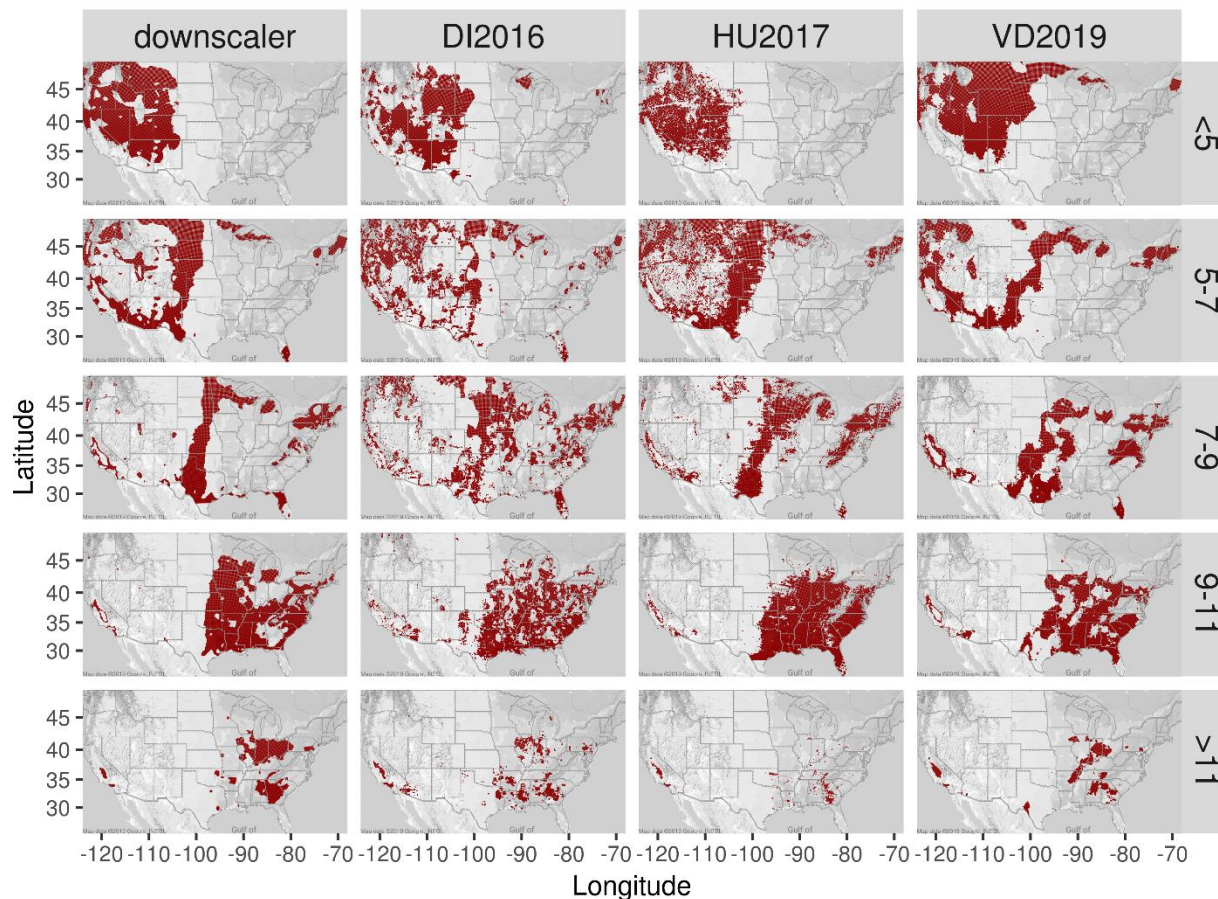
**Figure 2-33. (a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in percentage units for the four models in Figure 2-31. (b) Boxplot distributions of CV for grid cells binned by the average  $\text{PM}_{2.5}$  concentration for the four models. (Note: The box brackets the interquartile range (IQR), the horizontal line within the box represents the median, the whiskers represent 1.5 times the IQR from either end of the box, and circles represent individual values less than and greater than the range of the whiskers.)**



**Figure 2-34. Distance from the center of the 12-km grid cells to the nearest  $\text{PM}_{2.5}$  monitoring site for  $\text{PM}_{2.5}$  measurements from the AQS database and IMPROVE network.**

Concentrations less than  $5 \mu\text{g m}^{-3}$  occur exclusively in the western U.S. for the downscaler and HU2017 methods, and the western U.S. plus a few areas along the northern U.S. border in the eastern U.S. for the DI2016 and VD2019 methods (Figure 2-35, top row). Concentrations between  $5$  and  $7 \mu\text{g m}^{-3}$  are predicted in the western U.S. and parts of New England for all methods and over Florida by the downscaler and DI2016 approaches (Figure 2-35, second row). The CV among methods increases with decreasing concentration (Figure 2-33 above, panel b), and the median CV is about 15% for grid cells with mean concentrations less than  $7 \mu\text{g m}^{-3}$ . As illustrated by Figure 2-33 and Figure 2-35, the low-concentration areas with

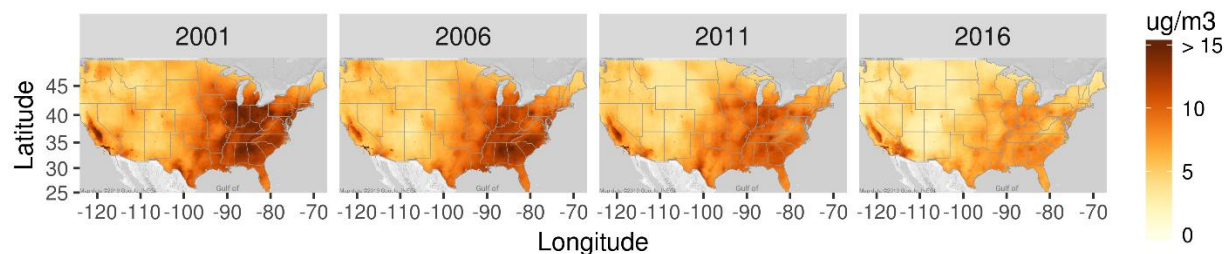
relatively large CVs are in the western U.S. and along the northern and southern border of the eastern U.S.



**Figure 2-35. Location of PM<sub>2.5</sub> predictions by range in annual average concentration for the four prediction methods at their native resolution.** (Note: Concentration ranges: < 5 µg/m<sup>3</sup>, 5-7 µg/m<sup>3</sup>, 7-9 µg/m<sup>3</sup>, 9-11 µg/m<sup>3</sup>, and >11 µg/m<sup>3</sup>.)

The comparison of PM<sub>2.5</sub> concentrations across approaches was based on the 2011 period due to the availability of predictions from multiple methods for that year. As discussed earlier in this chapter, PM<sub>2.5</sub> concentrations have declined over the U.S. in the last several decades. Annual mean PM<sub>2.5</sub> concentrations predicted by the VD2019 method for 2011 are compared with predictions for 2001, 2006, and 2016 in Figure 2-36. The VD2019 fields capture the trend of decreasing PM<sub>2.5</sub> over the U.S. during this period, and the areas with annual mean PM<sub>2.5</sub> concentration greater than 11 µg m<sup>-3</sup> in 2016 are limited to California and southwest Arizona.





**Figure 2-36. Annual mean PM<sub>2.5</sub> from the VD2019 method (van Donkelaar et al., 2019) for 2001, 2006, 2011, and 2016.**

#### 2.3.3.1.4 Summary

Hybrid PM<sub>2.5</sub> modeling methods have improved the ability to estimate PM<sub>2.5</sub> exposure for populations throughout the conterminous U.S. compared with the earlier approaches based on monitoring data alone. Excellent performance in cross-validation tests suggests that hybrid methods are reliable for estimating PM<sub>2.5</sub> exposure in many applications. As discussed in Chapter 3 of this PA, good agreement in health study results between monitor- and model-based methods for urban areas (McGuinn et al., 2017) and general consistency in results for the conterminous U.S. (Jerrett et al., 2017; Di et al., 2016) also suggests that the fields are reliable for use in health studies. However, there are also important limitations associated with the modeled fields. First, performance evaluations for the methods are weighted toward densely monitored urban areas at the scales of representation of the monitoring networks. Predictions at different scales or in sparsely monitored areas are relatively untested. Second, studies have reported heterogeneity in performance with relatively weak performance in parts of the western U.S., at low concentrations, at greater distance to monitors, and under conditions where the reliability and availability of key input datasets (e.g., satellite retrievals and air quality modeling) are limited. Differences in predictions among different hybrid methods have also been reported and tend to be most important under conditions with the performance issues just noted. Differences in predictions could also be related to the different approaches used to create long-term PM<sub>2.5</sub> fields (e.g., averaging daily PM<sub>2.5</sub> fields vs. developing long-term average fields), which is important due to variable monitoring schedules. More work on comprehensively characterizing the performance of modeled fields is warranted and will further inform our understanding of the implications of using these fields to estimate PM<sub>2.5</sub> exposures in health studies.

## 2.4 BACKGROUND PM

For the purposes of this assessment, we define background PM as all particles that are formed by sources or processes that cannot be influenced by actions within the jurisdiction of concern. For this document, U.S. background PM is defined as any PM formed from emissions

other than U.S. anthropogenic (i.e. manmade) emissions. Potential sources of U.S. background PM include both natural sources (i.e. PM that would exist in the absence of any anthropogenic emissions of PM or PM precursors) and transboundary sources originating outside U.S. borders.

Ambient monitoring networks provide long-term records of speciated PM concentrations across the U.S., which can inform estimates of individual source contributions to background PM levels in different parts of the country. However, even the most remote monitors within the U.S. can be periodically affected by U.S. anthropogenic emissions. Monitor data are also limited in more remote areas due to a sparser monitoring network where PM concentrations are more likely influenced by background sources. Chemical transport models (CTMs) offer complementary information to ambient monitor networks by providing more spatially and temporally comprehensive estimates of atmospheric composition. CTMs can also be applied to isolate contributions from specific emission sources to PM concentrations in different areas via source apportionment or “zero-out” modeling (i.e., estimating what the residual concentrations would be were emissions from the emission source of interest to be entirely removed).

At annual and national scales, estimated background PM concentrations in the U.S. are small compared to contributions from domestic anthropogenic emissions. For example, based on zero-out modeling in the last review of the PM NAAQS, annual background PM<sub>2.5</sub> concentrations were estimated to range from 0.5 - 3  $\mu\text{g}/\text{m}^3$  across the sites examined. The magnitude and sources of background PM can vary widely by region and time of year. Coastal sites may experience a consistent contribution of PM from sea spray aerosol, while other areas covered with dense vegetation may be impacted by biogenic aerosol production during the summertime. Sources of background PM also operate across a range of time scales. While some sources like biogenic aerosol vary at monthly to seasonal scales, many sources of background PM are episodic in nature. These episodic sources (e.g. large wildfires) can be characterized by infrequent contributions to high-concentration events occurring over shorter periods of time (e.g., hours to several days). Such episodic events are sporadic and do not necessarily occur in all years. While these exceptional episodes can lead to violations of the daily PM<sub>2.5</sub> standard (35  $\mu\text{g}/\text{m}^3$ ) in some cases (Schweizer et al., 2017), such events are routinely screened for and usually identifiable in the monitoring data. As described further below, contributions to background PM in the U.S. result mainly from sources within North America. Contributions from intercontinental events have also been documented (e.g., transport from dust storms occurring in deserts in North Africa and Asia), but these events are less common and represent a relatively small fraction of background PM in most places.

While the potential sources of background PM discussed above include sources of both fine (PM<sub>2.5</sub>) and coarse (PM<sub>10</sub>) particles, background contributions to ambient UFP are less well characterized and are not discussed here due to lack of information. Section 2.4.1 below further

discusses background PM from natural sources inside the U.S. Section 2.4.2 characterizes the role of international transport of PM from sources outside U.S. borders.

#### **2.4.1 Natural Sources**

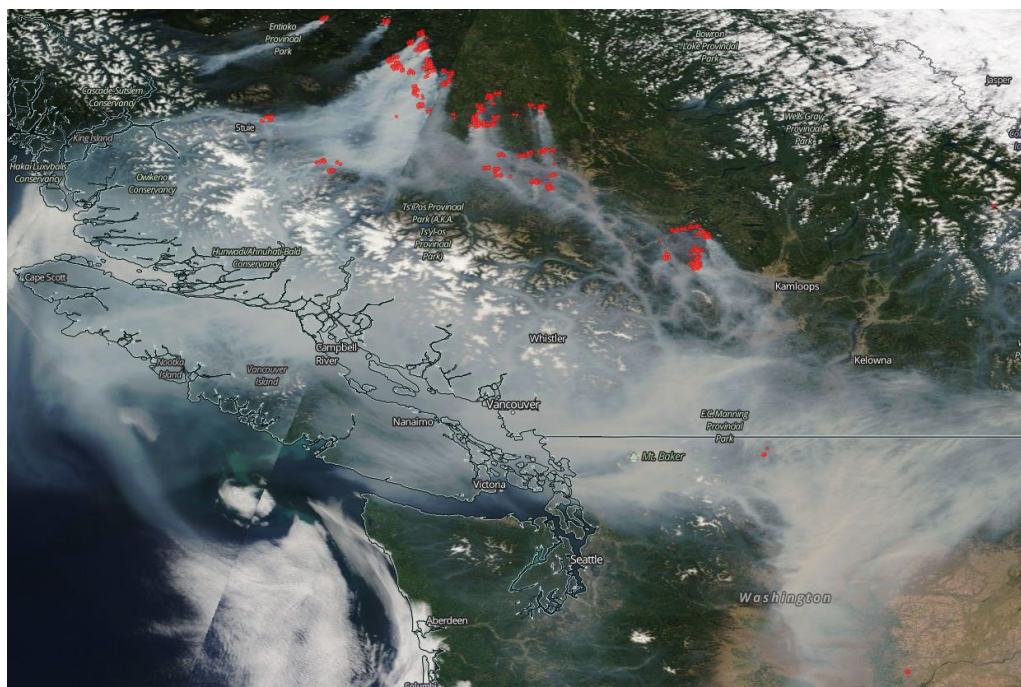
As noted in section 2.1.1, sources that contribute to natural background PM include dust from the wind erosion of natural surfaces, sea salt, wildland fires, primary biological aerosol particles (PBAP) such as bacteria and pollen, oxidation of biogenic hydrocarbons such as isoprene and terpenes to produce SOA, and geogenic sources such as sulfate formed from volcanic production of SO<sub>2</sub> and oceanic production of dimethyl-sulfide (DMS). While most of the above sources release or contribute predominantly to fine aerosol, some sources including windblown dust, and sea salt also produce particles in the coarse size range (U.S. EPA, 2019a, section 2.3.3).

Biogenic emissions from plants are perhaps the most ubiquitous sources of background PM in the U.S. Certain species of plants and trees can release large amounts of VOCs such as isoprene and monoterpenes that are oxidized in the atmosphere to form organic aerosol. SOA production from biogenic emissions is largest in the southeastern U.S., where conditions are warm, humid, and sunny for much of the year. Many of the processes involved with biogenic SOA formation are complex and remain highly uncertain. Results from radiocarbon techniques applied to distinguish modern (biogenic or fires) from fossil (anthropogenic) carbon fractions in organic aerosol have suggested comparable contributions from both carbon types in the Southeast where SOA concentrations are high (Schichtel et al., 2008). However, SOA formation from biogenic emission sources can also be facilitated by the presence of anthropogenic precursors (Xu et al., 2015). More work characterizing the interactions of anthropogenic and biogenic emissions is needed to determine the implications of such processes for background PM concentrations.

Soil dust and sea salt have been estimated to account for less than 10% of urban PM<sub>2.5</sub> on average in the U.S. (Karagulian et al., 2015), although episodic contributions from these sources can be much higher in some locations. For example, during a dust storm affecting Phoenix in July of 2011, peak hourly average PM<sub>10</sub> concentrations were greater than 5,000 µg/m<sup>3</sup>, with area-wide average hourly concentrations ranging from a few hundred to a few thousand µg/m<sup>3</sup> (Vukovic et al., 2014). Dust can also account for much of the PM that originates from outside the U.S., which we discuss further below (U.S. EPA, 2019a, section 2.5.4.2). In addition to sea salt aerosol, biological production of the sulfate precursor DMS can also occur in some marine environments, although the impact of DMS emissions on annual mean sulfate concentrations is likely very small in the U.S. (<0.2 µg/m<sup>3</sup>) and confined to coastal areas (Sarwar et al., 2018).

Wildfires release large amounts of particles and gaseous PM precursors. Invasive species, historical fire management practices, frequency of drought, and extreme heat have resulted in longer fire seasons (Jolly et al., 2015) and more large fires (Dennison et al., 2014) over time. In addition to emissions from fires in the U.S., emissions from fires in other countries can be transported to the U.S. Transport of smoke from fires in Canada, Mexico, Central America, and Siberia have been documented in multiple studies (U.S. EPA, 2009). According to the NEI, wildfire smoke contributes between 10 and 20% of primary PM emissions in the U.S. per year (U.S. EPA, 2019a, section 2.3.1), with much higher localized contributions near fire-affected areas.

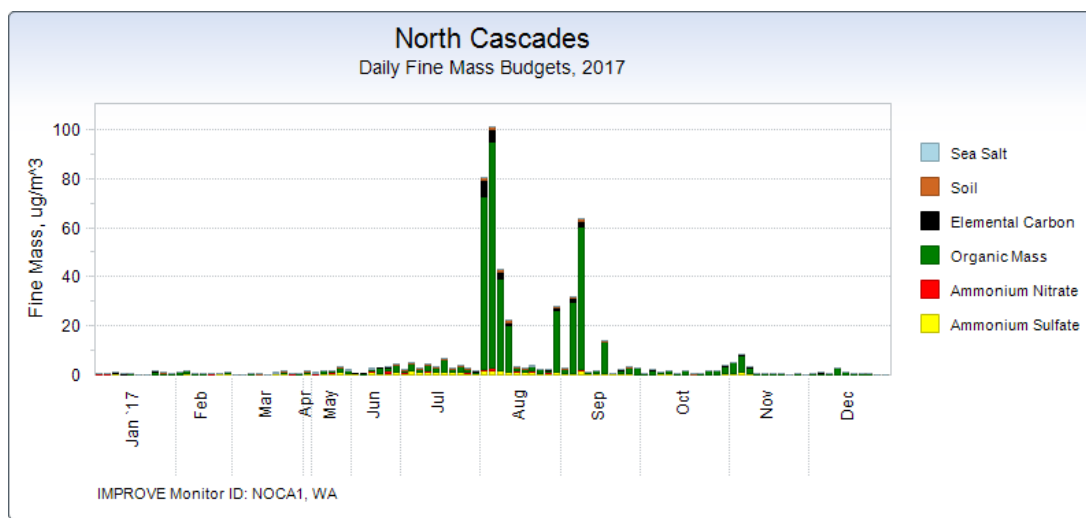
To illustrate how episodic impacts from a large natural source can affect PM concentrations in the U.S., Figure 2-37 and Figure 2-38 show an example from a recent wildfire event. In summer 2017, smoke from wildfires in British Columbia, Canada led to severe air quality degradation in parts of the Pacific Northwest. A NASA Worldview<sup>38</sup> image from August 4, 2017 (Figure 2-37) shows smoke from multiple fire detections across southern British Columbia crossing into northern Washington state. Smoke from these fires was also captured at the North Cascades IMPROVE monitor (Figure 2-38), where daily fine PM concentrations were increased from a typical baseline of less than  $10 \mu\text{g}/\text{m}^3$  to  $\sim 100 \mu\text{g}/\text{m}^3$  during this time.



**Figure 2-37. Smoke and fire detections observed by the MODIS instrument onboard the Aqua satellite on August 4<sup>th</sup>, 2017 accessed through NASA Worldview.**

<sup>38</sup> Available from <https://worldview.earthdata.nasa.gov>.





**Figure 2-38. Fine PM mass time series during 2017 from the North Cascades IMPROVE site in north central Washington state.<sup>39</sup>**

Later in August and September 2017, many other wildfires occurred in Washington state and Oregon, making this fire season one of the worst for the Pacific Northwest in recent history. The severe fires in British Columbia, Washington and Oregon during 2017 have been linked to the combination of usually hot temperatures in August/September in the region following a very wet preceding winter season. While many of the most severe wildfire events in the U.S. occur in the western part of the country during the late summer, most of the contiguous U.S. is affected by wildfire smoke during some part of the year (Kaulfus et al., 2017).

## 2.4.2 International Transport

Background PM contributions from international sources include PM that is both natural and anthropogenic in origin crossing into U.S. borders from Canada and Mexico or from longer range intercontinental transport. While in general the biggest contributions to U.S. background PM from international sources come from nearby Canada and Mexico, large episodic events from intercontinental sources can sometimes occur (e.g., windblown dust from Asia or Africa). This section discusses transboundary PM transport within North America (section 2.4.2.1) as well as long range intercontinental transport from anthropogenic (section 2.4.2.2) and natural (section 2.4.2.3) sources.

<sup>39</sup> Available at [http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF\\_PmHazeComp](http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_PmHazeComp).

#### **2.4.2.1 Transboundary Transport in North America**

As discussed above, some of the largest potential international sources of U.S. background PM originate elsewhere in North America. PM produced from fires in both Canada and Mexico can affect air quality in the U.S., particularly in border states (Park et al., 2007; Miller et al., 2011; Wang et al., 2018a). Anthropogenic emissions from Canada and Mexico can also influence U.S. PM air quality. An inverse modeling study by Henze et al. (2009) estimated that in 2001 anthropogenic SO<sub>x</sub> emissions from Canada and Mexico accounted for 6% and 4% respectively of total daily inorganic PM<sub>2.5</sub> in the U.S. These authors also estimated that SO<sub>x</sub> emissions related to international shipping accounted for approximately 2% of total inorganic PM in the U.S.

#### **2.4.2.2 Long Range Transport from Anthropogenic Sources**

Due to the relatively short atmospheric lifetime of particles (~days to weeks), long range transport of aerosols does not contribute significant PM mass to the U.S. Heald et al. (2006) estimated that transport from Asia accounted for less than 0.2 µg/m<sup>3</sup> of sulfate PM<sub>2.5</sub> in the Northwestern U.S. in spring, and Leibensperger et al. (2011) estimated intercontinental contributions from Asian anthropogenic SO<sub>2</sub> and NO<sub>x</sub> emissions of 0.1 - 0.25 µg/m<sup>3</sup> annually in the western U.S. Leibensperger et al. (2011) also concluded that much of the intercontinental influence captured by the GEOS-Chem model was in fact local PM production attributable to domestic emissions in receptor countries arising from changes in global oxidant budgets, rather than impacts from PM directly transported across geopolitical boundaries. The studies above are also consistent with findings from other analyses. A report from the United Nations on global air quality synthesizing results across many studies estimated an annual average contribution of approximately 0.1 µg/m<sup>3</sup> sulfate PM in North America due to transport from East Asia (TFHTAP, 2006).

#### **2.4.2.3 Long Range Transport from Natural Sources**

Long range transport of dust from both Asia (Vancuren and Cahill, 2002; Yu et al., 2008) and North Africa (Prospero, 1999a; Prospero, 1999b; Chiapello et al., 2005; McKendry et al., 2007) has been shown to occasionally contribute to surface PM concentrations in some regions of the U.S. The likelihood of such long-range dust transport events depends on large-scale meteorological patterns, which can vary significantly across seasons and between years. Yu et al. (2015) found that the transport of North African dust across the Atlantic Ocean is strongly negatively correlated with precipitation in the Sahel during the preceding year. Dust from Africa has also shown a decreasing trend of approximately 10% per decade from 1982 to 2008 based on measurements of aerosol optical depth and surface concentrations in Barbados. This trend was attributed to a corresponding decrease in surface winds over source regions (Ridley et al., 2014).

Variability in springtime Asian dust transport to the U.S. has been linked to north-south shifts in trans-Pacific flow modulated by the El Nino-Southern Oscillation (Achakulwisut et al., 2017), as well as to variations in regional precipitation affecting both dust emissions in Asia and atmospheric residence times during transport (Fischer et al., 2009).

On average, intercontinental dust transport is estimated to contribute about 1-2  $\mu\text{g}/\text{m}^3$  to annual  $\text{PM}_{2.5}$  at some U.S. sites (Jaffe et al., 2005; TFHTAP, 2006; Creamean et al., 2014). However, daily concentrations can be substantially larger for individual events, especially for coarser particles. For example, Jaffe et al. (2003) found evidence of Asian dust events in 1998 and 2001 contributing 30-40  $\mu\text{g}/\text{m}^3$  to daily  $\text{PM}_{10}$  at sites throughout the U.S., although the authors also note that large events of this scale are rare and only occurred twice during their 15-year study period. Similar magnitudes have also been reported for individual North African events; analysis of a multidecadal record of African dust reaching Miami indicated concentrations of PM ranging from ~10 to 120  $\mu\text{g}/\text{m}^3$  (Prospero, 1999b; Prospero, 1999a).<sup>40</sup>

### 2.4.3 Estimating Background PM with Recent Data

As discussed above, the 2009 PM ISA estimated background PM concentrations at several remote IMPROVE sites in different regions of the U.S. for 2004 using a combination of monitor data and zero-out air quality modeling. Revisiting the speciated IMPROVE PM data that the monitors included in the last assessment provides some insights into how contributions from different PM sources may have changed, and what those changes (or lack thereof) mean for our current understanding of background PM in the U.S.

Figure 2-39 shows observed annual average  $\text{PM}_{2.5}$  in 2004 and 2016 at the same remote monitors examined in the last ISA. The comparisons show decreases in both total  $\text{PM}_{2.5}$  and ammonium sulfate across all sites examined, consistent with decreases in anthropogenic  $\text{SO}_2$  and other PM precursors observed over this time period. It is likely that most of the remaining ammonium sulfate observed at these sites is also a result of domestic anthropogenic emissions and therefore not relevant for assessments of background PM.

Sea salt and dust aerosol are likely natural in origin at these remote sites. With the exception of REDW1, a coastal site in California, soil and sea salt aerosol together account for less than about 0.5  $\mu\text{g}/\text{m}^3$  of the annual average  $\text{PM}_{2.5}$  at all monitors examined here, which is below the values cited from the literature for long range dust contributions discussed above. Contributions from ammonium nitrate and elemental carbon could be from either anthropogenic or natural sources, but together represent less than about 0.5  $\mu\text{g}/\text{m}^3$  at most of the sites in 2016.

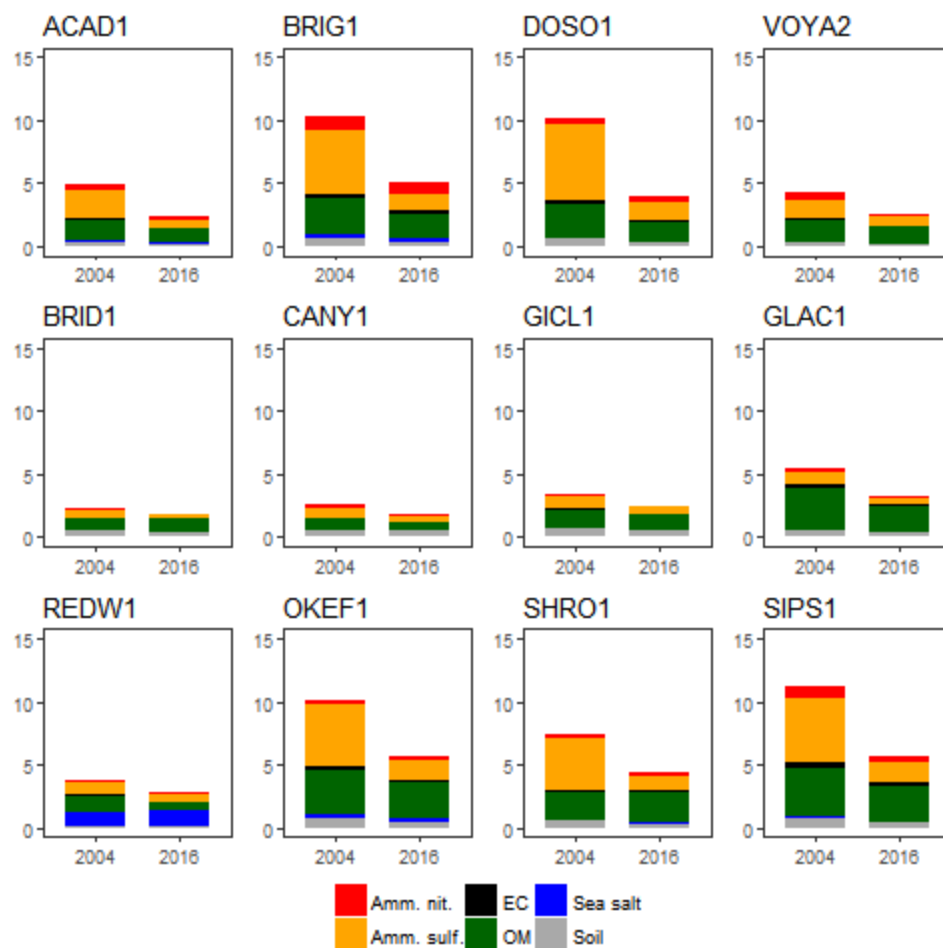
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<sup>40</sup> Sample collection began in 1974, before network  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  samplers were developed, and no size cut was specified (Prospero, 1999b).

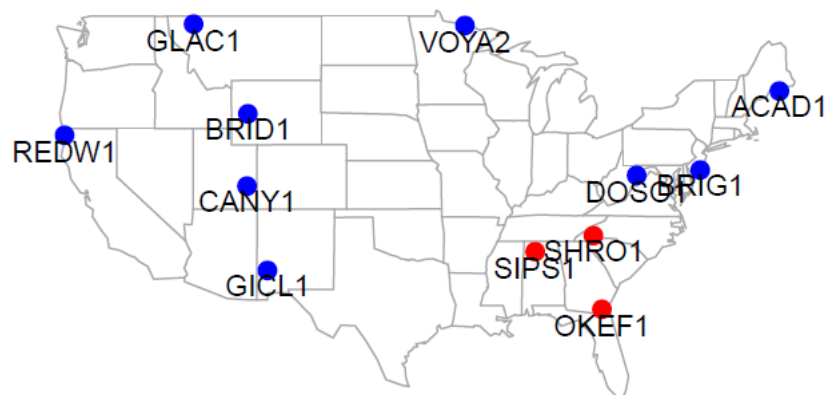
The largest contribution from nitrate occurs at the BRIG1 monitor in New Jersey and is likely anthropogenic given the high density of NO<sub>x</sub> from vehicle emissions in that region.

After ammonium sulfate, the next largest contributing species for most of the sites is organic matter, which for many of the monitors in Figure 2-39 represents 50% or more of total PM in both 2004 and 2016. In addition to the IMPROVE sites from the last ISA, Figure 2-31 also shows comparisons for three sites in the Southeast U.S. As a region, the Southeast has the highest levels of biogenic aerosol production in the country, so the organic matter contribution at these three sites likely represents an upper bound for the country of what natural biogenic organic aerosol production could be under present atmospheric conditions. The organic aerosol components shown in Figure 2-39 will also include the influence of fires for some monitors. The highest organic matter contribution for any of the sites shown in Figure 2-39, including the three Southeast monitors, is approximately 2 µg/m<sup>3</sup>. While contributions from ammonium sulfate have decreased substantially at some of the monitors, particularly the eastern sites, contributions from organic aerosol are roughly consistent between 2004 and 2016, as are the contributions from the other species assumed to be mostly natural in origin (soil and sea salt). Therefore, while no new zero-out modeling was done for the current review, revisiting these monitors with more recent data suggests that estimates of background concentrations at these monitors are still around 1-3 µg/m<sup>3</sup> and have not changed significantly since the last PM NAAQS Review.

While estimates of total annual background concentrations have generally not changed significantly since the last review, our scientific understanding of organic aerosol formation has evolved. Organic aerosol can be produced from a variety of natural and anthropogenic processes, which presents a challenge for source attribution techniques. Additionally, new research over the past decade has identified a host of new sources and chemical pathways for SOA formation that have only recently begun to be implemented into CTMs. Further research implementing these new sources and pathways into CTMs is needed to understand 1) the behavior of these different algorithms under a range of possible atmospheric conditions, and 2) what the implications are for understanding SOA formation in the U.S.



**Figure 2-39. Speciated annual average IMPROVE PM<sub>2.5</sub> in µg/m³ at select remote monitors during 2004 and 2016.** (Note: Monitor locations are shown in Figure 2-40.)



**Figure 2-40. Site locations for the IMPROVE monitors in Figure 2-39.** (Note: Monitors also assessed in the 2009 ISA are shown in blue. Monitors only examined in this assessment are shown in red.)

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### 3 REVIEW OF THE PRIMARY STANDARDS FOR PM<sub>2.5</sub>

This chapter presents our key policy-relevant considerations and conclusions regarding the public health protection provided by the current suite of primary PM<sub>2.5</sub> standards and the protection that could be provided by potential alternative standards. These considerations and conclusions are framed by a series of policy-relevant questions, including the following overarching questions:

- **Does the currently available scientific evidence, air quality and quantitative risk information support or call into question the adequacy of the public health protection afforded by the current annual and 24-hour PM<sub>2.5</sub> standards?**
- **What range of potential alternative standards could be supported by the available scientific evidence, air quality and risk information?**

The answers to these questions are informed by our evaluation of a series of more specific policy-relevant questions, which expand upon those presented at the outset of this review in the IRP (U.S. EPA, 2016). Answers to these questions are intended to inform decisions by the Administrator on whether, and if so how, to revise the current suite of primary fine particle standards.

Section 3.1 presents our approach for reviewing the primary standards for PM<sub>2.5</sub>. Sections 3.2 and 3.3 present our consideration of the available scientific evidence and our consideration of information from the PM<sub>2.5</sub> risk assessment, respectively. Section 3.4 summarizes CASAC advice and public comments and section 3.5 summarizes our conclusions regarding the adequacy of the public health protection provided by the current primary PM<sub>2.5</sub> standards and the protection that could be provided by potential alternative standards. Section 3.6 discusses areas for future research and data collection to improve our understanding of fine particle-related health effects in future reviews.

#### 3.1 APPROACH

##### 3.1.1 Approach Used in the Last Review

The last review of the primary PM NAAQS was completed in 2012 (78 FR 3086, January 15, 2013). As noted above (section 1.3), in the last review the EPA lowered the level of the primary annual PM<sub>2.5</sub> standard from 15.0 to 12.0 µg/m<sup>3</sup>,<sup>1</sup> and retained the existing 24-hour PM<sub>2.5</sub> standard with its level of 35 µg/m<sup>3</sup>. The 2012 decision to strengthen the suite of primary PM<sub>2.5</sub>

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<sup>1</sup> The Agency also eliminated spatial averaging provisions as part of the form of the annual standard.

standards was based on the Administrator's consideration of the extensive body of scientific evidence assessed in the 2009 ISA (U.S. EPA, 2009); the quantitative risk analyses presented in the 2010 HREA (U.S. EPA, 2010);<sup>2</sup> the advice and recommendations of the CASAC (e.g., Samet, 2009; Samet, 2010c; Samet, 2010b); and public comments on the proposed rule (78 FR 3086, January 15, 2013; U.S. EPA, 2012). The Administrator particularly noted the "strong and generally robust body of evidence of serious health effects associated with both long- and short-term exposures to PM<sub>2.5</sub>" (78 FR 3120, January 15, 2013). This included epidemiologic studies reporting health effect associations based on long-term average PM<sub>2.5</sub> concentrations ranging from about 15.0 µg/m<sup>3</sup> or above (i.e., at or above the level of the then-existing annual standard) to concentrations "significantly below the level of the annual standard" (78 FR 3120, January 15, 2013). The Administrator further observed that such studies were part of an overall pattern across a broad range of studies reporting positive associations, which were frequently statistically significant. Based on her "confidence in the association between exposure to PM<sub>2.5</sub> and serious public health effects, combined with evidence of such an association in areas that would meet the current standards" (78 FR 3120, January 15, 2013), the Administrator concluded that revision of the suite of primary PM<sub>2.5</sub> standards was necessary in order to provide increased public health protection. Specifically, she concluded that the then-existing suite of primary PM<sub>2.5</sub> standards was not sufficient, and thus not requisite, to protect public health with an adequate margin of safety. This decision was consistent with advice received from the CASAC (Samet, 2010c).

The Administrator next considered what specific revisions to the existing primary PM<sub>2.5</sub> standards were appropriate, given the available evidence and quantitative risk information. She considered both the annual and 24-hour PM<sub>2.5</sub> standards, focusing on the basic elements of those standards (i.e., indicator, averaging time, form, and level). These considerations, and the Administrator's conclusions, are summarized in sections 3.1.1.1 to 3.1.1.4 below.

#### **3.1.1.1 Indicator**

In initially setting standards for fine particles in 1997, the EPA concluded it was appropriate to control fine particles as a group, based on PM<sub>2.5</sub> mass, rather than singling out any particular component or class of fine particles (62 FR 38667, July 18, 1997). In the review completed in 2006, based on similar considerations, the EPA concluded that the available information supported retaining the PM<sub>2.5</sub> indicator and remained too limited to support a distinct

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<sup>2</sup> In the last review, the EPA generated a quantitative health risk assessment for PM, and did not conduct a microenvironmental exposure assessment (U.S. EPA, 2010). To be consistent with our general process for reviewing the NAAQS (section 1.2, above), and with our discussion of potential quantitative analyses in the current review, we refer to the 2010 health risk assessment as the 2010 HREA.

standard for any specific PM<sub>2.5</sub> component or group of components associated with particular source categories of fine particles (71 FR 61162 to 61164, October 17, 2006).

In the last review, the EPA again considered issues related to the appropriate indicator for fine particles, with a focus on evaluating support for the existing PM<sub>2.5</sub> mass-based indicator and for potential alternative indicators based on the ultrafine particle fraction or on fine particle composition (78 FR 3121, January 15, 2013).<sup>3</sup> With regard to PM<sub>2.5</sub> mass, as in the 1997 and 2006 reviews, the health studies available during the last review continued to link adverse health outcomes (e.g., premature mortality, hospital admissions, emergency department visits) with long- and short-term exposures to fine particles indexed largely by PM<sub>2.5</sub> mass (78 FR 3121, January 15, 2013). With regard to the ultrafine fraction of ambient PM, the PA noted the limited body of health evidence assessed in the ISA (summarized in U.S. EPA, 2009, section 2.3.5 and Table 2–6) and the limited monitoring information available to characterize ambient concentrations of ultrafine particles (U.S. EPA, 2011, section 1.3.2). With regard to PM composition, the ISA concluded that “the evidence is not yet sufficient to allow differentiation of those constituents or sources that are more closely related to specific health outcomes” (U.S. EPA, 2009, pp. 2-26 and 6-212; 78 FR 3123, January 15, 2013). The PA further noted that “many different constituents of the fine particle mixture as well as groups of components associated with specific source categories of fine particles are linked to adverse health effects” (U.S. EPA, 2011, p. 2–55; 78 FR 3123, January 15, 2013). Consistent with the considerations and conclusions in the PA, the CASAC advised that it was appropriate to consider retaining PM<sub>2.5</sub> as the indicator for fine particles. The CASAC specifically stated that “[t]here [is] insufficient peer-reviewed literature to support any other indicator at this time” (Samet, 2010a, p. 12). In light of the evidence and the CASAC’s advice, the Administrator concluded that it was “appropriate to retain PM<sub>2.5</sub> as the indicator for fine particles” (78 FR 3123, January 15, 2013).

### **3.1.1.2 Averaging Time**

In 1997, the EPA set an annual PM<sub>2.5</sub> standard to provide protection from health effects associated with long- and short-term exposures to PM<sub>2.5</sub>, and a 24-hour standard to supplement the protection afforded by the annual standard (62 FR 38667 to 38668, July, 18, 1997). In the 2006 review, the EPA retained both annual and 24-hour averaging times (71 FR 61164, October 17, 2006).

In the last review, the EPA again considered issues related to the appropriate averaging times for PM<sub>2.5</sub> standards, with a focus on evaluating support for the existing annual and 24-hour

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<sup>3</sup> In the last review, the ISA defined ultrafine particles as generally including particles with a mobility diameter less than or equal to 0.1 µm. Mobility diameter is defined as the diameter of a particle having the same diffusivity or electrical mobility in air as the particle of interest, and is often used to characterize particles of 0.5 µm or smaller (U.S. EPA, 2009, pp. 3-2 to 3-3).

averaging times and for potential alternative averaging times based on sub-daily or seasonal metrics. Based on the evidence assessed in the ISA, the PA noted that the overwhelming majority of studies that had been conducted since the 2006 review continued to utilize annual (or multi-year) or 24-hour PM averaging periods (U.S. EPA, 2011, section 2.3.2). With regard to potential support for an averaging time shorter than 24-hours, the PA noted that studies of cardiovascular effects associated with sub-daily PM concentrations had evaluated a variety of PM metrics (e.g., PM<sub>2.5</sub>, PM<sub>10</sub>, PM<sub>10-2.5</sub>, ultrafine particles), averaging periods (e.g., 1, 2, and 4 hours), and health outcomes (U.S. EPA, 2011, section 2.3.2). The PA concluded that this evidence, when viewed as a whole, was too uncertain to serve as a basis for establishing a primary PM<sub>2.5</sub> standard with an averaging time shorter than 24-hours (U.S. EPA, 2011, p. 2-57).<sup>4</sup> With regard to potential support for a seasonal averaging time, few studies were available to deduce a general pattern in PM<sub>2.5</sub>-related risk across seasons, and these studies did not provide information on health effects associated with season-long exposures to PM<sub>2.5</sub> (U.S. EPA, 2011, p. 2-58; 78 FR 3124, January 15, 2013).

The PA reached the overall conclusions that the available information provided strong support for considering retaining the current annual and 24-hour averaging times and did not provide support for considering alternative averaging times (U.S. EPA, 2011, p. 2-58). The CASAC agreed that these conclusions were reasonable (Samet, 2010a, p. 13). The Administrator concurred with the PA conclusions and with the CASAC's advice. Specifically, she judged that it was "appropriate to retain the current annual and 24-hour averaging times for the primary PM<sub>2.5</sub> standards to protect against health effects associated with long- and short-term exposure periods" (78 FR 3124, January 15, 2013).

### **3.1.1.3 Form**

In 1997, the EPA established the form of the annual PM<sub>2.5</sub> standard as an annual arithmetic mean, averaged over 3 years, from single or multiple community-oriented monitors.<sup>5</sup> That is, the level of the annual standard was to be compared to measurements made at each community-oriented monitoring site or, if specific criteria were met, measurements from multiple community-oriented monitoring sites could be averaged together (i.e., spatial

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<sup>4</sup> For respiratory effects specifically, the Administrator further noted the ISA conclusion that the strongest associations were observed with 24-hour average or longer exposures, not with exposures less than 24-hours (U.S. EPA, 2009, section 6.3).

<sup>5</sup> As noted above (section 1.3), in the last review the EPA replaced the term "community-oriented" monitor with the term "area-wide" monitor. *Area-wide* monitors are those sited at the neighborhood scale or larger, as well as those monitors sited at micro- or middle scales that are representative of many such locations in the same core-based statistical area (CBSA; 78 FR 3236, January 15, 2013). CBSAs are required to have at least one area-wide monitor sited in the area of expected maximum PM<sub>2.5</sub> concentration.

averaging)<sup>6</sup> (62 FR 38671 to 38672, July 18, 1997). In the 1997 review, the EPA also established the form of the 24-hour PM<sub>2.5</sub> standard as the 98<sup>th</sup> percentile of 24-hour concentrations at each monitor within an area (i.e., no spatial averaging), averaged over three years (62 FR at 38671 to 38674, July 18, 1997). In the 2006 review, the EPA retained these standard forms but tightened the criteria for using spatial averaging with the annual standard (71 FR 61167, October 17, 2006).<sup>7</sup>

In the last review, the EPA's consideration of the form of the annual PM<sub>2.5</sub> standard again included a focus on the issue of spatial averaging. An analysis of air quality and population demographic information indicated that the highest PM<sub>2.5</sub> concentrations in a given area tended to be measured at monitors in locations where the surrounding populations were more likely to live below the poverty line and to include larger percentages of racial and ethnic minorities (U.S. EPA, 2011, p. 2-60). Based on this analysis, the PA concluded that spatial averaging could result in disproportionate impacts in at-risk populations, including minority populations and populations with lower socioeconomic status (SES). Therefore, the PA concluded that it was appropriate to consider revising the form of the annual PM<sub>2.5</sub> standard such that it did not allow for the use of spatial averaging across monitors (U.S. EPA, 2011, p. 2-60). The CASAC agreed with the PA conclusions that it was "reasonable" for the EPA to eliminate the spatial averaging provisions (Samet, 2010c, p. 2), stating the following: "Given mounting evidence showing that persons with lower SES levels are a susceptible group for PM-related health risks, [the] CASAC recommends that the provisions that allow for spatial averaging across monitors be eliminated" (Samet, 2010a, p. 13).

The Administrator concluded that public health would not be protected with an adequate margin of safety in all locations, as required by law, if disproportionately higher PM<sub>2.5</sub> concentrations in low income and minority communities were averaged together with lower concentrations measured at other sites in a large urban area. Therefore, she concluded that the form of the annual PM<sub>2.5</sub> standard should be revised to eliminate spatial averaging provisions (78 FR 3124, January 15, 2013). Thus, the level of the annual PM<sub>2.5</sub> standard established in the last review is to be compared with measurements from each appropriate monitor in an area, with no allowance for spatial averaging.

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<sup>6</sup> The original criteria for spatial averaging included: (1) the annual mean concentration at each site shall be within 20% of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.6 for each calendar quarter (62 FR 38671 to 38672, July 18, 1997).

<sup>7</sup> Specifically, the Administrator revised spatial averaging criteria such that "(1) [t]he annual mean concentration at each site shall be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.9 for each calendar quarter (71 FR 61167, October 17, 2006).



In the last review, the EPA also considered the form of the 24-hour PM<sub>2.5</sub> standard. The Agency recognized that the existing 98<sup>th</sup> percentile form for the 24-hour standard was originally selected to provide a balance between limiting the occurrence of peak 24-hour PM<sub>2.5</sub> concentrations and identifying a stable target for risk management programs.<sup>8</sup> Updated air quality analyses in the last review provided additional support for the increased stability of the 98<sup>th</sup> percentile PM<sub>2.5</sub> concentration, compared to the 99<sup>th</sup> percentile (U.S. EPA, 2011, Figure 2-2, p. 2-62). Consistent with the PA conclusions based on this analysis, the Administrator concluded that it was appropriate to retain the 98<sup>th</sup> percentile form for the 24-hour PM<sub>2.5</sub> standard (78 FR 3127, January 15, 2013).

#### **3.1.1.4 Level**

The EPA's approach to considering alternative levels of the PM<sub>2.5</sub> standards in the last review was based on evaluating the public health protection afforded by the annual and 24-hour standards, taken together, against mortality and morbidity effects associated with long-term or short-term PM<sub>2.5</sub> exposures. This approach recognized that there is no bright line clearly directing the choice of level. Rather, the choice of what is appropriate is a public health policy judgment entrusted to the Administrator. In the last review, this judgment included consideration of the strengths and limitations of the evidence and the appropriate inferences to be drawn from the evidence and the risk assessments.

In evaluating alternative standards, the Agency considered the extent to which potential alternative annual and 24-hour standard levels would be expected to reduce the mortality and morbidity risks associated with both long-term and short-term PM<sub>2.5</sub> exposures. Results of the 2010 HREA indicated that, compared to revising the 24-hour standard level, lowering the level of the annual standard would result in more consistent risk reductions across urban study areas, thereby potentially providing a more consistent degree of public health protection across the U.S. (U.S. EPA, 2010, pp. 5-15 to 5-17; 78 FR 3128, January 15, 2013). Based on risk results, together with the available evidence, the Administrator concluded that it was appropriate to lower the level of the annual standard in order to increase protection against both long- and short-term PM<sub>2.5</sub> exposures. She further concluded that it was appropriate to retain the 24-hour standard in order to provide supplemental protection, particularly for areas with high peak-to-mean ratios of 24-hour PM<sub>2.5</sub> concentrations (e.g., areas with important local or seasonal sources) and for PM<sub>2.5</sub>-related effects that may be associated with shorter-than daily exposure periods. The Administrator judged that this approach was the "most effective and efficient way to reduce

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<sup>8</sup> See *ATA III*, 283 F.3d at 374–376 which concludes that it is legitimate for the EPA to consider overall stability of the standard and its resulting promotion of overall effectiveness of NAAQS control programs in setting a standard that is requisite to protect the public health.

total PM<sub>2.5</sub>-related population risk and to protect public health with an adequate margin of safety” (78 FR 3158, January 15, 2013).

In selecting the level of the annual PM<sub>2.5</sub> standard, the Administrator recognized the substantial increase in the number and diversity of studies available in the last review, including extended analyses of seminal studies of long-term PM<sub>2.5</sub> exposures (i.e., American Cancer Society (ACS) and Harvard Six Cities studies), important new long-term exposure studies, and new U.S. multi-city epidemiologic studies that greatly expanded and reinforced our understanding of mortality and morbidity effects associated with short-term PM<sub>2.5</sub> exposures. She placed the greatest emphasis on health endpoints for which the evidence was strongest, based on the assessment of the evidence in the ISA and on the ISA’s causality determinations (U.S. EPA, 2009, section 2.3.1). She particularly noted that the evidence was sufficient to conclude a causal relationship exists between PM<sub>2.5</sub> exposures and mortality and cardiovascular effects (i.e., for both long- and short-term exposures) and that the evidence was sufficient to conclude a causal relationship is “likely” to exist between PM<sub>2.5</sub> exposures and respiratory effects (i.e., for both long- and short-term exposures). The Administrator also noted additional, but more limited, evidence for a broader range of health endpoints, including evidence “suggestive of a causal relationship” between long-term exposures and developmental and reproductive effects as well as carcinogenic effects (78 FR 3158, January 15, 2013).

Based on information discussed and presented in the ISA, the Administrator recognized that health effects may occur over the full range of ambient PM<sub>2.5</sub> concentrations observed in epidemiologic studies, since no discernible population-level threshold could be identified based on the evidence available in the last review (78 FR 3158, January 15, 2013; U.S. EPA, 2009, section 2.4.3). To inform her decisions on an appropriate level for the annual standard in the absence of a discernible population-level threshold, the Administrator considered the degree to which epidemiologic studies indicate confidence in the reported health effect associations over distributions of ambient PM<sub>2.5</sub> concentrations. In doing so, she recognized that epidemiologic studies provide greater confidence in the observed associations for the part of the air quality distribution corresponding to the bulk of the health events evaluated, generally at and around the long-term mean PM<sub>2.5</sub> concentrations. Accordingly, the Administrator weighed most heavily the long-term mean concentrations reported in key multi-city epidemiologic studies. She also took into account additional population-level information from a subset of studies, beyond the long-term mean concentrations, to identify a broader range of PM<sub>2.5</sub> concentrations to consider in

judging the need for public health protection.<sup>9</sup> In doing so, the Administrator recognized that studies indicate diminished confidence in the magnitude and significance of observed associations in the lower part of the air quality distribution, corresponding to where a relatively small proportion of the health events are observed.

In revising the level of the annual standard to  $12.0 \mu\text{g}/\text{m}^3$ , the Administrator noted that such a level was below the long-term mean  $\text{PM}_{2.5}$  concentrations reported in key epidemiologic studies that provided evidence of an array of serious health effects, including premature mortality and increased hospitalizations for cardiovascular and respiratory effects (78 FR 3161, January 15, 2013). The Administrator further noted that  $12.0 \mu\text{g}/\text{m}^3$  generally corresponded to the lower portions (i.e., about the 25<sup>th</sup> percentile) of distributions of health events in the limited number of epidemiologic studies for which population-level information was available. The Administrator viewed this population information as helpful in guiding her determination as to where her confidence in the magnitude and significance of the  $\text{PM}_{2.5}$  associations were reduced to such a degree that a standard set at a lower level was not warranted. The Administrator also recognized that a level of  $12.0 \mu\text{g}/\text{m}^3$  reflected placing some weight on studies of reproductive and developmental effects, for which the evidence was more uncertain (78 FR 3161-3162, January 15, 2013).<sup>10</sup>

In conjunction with a revised annual standard with a level of  $12.0 \mu\text{g}/\text{m}^3$ , the Administrator concluded that the evidence supported retaining the  $35 \mu\text{g}/\text{m}^3$  level of the 24-hour  $\text{PM}_{2.5}$  standard. Specifically, she judged that by lowering the level of the annual standard, the distribution of 24-hour  $\text{PM}_{2.5}$  concentrations would be lowered as well, affording additional protection against effects associated with short-term  $\text{PM}_{2.5}$  exposures.<sup>11</sup> She noted that the existing 24-hour standard, with its  $35 \mu\text{g}/\text{m}^3$  level and 98<sup>th</sup> percentile form, would provide supplemental protection, particularly for areas with high peak-to-mean ratios possibly associated with strong local or seasonal sources and for areas with  $\text{PM}_{2.5}$ -related effects that may be associated with shorter than daily exposure periods (78 FR 3163, January 15, 2013).

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<sup>9</sup> This information characterized the distribution of health events in the studies, and the corresponding long-term mean  $\text{PM}_{2.5}$  concentrations (78 FR 3130 to 3134, January 15, 2013). The additional population-level data helped inform the Administrator's judgment of how far below the long-term mean concentrations to set the level of the annual standard (78 FR 3160).

<sup>10</sup> With respect to cancer, mutagenic, and genotoxic effects, the Administrator observed that the  $\text{PM}_{2.5}$  concentrations reported in studies evaluating these effects generally included ambient concentrations that are equal to or greater than ambient concentrations observed in studies that reported mortality and cardiovascular and respiratory effects (U.S. EPA, 2009, section 7.5). Therefore, the Administrator concluded that, in selecting a standard level that provides protection from mortality and cardiovascular and respiratory effects, it is reasonable to anticipate that protection will also be provided for carcinogenic effects (78 FR 3161-3162, January 15, 2013).

<sup>11</sup> This judgment is supported by risk results presented in the 2010 HREA. For example, see section 4.2.2, and Figures 4-4 and 4-6 (U.S. EPA, 2010).

The Administrator recognized that uncertainties remained in the scientific information. She specifically noted uncertainties related to understanding the relative toxicity of the different components in the fine particle mixture, the role of PM<sub>2.5</sub> in the complex ambient mixture, exposure measurement errors in epidemiologic studies, and the nature and magnitude of estimated risks related to relatively low ambient PM<sub>2.5</sub> concentrations. Furthermore, the Administrator noted that epidemiologic studies had reported heterogeneity in responses both within and between cities and in geographic regions across the U.S. She recognized that this heterogeneity may be attributed, in part, to differences in fine particle composition in different regions and cities. With regard to evidence for reproductive and developmental effects, the Administrator recognized that there were a number of limitations associated with this body of evidence, including the following: the limited number of studies evaluating such effects; uncertainties related to identifying the relevant exposure time periods of concern; and limited toxicological evidence providing little information on the mode of action(s) or biological plausibility for an association between long-term PM<sub>2.5</sub> exposures and adverse birth outcomes.

On balance, the Administrator found that the available evidence, interpreted in light of the remaining uncertainties (noted above), did not justify an annual standard level set below 12.0 µg/m<sup>3</sup> as being “requisite” (i.e., neither more nor less stringent than necessary) to protect public health with an adequate margin of safety. Thus, the Administrator concluded that the available evidence and information supported an annual standard with a level of 12.0 µg/m<sup>3</sup>, combined with a 24-hour standard with a level of 35 µg/m<sup>3</sup>. She noted that this combination of standard levels was consistent with the CASAC’s advice to consider an annual standard level within the range of 13 to 11 µg/m<sup>3</sup> and a 24-hour standard level from 35 to 30 µg/m<sup>3</sup> (Samet, 2010c). Taken together, the Administrator concluded that the revised annual PM<sub>2.5</sub> standard, with its level of 12.0 µg/m<sup>3</sup> and a form that does not allow for spatial averaging, combined with the existing 24-hour standard, would be requisite to protect the public health with an adequate margin of safety from effects associated with long- and short-term PM<sub>2.5</sub> exposures.

### **3.1.2 General Approach in the Current Review**

The approach for this review builds on the substantial body of work completed during the last review, taking into account the more recent scientific information and air quality data now available to inform our understanding of the key policy-relevant issues. The approach summarized below is most fundamentally based on using the EPA’s assessment of the current scientific evidence for health effects attributable to fine particle exposures (i.e., in the ISA, U.S. EPA, 2019), along with quantitative assessments of PM<sub>2.5</sub>-associated health risks and analyses of PM<sub>2.5</sub> air quality, and CASAC advice, to inform the Administrator’s judgments regarding the primary standards for fine particles that are requisite to protect the public health with an adequate

margin of safety. In this PA, we seek to provide as broad an array of policy options as is supportable by the available scientific and technical information, recognizing that the selection of a specific approach to reaching final decisions on the primary PM<sub>2.5</sub> standards will reflect the judgments of the Administrator as to what weight to place on the various types of information and associated uncertainties.

In considering the public health protection provided by the current primary PM<sub>2.5</sub> standards, and the protection that could be provided by alternatives, we emphasize health outcomes for which the ISA determines that the evidence supports either a “causal” or a “likely to be causal” relationship with PM<sub>2.5</sub> exposures (U.S. EPA, 2019). We consider the PM<sub>2.5</sub>-related health effects documented in studies that support these causality determinations and, together with other analyses (i.e., air quality analyses, risk assessment), what they may indicate regarding the primary PM<sub>2.5</sub> standards. In doing so, we specifically focus on information from key epidemiologic and controlled human exposure studies.

Epidemiologic studies represent a large part of the evidence base supporting several of the ISA’s “causal” and “likely to be causal” determinations. As discussed below in section 3.2.3.2, the use of information from epidemiologic studies to inform conclusions on the primary PM<sub>2.5</sub> standards is complicated by the fact that such studies evaluate associations between distributions of ambient PM<sub>2.5</sub> and health outcomes and do not identify the specific exposures that cause reported effects. Rather, health effects can occur over the entire distributions of ambient PM<sub>2.5</sub> concentrations evaluated, and epidemiologic studies do not identify a population-level threshold below which it can be concluded with confidence that PM-associated health effects do not occur (U.S. EPA, 2019, section 1.5.3). In the absence of a discernible threshold, we use two approaches to consider information from epidemiologic studies (section 3.2.3.2).

In one approach, we evaluate the PM<sub>2.5</sub> air quality distributions over which epidemiologic studies support health effect associations and the degree to which such distributions are likely to occur in areas meeting the current (or alternative) standards. As discussed further in section 3.2.3.2.1, epidemiologic studies generally provide the strongest support for reported health effect associations over the part of the air quality distribution corresponding to the bulk of the underlying data (i.e., estimated exposures and/or health events), often falling in the middle part of the distribution (i.e., rather than at the extreme upper or lower ends). In support of this, a number of epidemiologic studies report that confidence intervals around concentration-response functions are relatively narrow around the overall means of the PM<sub>2.5</sub> concentrations examined and wider at the extreme upper and lower ends of the distributions. The observed narrowing of confidence intervals over the middle portions of these distributions likely reflects the relatively large amount of data available (i.e., the numerous “typical” daily or annual PM<sub>2.5</sub> exposures estimated). As described in greater detail in section 3.2.3.2.1, in using PM<sub>2.5</sub> air quality data from

epidemiologic studies to inform conclusions on standards we evaluate study-reported means (or medians) of daily and annual average PM<sub>2.5</sub> concentrations as proxies for the middle portions of the air quality distributions that support reported associations. When data are available, we also consider the broader PM<sub>2.5</sub> air quality distributions around the overall mean concentrations, with a focus on the lower quartiles of data, to provide insight into the concentrations below which data supporting reported associations become relatively sparse.

A key uncertainty in using study-reported PM<sub>2.5</sub> concentrations to inform conclusions on the primary PM<sub>2.5</sub> standards is that they reflect the averages of daily or annual PM<sub>2.5</sub> air quality concentrations or exposure estimates in the study population over the years examined by the study, and are not the same as the PM<sub>2.5</sub> design values used by the EPA to determine whether areas meet the NAAQS (section 3.2.3.2.1).<sup>12</sup> Therefore, as described in section 3.2.3.2.2, in this review we also consider a second approach to evaluating information from epidemiologic studies. In this approach, we calculate study area air quality metrics similar to PM<sub>2.5</sub> design values (i.e., referred to in this PA as “pseudo-design values”) and consider the degree to which such metrics indicate that study area air quality would likely have met or violated the current or alternative standards during study periods. When pseudo-design values in individual study locations are linked with the populations living in those locations, or with the number of study-specific health events recorded in those locations, these values can provide insight into the degree to which reported health effect associations are based on air quality likely to have met or violated the current (or alternative) primary PM<sub>2.5</sub> standards.

To the extent the application of these two approaches indicates that health effect associations are based on PM<sub>2.5</sub> air quality likely to have met the current or alternative standards, those standards are likely to allow the daily or annual average PM<sub>2.5</sub> exposures that provide the foundation for reported associations. Alternatively, to the extent reported health effect associations reflect air quality violating the current or alternative standards, there is greater uncertainty in the degree to which those standards would allow the PM<sub>2.5</sub> exposures that provide the foundation for reported associations. Sections 3.2.3.2.1 and 3.2.3.2.2 discuss each of these approaches in detail, and present our key observations based on their application.

Beyond epidemiologic studies, we additionally consider what controlled human exposure studies may indicate regarding the current and alternative primary PM<sub>2.5</sub> standards. Controlled human exposure studies examine short-term PM<sub>2.5</sub> exposures (i.e., up to several hours) under

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<sup>12</sup> The design value is a statistic that describes the air quality status of a given area relative to the NAAQS. As discussed further in section 3.2.3.2.1, to determine whether areas meet or violate the NAAQS, the EPA measures air pollution concentrations at individual monitors (i.e., concentrations are not averaged across monitors) and calculates design values at monitors meeting appropriate data quality and completeness criteria. For an area to meet the NAAQS, all valid design values in that area, including the highest annual and 24-hour monitored values, must be at or below the levels of the standards.

carefully controlled laboratory conditions. Drawing from the ISA, such studies report PM<sub>2.5</sub>-induced changes in markers of cardiovascular function and provide strong support for the biological plausibility of the more serious cardiovascular-related outcomes observed in epidemiologic studies (sections 3.2.1 and 3.2.3.1). Unlike most epidemiologic studies, available controlled human exposure studies provide support for effects following single, short-term PM<sub>2.5</sub> exposures to concentrations that typically correspond to the upper end of the PM<sub>2.5</sub> air quality distribution in the U.S. (i.e., “peak” concentrations). In evaluating what such controlled human exposure studies may indicate regarding the primary standards, we consider the effects reported following PM<sub>2.5</sub> exposures, the exposure concentrations/durations reported to cause those effects, and the degree to which air quality analyses indicate that such exposures are likely to occur in areas meeting the current or alternative PM<sub>2.5</sub> standards.<sup>13</sup>

Consideration of the evidence and related air quality analyses, as summarized above, informs our evaluation of the public health protection provided by the combination of the current annual and 24-hour primary PM<sub>2.5</sub> standards, as well as the protection that could be provided by alternative annual and 24-hour standards with revised levels (section 3.4). There are various ways to combine an annual standard (based on arithmetic mean concentrations) and a 24-hour standard (based on 98<sup>th</sup> percentile concentrations), to achieve an appropriate degree of public health protection. The extent to which the standards are interrelated in any given area depends in large part on the relative levels of the standards, the peak-to-mean ratios that characterize air quality patterns in the area, and whether changes in air quality designed to meet a given suite of standards are likely to be of a more regional or more localized nature. In considering the combined effects of the standards, we recognize that changes in PM<sub>2.5</sub> air quality designed to meet an annual standard would likely result not only in lower short- and long-term PM<sub>2.5</sub> concentrations near the middle of the air quality distribution (i.e., around the mean of the distribution), but also in fewer and lower short-term peak PM<sub>2.5</sub> concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98<sup>th</sup> percentile form, would result not only in fewer and lower peak 24-hour PM<sub>2.5</sub> concentrations, but also in lower annual average PM<sub>2.5</sub> concentrations.

However, while either standard could be viewed as providing some measure of protection against both average exposures and peak exposures, the 24-hour and annual standards are not expected to be equally effective at limiting both types of exposures. Specifically, the 24-hour standard (with its 98<sup>th</sup> percentile form) is more directly tied to short-term peak PM<sub>2.5</sub> concentrations than to the more typical concentrations that make up the middle portion of the air

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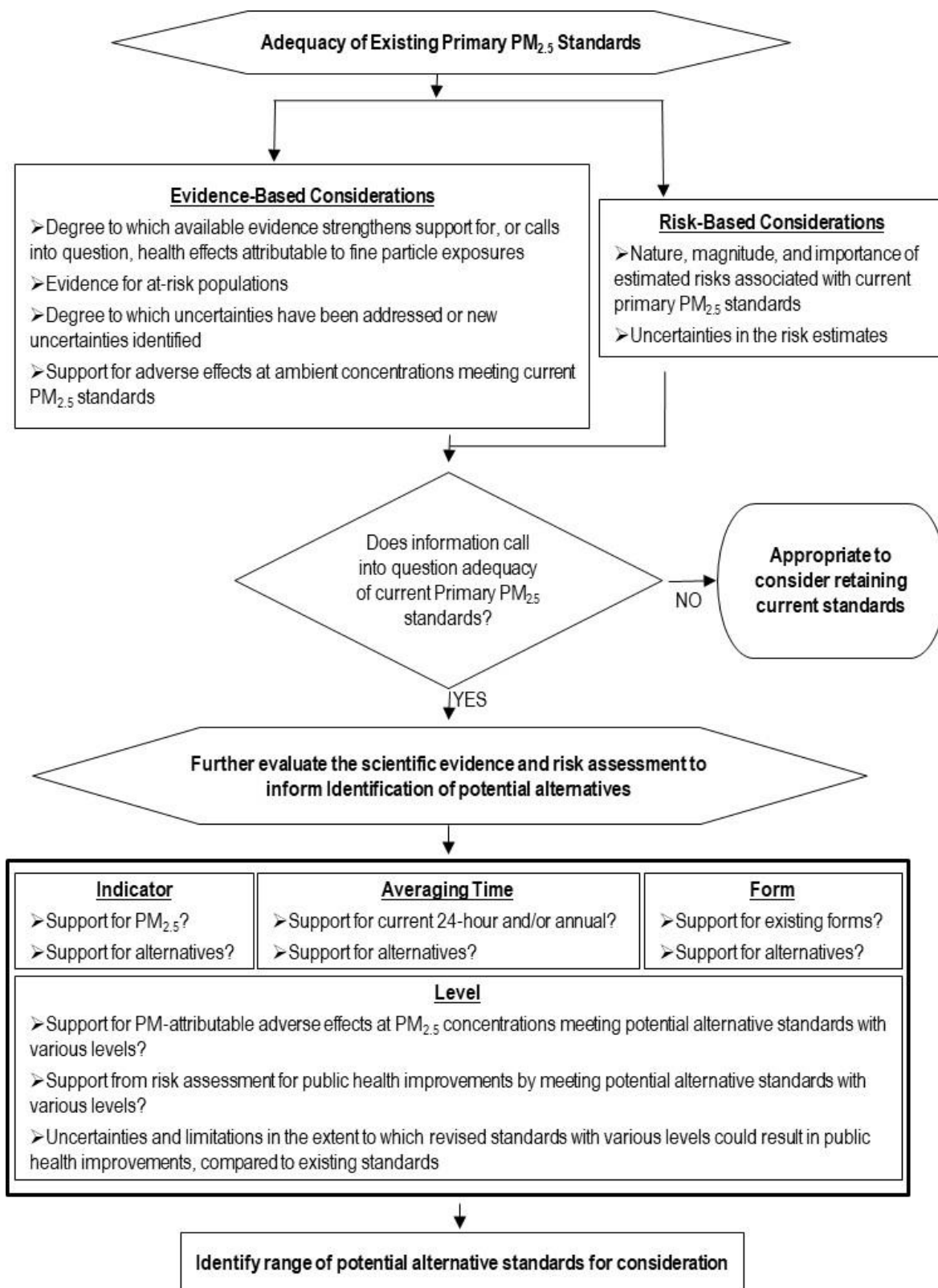
<sup>13</sup> As discussed further in section 3.2.3.1, animal toxicology studies can be similarly evaluated, though there is greater uncertainty in extrapolating the effects seen in animals, and the PM<sub>2.5</sub> exposures and doses that cause those effects, to human populations.

quality distribution, and thus more likely to appropriately limit exposures to peak concentrations. Compared to a standard that is directly tied to the middle of the air quality distribution, the 24-hour standard is less likely to appropriately limit the typical exposures that are most strongly associated with the health effects observed in epidemiologic studies. In contrast, the annual standard, with its form based on the arithmetic mean concentration, is more likely to effectively limit the PM<sub>2.5</sub> concentrations that comprise the middle portion of the air quality distribution, affording protection against the daily and annual PM<sub>2.5</sub> exposures that strongly support associations with the most serious PM<sub>2.5</sub>-related effects in epidemiologic studies (e.g., mortality, hospitalizations).

For these reasons, as in the last review (78 FR 3161-3162, January 15, 2013), we focus on the annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures, and thus protecting against the exposures that provide strong support for associations with mortality and morbidity in key epidemiologic studies. We additionally consider the 24-hour standard, with its 98<sup>th</sup> percentile form, as a means of providing supplemental protection against the short-term exposures to peak PM<sub>2.5</sub> concentrations that can occur in areas with strong contributions from local or seasonal sources, even when overall mean PM<sub>2.5</sub> concentrations remain relatively low (section 3.4).

Figure 3-1 summarizes our general approach to informing conclusions on the current primary standards and on potential alternatives. Subsequent sections of this chapter provide additional detail on this general approach.





**Figure 3-1. Overview of general approach for review of primary PM<sub>2.5</sub> standards.**

In adopting the approach outlined above, we recognize that decisions on the primary PM<sub>2.5</sub> standards are largely public health policy judgments to be made by the Administrator. The Administrator's final decisions will draw upon the scientific evidence for PM-related health effects, information from the quantitative assessment of population health risks, information from analyses of air quality, and judgments about how to consider the uncertainties and limitations that are inherent in the evidence and information. To inform the Administrator's public health policy judgments and decisions, the PA considers support for, and the potential implications of, placing more or less weight on various aspects of this evidence, air quality and risk information, and associated uncertainties and limitations.

This approach is consistent with the requirements of the NAAQS provisions of the CAA and with how the EPA and the courts have historically interpreted these CAA provisions. The CAA requires primary standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety. In setting primary standards that are "requisite" to protect public health, the EPA's task is to establish standards that are neither more nor less stringent than necessary for this purpose. The requirement that primary standards provide an "adequate margin of safety" is meant to address uncertainties associated with inconclusive scientific and technical information. Thus, as discussed in section 1.1 of this PA, the CAA does not require that primary standards be set at a zero-risk level, but rather at a level that, in the judgment of the Administrator, limits risk sufficiently so as to protect public health with an adequate margin of safety.

## 3.2 EVIDENCE-BASED CONSIDERATIONS

In this section, we draw from the EPA's synthesis and assessment of the scientific evidence presented in the ISA (U.S. EPA, 2019) to consider the following policy-relevant question:

- **To what extent does the currently available scientific evidence, as assessed in the ISA, support or call into question the public health protection afforded by the current suite of PM<sub>2.5</sub> standards?**

The ISA uses a weight-of-evidence framework for characterizing the strength of the available scientific evidence for health effects attributable to PM exposures (U.S. EPA, 2015, Preamble, Section 5). This framework provides the basis for robust, consistent, and transparent evaluation of the scientific evidence, including its uncertainties, and for drawing conclusions on PM-related health effects. As in the last review (U.S. EPA, 2009), the ISA for this review has adopted a five-level hierarchy to classify the overall weight of evidence into one of the following categories: causal relationship; likely to be a causal relationship; suggestive of, but not sufficient to infer, a causal relationship; inadequate to infer a causal relationship; and not likely to be a causal

relationship (U.S. EPA, 2015, Preamble Table II). In using the weight-of-evidence approach to inform judgments about the likelihood that various health effects are caused by PM exposures, evidence is evaluated for major outcome categories or groups of related outcomes (e.g., respiratory effects), integrating evidence from across disciplines, including epidemiologic, controlled human exposure, and animal toxicological studies and evaluating the coherence of evidence across a spectrum of related endpoints (U.S. EPA, 2015, Preamble, Section 5.c.). In this PA, we consider the full body of health evidence, placing the greatest emphasis on the health effects for which the evidence has been judged in the ISA to demonstrate a “causal” or a “likely to be causal” relationship with PM exposures. The ISA defines these causality determinations as follows (U.S. EPA, 2019, p. p-20):

- Causal relationship: the pollutant has been shown to result in health effects at relevant exposures based on studies encompassing multiple lines of evidence and chance, confounding, and other biases can be ruled out with reasonable confidence.
- Likely to be a causal relationship: there are studies in which results are not explained by chance, confounding, or other biases, but uncertainties remain in the health effects evidence overall. For example, the influence of co-occurring pollutants is difficult to address, or evidence across scientific disciplines may be limited or inconsistent.

In the sections below, we consider the nature of the health effects attributable to long- and short-term fine particle exposures (Section 3.2.1), the populations potentially at increased risk for PM-related effects (Section 3.2.2), and the PM<sub>2.5</sub> concentrations at which effects have been shown to occur (Section 3.2.3).

### 3.2.1 Nature of Effects

In considering the available evidence for health effects attributable to PM<sub>2.5</sub> exposures presented in the ISA, this section poses the following policy-relevant questions:

- **To what extent does the currently available scientific evidence strengthen, or otherwise alter, our conclusions from the last review regarding health effects attributable to long- or short-term fine particle exposures? Have previously identified uncertainties been reduced? What important uncertainties remain and have new uncertainties been identified?**

In answering these questions, as noted above, we consider the full body of evidence assessed in the ISA, placing particular emphasis on health outcomes for which the evidence supports either a “causal” or a “likely to be causal” relationship. While the strongest evidence focuses on PM<sub>2.5</sub>, the ISA also assesses the evidence for the ultrafine fraction of PM<sub>2.5</sub> (ultrafine particles or UFP),

generally considered as particulates with a diameter less than or equal to  $0.1\ \mu\text{m}$ <sup>14</sup> (typically based on physical size, thermal diffusivity or electrical mobility) (U.S. EPA, 2019, Preface, p. 11). Table 3-1 lists the health outcomes for which the ISA concludes the evidence supports either a causal, a likely to be causal, or a suggestive relationship (adapted from U.S. EPA, 2019, Table 1-4).

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<sup>14</sup> Though definitions of UFP vary across the scientific literature and, as discussed in sections 3.2.1.5 and 3.2.1.6, UFP exposures in animal toxicological and controlled human exposure studies typically use a particle concentrator, which can result in exposures to particles  $> 0.1\ \mu\text{m}$  in diameter in some studies of UFP-related health effects.

**Table 3-1. Key causality determinations for PM<sub>2.5</sub> and UFP exposures.**

Health Outcome	Size Fraction	Exposure Duration	2009 PM ISA	2019 PM ISA
Mortality	PM <sub>2.5</sub>	Long-term	Causal	Causal
		Short-term		
Cardiovascular effects	PM <sub>2.5</sub>	Long-term	Causal	Causal
		Short-term		
	UFP	Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Respiratory effects	PM <sub>2.5</sub>	Long-term	Likely to be causal	Likely to be causal
		Short-term		
	UFP	Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Cancer	PM <sub>2.5</sub>	Long-term	Suggestive of, but not sufficient to infer	Likely to be causal
Nervous System effects	PM <sub>2.5</sub>	Long-term	---	Likely to be causal
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
	UFP	Long-term	---	Suggestive of, but not sufficient to infer
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
Metabolic effects	PM <sub>2.5</sub>	Long-term	---	Suggestive of, but not sufficient to infer
		Short-term	---	Suggestive of, but not sufficient to infer
Reproduction and Fertility	PM <sub>2.5</sub>	Long-, Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Pregnancy and Birth Outcomes				

Table 3-1 lists the health outcomes for which the ISA concludes the evidence supports either a causal, a likely to be causal, or a suggestive relationship. For other health outcomes, the ISA concludes the evidence is inadequate to infer a causal relationship (U.S. EPA, 2019, Table 1-4).

The 2009 ISA (U.S. EPA, 2009) made causality determinations for the broad category of “Reproductive and Developmental Effects.” Causality determinations for 2009 represent this broad category and not specifically for “Male and Female Reproduction and Fertility” and “Pregnancy and Birth Outcomes”.

For reproductive and developmental effects, the ISA’s causality determinations reflect the combined evidence for both short- and long-term exposures (U.S. EPA, 2019, Chapter 9).

Sections 3.2.1.1 to 3.2.1.5 summarize the evidence supporting the ISA’s “causal” and “likely to be causal” determinations for PM<sub>2.5</sub> (bold, italics in Table 3-1). Section 3.2.1.6 briefly summarizes the evidence supporting the ISA’s “suggestive” determinations. Each of these sections focuses on addressing the policy-relevant questions posed above. Section 3.2.1.7 summarizes the evidence in preceding sections and revisits the policy-relevant questions posed above.

### **3.2.1.1 Mortality**

#### Long-term PM<sub>2.5</sub> exposures

In the last review, the 2009 PM ISA reported that the evidence was “sufficient to conclude that the relationship between long-term PM<sub>2.5</sub> exposures and mortality is causal” (U.S. EPA, 2009, p. 7-96). The strongest evidence supporting this conclusion was provided by epidemiologic studies, particularly those examining two seminal cohort, the American Cancer Society (ACS) and the Harvard Six Cities cohorts. Analyses of the Harvard Six Cities cohort included demonstrations that reductions in ambient PM<sub>2.5</sub> concentrations are associated with reduced mortality risk (Laden et al., 2006) and with increases in life expectancy (Pope et al., 2009). Further support was provided by other cohort studies conducted in North America and Europe that also reported positive associations between long-term PM<sub>2.5</sub> exposures and risk of mortality (U.S. EPA, 2009).

Recent cohort studies, which have become available since the 2009 ISA, continue to provide consistent evidence of positive associations between long-term PM<sub>2.5</sub> exposures and mortality. These studies add support for associations with total and non-accidental mortality,<sup>15</sup> as well as with specific causes of death, including cardiovascular disease and respiratory disease (U.S. EPA, 2019, section 11.2.2). Many of these recent studies have extended the follow-up periods originally evaluated in the ACS and Harvard Six Cities cohorts and continue to observe positive associations between long-term PM<sub>2.5</sub> exposures and mortality (U.S. EPA, 2019, section 11.2.2.1; Figures 11-18 and 11-19). Adding to recent evaluations of the ACS and Six Cities cohorts, studies conducted in other cohorts also demonstrate consistent, positive associations between long-term PM<sub>2.5</sub> exposure and mortality across various demographic groups (e.g., age, sex, occupation), spatial and temporal extents, exposure assessment metrics, and statistical techniques (U.S. EPA, 2019, sections 11.2.2.1, 11.2.5). This includes some of the largest cohort studies conducted to date, with analyses of the U.S. Medicare cohort that include nearly 61 million enrollees (Di et al., 2017b) and studies that control for a range of individual and

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<sup>15</sup> The majority of these studies examined non-accidental mortality outcomes, though some Medicare studies lack cause-specific death information and, therefore, examine total mortality.

ecological covariates, such as race, age, socioeconomic status, smoking status, body mass index, and annual weather variables (e.g., temperature, humidity).

A recent series of retrospective studies has additionally tested the hypothesis that past reductions in ambient PM<sub>2.5</sub> concentrations have been associated with increased life expectancy or a decreased mortality rate (U.S. EPA, 2019, section 11.2.2.5). In their original study, Pope et al. (2009) used air quality data in a cross-sectional analysis from 51 metropolitan areas across the U.S., beginning in the 1970s through the early 2000s, to demonstrate that a 10 µg/m<sup>3</sup> decrease in long-term PM<sub>2.5</sub> concentration was associated with a 0.61-year increase in life expectancy. In a subsequent analysis, these authors extended the period of analysis to include 2000 to 2007 (Correia et al., 2013), a time period with lower ambient PM<sub>2.5</sub> concentrations. In this follow-up study, a decrease in long-term PM<sub>2.5</sub> concentration continued to be associated with an increase in life expectancy, though the magnitude of the increase was smaller than during the earlier time period (i.e., a 10 µg/m<sup>3</sup> decrease in long-term PM<sub>2.5</sub> concentration was associated with a 0.35-year increase in life expectancy). Additional studies conducted in the U.S. or Europe similarly report that reductions in ambient PM<sub>2.5</sub> are associated with improvements in longevity (U.S. EPA, 2019, section 11.2.2.5).

The ISA specifically evaluates the degree to which recent studies that examine the relationship between long-term PM<sub>2.5</sub> exposure and mortality have addressed key policy-relevant issues and/or previously identified data gaps in the scientific evidence. For example, based on its assessment of the evidence, the ISA concludes that positive associations between long-term PM<sub>2.5</sub> exposures and mortality are robust across recent analyses using various approaches to estimate PM<sub>2.5</sub> exposures (e.g., based on monitors, modeling, satellites, or hybrid methods that combine information from multiple sources) (U.S. EPA, 2019, section 11.2.5.1). This includes a recent study (Hart et al. (2015) reporting that correction for bias due to exposure measurement error increases the magnitude of the hazard ratios (confidence intervals widen but the association remains statistically significant), suggesting that failure to correct for exposure measurement error could result in attenuation or underestimation of risk estimates. The ISA additionally concludes that positive associations between long-term PM<sub>2.5</sub> exposures and mortality are robust across statistical models that use different approaches to control for confounders or different sets of confounders (U.S. EPA, 2019, sections 11.2.3 and 11.2.5), across diverse geographic regions and populations, and across a range of temporal periods including the periods of declining PM concentrations (U.S. EPA, 2019, sections 11.2.2.5 and 11.2.5.3). Recent evidence further demonstrates that associations with mortality remain robust in copollutants analyses (U.S. EPA, 2019, section 11.2.3), and that associations persist in analyses restricted to long-term exposures below 12 µg/m<sup>3</sup> (Di et al., 2017b) or 10 µg/m<sup>3</sup> (Shi et al., 2016) (i.e., indicating that risks are not disproportionately driven by the upper portions of the air quality distribution).

An emerging group of studies explores the use of causal inference methods to further evaluate the causal nature of relationships between long-term PM<sub>2.5</sub> exposure and mortality (U.S. EPA, 2019, section 11.2.2.4). The goal of these methods is to “estimate the difference (or ratio) in the expected value of [an] outcome in the population under the exposure they received versus what it would have been had they received an alternative exposure” (Schwartz et al., 2015). For example, Wang et al. (2016)) observe a positive and statistically significant relationship between long-term exposure to PM<sub>2.5</sub> and total (nonaccidental) mortality in New Jersey using a difference-in-difference approach to control for geographical differences, long-term temporal trends, and temperature. Additionally, a few recent studies use statistical techniques to reduce uncertainties related to potential confounding in order to further inform conclusions on causality for long-term PM<sub>2.5</sub> exposure and mortality. For example, studies by Greven et al. (2011) and Pun et al. (2017) decompose ambient PM<sub>2.5</sub> into “spatial” and “spatiotemporal” components in order to evaluate the potential for bias due to unmeasured spatial confounding. The results of these analyses suggest the presence of unmeasured confounding for several health outcomes, though they do not indicate the direction or magnitude of the bias that could result.<sup>16 17</sup>

An additional important consideration in characterizing the public health impacts associated with PM<sub>2.5</sub> exposure is whether concentration-response relationships are linear across the range of concentrations or if nonlinear relationships exist along any part of this range. Several recent studies examine this issue, and continue to provide evidence of linear, no-threshold relationships between long-term PM<sub>2.5</sub> exposures and all-cause and cause-specific mortality (U.S. EPA, 2019, section 11.2.4). Though available studies have not systematically evaluated alternatives to a linear fitted model of concentration-response relationships, potential deviations from linearity have been assessed in individual studies using a variety of approaches (U.S. EPA, 2019, Table 11-7). However, interpreting the shapes of these relationships, particularly at PM<sub>2.5</sub> concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources

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<sup>16</sup> In public comments on the draft PA, the authors of the Pun et al. study further note that “the presence of unmeasured confounding...was expected given that we did not control for several potential confounders that may impact PM<sub>2.5</sub>-mortality associations, such as smoking, socio-economic status (SES), gaseous pollutants, PM<sub>2.5</sub> components, and long-term time trends in PM<sub>2.5</sub>” and that “spatial confounding may bias mortality risks both towards and away from the null” (Docket ID EPA-HQ-OAR-2015-0072-0065; accessible in <https://www.regulations.gov/>)

<sup>17</sup> In its letter on the draft PA, the CASAC cites the study by Eum et al. (2018), which evaluates approaches similar to those in Greven et al. (2011) and Pun et al. (2017). Eum et al. (2018) concludes that associations between 1-year PM<sub>2.5</sub> exposures and mortality “were likely confounded by long-term temporal trends in PM<sub>2.5</sub>” but that controlling for this confounding still resulted in a statistically significant “11.7% increase in all-cause mortality among Medicare beneficiaries for a 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>.”



of variability and uncertainty tend to smooth and “linearize” population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015, section 6.c).

The biological plausibility of PM<sub>2.5</sub>-attributable mortality is supported by the coherence of effects across scientific disciplines (i.e., animal toxicological, controlled human exposure studies, and epidemiologic), including in recent studies evaluating the morbidity effects that are the largest contributors to total (nonaccidental) mortality. The ISA outlines the available evidence for plausible pathways by which inhalation exposure to PM<sub>2.5</sub> could progress from initial events (e.g., pulmonary inflammation, autonomic nervous system activation) to endpoints relevant to population outcomes, particularly those related to cardiovascular diseases such as ischemic heart disease, stroke and atherosclerosis (U.S. EPA, 2019, section 6.2.1), and to metabolic disease and diabetes (U.S. EPA, 2019, section 7.3.1). The ISA notes “more limited evidence from respiratory morbidity” (U.S. EPA, 2019, p. 11-101) such as exacerbation of COPD (U.S. EPA, 2019, section 5.2.1) to support the biological plausibility of mortality due to long-term PM<sub>2.5</sub> exposures (U.S. EPA, 2019, section 11.2.1).

Taken together, recent studies reaffirm and further strengthen the body of evidence from the 2009 ISA for the relationship between long-term PM<sub>2.5</sub> exposure and mortality. Recent epidemiologic studies consistently report positive associations with mortality across different geographic locations, populations, and analytic approaches. Such studies reduce key uncertainties identified in the last review, including those related to potential copollutant confounding, and provide additional information on the shape of the concentration-response curve. Recent experimental and epidemiologic evidence for cardiovascular effects, and respiratory effects to a more limited degree, supports the plausibility of mortality due to long-term PM<sub>2.5</sub> exposures. The ISA concludes that, “collectively, this body of evidence is sufficient to conclude that a causal relationship exists between long-term PM<sub>2.5</sub> exposure and total mortality” (U.S. EPA, 2019, section 11.2.7; p. 11-102).

#### Short-term PM<sub>2.5</sub> exposures

The 2009 PM ISA concluded that “a causal relationship exists between short-term exposure to PM<sub>2.5</sub> and mortality” (U.S. EPA, 2009). This conclusion was based on the evaluation of both multi- and single-city epidemiologic studies that consistently reported positive associations between short-term PM<sub>2.5</sub> exposure and non-accidental mortality. These associations were strongest, in terms of magnitude and precision, primarily at lags of 0 to 1 days. Examination of the potential confounding effects of gaseous copollutants was limited, though evidence from single-city studies indicated that gaseous copollutants have minimal effect on the PM<sub>2.5</sub>-mortality relationship (i.e., associations remain robust to inclusion of other pollutants in copollutant models). The evaluation of cause-specific mortality found that effect estimates were

larger in magnitude, but also had larger confidence intervals, for respiratory mortality compared to cardiovascular mortality. Although the largest mortality risk estimates were for respiratory mortality, the interpretation of the results was complicated by the limited coherence from studies of respiratory morbidity. However, the evidence from studies of cardiovascular morbidity provided both coherence and biological plausibility for the relationship between short-term PM<sub>2.5</sub> exposure and cardiovascular mortality.

Recent multicity studies evaluated since the 2009 ISA continue to provide evidence of primarily positive associations between daily PM<sub>2.5</sub> exposures and mortality, with percent increases in total mortality ranging from 0.19% (Lippmann et al., 2013) to 2.80% (Kloog et al., 2013)<sup>18</sup> at lags of 0 to 1 days in single-pollutant models. Whereas most studies rely on assigning exposures using data from ambient monitors, associations are also reported in recent studies that employ hybrid modeling approaches using additional PM<sub>2.5</sub> data (i.e., from satellites, land use information, and modeling, in addition to monitors), allowing for the inclusion of more rural locations in analyses (Kloog et al., 2013, Shi et al., 2016, Lee et al., 2015).

Some recent studies have expanded the examination of potential confounders, including long-term temporal trends, weather, and co-occurring pollutants. Mortality associations were found to remain positive, although in some cases were attenuated, when using different approaches to account for temporal trends or weather covariates (U.S. EPA, 2019, section 11.1.5.1). For example, Sacks et al. (2012) examined the influence of model specification using the approaches for confounder adjustment from models employed in several recent multicity studies within the context of a common data set (U.S. EPA, 2019, section 11.1.5.1). These models use different approaches to control for long-term temporal trends and the potential confounding effects of weather. The authors report that associations between daily PM<sub>2.5</sub> and cardiovascular mortality were similar across models, with the percent increase in mortality ranging from 1.5–2.0% (U.S. EPA, 2019, Figure 11-4). Thus, alternative approaches to controlling for long-term temporal trends and for the potential confounding effects of weather may influence the magnitude of the association between PM<sub>2.5</sub> exposures and mortality but have not been found to influence the direction of the observed association (U.S. EPA, 2019, section 11.1.5.1). Taken together, the ISA concludes that recent multicity studies conducted in the U.S., Canada, Europe, and Asia continue to provide consistent evidence of positive associations between short-term PM<sub>2.5</sub> exposures and total mortality across studies that use different approaches to control for the potential confounding effects of weather (e.g., temperature) (U.S. EPA, 2019, section 1.4.1.5.1).

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<sup>18</sup> As detailed in the Preface to the ISA, risk estimates are for a 10 µg/m<sup>3</sup> increase in 24-hour avg PM<sub>2.5</sub> concentrations, unless otherwise noted (U.S. EPA, 2019).

With regard to copollutants, recent studies provide additional evidence that associations between short-term PM<sub>2.5</sub> exposures and mortality remain positive and relatively unchanged in copollutant models with both gaseous pollutants and PM<sub>10-2.5</sub> (U.S. EPA, 2019, Section 11.1.4). Additionally, the low ( $r < 0.4$ ) to moderate correlations ( $r = 0.4-0.7$ ) between PM<sub>2.5</sub> and gaseous pollutants and PM<sub>10-2.5</sub> increase the confidence in PM<sub>2.5</sub> having an independent effect on mortality (U.S. EPA, 2019, section 11.1.4).

The generally positive associations reported with mortality are supported by a small group of studies employing causal inference or quasi-experimental statistical approaches (U.S. EPA, 2019, section 11.1.2.1). For example, two studies by Schwartz et al. (Schwartz et al., 2015; Schwartz et al., 2017) report associations between PM<sub>2.5</sub> instrumental variables and mortality (U.S. EPA, 2019, Table 11-2), including in an analysis limited to days with 24-hour average PM<sub>2.5</sub> concentrations  $<30 \mu\text{g}/\text{m}^3$  (Schwartz et al., 2017). In addition to the main analyses, these studies conducted Granger-like causality tests as sensitivity analyses to examine whether there was evidence of an association between mortality and PM<sub>2.5</sub> after the day of death, which would support the possibility that unmeasured confounders were not accounted for in the statistical model. Neither study reports evidence of an association with PM<sub>2.5</sub> after death (i.e., they do not indicate unmeasured confounding). A recent quasi-experimental study examines whether a specific regulatory action in Tokyo, Japan (i.e., a diesel emission control ordinance) resulted in a subsequent reduction in daily mortality (Yorifuji et al., 2016). The authors report a reduction in mortality in Tokyo due to the ordinance, compared to Osaka, which did not have a similar diesel emission control ordinance in place.

The positive associations for total mortality reported across the majority of studies evaluated are further supported by analyses reporting generally consistent, positive associations with both cardiovascular and respiratory mortality (U.S. EPA, 2019, section 11.1.3). For both cardiovascular and respiratory mortality, there has been only limited assessment of potential copollutant confounding, though initial evidence indicates that associations remain positive and relatively unchanged in models with gaseous pollutants and PM<sub>10-2.5</sub>. This evidence further supports the copollutant analyses conducted for total mortality. The strong evidence for ischemic events and heart failure, as detailed in the assessment of cardiovascular morbidity (U.S. EPA, 2019, Chapter 6), provides biological plausibility for PM<sub>2.5</sub>-related cardiovascular mortality, which comprises the largest percentage of total mortality (i.e., ~33%) (NHLBI, 2017). Although there is evidence for exacerbations of COPD and asthma, the collective body of respiratory morbidity evidence provides only limited biological plausibility for PM<sub>2.5</sub>-related respiratory mortality (U.S. EPA, 2019, Chapter 5).

In the 2009 ISA, one of the main uncertainties identified was the regional and city-to-city heterogeneity in PM<sub>2.5</sub>-mortality associations. Recent studies examine both city-specific as well

as regional characteristics to identify the underlying contextual factors that could contribute to this heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Analyses focusing on effect modification of the PM<sub>2.5</sub>-mortality relationship by PM<sub>2.5</sub> components, regional patterns in PM<sub>2.5</sub> components and city-specific differences in composition and sources indicate some differences in the PM<sub>2.5</sub> composition and sources across cities and regions, but these differences do not fully explain the observed heterogeneity. Additional studies find that factors related to potential exposure differences, such housing stock and commuting, as well as city-specific factors (e.g., land-use, port volume, and traffic information), may explain some of the observed heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Collectively, recent studies indicate that the heterogeneity in PM<sub>2.5</sub>-mortality risk estimates cannot be attributed to one factor, but instead a combination of factors including, but not limited to, PM composition and sources as well as community characteristics that could influence exposures (U.S. EPA, 2019, section 11.1.12).

A number of recent studies conducted systematic evaluations of the lag structure of associations for the PM<sub>2.5</sub>-mortality relationship by examining either a series of single-day or multiday lags and these studies continue to support an immediate effect (i.e., lag 0 to 1 days) of short-term PM<sub>2.5</sub> exposures on mortality (U.S. EPA, 2019, section 11.1.8.1). Recent studies also conducted analyses comparing the traditional 24-hour average exposure metric with a sub-daily metric (i.e., 1-hour max). These initial studies provide evidence of a similar pattern of associations for both the 24-hour average and 1-hour max metric, with the association larger in magnitude for the 24-hour average metric.

Recent multicity studies indicate that positive and statistically significant associations with mortality persist in analyses restricted to short-term PM<sub>2.5</sub> exposures below 35 µg/m<sup>3</sup> (Lee et al., 2015),<sup>19</sup> below 30 µg/m<sup>3</sup> (Shi et al., 2016), and below 25 µg/m<sup>3</sup> (Di et al., 2017a), indicating that risks associated with short-term PM<sub>2.5</sub> exposures are not disproportionately driven by the peaks of the air quality distribution. Additional studies examine the shape of the concentration-response relationship and whether a threshold exists specifically for PM<sub>2.5</sub> (U.S. EPA, 2019, section 11.1.10). These studies have used various statistical approaches and consistently demonstrate a linear relationship with no evidence of a threshold. Recent analyses provide initial evidence indicating that PM<sub>2.5</sub>-mortality associations persist and may be stronger (i.e., a steeper slope) at lower concentrations (e.g., Di et al., 2017a; Figure 11-12 in U.S. EPA, 2019). However, given the limited data available at the lower end of the distribution of ambient PM<sub>2.5</sub> concentrations, the shape of the concentration-response curve remains uncertain at these low concentrations and, to date, studies have not conducted extensive analyses exploring

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<sup>19</sup> Lee et al. (2015) also report that positive and statistically significant associations between short-term PM<sub>2.5</sub> exposures and mortality persist in analyses restricted to areas with long-term concentrations below 12 µg/m<sup>3</sup>.

alternatives to linearity when examining the shape of the PM<sub>2.5</sub>-mortality concentration-response relationship.

Overall, recent epidemiologic studies build upon and extend the conclusions of the 2009 ISA for the relationship between short-term PM<sub>2.5</sub> exposures and total mortality. Supporting evidence for PM<sub>2.5</sub>-related cardiovascular morbidity, and more limited evidence from respiratory morbidity, provides biological plausibility for mortality due to short-term PM<sub>2.5</sub> exposures. The primarily positive associations observed across studies conducted in diverse geographic locations is further supported by the results from co-pollutant analyses indicating robust associations, along with evidence from analyses of the concentration-response relationship. The ISA states that, collectively, “this body of evidence is sufficient to conclude that a causal relationship exists between short-term PM<sub>2.5</sub> exposure and total mortality” (U.S. EPA, 2019, pp. 11-58).

### **3.2.1.2 Cardiovascular Effects**

#### Long-term PM<sub>2.5</sub> exposures

The scientific evidence reviewed in the 2009 PM ISA was “sufficient to infer a causal relationship between long-term PM<sub>2.5</sub> exposure and cardiovascular effects” (U.S. EPA, 2009). The strongest line of evidence comprised findings from several large epidemiologic studies of U.S. cohorts that consistently showed positive associations between long-term PM<sub>2.5</sub> exposure and cardiovascular mortality (Pope et al., 2004, Krewski et al., 2009, Miller et al., 2007, Laden et al., 2006). Studies of long-term PM<sub>2.5</sub> exposure and cardiovascular morbidity were limited in number. Biological plausibility and coherence with the epidemiologic findings were provided by studies using genetic mouse models of atherosclerosis demonstrating enhanced atherosclerotic plaque development and inflammation, as well as changes in measures of impaired heart function, following 4- to 6-month exposures to PM<sub>2.5</sub> concentrated ambient particles (CAPs), and by a limited number of studies reporting CAPs-induced effects on coagulation factors, vascular reactivity, and worsening of experimentally induced hypertension in mice (U.S. EPA, 2009).

Consistent with the evidence assessed in the 2009 PM ISA, the 2019 ISA concludes that recent studies, together with the evidence available in previous reviews, support a causal relationship between long-term exposure to PM<sub>2.5</sub> and cardiovascular effects. As discussed above (section 3.2.1.1), results from recent U.S. and Canadian cohort studies consistently report positive associations between long-term PM<sub>2.5</sub> exposure and cardiovascular mortality (U.S. EPA, 2019, Figure 6-19) in evaluations conducted at varying spatial scales and employing a variety of exposure assessment and statistical methods (U.S. EPA, 2019, section 6.2.10). Positive associations between long-term PM<sub>2.5</sub> exposures and cardiovascular mortality are generally robust in copollutant models adjusted for ozone, NO<sub>2</sub>, PM<sub>10-2.5</sub>, or SO<sub>2</sub>. In addition, most of the results from analyses examining the shape of the concentration-response relationship for

cardiovascular mortality support a linear relationship with long-term PM<sub>2.5</sub> exposures and do not identify a threshold below which effects do not occur (U.S. EPA, 2019, section 6.2.16; Table 6-52).<sup>20</sup>

The body of literature examining the relationship between long-term PM<sub>2.5</sub> exposure and cardiovascular morbidity has greatly expanded since the 2009 PM ISA, with positive associations reported in several cohorts (U.S. EPA, 2019, section 6.2). Though results for cardiovascular morbidity are less consistent than those for cardiovascular mortality (U.S. EPA, 2019, section 6.2), recent studies provide some evidence for associations between long-term PM<sub>2.5</sub> exposures and the progression of cardiovascular disease. Positive associations with cardiovascular morbidity (e.g., coronary heart disease, stroke) and atherosclerosis progression are observed in several epidemiologic studies (U.S. EPA, 2019, sections 6.2.2. to 6.2.9). Associations in such studies are supported by toxicological evidence for increased plaque progression in mice following long-term exposure to PM<sub>2.5</sub> collected from multiple locations across the U.S. (U.S. EPA, 2019, section 6.2.4.2). A small number of epidemiologic studies also report positive associations between long-term PM<sub>2.5</sub> exposure and heart failure, changes in blood pressure, and hypertension (U.S. EPA, 2019, sections 6.2.5 and 6.2.7). Associations with heart failure are supported by animal toxicological studies demonstrating decreased cardiac contractility and function, and increased coronary artery wall thickness following long-term PM<sub>2.5</sub> exposure (U.S. EPA, 2019, section 6.2.5.2). Similarly, a limited number of animal toxicological studies demonstrating a relationship between long-term exposure to PM<sub>2.5</sub> and consistent increases in blood pressure in rats and mice are coherent with epidemiologic studies reporting positive associations between long-term exposure to PM<sub>2.5</sub> and hypertension.

Longitudinal epidemiologic analyses also report positive associations with markers of systemic inflammation (U.S. EPA, 2019, section 6.2.11), coagulation (U.S. EPA, 2019, section 6.2.12), and endothelial dysfunction (U.S. EPA, 2019, section 6.2.13). These results are coherent with animal toxicological studies generally reporting increased markers of systemic inflammation, oxidative stress, and endothelial dysfunction (U.S. EPA, 2019, section 6.2.12.2 and 6.2.14).

In summary, the ISA concludes that there is consistent evidence from multiple epidemiologic studies illustrating that long-term exposure to PM<sub>2.5</sub> is associated with mortality from cardiovascular causes. Associations with CHD, stroke and atherosclerosis progression were observed in several additional epidemiologic studies providing coherence with the mortality findings. Results from copollutant models generally support the independence of the PM<sub>2.5</sub> associations. Additional evidence of the independent effect of PM<sub>2.5</sub> on the cardiovascular

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<sup>20</sup> As noted above for mortality, uncertainty in the shape of the concentration-response relationship increases near the upper and lower ends of the distribution due to limited data.

system is provided by experimental studies in animals, which demonstrate biologically plausible pathways by which long-term inhalation exposure to PM<sub>2.5</sub> could potentially result in outcomes such as CHD, stroke, CHF and cardiovascular mortality. The combination of epidemiologic and experimental evidence results in the ISA conclusion that “a causal relationship exists between long-term exposure to PM<sub>2.5</sub> and cardiovascular effects” (U.S. EPA, 2019, section 6.2.18).

#### Short-term PM<sub>2.5</sub> exposures

The 2009 PM ISA concluded that “a causal relationship exists between short-term exposure to PM<sub>2.5</sub> and cardiovascular effects” (U.S. EPA, 2009). The strongest evidence in the 2009 PM ISA was from epidemiologic studies of ED visits and hospital admissions for IHD and HF, with supporting evidence from epidemiologic studies of cardiovascular mortality (U.S. EPA, 2009). Animal toxicological studies provided coherence and biological plausibility for the positive associations reported with myocardial ischemia ED visit and hospital admissions. These included studies reporting reduced myocardial blood flow during ischemia and studies indicating altered vascular reactivity. In addition, effects of PM<sub>2.5</sub> exposure on a potential indicator of ischemia (i.e., ST segment depression on an electrocardiogram) were reported in both animal toxicological and epidemiologic panel studies.<sup>21</sup> Key uncertainties from the last review resulted from inconsistent results across disciplines with respect to the relationship between short-term exposure to PM<sub>2.5</sub> and changes in blood pressure, blood coagulation markers, and markers of systemic inflammation. In addition, while the 2009 PM ISA identified a growing body of evidence from controlled human exposure and animal toxicological studies, uncertainties remained with respect to biological plausibility.

A large body of recent evidence confirms and extends the evidence from the 2009 ISA indicating that there is a causal relationship between short-term PM<sub>2.5</sub> exposure and cardiovascular effects. This includes generally positive associations observed in multicity epidemiologic studies of emergency department visits and hospital admissions for ischemic heart disease (IHD), heart failure (HF), and combined cardiovascular-related endpoints. In particular, nationwide studies of older adults (65 years and older) using Medicare records report positive associations between PM<sub>2.5</sub> exposures and hospital admissions for HF (U.S. EPA, 2019, section 6.1.3.1). Additional multicity studies conducted in the northeast U.S. report positive associations between short-term PM<sub>2.5</sub> exposures and emergency department visits or hospital admissions for IHD (U.S. EPA, 2019, section 6.1.2.1) while studies conducted in the U.S. and Canada reported positive associations between short-term PM<sub>2.5</sub> exposures and emergency department visits for HF. Epidemiologic studies conducted in single cities contribute some

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<sup>21</sup> Some animal studies included in the 2009 PM ISA examined exposures to mixtures, such as motor vehicle exhaust or woodsmoke. In these studies, it was unclear if the resulting cardiovascular effects could be attributed specifically to the particulate components of the mixture.

support, though associations reported in single-city studies are less consistently positive than in multicity studies, and include a number of studies reporting null associations (U.S. EPA, 2019, sections 6.1.2 and 6.1.3). When considered as a whole; however, the recent body of IHD and HF epidemiologic evidence supports the evidence from previous ISAs reporting mainly positive associations between short-term PM<sub>2.5</sub> concentrations and emergency department visits and hospital admissions.

In addition, a number of more recent controlled human exposure, animal toxicological, and epidemiologic panel studies provide evidence that PM<sub>2.5</sub> exposure could plausibly result in IHD or HF through pathways that include endothelial dysfunction, arterial thrombosis, and arrhythmia (U.S. EPA, 2019, section 6.1.1). The most consistent evidence from recent controlled human exposure studies is for endothelial dysfunction, as measured by changes in brachial artery diameter or flow mediated dilation. All but one of the available controlled human exposure studies examining the potential for endothelial dysfunction report an effect of PM<sub>2.5</sub> exposure on measures of blood flow (U.S. EPA, 2019, section 6.1.13.2). These studies report variable results regarding the timing of the effect and the mechanism by which reduced blood flow occurs (i.e., availability vs sensitivity to nitric oxide). Some controlled human exposure studies using CAPs report evidence for small increases in blood pressure (U.S. EPA, 2019, section 6.1.6.3). In addition, although not entirely consistent, there is also some evidence across controlled human exposure studies for conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (HRV) (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). Thus, when taken as a whole, controlled human exposure studies are coherent with epidemiologic studies in that they demonstrate short-term exposures to PM<sub>2.5</sub> may result in the types of cardiovascular endpoints that could lead to emergency department visits and hospital admissions in some people.

Animal toxicological studies published since the 2009 ISA also support a relationship between short-term PM<sub>2.5</sub> exposure and cardiovascular effects. A recent study demonstrating decreased cardiac contractility and left ventricular pressure in mice is coherent with the results of epidemiologic studies reporting associations between short-term PM<sub>2.5</sub> exposure and heart failure (U.S. EPA, 2019, section 6.1.3.3). In addition, and as with controlled human exposure studies, there is generally consistent evidence in animal toxicological studies for indicators of endothelial dysfunction (U.S. EPA, 2019, section 6.1.13.3). Studies in animals also provide evidence for changes in a number of other cardiovascular endpoints following short-term PM<sub>2.5</sub> exposure. Although not entirely consistent, these studies provide some evidence of conduction abnormalities and arrhythmia (U.S. EPA, 2019, section 6.1.4.4), changes in HRV (U.S. EPA,



2019, section 6.1.10.3), changes in blood pressure (U.S. EPA, 2019, section 6.1.6.4), and evidence for systemic inflammation and oxidative stress (U.S. EPA, 2019, section 6.1.11.3).

In summary, recent evidence further supports and extends the conclusions of the evidence base reported in the 2009 ISA. In support of epidemiologic studies reporting robust associations in copollutant models, direct evidence for an independent effect of PM<sub>2.5</sub> on cardiovascular effects can be found in a number of controlled human exposure and animal toxicological studies. Coherent with these results are epidemiologic panel studies reporting that PM<sub>2.5</sub> exposure is associated with some of the same cardiovascular endpoints reported in experimental studies. For these effects, there are inconsistencies in results across some animal toxicological, controlled human exposure, and epidemiologic panel studies, though this may be due to substantial differences in study design and/or study populations. Overall, the results from epidemiologic panel, controlled human exposure, and animal toxicological studies, in particular those related to endothelial dysfunction, impaired cardiac function, ST segment depression, thrombosis, conduction abnormalities, and changes in blood pressure provide coherence and biological plausibility for the consistent results from epidemiologic studies observing positive associations between short-term PM<sub>2.5</sub> concentrations and IHD and HF, and ultimately cardiovascular mortality. The ISA concludes that, overall, “there continues to be sufficient evidence to conclude that a causal relationship exists between short-term PM<sub>2.5</sub> exposure and cardiovascular effects” (U.S. EPA, 2019, p. 6-138).

### **3.2.1.3 Respiratory Effects**

#### Long-term PM<sub>2.5</sub> exposures

The 2009 PM ISA concluded that “a causal relationship is likely to exist between long-term PM<sub>2.5</sub> exposure and respiratory effects” (U.S. EPA, 2009). This conclusion was based mainly on epidemiologic evidence demonstrating associations between long-term PM<sub>2.5</sub> exposure and changes in lung function or lung function growth in children. Biological plausibility was provided by a single animal toxicological study examining pre- and post-natal exposure to PM<sub>2.5</sub> CAPs, which found impaired lung development. Epidemiologic evidence for associations between long-term PM<sub>2.5</sub> exposure and other respiratory outcomes, such as the development of asthma, allergic disease, and COPD; respiratory infection; and the severity of disease was limited, both in the number of studies available and the consistency of the results. Experimental evidence for other outcomes was also limited, with one animal toxicological study reporting that long-term exposure to PM<sub>2.5</sub> CAPs results in morphological changes in nasal airways of healthy animals. Other animal studies examined exposure to mixtures, such as motor vehicle exhaust and woodsmoke, and effects were not attributed specifically to the particulate components of the mixture.

Recent cohort studies provide additional support for the relationship between long-term PM<sub>2.5</sub> exposure and decrements in lung function growth (as a measure of lung development), indicating a robust and consistent association across study locations, exposure assessment methods, and time periods (U.S. EPA, 2019, section 5.2.13). This relationship is further supported by a recent retrospective study that reports an association between declining PM<sub>2.5</sub> concentrations and improvements in lung function growth in children (U.S. EPA, 2019, section 5.2.11). Epidemiologic studies also examine asthma development in children (U.S. EPA, 2019, section 5.2.3), with recent prospective cohort studies reporting generally positive associations, though several are imprecise (i.e., they report wide confidence intervals). Supporting evidence is provided by studies reporting associations with asthma prevalence in children, with childhood wheeze, and with exhaled nitric oxide, a marker of pulmonary inflammation (U.S. EPA, 2019, section 5.2.13). A recent animal toxicological study showing the development of an allergic phenotype and an increase in a marker of airway responsiveness provides biological plausibility for allergic asthma (U.S. EPA, 2019, section 5.2.13). Other epidemiologic studies report a PM<sub>2.5</sub>-related acceleration of lung function decline in adults, while improvement in lung function was observed with declining PM<sub>2.5</sub> concentrations (U.S. EPA, 2019, section 5.2.11). A recent longitudinal study found declining PM<sub>2.5</sub> concentrations are also associated with an improvement in chronic bronchitis symptoms in children, strengthening evidence reported in the 2009 ISA for a relationship between increased chronic bronchitis symptoms and long-term PM<sub>2.5</sub> exposure (U.S. EPA, 2019, section 5.2.11). A common uncertainty across the epidemiologic evidence is the lack of examination of copollutants to assess the potential for confounding. While there is some evidence that associations remain robust in models with gaseous pollutants, a number of these studies examining copollutant confounding were conducted in Asia, and thus have limited generalizability due to high annual pollutant concentrations.

When taken together, the ISA concludes that the “epidemiologic evidence strongly supports a relationship with decrements in lung function growth in children” and “with asthma development in children, with increased bronchitic symptoms in children with asthma, with an acceleration of lung function decline in adults, and with respiratory mortality and cause-specific respiratory mortality for COPD and respiratory infection” (U.S. EPA, 2019, p. 1-34). In support of the biological plausibility of such associations reported in epidemiologic studies of respiratory health effects, animal toxicological studies continue to provide direct evidence that long-term exposure to PM<sub>2.5</sub> results in a variety of respiratory effects. Recent animal studies show pulmonary oxidative stress, inflammation, and morphologic changes in the upper (nasal) and lower airways. Other results show that changes are consistent with the development of allergy and asthma, and with impaired lung development. Overall, the ISA concludes that “the collective

evidence is sufficient to conclude that a causal relationship is likely to exist between long-term PM<sub>2.5</sub> exposure and respiratory effects” (U.S. EPA, 2019, section 5.2.13).

#### Short-term PM<sub>2.5</sub> exposures

The 2009 PM ISA (U.S. EPA, 2009) concluded that a “causal relationship is likely to exist” between short-term PM<sub>2.5</sub> exposure and respiratory effects. This conclusion was based mainly on the epidemiologic evidence demonstrating positive associations with various respiratory effects. Specifically, the 2009 ISA described epidemiologic evidence as consistently showing PM<sub>2.5</sub>-associated increases in hospital admissions and emergency department visits for chronic obstructive pulmonary disease (COPD) and respiratory infection among adults or people of all ages, as well as increases in respiratory mortality. These results were supported by studies reporting associations with increased respiratory symptoms and decreases in lung function in children with asthma, though the epidemiologic evidence was inconsistent for hospital admissions or emergency department visits for asthma. Studies examining copollutant models showed that PM<sub>2.5</sub> associations with respiratory effects were robust to inclusion of CO or SO<sub>2</sub> in the model, but often were attenuated (though still positive) with inclusion of O<sub>3</sub> or NO<sub>2</sub>. In addition to the copollutant models, evidence supporting an independent effect of PM<sub>2.5</sub> exposure on the respiratory system was provided by animal toxicological studies of PM<sub>2.5</sub> CAPs demonstrating changes in some pulmonary function parameters, as well as inflammation, oxidative stress, injury, enhanced allergic responses, and reduced host defenses. Many of these effects have been implicated in the pathophysiology for asthma exacerbation, COPD exacerbation, or respiratory infection. In the few controlled human exposure studies conducted in individuals with asthma or COPD, PM<sub>2.5</sub> exposure mostly had no effect on respiratory symptoms, lung function, or pulmonary inflammation. Available studies in healthy people also did not clearly demonstrate respiratory effects following short-term PM<sub>2.5</sub> exposures.

Recent epidemiologic studies provide evidence for a relationship between short-term PM<sub>2.5</sub> exposure and several respiratory-related endpoints, including asthma exacerbation (U.S. EPA, 2019, section 5.1.2.1), COPD exacerbation (U.S. EPA, 2019, section 5.1.4.1), and combined respiratory-related diseases (U.S. EPA, 2019, section 5.1.6), particularly from studies examining emergency department visits and hospital admissions. The generally positive associations between short-term PM<sub>2.5</sub> exposure and asthma and COPD emergency department visits and hospital admissions are supported by epidemiologic studies demonstrating associations with other respiratory-related effects such as symptoms and medication use that are indicative of asthma and COPD exacerbations (U.S. EPA, 2019, sections 5.1.2.2 and 5.4.1.2). The collective body of epidemiologic evidence for asthma exacerbation is more consistent in children than in adults. Additionally, epidemiologic studies examining the relationship between short-term PM<sub>2.5</sub>

exposure and respiratory mortality provide evidence of consistent positive associations, demonstrating a continuum of effects (U.S. EPA, 2019, section 5.1.9).

Building off the studies evaluated in the 2009 ISA, recent epidemiologic studies expand the assessment of potential copollutant confounding. There is some evidence that PM<sub>2.5</sub> associations with asthma exacerbation, combined respiratory-related diseases, and respiratory mortality remain relatively unchanged in copollutant models with gaseous pollutants (i.e., O<sub>3</sub>, NO<sub>2</sub>, SO<sub>2</sub>, with more limited evidence for CO) and other particle sizes (i.e., PM<sub>10-2.5</sub>) (U.S. EPA, 2019, section 5.1.10.1).

The uncertainty related to whether there is an independent effect of PM<sub>2.5</sub> on respiratory health is also partially addressed by findings from animal toxicological studies. Specifically, short-term exposure to PM<sub>2.5</sub> enhanced asthma-related responses in an animal model of allergic airways disease and enhanced lung injury and inflammation in an animal model of COPD (U.S. EPA, 2019, sections 5.1.2.4.4 and 5.1.4.4.3). The experimental evidence provides biological plausibility for some respiratory-related endpoints, including limited evidence of altered host defense and greater susceptibility to bacterial infection as well as consistent evidence of respiratory irritant effects. Animal toxicological evidence for other respiratory effects is inconsistent.

The ISA concludes that “[t]he strongest evidence of an effect of short-term PM<sub>2.5</sub> exposure on respiratory effects is provided by epidemiologic studies of asthma and COPD exacerbation. While animal toxicological studies provide biological plausibility for these findings, some uncertainty remains with respect to the independence of PM<sub>2.5</sub> effects” (U.S. EPA, 2019, p. 5-155). When taken together, the ISA concludes that this evidence “is sufficient to conclude that a causal relationship is likely to exist between short-term PM<sub>2.5</sub> exposure and respiratory effects” (U.S. EPA, 2019, p. 5-155).

#### **3.2.1.4 Cancer – Long-term PM<sub>2.5</sub> Exposures**

The 2009 ISA concluded that the overall body of evidence was “suggestive of a causal relationship between relevant PM<sub>2.5</sub> exposures and cancer” (U.S. EPA, 2009). This conclusion was based primarily on positive associations observed in a limited number of epidemiologic studies of lung cancer mortality. The few epidemiologic studies that had evaluated PM<sub>2.5</sub> exposure and lung cancer incidence or cancers of other organs and systems generally did not show evidence of an association. Toxicological studies did not focus on exposures to specific PM size fractions, but rather investigated the effects of exposures to total ambient PM, or other source-based PM such as wood smoke. Collectively, results of in vitro studies were consistent with the larger body of evidence demonstrating that ambient PM and PM from specific combustion sources are mutagenic and genotoxic. However, animal inhalation studies found

little evidence of tumor formation in response to chronic exposures. A small number of studies provided preliminary evidence that PM exposure can lead to changes in methylation of DNA, which may contribute to biological events related to cancer.

Since the 2009 ISA, additional cohort studies provide evidence that long-term PM<sub>2.5</sub> exposure is positively associated with lung cancer mortality and with lung cancer incidence, and provide initial evidence for an association with reduced cancer survival (U.S. EPA, 2019, section 10.2.5). Reanalyses of the ACS cohort using different years of PM<sub>2.5</sub> data and follow-up, along with various exposure assignment approaches, provide consistent evidence of positive associations between long-term PM<sub>2.5</sub> exposure and lung cancer mortality (U.S. EPA, 2019, Figure 10-3). Additional support for positive associations with lung cancer mortality is provided by recent epidemiologic studies using individual-level data to control for smoking status, by studies of people who have never smoked (though such studies generally report wide confidence intervals due to the small number of lung cancer mortality cases within this population), and in analyses of cohorts that relied upon proxy measures to account for smoking status (U.S. EPA, 2019, section 10.2.5.1.1). Although studies that have evaluated lung cancer incidence, including studies of people who have never smoked, are limited in number, recent studies generally report positive associations with long-term PM<sub>2.5</sub> exposures (U.S. EPA, 2019, section 10.2.5.1.2). A subset of the studies focusing on lung cancer incidence also examined histological subtype, providing some evidence of positive associations for adenocarcinomas, the predominate subtype of lung cancer observed in people who have never smoked (U.S. EPA, 2019, section 10.2.5.1.2). Associations between long-term PM<sub>2.5</sub> exposure and lung cancer incidence were found to remain relatively unchanged, though in some cases confidence intervals widened, in analyses that attempted to reduce exposure measurement error by accounting for length of time at residential address or by examining different exposure assignment approaches (U.S. EPA, 2019, section 10.2.5.1.2).

The ISA evaluates the degree to which recent epidemiologic studies have addressed the potential for confounding by copollutants and the shape of the concentration-response relationship. To date, relatively few studies have evaluated the potential for copollutant confounding of the relationship between long-term PM<sub>2.5</sub> exposure and lung cancer mortality or incidence. The small number of such studies have generally focused on O<sub>3</sub> and report that PM<sub>2.5</sub> associations remain relatively unchanged in copollutant models (U.S. EPA, 2019, section 10.2.5.1.3). However, available studies have not systematically evaluated the potential for copollutant confounding by other gaseous pollutants or by other particle size fractions (U.S. EPA, 2019, section 10.2.5.1.3). Compared to total (non-accidental) mortality (see section 3.2.1.1), fewer studies have examined the shape of the concentration-response curve for cause-specific mortality outcomes, including lung cancer. Several studies have reported no

evidence of deviations from linearity in the shape of the concentration-response relationship (Lepeule et al., 2012; Raaschou-Nielsen et al., 2013; Puett et al., 2014), though authors provided only limited discussions of results (U.S. EPA, 2019, section 10.2.5.1.4).

In support of the biological plausibility of an independent effect of PM<sub>2.5</sub> on cancer, the ISA notes evidence from recent experimental and epidemiologic studies demonstrating that PM<sub>2.5</sub> exposure can lead to a range of effects indicative of mutagenicity, genotoxicity, and carcinogenicity, as well as epigenetic effects (U.S. EPA, 2019, section 10.2.7). For example, both in vitro and in vivo toxicological studies have shown that PM<sub>2.5</sub> exposure can result in DNA damage (U.S. EPA, 2019, section 10.2.2). Although such effects do not necessarily equate to carcinogenicity, the evidence that PM exposure can damage DNA, and elicit mutations, provides support for the plausibility of epidemiologic associations with lung cancer mortality and incidence. Additional supporting studies indicate the occurrence of micronuclei formation and chromosomal abnormalities (U.S. EPA, 2019, section 10.2.2.3), and differential expression of genes that may be relevant to cancer pathogenesis, following PM exposures. Experimental and epidemiologic studies that examine epigenetic effects indicate changes in DNA methylation, providing some support for PM<sub>2.5</sub> exposure contributing to genomic instability (U.S. EPA, 2019, section 10.2.3).

Epidemiologic evidence for associations between PM<sub>2.5</sub> and lung cancer mortality and incidence, together with evidence supporting the biological plausibility of such associations, contributes to the ISA's conclusion that the evidence "is sufficient to conclude that a causal relationship is likely to exist between long-term PM<sub>2.5</sub> exposure and cancer" (U.S. EPA, 2019, section 10.2.7).

### **3.2.1.5 Nervous System Effects**

#### **Long-term PM<sub>2.5</sub> exposures**

Reflecting the very limited evidence available in the last review, the 2009 ISA did not make a causality determination for long-term PM<sub>2.5</sub> exposures and nervous system effects (U.S. EPA, 2009). Since the last review, this body of evidence has grown substantially (U.S. EPA, 2019, section 8.2). Recent animal toxicology studies report that long-term PM<sub>2.5</sub> exposures can lead to morphologic changes in the hippocampus and to impaired learning and memory. This evidence is consistent with epidemiologic studies reporting that long-term PM<sub>2.5</sub> exposure is associated with reduced cognitive function (U.S. EPA, 2019, section 8.2.5). Further, while the evidence is limited, the presence of early markers of Alzheimer's disease pathology has been demonstrated in rodents following long-term exposure to PM<sub>2.5</sub> CAPs. These findings support reported associations with neurodegenerative changes in the brain (i.e., decreased brain volume), all-cause dementia, or hospitalization for Alzheimer's disease in a small number of

epidemiologic studies (U.S. EPA, 2019, section 8.2.6). Additionally, loss of dopaminergic neurons in the substantia nigra, a hallmark of Parkinson disease, has been reported in mice (U.S. EPA, 2019, section 8.2.4), though epidemiologic studies provide only limited support for associations with Parkinson's disease (U.S. EPA, 2019, section 8.2.6). Overall, the lack of consideration of copollutant confounding introduces some uncertainty in the interpretation of epidemiologic studies of nervous system effects, but this uncertainty is partly addressed by the evidence for an independent effect of PM<sub>2.5</sub> exposures provided by experimental animal studies.

In addition to the findings described above, which are most relevant to older adults, several recent studies of neurodevelopmental effects in children have also been conducted. Positive associations between long-term exposure to PM<sub>2.5</sub> during the prenatal period and autism spectrum disorder (ASD) are observed in multiple epidemiologic studies (U.S. EPA, 2019, section 8.2.7.2), while studies of cognitive function provide little support for an association (U.S. EPA, 2019, section 8.2.5.2). Interpretation of these epidemiologic studies is limited due to the small number of studies, their lack of control for potential confounding by copollutants, and uncertainty regarding the critical exposure windows. Biological plausibility is provided for the ASD findings by a study in mice that found inflammatory and morphologic changes in the corpus collosum and hippocampus, as well as ventriculomegaly (i.e., enlarged lateral ventricles) in young mice following prenatal exposure to PM<sub>2.5</sub> CAPs.

Taken together, the ISA concludes that recent studies indicate long-term PM<sub>2.5</sub> exposures can lead to effects on the brain associated with neurodegeneration (i.e., neuroinflammation and reductions in brain volume), as well as cognitive effects in older adults (U.S. EPA, 2019, Table 1-2). Animal toxicology studies provide evidence for a range of nervous system effects in adult animals, including neuroinflammation and oxidative stress, neurodegeneration, and cognitive effects, and effects on neurodevelopment in young animals. The epidemiologic evidence is more limited but studies generally support associations between long-term PM<sub>2.5</sub> exposure and changes in brain morphology, cognitive decrements and dementia. There is also initial, and limited, evidence for neurodevelopmental effects, particularly ASD. The consistency and coherence of the evidence supports the ISA's conclusion that "the collective evidence is sufficient to conclude that a causal relationship is likely to exist between long-term PM<sub>2.5</sub> exposure and nervous system effects" (U.S. EPA, 2019, section 8.2.9).

#### **3.2.1.6 Other Effects**

Compared to the health outcomes discussed above, the ISA concludes that there is greater uncertainty in the evidence linking PM<sub>2.5</sub>, or UFP, exposures with other health outcomes, reflected in conclusions that the evidence is "suggestive of, but not sufficient to infer, a causal relationship." The sections below summarize the daft ISA conclusions for these "suggestive"

outcomes for long-term (Section 3.2.1.6.1) and short-term (Section 3.2.1.6.2) PM<sub>2.5</sub> and UFP exposures.

#### **3.2.1.6.1 Long-term Exposures**

As indicated in Table 3-1 above, the ISA concludes that the evidence is “suggestive of, but not sufficient to infer, a causal relationship” between long-term PM<sub>2.5</sub> exposures and metabolic effects and reproductive and developmental effects (reproduction and fertility; pregnancy and birth outcomes). These conclusions reflect evidence that is “generally supportive but not entirely consistent or is limited overall” where “[c]hance, confounding, and other biases cannot be ruled out” (U.S. EPA, 2019, Preface, p. P-20). The basis for these causality determinations is summarized briefly below.

##### *PM<sub>2.5</sub> – Metabolic effects*

There were no causality determinations for long-term PM<sub>2.5</sub> exposure and metabolic effects in the 2009 ISA (U.S. EPA, 2009). However, the literature pertaining to the effect of long-term exposure to PM<sub>2.5</sub> and metabolic effects has expanded substantially since the 2009 ISA, and consists of both epidemiologic and experimental evidence (U.S. EPA, 2019, section 7.2). Epidemiologic studies report positive associations between long-term PM<sub>2.5</sub> exposure and diabetes-related mortality. In addition, although results were not consistent across cohorts, there is some evidence from epidemiologic studies for positive associations with incident diabetes, metabolic syndrome, and alterations in glucose and insulin homeostasis. Consideration of copollutant confounding was limited. In animal toxicologic studies, there is some support for a relationship between long-term PM<sub>2.5</sub> exposure and metabolic effects from experimental studies demonstrating increased blood glucose, insulin resistance, and inflammation and visceral adiposity but the experimental evidence was not entirely consistent. Based on this evidence, the ISA concludes that, “[o]verall, the collective evidence is suggestive of, but is not sufficient to infer, a causal relationship between long-term PM<sub>2.5</sub> exposure and metabolic effects” (U.S. EPA, 2019, p. 7-52).

##### *PM<sub>2.5</sub> – Reproductive and developmental effects*

The 2009 ISA determined that the evidence was “suggestive of a causal relationship” for the association between long-term PM<sub>2.5</sub> exposure and reproductive and developmental outcomes. The body of literature characterizing these relationships has grown since the 2009 ISA, with much of the evidence focusing on reproduction and fertility or pregnancy and birth outcomes, though important uncertainties persist (U.S. EPA, 2019, sections 9.1.1, 9.1.2, 9.1.5).

Effects of PM<sub>2.5</sub> exposure on sperm have been studied in both epidemiology and toxicology studies and shows the strongest evidence in epidemiologic studies for impaired sperm motility and in animal toxicological studies for impaired spermiation. Epidemiologic evidence on



sperm morphology have reported inconsistent results. Evidence for effects of PM<sub>2.5</sub> exposure on female reproduction also comes from both epidemiology and toxicology studies. In the epidemiologic literature, results on human fertility and fecundity is limited, but the evidence on in vitro fertilization indicates a modest association of PM<sub>2.5</sub> exposures with decreased odds of becoming pregnant. Studies in rodents have shown ovulation and estrus are affected by PM<sub>2.5</sub> exposure. Biological plausibility for outcomes related to male and female fertility and reproduction comes from laboratory animal studies demonstrating genetic and epigenetic changes in germ cells with PM<sub>2.5</sub> exposure. The ISA concludes that, “[c]ollectively, the evidence is suggestive of, but not sufficient to infer, a causal relationship between PM<sub>2.5</sub> exposure and male and female reproduction and fertility” (U.S. EPA, 2019, p. 9-43).

With regard to pregnancy and birth outcomes, while the collective evidence for many of the outcomes examined is not consistent, there are some animal toxicology and epidemiologic studies that indicate an association between PM<sub>2.5</sub> exposures and reduced fetal growth, low birth weight and preterm birth. Most of the epidemiologic studies do not control for co-pollutant confounding and do not identify a specific sensitive window of exposure, but results from animal toxicologic studies provide biological plausibility for these outcomes, as well as support for multiple sensitive windows for PM<sub>2.5</sub> exposure-associated outcomes. There is also epidemiologic evidence for congenital heart defects of different types, as well as biological plausibility to support this outcome from the animal toxicology literature. However, evidence for a relationship between PM<sub>2.5</sub> exposure and various pregnancy-related pathologies, including gestational hypertension, pre-eclampsia and gestational diabetes is inconsistent. Biological plausibility for effects of PM<sub>2.5</sub> exposure and various pregnancy and birth outcomes is provided by studies showing that PM<sub>2.5</sub> exposure in laboratory rodents resulted in impaired implantation and vascular endothelial dysfunction. Coherence with toxicological studies is provided by epidemiologic studies in humans reporting associations with epigenetic changes to the placenta and impaired fetal thyroid function. When taken together, the ISA concludes that the available evidence, including uncertainties that evidence, is “suggestive of, but not sufficient to infer, a causal relationship between exposure to PM<sub>2.5</sub> and pregnancy and birth outcomes” (U.S. EPA, 2019, p. 9-44).

#### *UFP – Nervous System Effects*

The 2009 ISA reported limited animal toxicological evidence of a relationship between long-term exposure to UFP and nervous system effects, with no supporting epidemiologic studies. Recent animal toxicological studies substantially add to this evidence base. Multiple toxicological studies of long-term UFP exposure conducted in adult mice provide consistent evidence of brain inflammation and oxidative stress in the whole brain, hippocampus, and cerebral cortex (U.S. EPA, 2019, section 8.6.3). Studies also found morphologic changes,

specifically neurodegeneration in specific regions of the hippocampus and pathologic changes characteristic of Alzheimer's disease, and initial evidence of behavioral effects in adult mice (U.S. EPA, 2019, sections 8.6.4 and 8.6.5). Toxicological studies examining pre- and post-natal UFP exposures provide extensive evidence for behavioral effects, altered neurotransmitters, neuroinflammation, and morphologic changes (U.S. EPA, 2019, section 8.6.6.2). Persistent ventriculomegaly was observed in male, but not female, mice exposed postnatally to UFP (U.S. EPA, 2019, section 8.6.6). Epidemiologic evidence is limited to a single study of school children that provides support for the experimental results. This study, which did not consider copollutant confounding, reports an association between long-term exposure to UFP, which was measured at the school, and decrements on tests of attention and memory. Uncertainty results from the lack of information on the spatial and temporal variability of UFP exposures on long-term UFP exposures at the population level. Based primarily on the animal toxicological evidence of neurotoxicity and altered neurodevelopment, the ISA concludes that the evidence is “suggestive of, but not sufficient to infer, a causal relationship” between long-term UFP exposure and nervous system effects (U.S. EPA, 2019, section 8.6.7).

#### **3.2.1.6.2 Short-term Exposures**

As indicated in Table 3-1 above, the ISA concludes that the evidence is “suggestive of, but not sufficient to infer, a causal relationship” between short-term PM<sub>2.5</sub> exposures and metabolic effects and nervous system effects. Additionally, the ISA concludes that the evidence is “suggestive” for short-term UFP exposures and cardiovascular effects, respiratory effects, and nervous system effects. As for the outcomes related to long-term exposures, discussed above, these conclusions reflect evidence that is “generally supportive but not entirely consistent or is limited overall” where “[c]hance, confounding, and other biases cannot be ruled out” (U.S. EPA, 2019, Preface, p.P-20). The basis for these causality determinations is summarized briefly below.

##### *PM<sub>2.5</sub> – Metabolic effects*

There were no studies of the effect of short-term PM<sub>2.5</sub> exposure and metabolic effects reviewed in the 2009 ISA (U.S. EPA, 2009). New evidence for a relationship between short-term PM<sub>2.5</sub> exposure and metabolic effects is based on a small number of epidemiologic and animal toxicological studies reporting effects on glucose and insulin homeostasis and other indicators of metabolic function such as inflammation in the visceral adipose tissue and liver (U.S. EPA, 2019, section 7.1). The ISA concludes that, overall, the collective evidence “is suggestive of, but not sufficient to infer, a causal relationship between short-term PM<sub>2.5</sub> exposure and metabolic effects” (U.S. EPA, 2019, p. 7-11).

##### *PM<sub>2.5</sub> – Nervous system effects*

The evidence reviewed in the 2009 ISA was characterized as "inadequate to infer" a causal relationship between short-term PM<sub>2.5</sub> exposure and nervous system effects (U.S. EPA, 2009), based on a small number of experimental animal studies. Recent studies strengthen the evidence that short-term exposure to PM<sub>2.5</sub> can affect the nervous system (U.S. EPA, 2019, section 8.1). The strongest evidence is provided by experimental studies in mice that show effects on the brain. These toxicological studies demonstrate changes in neurotransmitters in the hypothalamus that are linked to sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) stress axis activation, as well as upregulation of inflammation-related genes, changes in cytokine levels, and other changes that are indicative of brain inflammation. In addition, an association of short-term PM<sub>2.5</sub> exposure with hospital admissions for Parkinson's disease was observed indicating the potential for exacerbation of neurological diseases. The ISA concludes that, overall, the collective evidence "is suggestive of, but not sufficient to infer, a causal relationship between short-term exposure to PM<sub>2.5</sub> and nervous system effects" (U.S. EPA, 2019, p. 8-15).

#### *UFP – Cardiovascular effects*

In the 2009 ISA, the evidence from toxicological studies, many of which examined exposures to whole diesel exhaust or wood smoke rather than UFP alone, was suggestive of a causal relationship between short-term UFP exposure and cardiovascular effects. Since the 2009 ISA, there have been only a limited number of studies published describing the relationship between short-term UFP exposure and cardiovascular effects. This includes a small number of epidemiologic panel studies that have observed positive associations between short-term exposure to UFPs and measures of HRV (U.S. EPA, 2019, section 6.5.9.1) and markers of coagulation (U.S. EPA, 2019, section 6.5.11.1) although there are also studies that did not report such UFP-related effects. In addition, there is evidence from a single controlled human exposure study indicating decreases in the anticoagulant proteins plasminogen and thrombomodulin in individuals with metabolic syndrome (U.S. EPA, 2019, section 6.5.11.2). There is inconsistent evidence from controlled human exposure and epidemiologic panel studies for endothelial dysfunction, changes in blood pressure, and systemic inflammation following short-term exposure to UFPs. Notably, there is little evidence of an effect when considering short-term UFP exposure on other cardiovascular endpoints as well as cardiovascular-disease emergency department visits or hospital admissions. The assessment of study results across experimental and epidemiologic studies is complicated by differences in the size distributions examined between disciplines and by the nonuniformity in the exposure metrics examined (e.g., particle number concentration, surface area concentration, and mass concentration) (U.S. EPA, 2019, section 1.4.3). When considered as a whole, the ISA concludes that the evidence is "suggestive

of, but not sufficient to infer, a causal relationship between short-term exposure UFP exposure and cardiovascular effects” (U.S. EPA, 2019, p. 6-304).

#### *UFP – Respiratory effects*

A limited number of studies examining short-term exposure to UFPs and respiratory effects were reported in the 2009 ISA, which concluded that the relationship between short-term exposure to UFP and respiratory effects is “suggestive of a causal relationship.” This conclusion was based on epidemiologic evidence indicating associations with combined respiratory-related diseases, respiratory infection, and asthma exacerbation. In addition, personal exposures to ambient UFP were associated with lung function decrements in adults with asthma. The few available experimental studies provided limited coherence with epidemiologic findings for asthma exacerbation. Recent studies add to this evidence base and support epidemiologic evidence for asthma exacerbation and combined respiratory-related diseases but do not rule out chance, confounding, and other biases (U.S. EPA, 2019, section 5.5). For example, associations persist in one epidemiologic study with adjustment for NO<sub>2</sub>, but not in another. Additional supporting evidence, showing decrements in lung function and enhancement of allergic inflammation and other allergic responses, is provided by a controlled human exposure study in adults with asthma and by animal toxicological studies in an animal model of allergic airway disease. For combined respiratory-related diseases, recent findings add consistency for hospital admissions and emergency department visits and indicate lung function changes among adults with asthma or COPD. Uncertainty remains regarding the characterization of UFP exposures and the potential for copollutant confounding in epidemiologic studies, which limits inference about an independent effect of UFP exposures (U.S. EPA, 2019, section 5.5). The ISA concludes that, overall, the evidence is “suggestive of, but not sufficient to infer, a causal relationship between short-term UFP exposure and respiratory effects” (U.S. EPA, 2019, p. 5-303).

#### *UFP- Nervous system effects*

The 2009 ISA reported limited animal toxicological evidence of a relationship between short-term exposure to UFP and nervous system effects, without supporting epidemiologic studies. Several recent experimental studies add to this evidence base. In the current review, the strongest evidence for a relationship between short-term UFP exposure and nervous system effects is provided by animal toxicological studies that show inflammation and oxidative stress in multiple brain regions following exposure to UFP. There is a lack of evidence from epidemiologic studies (U.S. EPA, 2019, section 8.5). The ISA concludes that, overall, the collective evidence is “suggestive of, but not sufficient to infer, a causal relationship between short-term UFP exposure and nervous system effects” (U.S. EPA, 2019, p. 8-86).

### 3.2.1.7 Summary

Based on the evidence assessed in the ISA (U.S. EPA, 2019), and summarized in sections 3.2.1.1 to 3.2.1.6 above, we revisit the policy-relevant questions posed at the beginning of this section:

- **To what extent does the currently available scientific evidence strengthen, or otherwise alter, our conclusions from the last review regarding health effects attributable to long- or short-term fine particle exposures? Have previously identified uncertainties been reduced? What important uncertainties remain and have new uncertainties been identified?**

We consider these questions in the context of the evidence for effects of long- and short-term PM<sub>2.5</sub> exposures.

Studies conducted since the 2009 ISA have broadened our understanding of the health effects that can result from long-term PM<sub>2.5</sub> exposures and have reduced key uncertainties identified in the last review. Recent epidemiologic studies consistently report positive associations between long-term PM<sub>2.5</sub> exposures and a wide range of health outcomes, including total and cause-specific mortality, cardiovascular and respiratory morbidity, lung cancer, and nervous system effects. Such associations have been reported in analyses examining a variety of study designs, approaches to estimating PM<sub>2.5</sub> exposures, statistical models, and long-term exposure windows (i.e., the exposure period that is associated with the health outcome). Recent evidence also includes retrospective studies that demonstrate improvements in health outcomes, including increasing life expectancy, decreasing mortality, or decreasing respiratory effects, as a result of past declines in ambient PM<sub>2.5</sub> concentrations. Recent epidemiologic studies report that associations with mortality (total, cardiovascular, and respiratory) remain relatively unchanged in copollutant models, supporting the independence of these associations from co-occurring gases or coarse PM. Recent studies additionally report that associations (i.e., primarily with mortality) persist in analyses restricted to long-term PM<sub>2.5</sub> exposures in the lower portions of the air quality distribution, and such studies do not identify a threshold below which associations no longer occur. The biological plausibility of health effect associations reported in epidemiologic studies is supported by coherent results from experimental studies. Recent evidence from animal toxicology and/or controlled human exposure studies provides stronger support, compared to previous reviews, for potential biologic pathways by which long-term PM<sub>2.5</sub> exposures could lead to effects on the cardiovascular and respiratory systems, effects on the nervous system, and

to lung cancer.<sup>22 23</sup> In addition to providing insight into potential mechanisms, experimental studies also demonstrate direct effects of PM<sub>2.5</sub> exposures, providing further support for independent effects of particle exposures on health (i.e., not confounded by co-occurring pollutants). When taken together, the evidence available in this review (i.e., U.S. EPA, 2019) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of long-term PM<sub>2.5</sub> exposures.

As with the evidence for effects of long-term exposures, since the 2009 ISA, much progress has been made in assessing key uncertainties in our understanding of health effects associated with short-term PM<sub>2.5</sub> exposures. Recent epidemiologic studies build upon and further reaffirm those studies evaluated in the 2009 PM ISA, providing evidence of positive associations across a range of effects. The independence of the PM<sub>2.5</sub> effects reported in such studies is further supported by the results of copollutant analyses indicating that associations with short-term PM<sub>2.5</sub> remain robust. Some recent studies report that associations persist in analyses that exclude short-term PM<sub>2.5</sub> exposures near the upper end of the air quality distribution and that a threshold below which associations no longer occur is not identifiable from the available data. The plausibility of PM<sub>2.5</sub>-associated mortality is supported by associations with cardiovascular and respiratory morbidity. Direct evidence for PM<sub>2.5</sub> exposure-related cardiovascular effects can also be found in recent controlled human exposure and animal toxicological studies, supported by results of epidemiologic panel studies, reporting that PM<sub>2.5</sub> exposure can result in various cardiovascular effects, including endothelial dysfunction, impaired cardiac function, ST segment depression, thrombosis, conduction abnormalities, and increased blood pressure. Overall, the results from these studies provide coherence and biological plausibility for the consistent results from epidemiologic studies observing positive associations between short-term PM<sub>2.5</sub> concentrations and ischemic heart disease and heart failure, and ultimately cardiovascular mortality. While there are inconsistencies in results across some of the animal toxicological, controlled human exposure, and epidemiologic panel studies, this may be due to substantial differences in study design, study populations, or differences in PM composition across study locations. While recent epidemiologic studies also demonstrate associations between short-term PM<sub>2.5</sub> exposures and respiratory effects, particularly asthma and COPD exacerbations, and while animal toxicological studies provide biological plausibility for these findings, some uncertainty

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<sup>22</sup> For respiratory effects, nervous system effects, and cancer-related effects animal studies provide support for potential biologic pathways while controlled human exposure studies are more limited.

<sup>23</sup> Animal studies also provide stronger support in this review for effects following exposures to UFP (section 3.2.1), though important uncertainties remain (e.g., inconsistent UFP definitions across studies, various methods of administering UFP exposures in health studies, limited understanding of ambient UFP concentrations and distributions in epidemiologic studies), limiting the potential for these studies to inform policy-relevant conclusions.

remains with respect to the independence of PM<sub>2.5</sub> effects. Thus, when taken together, the evidence available in this review (U.S. EPA, 2019) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of short-term PM<sub>2.5</sub> exposures.

### **3.2.2 Potential At-Risk Populations**

The NAAQS are meant to protect the population as a whole, including groups that may be at increased risk for pollutant-related health effects. In the last review, based on the evidence assessed in the 2009 ISA (U.S. EPA, 2009), the 2011 PA focused on children, older adults, people with pre-existing heart and lung diseases, and those of lower socioeconomic status as populations that are “likely to be at increased risk of PM-related effects” (U.S. EPA, 2011, p. 2-31). In the current review, the ISA cites extensive evidence indicating that “both the general population as well as specific populations and lifestages are at risk for PM<sub>2.5</sub>-related health effects” (U.S. EPA, 2019, p. 12-1). For example, in support of its “causal” and “likely to be causal” determinations, the ISA cites substantial evidence for:

- PM-related mortality and cardiovascular effects in older adults (U.S. EPA, 2019, sections 11.1, 11.2, 6.1, and 6.2);
- PM-related cardiovascular effects in people with pre-existing cardiovascular disease (U.S. EPA, 2019, section 6.1);
- PM-related respiratory effects in people with pre-existing respiratory disease, particularly asthma (U.S. EPA, 2019, section 5.1); and
- PM-related impairments in lung function growth and asthma development in children (U.S. EPA, 2019, sections 5.1 and 5.2; 12.5.1.1).

The ISA additionally notes that stratified analyses (i.e., analyses that directly compare PM-related health effects across groups) provide strong evidence for racial and ethnic differences in PM<sub>2.5</sub> exposures and in PM<sub>2.5</sub>-related health risk. Such analyses indicate that minority populations such as Hispanic and non-Hispanic black populations have higher PM<sub>2.5</sub> exposures than non-Hispanic white populations, thus contributing to adverse health risk in non-white populations (U.S. EPA, 2019, section 12.5.4). Stratified analyses focusing on other groups also suggest that populations with pre-existing cardiovascular or respiratory disease, populations that are overweight or obese, populations that have particular genetic variants, and populations that are of low socioeconomic status could be at increased risk for PM<sub>2.5</sub>-related adverse health effects (U.S. EPA, 2019, Chapter 12).

Thus, the groups at risk of PM<sub>2.5</sub>-related health effects represent a substantial portion of the total U.S. population. In evaluating the primary PM<sub>2.5</sub> standards, an important consideration is the potential PM<sub>2.5</sub>-related public health impacts in these populations.

### 3.2.3 PM<sub>2.5</sub> Concentrations in Key Studies Reporting Health Effects

To inform conclusions on the adequacy of the public health protection provided by the current primary PM<sub>2.5</sub> standards, this section evaluates the PM<sub>2.5</sub> exposures and ambient concentrations (i.e., used as surrogates for exposures in epidemiologic studies) in studies reporting PM<sub>2.5</sub>-related health effects. We specifically consider the following overarching questions:

- **What are the short- or long-term PM<sub>2.5</sub> exposures that have been associated with health effects and to what extent does the evidence support the occurrence of such effects for air quality meeting the current primary PM<sub>2.5</sub> standards?**

In addressing these questions, we emphasize health outcomes for which the ISA has concluded the evidence supports a “causal” or a “likely to be causal” relationship with PM exposures. As discussed above, this includes mortality, cardiovascular effects, and respiratory effects associated with short- or long-term PM<sub>2.5</sub> exposures and cancer and nervous system effects associated with long-term PM<sub>2.5</sub> exposures. While the causality determinations in the ISA are informed by studies evaluating a wide range of PM<sub>2.5</sub> concentrations, this section considers the degree to which the evidence supports the occurrence of PM-related effects at concentrations relevant to informing conclusions on the primary PM<sub>2.5</sub> standards. Section 3.2.3.1 considers the exposure concentrations that have been evaluated in experimental studies and section 3.2.3.2 considers the ambient concentrations in locations evaluated by epidemiologic studies.

#### 3.2.3.1 PM Exposure Concentrations Evaluated In Experimental Studies

In the ISA, the evidence for a particular PM<sub>2.5</sub>-related health outcome is strengthened when results from experimental studies demonstrate biologically plausible mechanisms through which adverse human health outcomes could occur (U.S. EPA, 2015, Preamble p. 20). Two types of experimental studies are of particular importance in understanding the effects of PM exposures: controlled human exposure and animal toxicology studies. In such studies, investigators expose human volunteers or laboratory animals, respectively, to known concentrations of air pollutants under carefully regulated environmental conditions and activity levels. Thus, controlled human exposure and animal toxicology studies can provide information on the health effects of experimentally administered pollutant exposures under highly controlled laboratory conditions (U.S. EPA, 2015, Preamble, p. 11).

In this section, we consider the PM<sub>2.5</sub> exposure concentrations shown to cause effects in controlled human exposure studies and in animal toxicology studies. We particularly consider the consistency of specific PM<sub>2.5</sub>-related effects across studies, the potential adversity of such effects, and the degree to which exposures shown to cause effects are likely to occur in areas meeting the current primary standards. To address these issues, we consider the following question:



- **To what extent does the evidence from controlled human exposure or animal toxicology studies support the potential for adverse cardiovascular, respiratory, or other effects following PM<sub>2.5</sub> exposures likely to occur in areas meeting the current primary standards?**

#### Controlled Human Exposure Studies

As discussed in detail in the ISA (U.S. EPA, 2019, section 6.1), controlled human exposure studies have reported that PM<sub>2.5</sub> exposures lasting from less than one hour up to five hours can impact cardiovascular function.<sup>24</sup> The most consistent evidence from these studies is for impaired vascular function (U.S. EPA, 2019, section 6.1.13.2). In addition, although less consistent, the ISA notes that studies examining PM<sub>2.5</sub> exposures also provide evidence for increased blood pressure (U.S. EPA, 2019, section 6.1.6.3), conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). The ISA concludes that, when taken as a whole, controlled human exposure studies demonstrate that short-term exposure to PM<sub>2.5</sub> may impact cardiovascular function in ways that could lead to more serious outcomes (U.S. EPA, 2019, section 6.1.16). Thus, such studies can provide insight into the potential for specific PM<sub>2.5</sub> exposures to cause physiological changes that could increase the risk of more serious effects.

Table 3-2 below summarizes information from the ISA<sup>25</sup> on available controlled human exposure studies that evaluate effects on markers of cardiovascular function following exposures to PM<sub>2.5</sub>, either as concentrated ambient particles (CAP) or in unfiltered versus filtered exhaust.<sup>26</sup>

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<sup>24</sup> In contrast, controlled human exposure studies provide little evidence for respiratory effects following short-term PM<sub>2.5</sub> exposures (U.S. EPA, 2019, section 5.1, Table 5-18). Therefore, this section focuses on cardiovascular effects evaluated in controlled human exposure studies of PM<sub>2.5</sub> exposure.

<sup>25</sup> Table 3-2 includes the controlled human exposure studies, and the endpoints from each study, that are discussed in the ISA.

<sup>26</sup> Table 3-2 identifies controlled human exposure studies included in the ISA that examine the potential for PM<sub>2.5</sub> exposures to alter markers of cardiovascular function. Studies that focus on specific components of PM<sub>2.5</sub> (e.g., endotoxin), or studies that evaluated PM<sub>2.5</sub> exposures only in the presence of an intervention (e.g., dietary intervention) or other pollutant (e.g., ozone), are not included.

**Table 3-2. Summary of information from PM<sub>2.5</sub> controlled human exposure studies.**

<b>Study</b>	<b>Population</b>	<b>Exposure Details (average concentration; duration)</b>	<b>Results</b>
Bräuner et al., 2008	Healthy adults	10.5 µg/m <sup>3</sup> PM <sub>2.5</sub> (unfiltered) vs below detection (filtered); 24 h	No significant effect on markers of vascular function
Hemmingsen et al., 2015a, Hemmingsen et al., 2015b	Healthy, overweight older adults	24 µg/m <sup>3</sup> (unfiltered) vs 3.0 µg/m <sup>3</sup> (filtered) Copenhagen PM; 5 h	Impaired vascular function and altered heart rate variability; no significant changes in blood pressure or markers of inflammation or oxidative stress
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	64 µg/m <sup>3</sup> CAP (lower exposure); 2 h	No significant change in blood markers of inflammation or oxidative stress
Huang et al., 2012	Healthy adults	90 µg/m <sup>3</sup> CAP; 2 h	No significant changes in heart rate variability
Devlin et al., 2003	Healthy older adults	99 µg/m <sup>3</sup> CAP <sup>27</sup> ; 2 h	Decreased heart rate variability
Hazucha et al., 2013	Adult current and former smokers	109 µg/m <sup>3</sup> CAP; 2 h	No significant changes in markers of inflammation or coagulation
Ghio et al., 2000	Healthy young adults	120 µg/m <sup>3</sup> CAP; 2 h	Increased fibrinogen (coagulation)
Ghio et al., 2003	Healthy young adults	120 µg/m <sup>3</sup> CAP; 2 h	Increased fibrinogen; no significant effect on markers of inflammation
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	140 µg/m <sup>3</sup> CAP (higher exposure); 2 h	Increased blood inflammatory markers
Brook et al., 2009	Healthy adults	149 µg/m <sup>3</sup> CAP; 2 h	Impaired vascular function, increased blood pressure; no significant change in markers of inflammation (compared to filtered air)
Ramanathan et al., 2016	Healthy adults	149 µg/m <sup>3</sup> CAP; 2 h	Decreased anti-oxidant/anti-inflammatory capacity when baseline capacity was low

<sup>27</sup> The published study reports an average CAP concentration of 41 µg/m<sup>3</sup>, but communication with the study authors revealed an error in that reported concentration (Jenkins, 2016).

Sivagangabalan et al., 2011	Healthy adults	150 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Increase in indicator of possible arrhythmia; no significant effect on heart rate
Kusha et al., 2012	Healthy adults	154 $\mu\text{g}/\text{m}^3$ CAP; 2 h	No significant effect on indicator of possible arrhythmia
Gong et al., 2003	Adults with and without asthma	174 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Increased heart rate; No significant effect on indicators of arrhythmia, inflammation, coagulation; inconsistent effects on blood pressure
Gong et al., 2004	Older adults with and without COPD	200 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Decreased heart rate variability, increase in markers of inflammation (without COPD only); inconsistent effect on arrhythmia; no significant effect on markers of blood coagulation
Liu et al., 2015	Healthy adults	238 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increase in urinary markers of oxidative stress and vascular dysfunction; no significant effect on blood markers of oxidative stress, vascular function, or inflammation
Bellavia et al., 2013	Healthy adults	~242 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increased blood pressure
Behbod et al., 2013	Healthy adults	~250 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increase in markers of inflammation
Tong et al., 2015	Healthy older adults	253 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Impaired vascular function and increased blood pressure; no significant change in markers of inflammation or coagulation
Lucking et al., 2011	Healthy young men	320 $\mu\text{g}/\text{m}^3$ (unfiltered) vs 7.2 $\mu\text{g}/\text{m}^3$ (filtered); 1 h	Impaired vascular function and increased potential for coagulation; no significant effect on blood pressure, markers of inflammation, or arterial stiffness
Vieira et al., 2016a, Vieira et al., 2016b	Healthy adults; Heart failure patients	325 $\mu\text{g}/\text{m}^3$ (unfiltered) vs 25 $\mu\text{g}/\text{m}^3$ (filtered) diesel exhaust; 21-min	Increase in marker of potential impairment in heart function, impaired vascular function (heart failure patients); no significant effect on blood pressure, heart rate or heart rate variability, markers of inflammation, markers of coagulation, or arterial stiffness

Most of the controlled human exposure studies in Table 3-2 have evaluated average  $\text{PM}_{2.5}$  exposure concentrations at or above about  $100 \mu\text{g}/\text{m}^3$ , with exposure durations typically up to about two hours. Statistically significant effects on one or more indicators of cardiovascular function are often, though not always, reported following 2-hour exposures to average  $\text{PM}_{2.5}$  concentrations at and above about  $120 \mu\text{g}/\text{m}^3$ , with less consistent evidence for effects following exposures to lower concentrations. Impaired vascular function, the effect identified in the ISA as

the most consistent across studies (U.S. EPA, 2019, section 6.1.13.2), is shown following 2-hour exposures to PM<sub>2.5</sub> concentrations at and above 149 µg/m<sup>3</sup>. Mixed results are reported in the three studies that evaluate longer exposure durations (i.e., longer than 2 hours) and lower PM<sub>2.5</sub> concentrations, with significant effects on some outcomes reported following 5-hour exposures to 24 µg/m<sup>3</sup> in Hemmingsen et al. (2015b), but not for other outcomes following 5-hour exposures in Hemmingsen et al. (2015a) and not following 24-hour exposures to 10.5 µg/m<sup>3</sup> in Bräuner et al. (2008).

To provide some insight into what these studies may indicate regarding the primary PM<sub>2.5</sub> standards, we consider the degree to which 2-hour ambient PM<sub>2.5</sub> concentrations in locations meeting the current primary standards are likely to exceed the 2-hour exposure concentrations at which statistically significant effects are reported in multiple studies for one or more indicators of cardiovascular function. To this end, we refer to Figure 2-14 (Chapter 2, section 2.3.2.2.3), which presents the frequency distribution of 2-hour average PM<sub>2.5</sub> concentrations from all FEM PM<sub>2.5</sub> monitors in the U.S. for 2015-2017. At sites meeting the current primary PM<sub>2.5</sub> standards, most 2-hour concentrations are below 11 µg/m<sup>3</sup>, and almost never exceed 32 µg/m<sup>3</sup>. The extreme upper end of the distribution of 2-hour PM<sub>2.5</sub> concentrations is shifted higher during the warmer months (April to September, denoted by red bars in Figure 2-14), generally corresponding to the period of peak wildfire frequency in the U.S. At sites meeting the current primary standards, the highest 2-hour concentrations measured almost never occur outside of the period of peak wildfire frequency (i.e., 99.9<sup>th</sup> percentile of 2-hour concentrations is 68 µg/m<sup>3</sup> during the warm season). Most of the sites measuring these very high concentrations are in the northwestern U.S. and California (see Appendix A, Figure A-1), where wildfires have been relatively common in recent years. When the typical fire season is excluded from the analysis (blue in Figure 2-14), the extreme upper end of the distribution is reduced (i.e., 99.9<sup>th</sup> percentile of 2-hour concentrations is 59 µg/m<sup>3</sup>).<sup>28</sup>

Thus, while controlled human exposure studies support the plausibility of the serious cardiovascular effects that have been linked with ambient PM<sub>2.5</sub> exposures (U.S. EPA, 2019, Chapter 6), the PM<sub>2.5</sub> exposure concentrations evaluated in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards. Therefore, controlled human exposure studies are of limited utility in informing conclusions on the adequacy of the public health protection provided by the current standards. Additional controlled human exposure studies that examine longer exposure periods (e.g., 24-hour as in Bräuner et al. (2008); 5-hour as in Hemmingsen et al. (2015b)), or repeated exposures,

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<sup>28</sup> Similar analyses of 5-hour PM<sub>2.5</sub> concentrations are presented in Appendix A, Figure A-2.

to concentrations typical in the ambient air across much of the U.S. may provide additional insight into this issue in future reviews.

### Animal Toxicology Studies

The ISA relies on animal toxicology studies to support the plausibility of a wide range of PM<sub>2.5</sub>-related health effects. While animal toxicology studies often examine more severe health outcomes and longer exposure durations than controlled human exposure studies, there is uncertainty in extrapolating the effects seen in animals, and the PM<sub>2.5</sub> exposures and doses that cause those effects, to human populations. We consider these uncertainties when evaluating what the available animal toxicology studies may indicate with regard to the current primary PM<sub>2.5</sub> standards.

Most of the animal toxicology studies assessed in the ISA have examined effects following exposures to PM<sub>2.5</sub> concentrations well-above the concentrations likely to be allowed by the current PM<sub>2.5</sub> standards. Such studies have generally examined short-term exposures to PM<sub>2.5</sub> concentrations from 100 to >1,000 µg/m<sup>3</sup> and long-term exposures to concentrations from 66 to >400 µg/m<sup>3</sup> (e.g., see U.S. EPA, 2019, Table 1-2). Two exceptions are a study reporting impaired lung development following long-term exposures (i.e., 24 hours per day for several months prenatally and postnatally) to an average PM<sub>2.5</sub> concentration of 16.8 µg/m<sup>3</sup> (Mauad et al., 2008) and a study reporting increased carcinogenic potential following long-term exposures (i.e., 2 months) to an average PM<sub>2.5</sub> concentration of 17.7 µg/m<sup>3</sup> (Cangerana Pereira et al., 2011). These two studies demonstrate serious effects following long-term exposures to PM<sub>2.5</sub> concentrations similar to the ambient concentrations reported in some PM<sub>2.5</sub> epidemiologic studies (U.S. EPA, 2019, Table 1-2), though still above the ambient concentrations likely to occur in areas meeting the current primary standards. Thus, as is the case with controlled human exposure studies, animal toxicology studies support the plausibility of various adverse effects that have been linked to ambient PM<sub>2.5</sub> exposures (U.S. EPA, 2019), but have not evaluated PM<sub>2.5</sub> exposures likely to occur in areas meeting the current primary standards. Given this, and the additional uncertainty of extrapolating from effects in animals to those in human populations, animal toxicology studies are of limited utility in informing conclusions on the public health protection provided by the current or alternative primary PM<sub>2.5</sub> standards.

#### **3.2.3.2 Ambient PM Concentrations in Locations of Epidemiologic Studies**

As summarized in section 3.2.1 above, epidemiologic studies examining associations between daily or annual average PM<sub>2.5</sub> exposures and mortality or morbidity represent a large part of the evidence base supporting several of the ISA's "causal" and "likely to be causal" determinations. In this section, we consider the ambient PM<sub>2.5</sub> concentrations present in areas where epidemiologic studies have evaluated associations with mortality or morbidity, and what

such concentrations may indicate regarding the primary PM<sub>2.5</sub> standards. The approaches discussed in this section are also summarized above in section 3.1.2.

As noted in section 3.1.2, the use of information from epidemiologic studies to inform conclusions on the primary PM<sub>2.5</sub> standards is complicated by the fact that such studies evaluate associations between distributions of ambient PM<sub>2.5</sub> and health outcomes, and do not identify the specific exposures that cause reported effects. Rather, health effects can occur over the entire distributions of ambient PM<sub>2.5</sub> concentrations evaluated, and epidemiologic studies do not identify a population-level threshold below which it can be concluded with confidence that PM-associated health effects do not occur (U.S. EPA, 2019, section 1.5.3).

In the absence of discernible thresholds, we use two approaches to consider information from epidemiologic studies. In one approach, we evaluate the PM<sub>2.5</sub> air quality distributions reported by key epidemiologic studies (i.e., and used to estimate exposures in these studies) and the degree to which such distributions are likely to occur in areas meeting the current (or alternative) standards (section 3.2.3.2.1). We recognize uncertainty in using this approach to inform conclusions on the primary standards because study-reported PM<sub>2.5</sub> concentrations are not the same as the design values used by the EPA to determine whether areas meet the NAAQS (discussed further below). Therefore, in an additional approach, we calculate study area air quality metrics similar to PM<sub>2.5</sub> design values and consider the degree to which such metrics indicate that study area air quality would likely have met or violated the current or alternative standards during study periods (section 3.2.3.2.2).

To the extent these approaches indicate that health effect associations are based on PM<sub>2.5</sub> air quality likely to have met the current or alternative standards, such standards are likely to allow the daily or annual average PM<sub>2.5</sub> exposures that provide the foundation for reported associations. Alternatively, to the extent reported health effect associations reflect air quality violating the current or alternative standards, there is greater uncertainty in the degree to which such standards would allow the PM<sub>2.5</sub> exposures that provide the foundation for reported associations. The sections below (i.e., 3.2.3.2.1, 3.2.3.2.2) discuss each of these approaches in more detail, and present our key observations based on their application. The potential implications of these observations for the current and alternative primary PM<sub>2.5</sub> standards are discussed below in section 3.4.

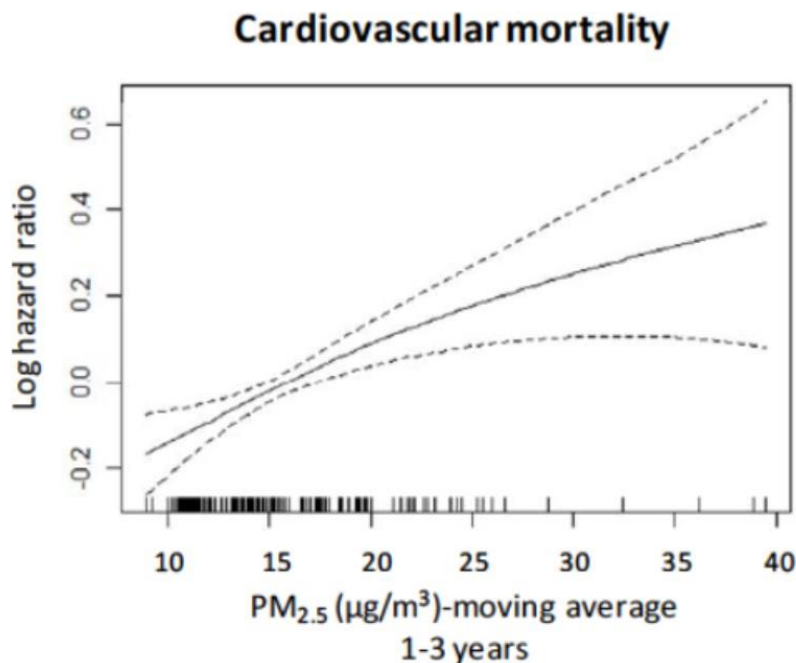
#### **3.2.3.2.1 PM<sub>2.5</sub> Air Quality Distributions Associated with Mortality or Morbidity in Key Epidemiologic Studies**

In this section, we consider the PM<sub>2.5</sub> air quality distributions associated with mortality or morbidity in key epidemiologic studies, with a focus on the parts of the distributions over which those studies provide the strongest support for reported associations. As discussed further below, while health effects may occur at PM<sub>2.5</sub> concentrations across the air quality distribution,

epidemiologic studies often provide the strongest support for reported health effect associations over the part of the distribution corresponding to the bulk of the underlying data (i.e., estimated exposures and/or health events). This is the case both for studies of daily PM<sub>2.5</sub> exposures and for studies of annual average PM<sub>2.5</sub> exposures.

Studies of daily PM<sub>2.5</sub> exposures examine associations between day-to-day variation in PM<sub>2.5</sub> concentrations and health outcomes, often over several years. While there can be considerable variability in daily exposures over a multi-year study period, most of the estimated exposures reflect days with ambient PM<sub>2.5</sub> concentrations around the middle of the air quality distributions examined (i.e., “typical” days rather than days with extremely high or extremely low concentrations). Similarly, for studies of annual PM<sub>2.5</sub> exposures, most of the estimated exposures reflect annual average PM<sub>2.5</sub> concentrations around the middle of the air quality distributions examined. In both cases, epidemiologic studies provide the strongest support for reported health effect associations for this middle portion of the PM<sub>2.5</sub> air quality distribution, which corresponds to the bulk of the underlying data, rather than the extreme upper or lower ends of the distribution. Consistent with this, as noted above in section 3.2.1.1, several epidemiologic studies report that associations persist in analyses that exclude the upper portions of the distributions of estimated PM<sub>2.5</sub> exposures, indicating that “peak” PM<sub>2.5</sub> exposures are not disproportionately responsible for reported health effect associations.

An example of the relationship between data density and reported health effect associations is illustrated in Figure 3-2 below (from Lepeule et al., 2012, Figure 1 in supplemental material; U.S. EPA, 2019, Figure 6-26). For the years 1974 to 2009, Lepeule et al. (2012) report a positive and statistically significant association between estimated long-term PM<sub>2.5</sub> exposures and cardiovascular mortality in six U.S. cities. Based on a visual inspection of the concentration-response function reported in this study (i.e., presented in Figure 3-2), 95% confidence intervals are narrowest for long-term PM<sub>2.5</sub> concentrations near the overall mean concentration reported in the study (i.e., 15.9 µg/m<sup>3</sup>). Confidence intervals widen at lower and higher long-term PM<sub>2.5</sub> concentrations, particularly at concentrations ≤ ~10 µg/m<sup>3</sup> and ≥ ~20 µg/m<sup>3</sup>. This widening in the confidence intervals is likely due in part to the comparative lack of data at concentrations approaching the lower and upper ends of the air quality distribution (i.e., exposure estimates are indicated by hash marks on the horizontal axis).



**Figure 3-2. Estimated concentration-response function and 95% confidence intervals between PM<sub>2.5</sub> and cardiovascular mortality in the Six Cities Study (1974-2009) (from Lepeule et al., 2012, supplemental material, figure 1; Figure 6-26 in U.S. EPA, 2019).**

Similar to the information presented in Figure 3-2, other recent studies have also reported that confidence intervals around concentration-response functions are relatively narrow at PM<sub>2.5</sub> concentrations around the overall mean concentrations reported by those studies, likely reflecting high data density in the middle portions of the distributions (e.g., Crouse et al., 2015; Villeneuve et al., 2015; Shi et al., 2016 as discussed in U.S. EPA, 2019, section 11.2.4). Thus, consistent with the approach in the last review (78 FR 3161, January 15, 2013; U.S. EPA, 2011, sections 2.1.3 and 2.3.4.1), we use study-reported means (or medians) of daily and annual average PM<sub>2.5</sub> concentrations as proxies for the middle portions of the air quality distributions, over which studies generally provide strong support for reported associations. As described further below, when considering the PM<sub>2.5</sub> air quality distributions in epidemiologic studies in this section, we focus on PM<sub>2.5</sub> concentrations around these overall means (including concentrations somewhat below means).

To evaluate the PM<sub>2.5</sub> air quality distributions in key studies in this review, we first identify the epidemiologic studies assessed in the ISA that have the potential to be most informative in reaching conclusions on the primary PM<sub>2.5</sub> standards. As for the experimental studies discussed above, we focus on epidemiologic studies that provide strong support for “causal” or “likely to be causal” relationships with PM<sub>2.5</sub> exposures in the ISA. We focus on the health effect associations that are determined in the ISA to be consistent across studies, coherent



with the broader body of evidence (e.g., including animal and controlled human exposure studies), and robust to potential confounding by co-occurring pollutants and other factors. We emphasize multicity studies that examine health effect associations in the U.S. or Canada, as such studies examine potential associations over large geographic areas with diverse atmospheric conditions and population demographics (e.g., U.S. EPA, 2019, sections 11.1 and 11.2). Additionally, studies examining associations outside the U.S. or Canada reflect air quality and exposure patterns that may be less typical of the U.S., and thus less likely to be informative for purposes of reviewing the NAAQS.<sup>29</sup>

Figure 3-3 to Figure 3-6 and Table 3-3 below summarize information from U.S. and Canadian studies that are assessed in the ISA and that meet these criteria. For each study, Figure 3-3 to Figure 3-6 present the cohort and/or geographic area examined, the approach used to estimate PM<sub>2.5</sub> exposures (i.e., monitored versus predicted with hybrid modeling methods<sup>30</sup>), the study years during which health events occurred, the years of PM<sub>2.5</sub> air quality data used to estimate exposures, and the effect estimate<sup>31</sup> with 95% confidence intervals (per 5 µg/m<sup>3</sup> for long-term exposures; 10 µg/m<sup>3</sup> for short-term exposures). When available, these figures also include the overall means (or medians if means are not available) of the short- or long-term PM<sub>2.5</sub> exposure estimates reported by the study.

Figure 3-3 and Figure 3-4 summarize information from studies of long-term PM<sub>2.5</sub> exposures. Figure 3-5 and Figure 3-6 summarize information from studies of short-term PM<sub>2.5</sub> exposures. Table 3-3 summarizes information from the smaller group of retrospective studies that have evaluated the potential for improvements in public health as ambient PM<sub>2.5</sub> concentrations have declined over time. It is important to note that these retrospective studies tend to focus on time periods during which ambient PM<sub>2.5</sub> concentrations were substantially higher than those measured more recently (e.g., see Chapter 2, Figure 2-8).

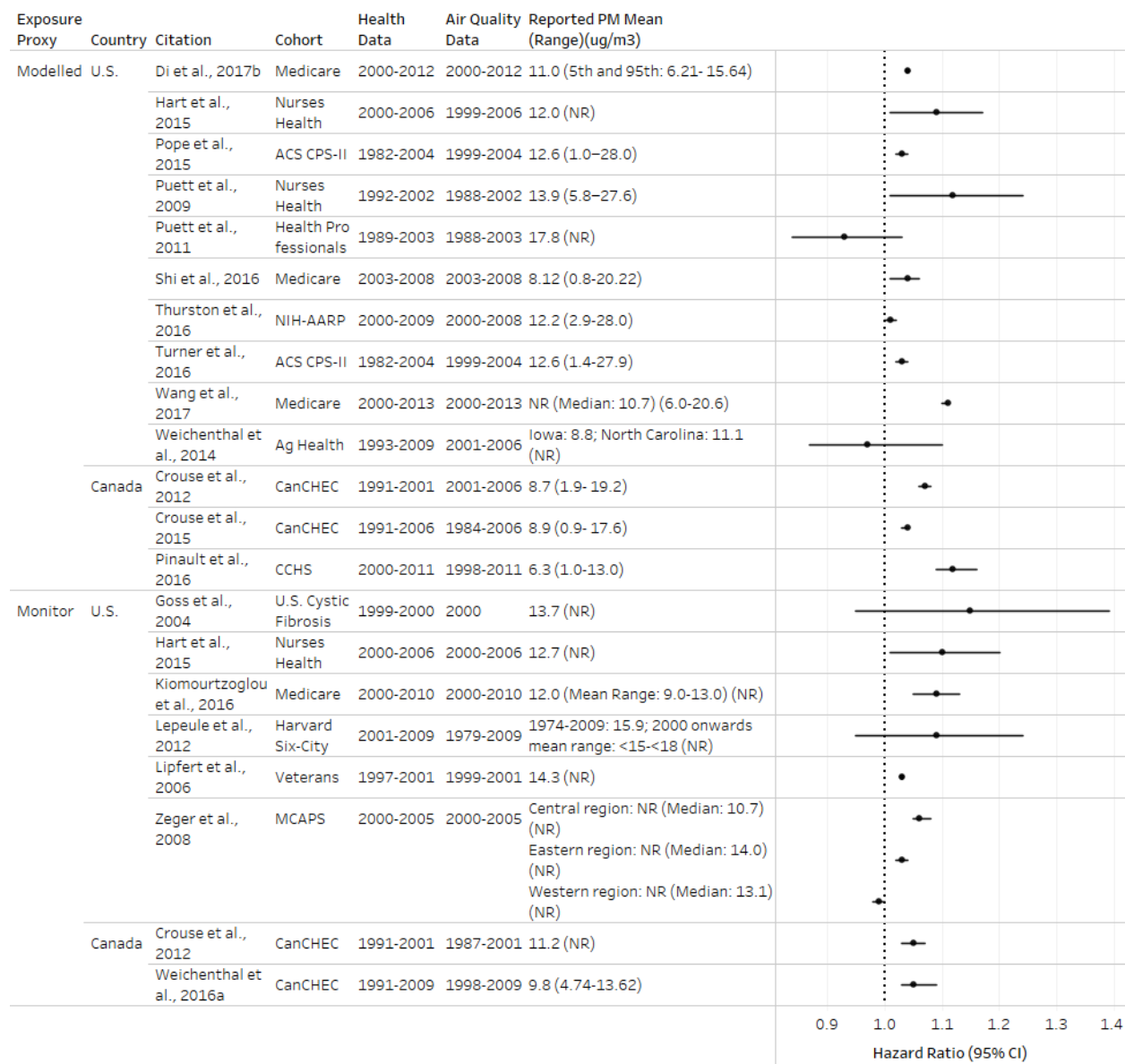
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<sup>29</sup> This emphasis on studies conducted in the U.S. or Canada is consistent with the approach in the last review of the PM NAAQS (U.S. EPA, 2011, section 2.1.3).

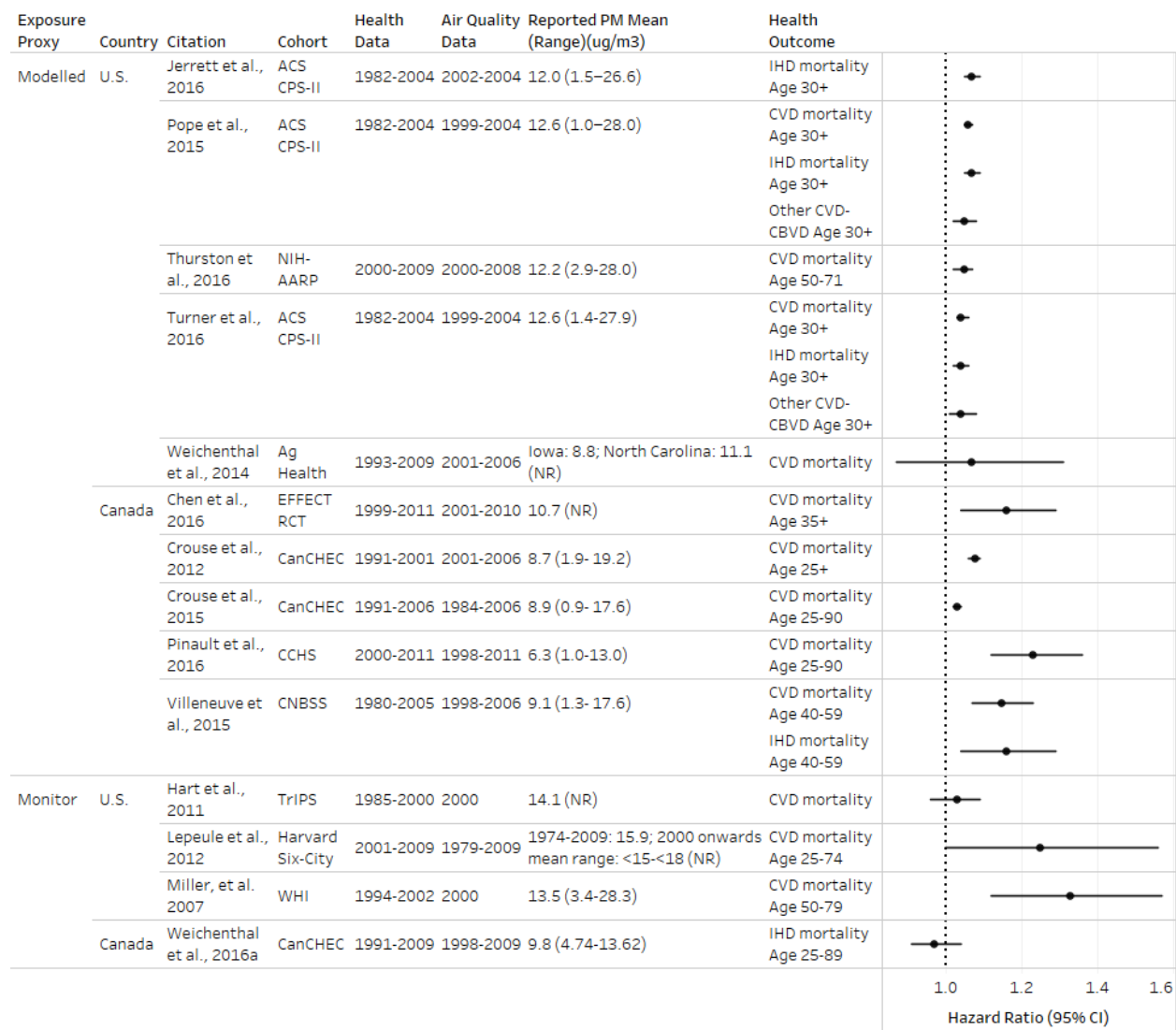
<sup>30</sup> As discussed further below, and in Chapter 2, hybrid methods incorporate data from several sources, often including satellites and models, in addition to ground-based monitors.

<sup>31</sup> The effect estimates presented in the forest plot figures (Figure 3-3 to Figure 3-6) show the associations of long or short-term PM<sub>2.5</sub> exposures with health endpoints presented either as hazard ratio or odds ratio or relative risk (for which the bold dotted vertical line is at 1), or as per unit or percent change (for which the bold dotted vertical line is at 0).

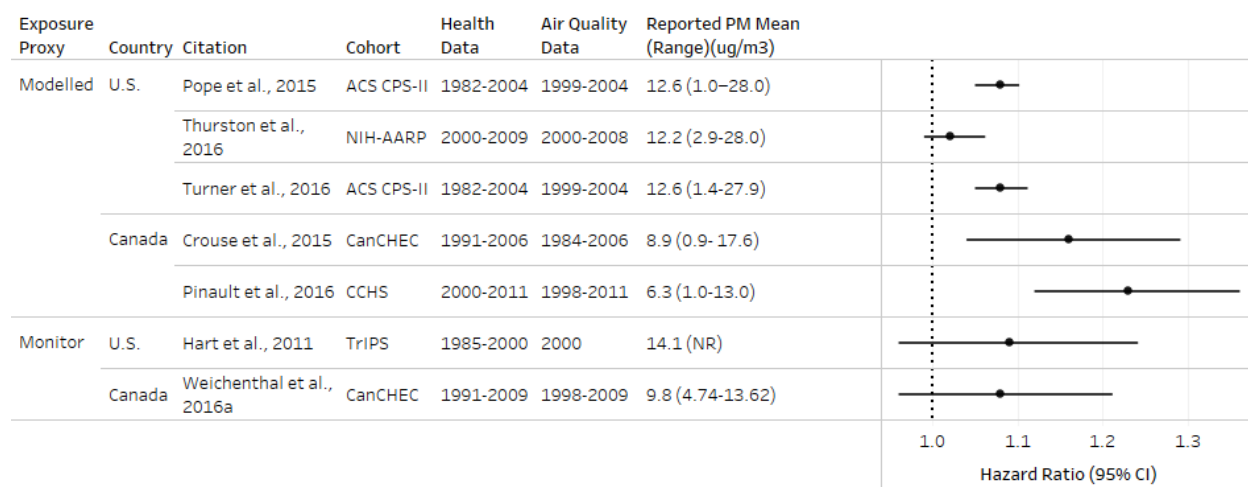
## All-cause mortality



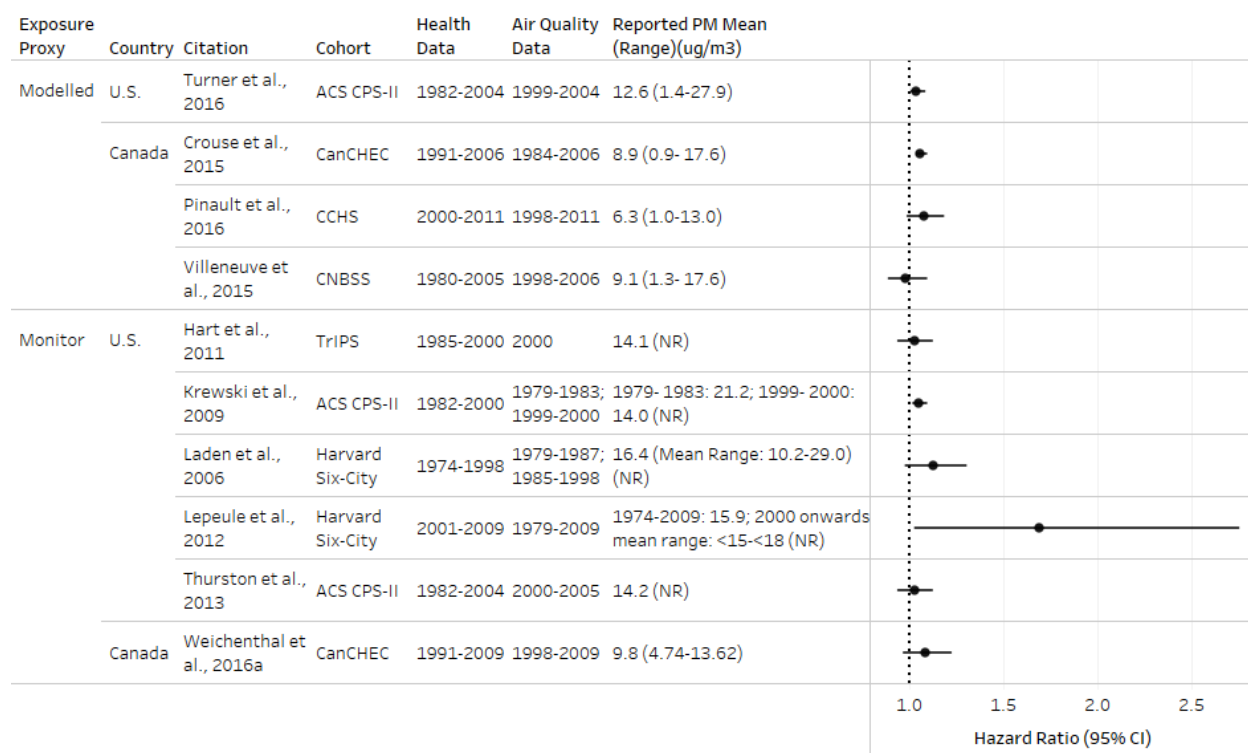
## CVD mortality



## Respiratory mortality

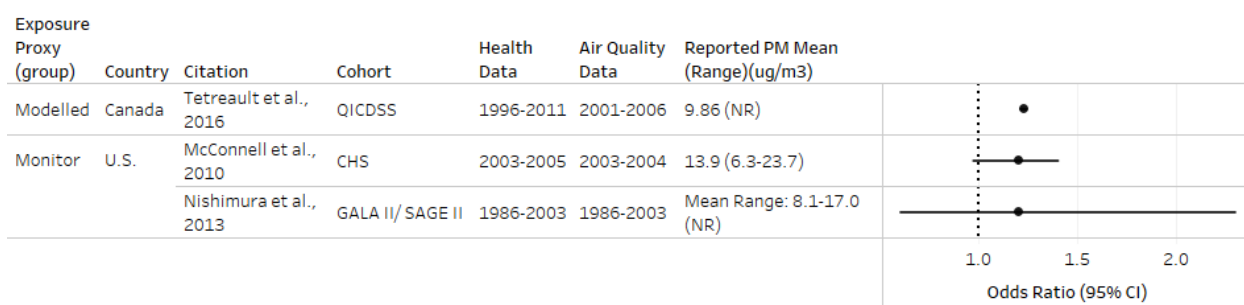


## Lung cancer mortality

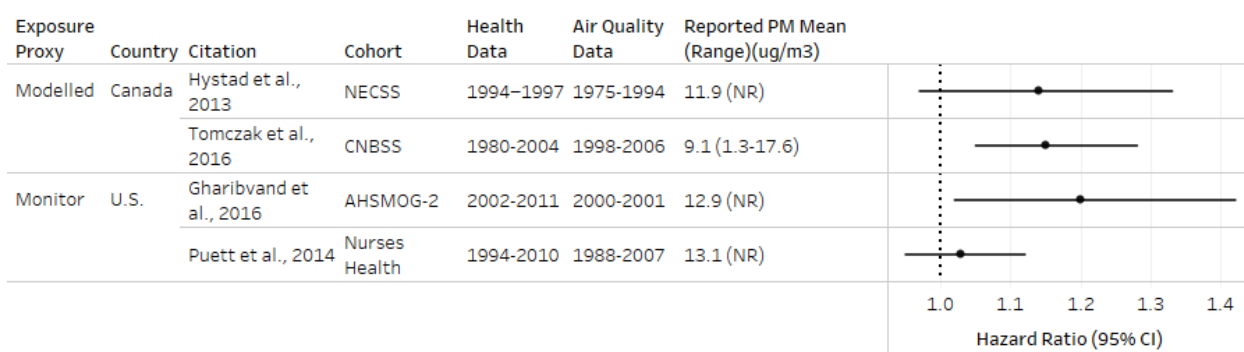


**Figure 3-3. Epidemiologic studies examining associations between long-term PM<sub>2.5</sub> exposures and mortality.**

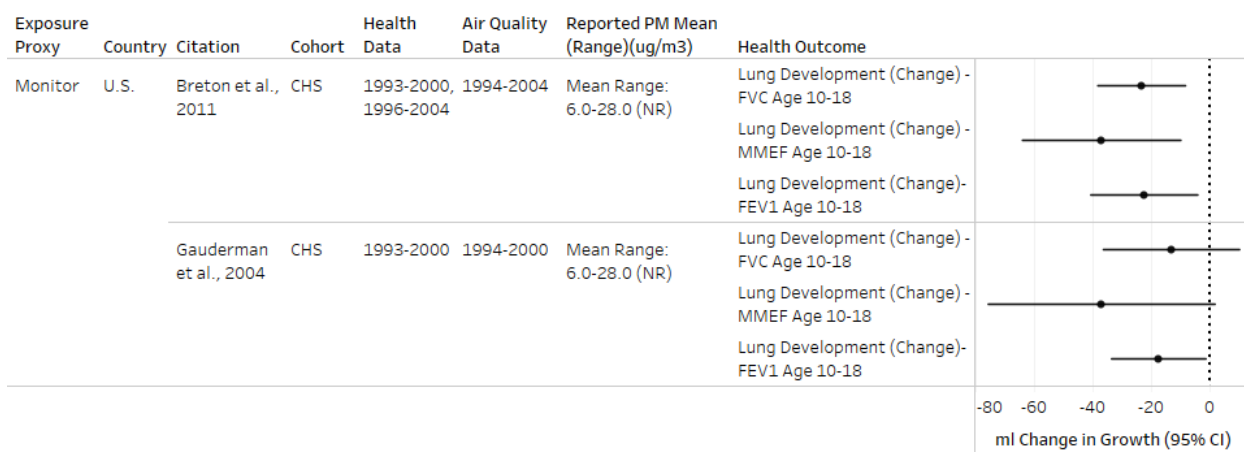
## Asthma incidence



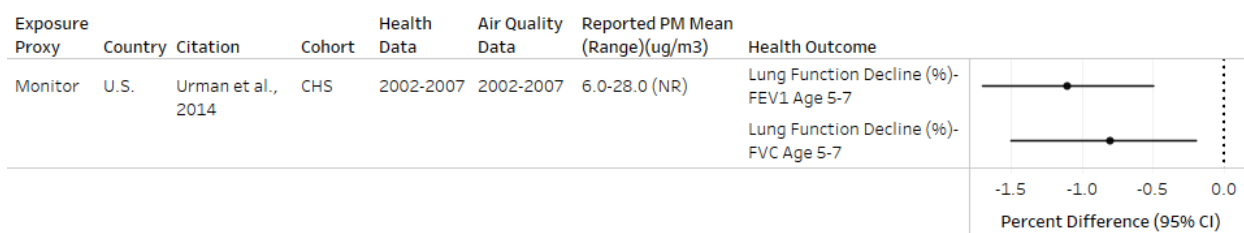
## Lung cancer incidence



## Lung development

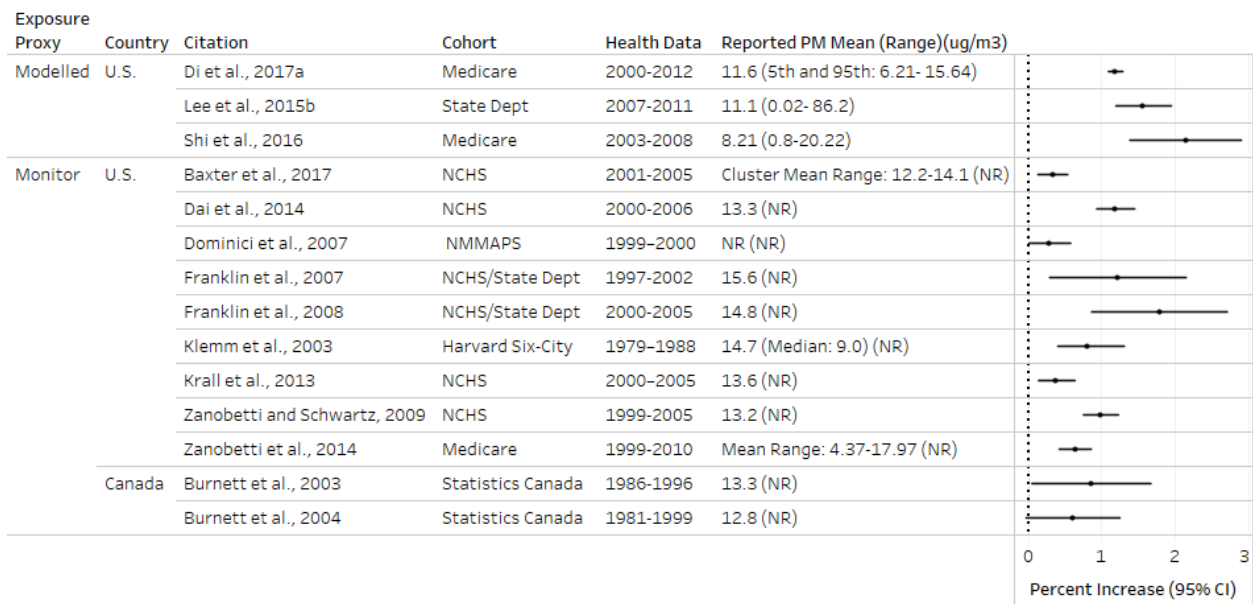


## Lung function

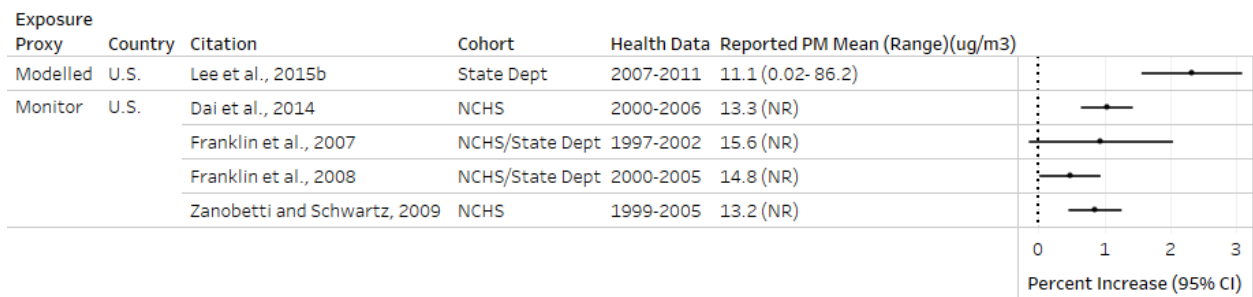


**Figure 3-4. Epidemiologic studies examining associations between long-term PM<sub>2.5</sub> exposures and morbidity.**

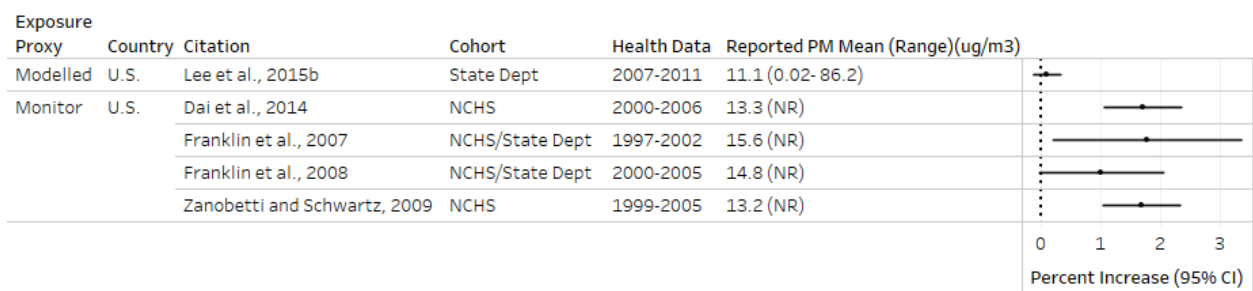
## All-cause mortality



## CVD mortality



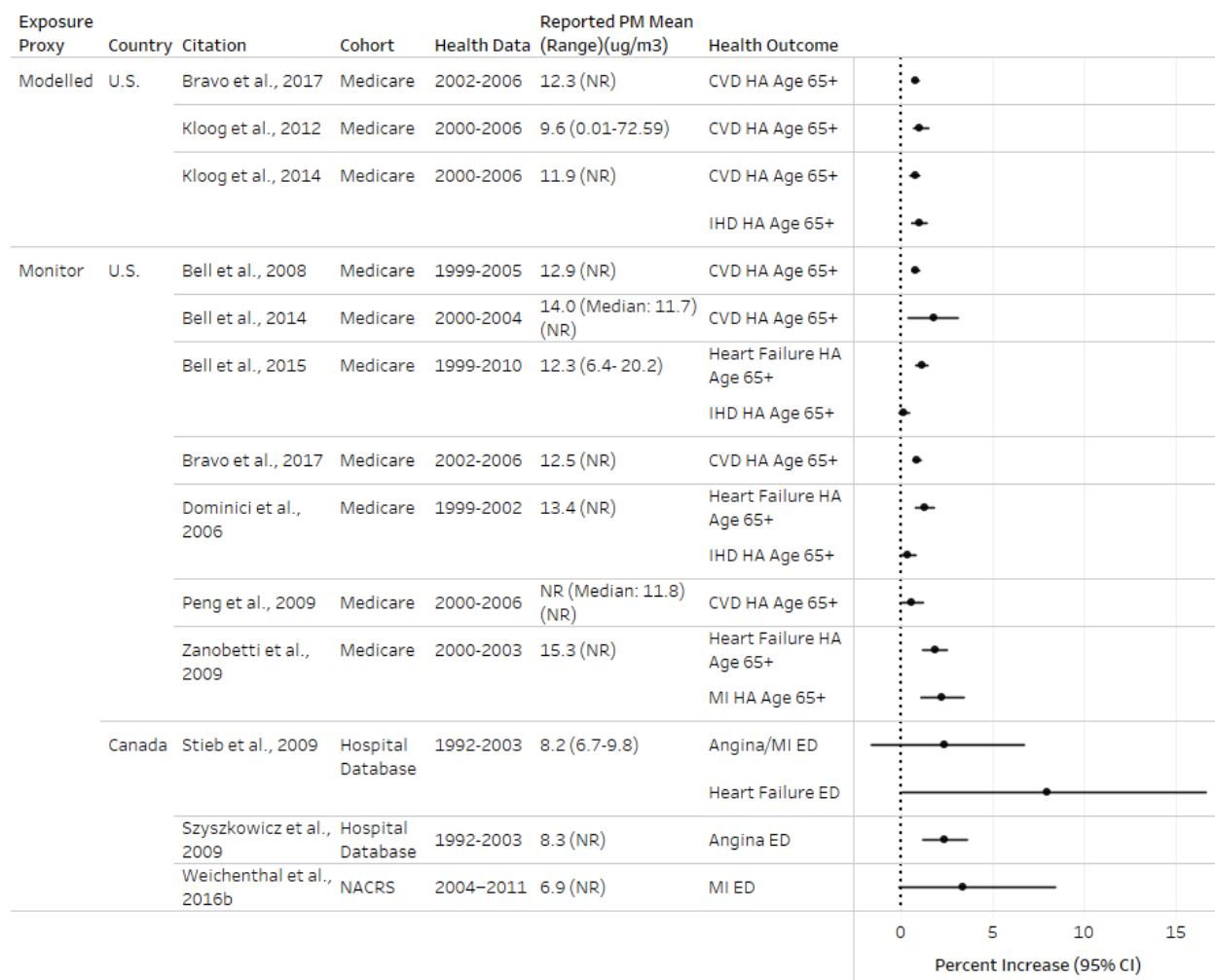
## Respiratory mortality



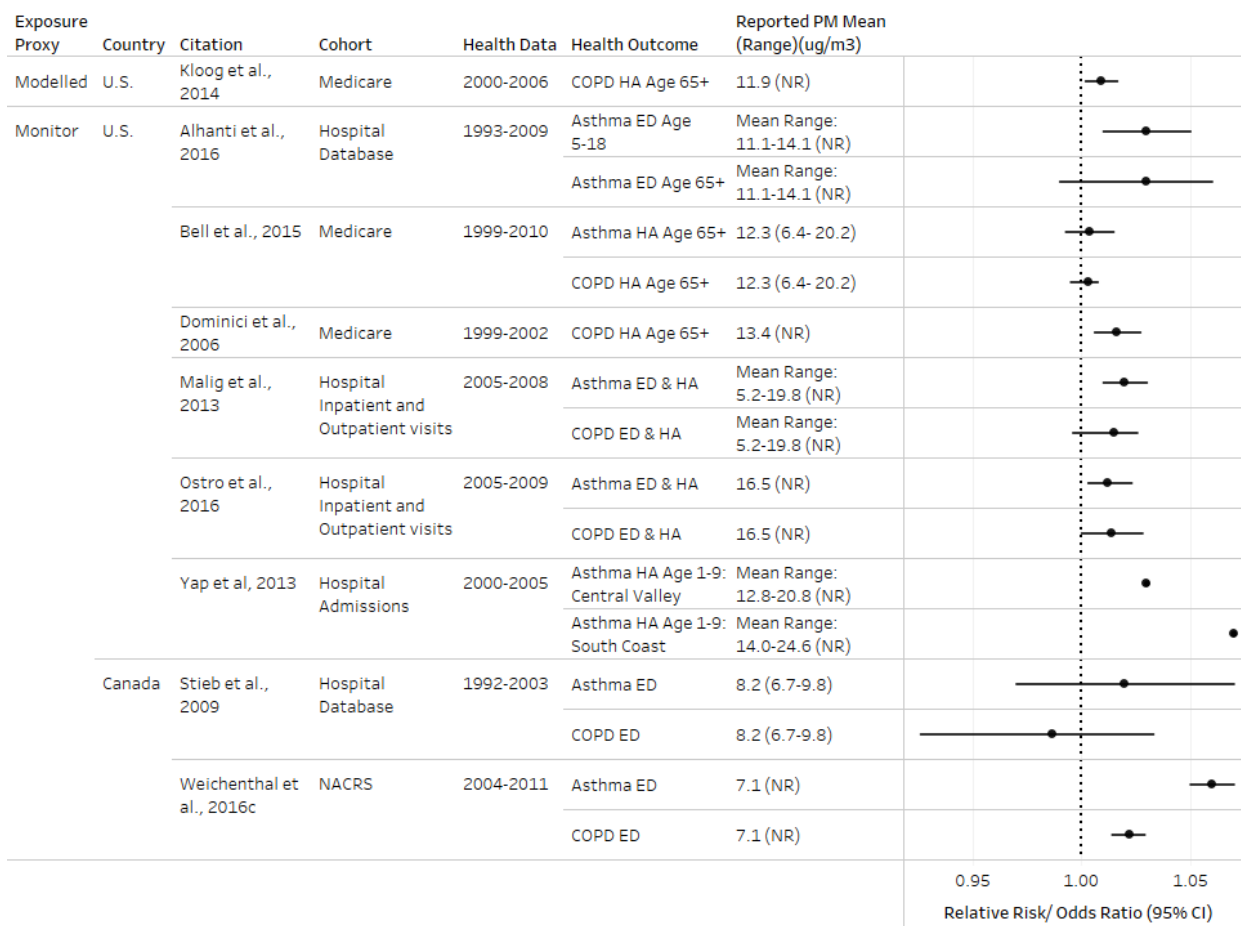
**Figure 3-5. Epidemiologic studies examining associations between short-term PM<sub>2.5</sub> exposures and mortality.<sup>32</sup>**

<sup>32</sup> As noted above, the overall mean PM<sub>2.5</sub> concentrations reported in studies of short-term (24-hour) exposures reflect averages across the study population and over the years of the study. Thus, mean concentrations reflect long-term averages of 24-hour PM<sub>2.5</sub> exposure estimates.

## CVD morbidity



## Respiratory morbidity



**Figure 3-6. Epidemiologic studies examining associations between short-term PM<sub>2.5</sub> exposures and morbidity.**



**Table 3-3. Epidemiologic studies examining the health impacts of long-term reductions in ambient PM<sub>2.5</sub> concentrations.**

Study Reference	Study Area	Years of PM <sub>2.5</sub> Air Quality (monitored)	Starting PM <sub>2.5</sub> Concentrations (mean)	Ending PM <sub>2.5</sub> concentrations (mean)	Study Results
Pope et al. (2009)	211 U.S. counties	1979-1983 compared to 1999-2000	20.6 µg/m <sup>3</sup>	14.1 µg/m <sup>3</sup>	Statistically significant association between declining ambient PM <sub>2.5</sub> and increasing life expectancy
Correia et al. (2013)	545 U.S. counties	2000 compared to 2007	13.2 µg/m <sup>3</sup>	11.6 µg/m <sup>3</sup>	Statistically significant association between declining ambient PM <sub>2.5</sub> and increasing life expectancy
Berhane et al. (2016)	4,602 children in 8 California communities	1992-2000; 1995-2003; 2002-2011	20.5 µg/m <sup>3</sup>	14.4 µg/m <sup>3</sup>	Statistically significant decrease in bronchitic symptoms in 10-year old children with and without asthma
Gauderman et al. (2015)	2,120 children in 5 California communities	1994-1997; 1997-2000; 2007-2010	21.3-31.5 µg/m <sup>3</sup>	11.9-17.8 µg/m <sup>3</sup>	Statistically significant improvements in 4-year growth of lung function

Based on the information in Figure 3-3 to Figure 3-6 and Table 3-3, key epidemiologic studies conducted in the U.S. or Canada indicate generally positive and statistically significant associations between estimated PM<sub>2.5</sub> exposures (short- or long-term) and mortality or morbidity across a wide range of ambient PM<sub>2.5</sub> concentrations. As discussed above, considering the PM<sub>2.5</sub> concentrations around (i.e., somewhat below to somewhat above) the overall means in these studies can provide insight into the part of the air quality distribution over which studies provide the strongest support for reported health effect associations. Evaluating whether such PM<sub>2.5</sub> air quality distributions would be likely to occur in areas meeting the current (or alternative) primary standards can inform conclusions on the degree to which those standards would limit the potential for the long-term and short-term PM<sub>2.5</sub> exposures that provide strong support for reported associations.

For a subset of key epidemiologic studies with available information, we characterize the broader distributions of ambient concentrations, with a particular focus on the concentrations below which data could become appreciably more limited (i.e., below which relatively few estimated exposures, and/or few health events, occurred). As noted above, confidence in reported health effect associations declines for portions of the air quality distribution accounting for

comparatively little data (i.e., concentrations approaching the lower and upper ends of the distribution). Thus, considering the concentrations below which data become relatively sparse can provide insight into the ambient PM<sub>2.5</sub> concentrations below which confidence in reported health effect associations may decrease notably. While there is no single concentration below which we lose confidence in reported associations, consistent with the approach in the last review (U.S. EPA, 2011, section 2.3.4.1), we identify the PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of health data (when available) or exposure estimates to provide insight into the concentrations that comprise the lower quartiles of the air quality distributions.<sup>33</sup>

To frame our evaluation of study-reported PM<sub>2.5</sub> concentrations, we specifically consider the following questions:

- **What are the overall mean PM<sub>2.5</sub> concentrations reported by key epidemiologic studies?**
- **For studies with available information on the broader distributions of exposure estimates and/or health events, what are the PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and/or 10<sup>th</sup> percentiles of those data?**

Answers to these questions can provide insight into the range of PM<sub>2.5</sub> concentrations, including those below the overall means, over which key studies provide strong support for reported associations. To this end, Figure 3-7 and Figure 3-8 below present information on the monitored (Figure 3-7) and hybrid model-predicted (Figure 3-8) ambient PM<sub>2.5</sub> concentrations used to estimate PM<sub>2.5</sub> exposures in key epidemiologic studies.

Drawing from the U.S. and Canadian multicity studies in Figure 3-3 to Figure 3-6 above,<sup>34</sup> the studies included in Figure 3-7 and Figure 3-8 are those that report overall mean (or median) PM<sub>2.5</sub> concentrations and for which the years of PM<sub>2.5</sub> air quality data used to estimate exposures overlap entirely with the years during which health events are reported. Regarding this latter issue, the PM<sub>2.5</sub> concentrations reported by studies that estimate exposures from air quality corresponding to only part of the study period, often including only the later years of the health data (e.g., Miller et al., 2007; Hart et al., 2011; Thurston et al., 2013; Weichenthal et al., 2014; Weichenthal et al., 2016a; Pope et al., 2015; Villeneuve et al., 2015; Turner et al., 2016), are not

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<sup>33</sup> In the last review of the PM NAAQS, the PA identified the long-term PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of health events, or study populations. In doing so, the PA noted that a range of one standard deviation around the mean represents approximately 68% of normally distributed data and, below the mean, falls between the 25<sup>th</sup> and 10<sup>th</sup> percentiles.

<sup>34</sup> Most of the studies included in Table 3-3 above (i.e., studies that examine relationships between declining ambient PM<sub>2.5</sub> concentrations and improving health) report mean ambient PM<sub>2.5</sub> concentrations well-above those in the studies highlighted in Figure 3-3 to Figure 3-6, and well-above the concentrations likely to be informative for conclusions on the current primary PM<sub>2.5</sub> standards. Therefore, our evaluation of mean concentrations focuses on the key studies identified in Figure 3-3 to Figure 3-6.

likely to reflect the full ranges of ambient PM<sub>2.5</sub> concentrations that contributed to reported associations.<sup>35</sup>

Figure 3-7 highlights the overall mean (or median) PM<sub>2.5</sub> concentrations reported in key studies that use ground-based monitors alone to estimate long- or short-term PM<sub>2.5</sub> exposures. For the subset of studies with available information on the broader distributions of underlying data, Figure 3-7 also identifies the study-period mean PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of health events<sup>36</sup> (see Appendix B, Section B.2 for more information).

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<sup>35</sup> This is an issue only for some studies of long-term PM<sub>2.5</sub> exposures. While this approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term PM<sub>2.5</sub> exposures, under the assumption that spatial patterns in PM<sub>2.5</sub> concentrations are not appreciably different during time periods for which air quality information is not available (e.g., Chen et al., 2016), our interest is in understanding the distribution of ambient PM<sub>2.5</sub> concentrations that could have contributed to reported health outcomes.

<sup>36</sup> That is, 25% of the total health events occurred in study locations with mean PM<sub>2.5</sub> concentrations (i.e., averaged over the study period) below the 25<sup>th</sup> percentiles identified in Figure 3-7 and 10% of the total health events occurred in study locations with mean PM<sub>2.5</sub> concentrations below the 10<sup>th</sup> percentiles identified.

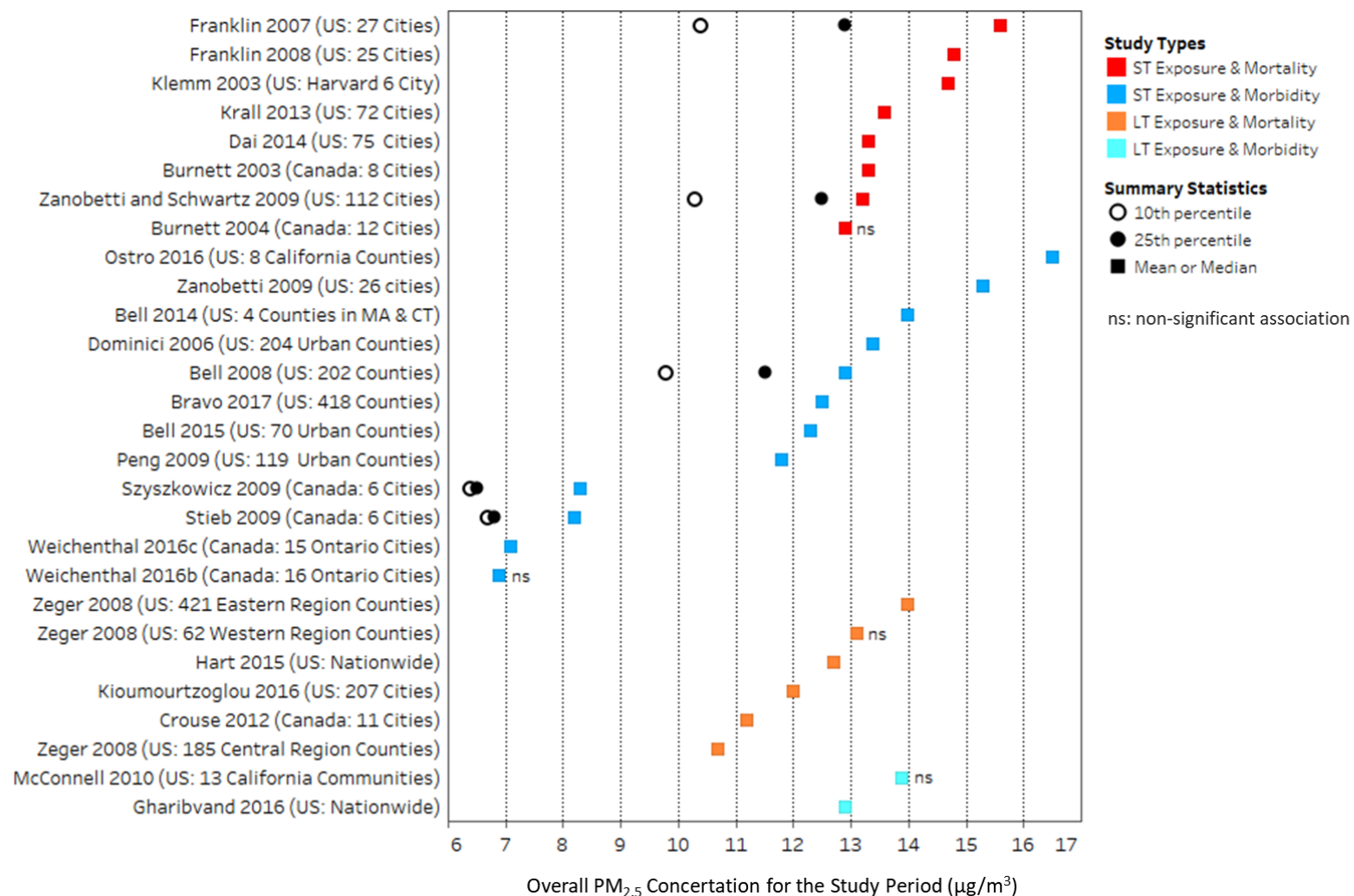


Figure 3-7. Monitored PM<sub>2.5</sub> concentrations in key epidemiologic studies.

We also consider the emerging body of studies that use predicted ambient PM<sub>2.5</sub> concentrations from hybrid modeling methods to estimate long- or short-term PM<sub>2.5</sub> exposures (Figure 3-8, below). As discussed in Chapter 2 of this PA (section 2.3.3), hybrid methods incorporate data from several sources, often including satellites and models in addition to ground-based monitors. Compared to ground-based monitors alone, hybrid methods have the potential to improve the characterization of PM<sub>2.5</sub> exposures in areas with relatively sparse monitoring networks (U.S. EPA, 2019, sections 3.3.2 to 3.3.5).

Figure 3-8 presents overall means of predicted PM<sub>2.5</sub> concentrations for key studies, and the concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of estimated exposures or health events<sup>37</sup> when available (see appendix B, section B.3 for additional information).<sup>38</sup> As for the monitor-based studies highlighted above, Figure 3-8 focuses on multicity studies that examine health outcomes supporting “causal” or “likely to be causal” determinations in the ISA and that use air quality data to estimate PM<sub>2.5</sub> exposures for the entire range of years during which health events occurred.<sup>39</sup> In addition to these criteria, we also consider the approach used to validate hybrid model predictions. In particular, the studies included in Figure 3-8 are those for which relatively robust model validation analyses are reported to have been conducted for the full range of years during which PM<sub>2.5</sub> exposures are estimated in the health study (e.g., regional or

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<sup>37</sup> For most studies in Figure 3-8, 25<sup>th</sup> percentiles of exposure estimates are presented. The exception is Di et al. (2017a), for which Figure 3-8 presents the short-term PM<sub>2.5</sub> exposure estimates corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of deaths in the study population (i.e., 25% and 10% of deaths occurred at concentrations below these concentrations). In addition, the authors of Di et al. (2017b) provided population-weighted exposure values (Chan, 2019). The 10<sup>th</sup> and 25<sup>th</sup> percentiles of these population-weighted exposure estimates are 7.9 and 9.5 µg/m<sup>3</sup>, respectively.

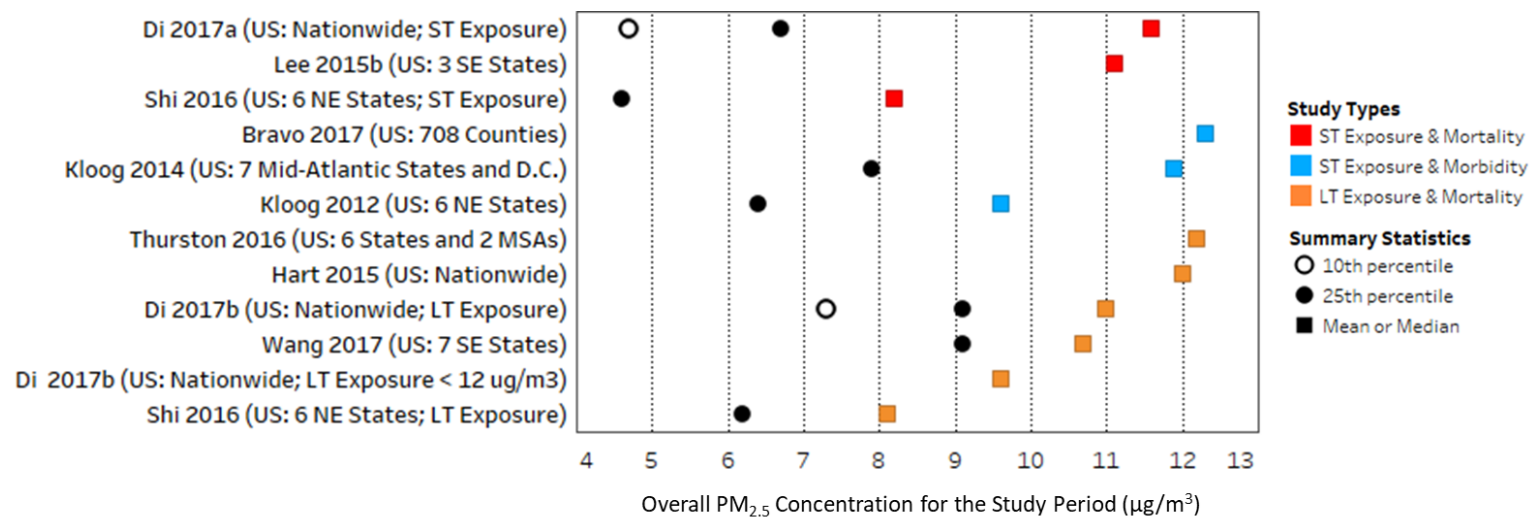
<sup>38</sup> In addition, 75<sup>th</sup> percentiles of exposure estimates are available for some studies. They are as follows: 14.4 µg/m<sup>3</sup> (Di et al., 2017a), 12.9 µg/m<sup>3</sup> (Di et al., 2017b), 11.7 µg/m<sup>3</sup> (Kloog et al., 2012), 10.7 µg/m<sup>3</sup> (Shi et al. (2016), short-term exposures), 10.0 µg/m<sup>3</sup> (Shi et al. (2016), long-term exposures), 12.9 µg/m<sup>3</sup> (Wang et al., 2017).

<sup>39</sup> All studies that meet the criteria for inclusion in Figure 3-8 were conducted in the U.S.

national 10-fold cross validation performance statistics reported for the same years that exposures are estimated).<sup>40</sup>

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<sup>40</sup> For example, due to lack of spatial field availability before 1998, Crouse et al. (2015) use median annual PM<sub>2.5</sub> concentrations for the 1998-2006 time period (van Donkelaar et al., 2010; van Donkelaar et al., 2015a; van Donkelaar et al., 2013) to predict exposures during the 1984-2006 period. Similarly, for Pinault et al. (2016), model validation is for 2004 to 2008 (van Donkelaar et al., 2015b) while exposures are estimated for 1998 to 2012. Paciorek et al. (2009), which presents the model validation results for Puett et al. (2009) and Puett et al. (2011), notes that PM<sub>2.5</sub> monitoring was sparse prior to 1999, with many of the available PM<sub>2.5</sub> monitors in rural and protected areas. Therefore, Paciorek et al. (2009) conclude that coverage in the validation set for most of the study period (1988-1998) is poor and that their model strongly underestimates uncertainty (Paciorek et al. (2009), p. 392 in published manuscript). Hystad et al. (2013) used exposure fields developed by calibrating satellite-based PM<sub>2.5</sub> surfaces from a recent period (van Donkelaar et al., 2010) to estimate exposure for the 1975 to 1994 (Hystad et al., 2012). Hystad et al. (2012) noted that a random effect model was used to estimate PM<sub>2.5</sub> based on TSP measurements and metropolitan indicator variables because only small number of PM<sub>2.5</sub> measurements were available, and no measurements were made prior to 1984. Thus, these studies from Figures 3-3 to 3-6 are not included in Figure 3-8.



**Figure 3-8. Hybrid model-predicted PM<sub>2.5</sub> concentrations in key epidemiologic studies.**

Taking the information in Figure 3-7 and Figure 3-8 together, key epidemiologic studies conducted in the U.S. or Canada report generally positive and statistically significant associations between estimated PM<sub>2.5</sub> exposures (short- or long-term) and mortality or morbidity across a wide range of monitored or hybrid-model-predicted ambient PM<sub>2.5</sub> concentrations. With regard to these studies, we particularly note the following:

- For the large majority of key studies, the PM<sub>2.5</sub> air quality distributions that support reported associations are characterized by overall mean (or median) PM<sub>2.5</sub> concentrations ranging from just above 8.0 µg/m<sup>3</sup> to just above 16.0 µg/m<sup>3</sup>. There is substantial overlap between mean concentrations based on monitoring alone and those based on hybrid modeling approaches.
  - Most key studies that use monitors alone to estimate PM<sub>2.5</sub> exposures, and all of the U.S. studies in this group, report overall mean PM<sub>2.5</sub> concentrations at or above 10.7 µg/m<sup>3</sup>.
  - Four Canadian studies that use monitors alone report lower overall mean concentrations. Two of these studies report overall means just above 8.0 µg/m<sup>3</sup> (both report positive and statistically significant associations) and two studies report overall means around 7.0 µg/m<sup>3</sup> (positive and statistically significant association in one of these studies).
  - Most key studies that use hybrid modeling approaches to estimate PM<sub>2.5</sub> exposures report overall mean concentrations at or above 9.6 µg/m<sup>3</sup>. All of these studies were conducted in the U.S. and report positive and statistically significant health effect associations.
  - The hybrid modeling study with the lowest PM<sub>2.5</sub> concentrations reports overall means just above 8.0 µg/m<sup>3</sup> (i.e., Shi et al., 2016). This study reports positive and statistically significant health effect associations with both short- and long-term PM<sub>2.5</sub> exposures.<sup>41</sup>
- Four U.S. studies examine health effect associations in analyses with the highest exposures excluded. Only one of these restricted analyses is reflected in Figure 3-8 (i.e., Di et al., 2017b; “LT exposure < 12 µg/m<sup>3</sup>”). In addition to this study, Lee et al. (2015), Di et al. (2017a) and Shi et al. (2016) also report positive and statistically significant associations in restricted analyses.
  - Lee et al. (2015) reports a positive and statistically significant association in an analysis restricted to zip codes with annual average PM<sub>2.5</sub> concentrations < 12 µg/m<sup>3</sup> and to days with 24-hour average PM<sub>2.5</sub> concentrations < 35 µg/m<sup>3</sup>. This study did not report an overall mean PM<sub>2.5</sub> concentration for the restricted analysis, though it was presumably somewhat below the mean reflected in Figure 3-8 (i.e., 11.1 µg/m<sup>3</sup>).

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<sup>41</sup> However, the authors report that, for associations with long-term PM<sub>2.5</sub> exposures, most deaths occurred at or above the 75<sup>th</sup> percentile of annual exposure estimates (i.e., 10 µg/m<sup>3</sup>) (see Tables 1 and 2 in published manuscript). Authors did not report this information for their analysis of short-term PM<sub>2.5</sub> exposures.



- Di et al. (2017a) reports a positive and statistically significant association in an analysis restricted to 24-hour PM<sub>2.5</sub> exposure estimates < 25 µg/m<sup>3</sup>. This study did not report an overall mean PM<sub>2.5</sub> concentration for the restricted analysis, though it was presumably somewhat below the mean reflected in Figure 3-8 (i.e., 11.6 µg/m<sup>3</sup>).
- Shi et al. (2016) report positive and statistically significant associations in analyses restricted to annual PM<sub>2.5</sub> exposure estimates < 10 µg/m<sup>3</sup> and in analyses restricted to 24-hour exposure estimates < 30 µg/m<sup>3</sup>. This study does not report the overall mean PM<sub>2.5</sub> concentrations in restricted analyses, though such means are presumably somewhat below those reflected in Figure 3-8 (i.e., 8.1 and 8.2 µg/m<sup>3</sup>).
- For some key studies, information on the broader distributions of PM<sub>2.5</sub> exposure estimates and/or health events is available.
  - In U.S. studies that use monitors alone to estimate PM<sub>2.5</sub> exposures, 25<sup>th</sup> percentiles of health events correspond to mean PM<sub>2.5</sub> concentrations (i.e., averaged over the study period for each study city) at or above 11.5 µg/m<sup>3</sup> and 10<sup>th</sup> percentiles of health events correspond to mean PM<sub>2.5</sub> concentrations at or above 9.8 µg/m<sup>3</sup> (i.e., 25% and 10% of health events, respectively, occur in study locations with mean PM<sub>2.5</sub> concentrations below these values).
  - In the Canadian studies that use monitors alone to estimate PM<sub>2.5</sub> exposures, 25<sup>th</sup> percentiles of health events correspond to mean PM<sub>2.5</sub> concentrations at or above 6.5 µg/m<sup>3</sup> and 10<sup>th</sup> percentiles of health events correspond to mean PM<sub>2.5</sub> concentrations at or above 6.4 µg/m<sup>3</sup>.
  - Of the key studies that use hybrid modeling approaches to estimate long-term PM<sub>2.5</sub> exposures, the ambient PM<sub>2.5</sub> concentrations corresponding to 25<sup>th</sup> percentiles of estimated exposures are 6.2 and 9.1 µg/m<sup>3</sup>. In the one study with data available on the 10<sup>th</sup> percentile of PM<sub>2.5</sub> exposure estimates, the concentration corresponding to that 10<sup>th</sup> percentile is 7.3 µg/m<sup>3</sup>.
  - In studies that use hybrid modeling approaches to estimate short-term PM<sub>2.5</sub> exposures, the ambient concentrations corresponding to 25<sup>th</sup> percentiles of estimated exposures, or health events, are generally at or above 6.4 µg/m<sup>3</sup>. In the one study with lower concentrations, the ambient PM<sub>2.5</sub> concentration corresponding to the 25<sup>th</sup> percentile of estimated exposures is 4.7 µg/m<sup>3</sup>.<sup>42</sup> In the one study with information available on the 10<sup>th</sup> percentile of health events, the ambient PM<sub>2.5</sub> concentration corresponding to that 10<sup>th</sup> percentile is 4.7 µg/m<sup>3</sup>.

The information in Figure 3-7 and Figure 3-8 indicates consistent support for generally positive and statistically significant health effect associations for PM<sub>2.5</sub> air quality distributions

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<sup>42</sup> As noted above, in this study (Shi et al., 2016), the authors report that most deaths occurred at or above the 75<sup>th</sup> percentile of annual exposure estimates (i.e., 10 µg/m<sup>3</sup>). The short-term exposure estimates accounting for most deaths are not presented in the published study.

characterized by overall mean (or median) concentrations above  $8.0 \mu\text{g}/\text{m}^3$ , with most studies (and all but one U.S. study) reporting overall mean (or median) concentrations at or above  $9.6 \mu\text{g}/\text{m}^3$ . While the ambient  $\text{PM}_{2.5}$  concentrations around these overall means generally reflect the part of the air quality distribution over which studies provide the strongest support for reported  $\text{PM}_{2.5}$  effect estimates, there are uncertainties in using these concentrations to inform conclusions on the primary  $\text{PM}_{2.5}$  standards. These uncertainties are summarized below and their potential implications for conclusions on the current and alternative standards are discussed further in section 3.5.

A key uncertainty in using study-reported mean  $\text{PM}_{2.5}$  concentrations to inform conclusions on the primary  $\text{PM}_{2.5}$  standards is that such concentrations are not the same as the ambient concentrations used by the EPA to determine whether areas meet or violate the PM NAAQS. As discussed above, the overall mean  $\text{PM}_{2.5}$  concentrations reported by key epidemiologic studies reflect averaging of short- or long-term  $\text{PM}_{2.5}$  exposure estimates across locations (i.e., across multiple monitors or across modeled grid cells) and over time (i.e., over several years). In contrast, to determine whether areas meet or violate the NAAQS, the EPA measures air pollution concentrations at individual monitors (i.e., concentrations are not averaged across monitors) and calculates “design values” at monitors meeting appropriate data quality and completeness criteria. For the annual  $\text{PM}_{2.5}$  standard, design values are calculated as the annual arithmetic mean  $\text{PM}_{2.5}$  concentration, averaged over 3 years. For the 24-hour standard, design values are calculated as the 98<sup>th</sup> percentile of the annual distribution of 24-hour  $\text{PM}_{2.5}$  concentrations, averaged over three years (described in Appendix N of 40 CFR Part 50). For an area to meet the NAAQS, all valid design values in that area, including the highest annual and 24-hour monitored values, must be at or below the levels of the standards.

Because of this approach to determining whether areas meet the NAAQS, and because monitors are often required in locations with relatively high  $\text{PM}_{2.5}$  concentrations (section 2.2.3), areas meeting a  $\text{PM}_{2.5}$  standard with a particular level would be expected to have average  $\text{PM}_{2.5}$  concentrations (i.e., averaged across space and over time in the area) somewhat below that standard level. In support of this, analyses of recent air quality in U.S. CBSAs indicate that maximum annual  $\text{PM}_{2.5}$  design values for a given three-year period are often 10% to 20% higher than average monitored concentrations (i.e., averaged across multiple monitors in the same CBSA) (Appendix B, section B.7). The difference between the maximum annual design value and average concentration in an area can be smaller or larger than this range, likely depending on factors such as the number of monitors, monitor siting characteristics, and the distribution of

ambient PM<sub>2.5</sub> concentrations.<sup>43</sup> When using this information to interpret key epidemiologic studies in the context of the primary standards, it is also important to note that such ratios may depend on how the average concentrations in a study are calculated (i.e., averaged across monitors versus across modeled grid cells). Thus, as discussed further in section 3.5 below, when evaluating what the mean PM<sub>2.5</sub> concentrations reported by key epidemiologic studies may indicate regarding the current or alternative PM<sub>2.5</sub> standards, we consider the broader relationships between mean PM<sub>2.5</sub> concentrations, averaged across space and over time, and PM<sub>2.5</sub> design values.<sup>44</sup>

Additional uncertainties in using the PM<sub>2.5</sub> concentrations reported by key epidemiologic studies to inform conclusions on the primary PM<sub>2.5</sub> standards include the following:

- Effects can occur over the full distributions of ambient PM<sub>2.5</sub> concentrations evaluated in epidemiologic studies, and the evidence does not identify a threshold concentration below which PM<sub>2.5</sub>-associated effects no longer occur. Thus, while conclusions on primary standards can be informed by comparing the PM<sub>2.5</sub> air quality distributions present in key studies with the distributions likely to occur in areas meeting the current or alternative standards, studies do not identify specific PM<sub>2.5</sub> exposures that result in health effects or exposures below which effects do not occur.
- For studies that use hybrid model predictions to estimate PM<sub>2.5</sub> exposures, the performance of the recently developed modeling approaches depends on the availability of monitoring data and varies by location. As noted in Chapter 2 (section 2.3.3), factors likely contributing to poorer model performance often coincide with relatively low ambient PM<sub>2.5</sub> concentrations, potentially accounting for the observations that model performance for hybrid models weakens by some metrics with decreasing PM<sub>2.5</sub> concentration and that the normalized variability between predictions based on different hybrid modeling approaches increases with decreasing concentrations. Thus, uncertainty in hybrid model predictions becomes an increasingly important consideration as lower predicted concentrations are considered.

The potential implications of these and other uncertainties for conclusions on the current and alternative primary PM<sub>2.5</sub> standards are discussed below in section 3.4.

### **3.2.3.2.2 PM<sub>2.5</sub> Pseudo-Design Values in Locations of Key Epidemiologic Studies**

In addition to considering the study-reported PM<sub>2.5</sub> concentrations discussed above, we also evaluate study area air quality using metrics more closely related to the design values

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<sup>43</sup> Given that higher PM<sub>2.5</sub> concentrations have been reported at some near-road monitoring sites, relative to the surrounding area (section 2.3.2.2.2), recent requirements for PM<sub>2.5</sub> monitoring at near-road locations in large urban areas (section 2.2.3) may increase the ratios of maximum annual design values to averaged concentrations in some areas.

<sup>44</sup> As discussed above in section 3.1.2, compared to the annual standard, the potential implications of overall mean PM<sub>2.5</sub> concentrations reported by key epidemiologic studies are less clear for the 24-hour PM<sub>2.5</sub> standard with its 98<sup>th</sup> percentile form (section 3.4).

employed by the EPA to determine whether areas meet or violate the primary PM<sub>2.5</sub> standards. To the extent these metrics suggest that reported health effect associations are based largely on PM<sub>2.5</sub> air quality that would have met the current or alternative standards during study periods, we have greater confidence that those standards would allow the PM<sub>2.5</sub> exposures that provide the basis for reported associations. In contrast, to the extent these metrics suggest that reported health effect associations are based largely on air quality that would have violated the current or alternative standards, there is greater uncertainty in the degree to which those standards would allow the PM<sub>2.5</sub> exposures that provide the basis for reported associations.

To evaluate this issue, we calculate metrics similar to PM<sub>2.5</sub> design values (referred to here as “pseudo-design values”) for the locations and time periods evaluated by key U.S. and Canadian epidemiologic studies. Pseudo-design values are calculated as follows:

- We first identify the study locations with one or more PM<sub>2.5</sub> monitors operating during the study period, and that have sufficient monitoring data available to calculate pseudo-design values.<sup>45</sup>
  - For key studies conducted in the U.S., study locations are defined as the counties included in the study.
  - For key studies conducted in Canada, study locations are defined as the cities included in the study.
- For each monitored study location, we then identify the highest annual and 24-hour PM<sub>2.5</sub> pseudo-design values for each 3-year period of the study and calculate the study-period average of these highest values.
- We also identify the number of people living in each study location or, when available, the number of health events that occurred in each location during the study period.<sup>46</sup>
- To evaluate the percentages of study area populations living in locations likely to have met the current standards over study periods (or the percentages of health events occurring in

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<sup>45</sup> Pseudo-design values are based on data from both FRM/FEM monitors and from high quality non-FRM/FEM monitors. The non-regulatory data used to calculate pseudo-design values come from monitors typically used for EPA applications like AirNow that are not FRM or FEM. Only monitors with 75% completeness for each of the 12 quarters in a 3-year design value period were included. Sensitivity analyses based only on data from FRM/FEM regulatory monitors gave similar results (Appendix B, section B.5). For the pseudo-design values at the Canadian sites, only sites with 75% completeness for each year of the 3-year design value period were included. These criteria are slightly different than that of actual design values which have strict rounding conventions and substitution tests for sites with less than 75% completeness for each quarter. Additional information on the approach and data sources used to identify pseudo-design values in study locations is provided in Appendix B (section B.4.3).

<sup>46</sup> When available, we use the number of health events in each study location. However, for most key studies, health event data was not available for each study location. For these studies, we evaluate the population living in each study location. Comparison of these approaches in the subset of studies for which health events are available demonstrate that distributions of annual pseudo-design values are comparable for the two approaches (Appendix B, section B.6).

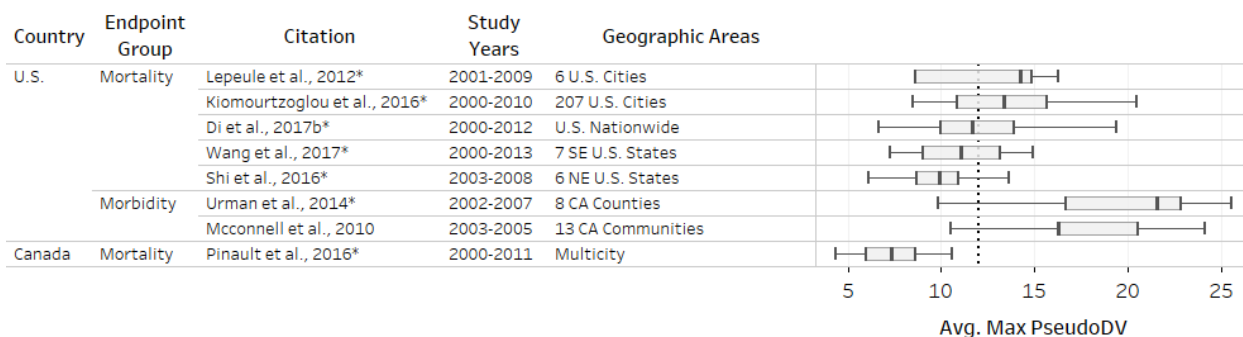
such locations), we identify the percentages in locations with study-period average pseudo-design values at or below the levels of the current annual (Figure 3-9; Appendix B, Tables B-5 and B-6) and 24-hour (Appendix B, Figure B-9) PM<sub>2.5</sub> standards.

In Figure 3-9, whiskers reflect annual PM<sub>2.5</sub> pseudo-design values corresponding to 5<sup>th</sup> and 95<sup>th</sup> percentiles of study area populations (or health events), boxes correspond to the 25<sup>th</sup> and 75<sup>th</sup> percentiles, and the vertical lines inside the boxes correspond to 50<sup>th</sup> percentiles. The vertical dotted line in Figure 3-9 is drawn at 12.0 µg/m<sup>3</sup>, the level of the current annual PM<sub>2.5</sub> standard. For studies with 25<sup>th</sup> percentiles ≤ 12.0 µg/m<sup>3</sup>, at least 25% of the study area population (i.e., in counties or cities with pseudo-design values) lived in locations likely to have met the current annual standard over the study period (or at least 25% of health events occurred in such locations).<sup>47</sup> Similarly, for studies with 50<sup>th</sup> or 75<sup>th</sup> percentiles ≤ 12.0 µg/m<sup>3</sup>, at least 50% or 75% of the study area population, respectively, lived in locations likely to have met the current annual standard over the study period (or at least 50% or 75% of health events occurred in such locations). The percentage of study area populations (or health events) in locations likely to have met the current 24-hour standard over study periods was typically larger than the percentage in locations likely to have met the current annual standard (i.e., Appendix B, Figure B-9).

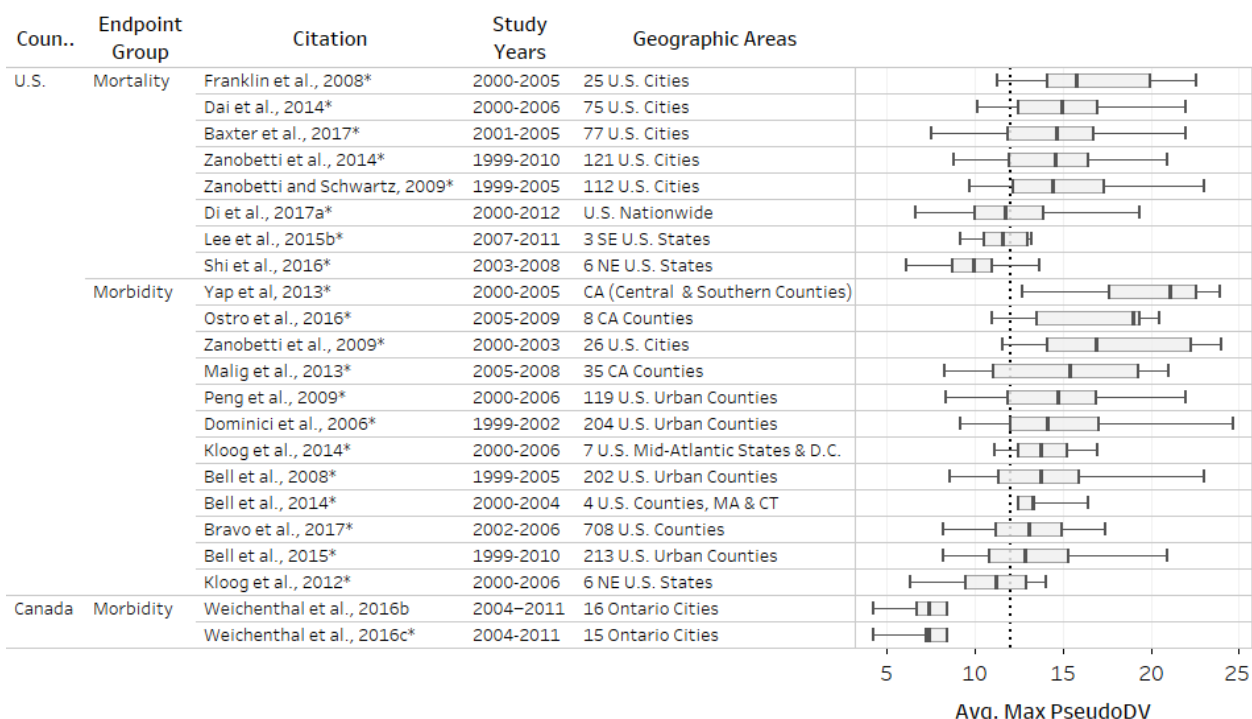
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<sup>47</sup> As discussed below, among study locations with averaged PM<sub>2.5</sub> pseudo-design values (i.e., averaged over the study period) at or below 12.0 µg/m<sup>3</sup>, almost all individual 3-year pseudo-design values are also at or below 12.0 µg/m<sup>3</sup> (i.e., 89% for Di et al. (2017b); 98% for Shi et al. (2016)— see Appendix B, section B.9).

### Long-term exposure studies



### Short-term exposure studies



**Figure 3-9. PM<sub>2.5</sub> annual pseudo-design values (in  $\mu\text{g}/\text{m}^3$ ) corresponding to various percentiles<sup>48</sup> of study area populations or health events for studies of long-term and short-term PM<sub>2.5</sub> exposures.<sup>49</sup>**

<sup>48</sup> Asterisks next to study citations denote statistically significant effect estimates.

<sup>49</sup> For most of the studies included in Figure 3-9, pseudo-design values are available for >70% of study area populations (or health events). Exceptions are Kloog et al. (2012), Lee et al. (2015), Pinault et al. (2016), and Wang et al. (2017), with pseudo-design values available for 67%, 56%, 51%, and 65% of study area populations, respectively.

Drawing from the information in Figure 3-9 (and Figure B-9 in Appendix B), we particularly note the following:

- For most of the key studies (i.e., 18 of the 29 in Figure 3-9<sup>50</sup>), about 25% or more of the study area populations (i.e., of those in areas with pseudo-design values) lived in locations with air quality likely to have met the current primary standards over study periods (or about 25% or more of health events occurred in locations with such air quality).
  - For the 15 U.S. studies included in this group, annual pseudo-design values from 8.7 to 11.9  $\mu\text{g}/\text{m}^3$  correspond to 25<sup>th</sup> percentiles of study area populations (or health events).
  - For the three Canadian studies included in this group, annual pseudo-design values from 6.0 to 7.2  $\mu\text{g}/\text{m}^3$  correspond to 25<sup>th</sup> percentiles of study area populations (or health events).
- For nine of the key studies, most of the study area population (i.e., > 50% of those living in areas with pseudo-design values) lived in locations with air quality likely to have met the current standards over study periods (or > 50% of health events occurred in locations with such air quality).
  - For the six U.S. studies included in this group, annual pseudo-design values from 9.9 to 11.7  $\mu\text{g}/\text{m}^3$  correspond to 50<sup>th</sup> percentiles of study area populations (or health events).
  - For the three Canadian studies included in this group, annual pseudo-design values from 7.3 to 7.4  $\mu\text{g}/\text{m}^3$  correspond to 50<sup>th</sup> percentiles of study area populations (or health events).
- For four of the key studies, the large majority of the study area population (i.e., >75% of those living in areas with pseudo-design values) lived in locations with air quality likely to have met the current standards over study periods (or >75% of health events occurred in locations with such air quality).
  - One of these studies (Shi et al., 2016) was conducted in the U.S. In this study, an annual pseudo-design value of 11.0  $\mu\text{g}/\text{m}^3$  corresponds to the 75<sup>th</sup> percentile of the study area population.<sup>51</sup>
  - Three of these studies (Pinault et al., 2016; Weichenthal et al., 2016c; and Weichenthal et al., 2016b) were conducted in Canada. In these studies, annual pseudo-design values from 8.4 to 8.6  $\mu\text{g}/\text{m}^3$  correspond to 75<sup>th</sup> percentiles of the study area populations (or health events).
- For the remaining 11 key studies, the large majority of the study area population (i.e., >75% of those living in areas with pseudo-design values) lived in locations with air quality likely to

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<sup>50</sup> Shi et al. (2016) separately examined long- and short-term PM<sub>2.5</sub> exposures and, therefore, is included twice in Figure 3-9 and Figure B-9.

<sup>51</sup> In Shi et al. (2016), 85% of all of the study areas with pseudo-design values would likely have met the current annual standard over the entire study period (i.e., annual pseudo-design values for every three-year period examined were  $\leq 12.0 \mu\text{g}/\text{m}^3$ ).

have violated one or both of the current standards during study periods (or >75% of health events occurred in locations with such air quality).

While the information in Figure 3-9 can inform conclusions regarding the degree to which air quality present in study locations and during study periods would likely have met the current primary PM<sub>2.5</sub> standards, there are important uncertainties to consider when using such information to inform conclusions on the primary PM<sub>2.5</sub> standards. These include the following:

- For most key multicity studies, some study locations would likely have met the current primary standards over study periods while others would likely have violated one or both standards. There is uncertainty in how to interpret such studies to inform conclusions on the NAAQS. However, the importance of this uncertainty is lessened for studies that report positive and statistically significant associations in populations that reside almost entirely in areas likely to have met the current standards (e.g., Pinault et al., 2016; Shi et al., 2016; Weichenthal et al., 2016c). This uncertainty is also lessened for key studies that report positive and statistically significant associations in analyses restricted long-term average PM<sub>2.5</sub> concentrations below 12 µg/m<sup>3</sup> (Di et al., 2017b) or 10 µg/m<sup>3</sup> (Shi et al., 2016), which account for about half of the total deaths in these studies (i.e., 54% in Di et al. (2017b), and 49% in Shi et al. (2016)). Effect estimates in these restricted analyses are slightly larger than those based on the entire cohort.
- For each study location, maximum 3-year pseudo-design values are averaged over study periods. Depending on the years of air quality evaluated by the study, for some locations those averages could reflect air quality that violated the current standards during part of the study period and met the current standards during part of the study period. However, analysis of this issue indicates that, among study locations with averaged PM<sub>2.5</sub> pseudo-design values (i.e., averaged over the study period) at or below 12.0 µg/m<sup>3</sup>, almost all individual 3-year pseudo-design values are also at or below 12.0 µg/m<sup>3</sup> (i.e., 89% for Di et al. (2017b); 98% for Shi et al. (2016)— see Appendix B, section B.9).
- Analyses identifying pseudo-design values in study locations necessarily focus on locations with at least one PM<sub>2.5</sub> monitor. While this approach can account for the large majority of study area populations for studies that use monitors alone to estimate PM<sub>2.5</sub> exposures, some recent key epidemiologic studies use hybrid modeling approaches to predict ambient PM<sub>2.5</sub> concentrations in locations with and without nearby ground-based monitors (i.e., Figure 3-8, above). For these studies, PM<sub>2.5</sub> pseudo-design values are not available for unmonitored study locations. For most of the key studies, pseudo-design values are available for locations accounting for more than 70% of the study population. However, for some studies, the percentages of study area populations living in locations with pseudo-design values are lower (Kloog et al., 2012; Lee et al., 2015; Pinault et al., 2016; Wang et al., 2017). To the extent unmonitored areas have generally lower ambient PM<sub>2.5</sub> concentrations than monitored areas, our analyses of pseudo-design values could be biased toward the higher values present in monitored locations.
- PM<sub>2.5</sub> monitoring requirements have changed since the study periods covered by key studies. In particular, PM<sub>2.5</sub> pseudo-design values during study periods do not reflect the near-road PM<sub>2.5</sub> monitors that are now required in many large urban areas (discussed in section 2.3.2.2.2 above). Had current requirements for near-road monitors been in place during study



periods, the maximum pseudo-design values in some counties could have been higher than those identified. Early data from near road monitors indicates that about half of urban areas with near-road monitors measured the highest annual design values at those monitors. Of the CBSAs with highest annual design values at near-road sites, those design values were, on average,  $0.7 \mu\text{g}/\text{m}^3$  higher than at the highest measuring non-near-road sites (range is 0.1 to  $2.0 \mu\text{g}/\text{m}^3$  higher at near-road sites) (Table 2-2 above).

The potential implications of these and other uncertainties for the primary  $\text{PM}_{2.5}$  standards are discussed in section 3.4 below.

### 3.2.3.3 Conclusions from the Evidence

In reaching conclusions based on the evidence considered in section 3.2.3, we revisit the questions posed at the beginning of the section:

- **What are the short- or long-term  $\text{PM}_{2.5}$  exposures that have been associated with health effects and to what extent does the evidence support the occurrence of such effects for air quality meeting the current primary  $\text{PM}_{2.5}$  standards?**

To answer these questions, we draw on information from experimental studies, as discussed in section 3.2.3.1, and information from epidemiologic studies, as discussed in section 3.2.3.2.

With regard to the experimental evidence, we note that available controlled human exposure and animal toxicology studies provide general support for the plausibility of many of the serious health outcomes associated with estimated  $\text{PM}_{2.5}$  exposures in epidemiologic studies (U.S. EPA, 2019, Chapters 5 to 11). However, the  $\text{PM}_{2.5}$  exposure concentrations consistently shown to elicit effects across these studies are considerably higher than the ambient concentrations typically measured in the U.S. in recent years, and higher than the concentrations likely to occur in areas meeting the current primary standards (section 3.2.3.1). A limited number of experimental studies report effects following exposures to lower  $\text{PM}_{2.5}$  concentrations (Mauad et al. (2008); Cangerana Pereira et al. (2011),<sup>52</sup> though still above typical ambient concentrations observed in locations meeting the current standards. Thus, while experimental studies support the plausibility of serious  $\text{PM}_{2.5}$ -associated health effects, these studies provide limited insight into the occurrence of effects following  $\text{PM}_{2.5}$  exposures likely to occur in the ambient air in areas meeting the current primary  $\text{PM}_{2.5}$  standards.

With regard to the epidemiologic evidence, we first note that key studies conducted in the U.S. or Canada indicate positive and often statistically significant associations between estimated

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<sup>52</sup> Mauad et al. (2008) and Cangerana Pereira et al. (2011) report respiratory and cancer-related effects, respectively, in animals following long-term exposures to  $16.8$  and  $17.7 \mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$ . Hemmingsen et al. (2015b) reports cardiovascular effects in human volunteers following 5-hour exposures to an average of  $24 \mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$ . Additionally, the controlled human exposure study by Bräuner et al. (2008) reports no change in markers of cardiovascular function following 24-hour PM exposures to an average  $\text{PM}_{2.5}$  concentration of  $10.5 \mu\text{g}/\text{m}^3$ .

PM<sub>2.5</sub> exposures (short- or long-term) and mortality or morbidity across a broad range of ambient concentrations. These include associations based on PM<sub>2.5</sub> air quality distributions lower than those in key studies from the last review.<sup>53</sup> Based on the information in Figure 3-7 and Figure 3-8, the large majority of key epidemiologic studies in the current review report health effect associations for air quality distributions characterized by overall mean PM<sub>2.5</sub> concentrations ranging from 8.1 µg/m<sup>3</sup> to 16.5 µg/m<sup>3</sup>, with mean concentrations in most of these studies (and all but one key U.S. study) at or above 9.6 µg/m<sup>3</sup>. These include studies that report associations in a wide variety of populations, including studies examining substantial portions of the U.S. population and studies examining groups that may be at comparatively high risk (e.g., older adults, children). These studies employ various study designs and examine a wide variety of health outcomes, geographic areas, approaches to estimating PM<sub>2.5</sub> exposures, and approaches to control for confounding. The evidence for associations at lower ambient concentrations (i.e., means < 8.0 µg/m<sup>3</sup>) is more limited, with two studies conducted in Ontario reporting positive associations (statistically significant in one study) for PM<sub>2.5</sub> air quality distributions characterized by overall mean concentrations around 7.0 µg/m<sup>3</sup> (Weichenthal et al., 2016c; Weichenthal et al., 2016b).

Considering the PM<sub>2.5</sub> concentrations around these overall means can provide insight into the part of the air quality distribution over which studies provide the strongest support for reported health effect associations. Evaluating whether such PM<sub>2.5</sub> air quality distributions would be likely to occur in areas meeting the current (or alternative) primary standards can inform conclusions on the degree to which those standards would limit the potential for the long- and short-term PM<sub>2.5</sub> exposures that support reported health effect associations. However, a limitation of considering study-reported mean PM<sub>2.5</sub> concentrations to inform conclusions on the primary PM<sub>2.5</sub> standards is that such concentrations, by themselves, do not indicate whether study areas would likely have met or violated the current standards (or alternatives).

As discussed above (sections 3.2.3.2.1 and 3.2.3.2.2), the EPA uses design values at individual monitors to determine whether areas meet the NAAQS. Based on analyses of recent air quality in U.S. CBSAs, maximum annual PM<sub>2.5</sub> design values for a given three-year period are often 10% to 20% higher than average concentrations over that period (i.e., averaged across monitors in the same CBSA) (Appendix B, Figure B-7 and Table B-9). These relationships suggest that areas with maximum annual PM<sub>2.5</sub> design values of 12.0 µg/m<sup>3</sup> (i.e., just meeting the current annual standard) are likely to have long-term mean PM<sub>2.5</sub> concentrations (i.e., averaged

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<sup>53</sup> In the last review key epidemiologic studies supporting “causal” or “likely to be causal” determinations examined distributions of ambient PM<sub>2.5</sub> with overall mean concentrations at or above 12.8 µg/m<sup>3</sup> (U.S. EPA, 2011, Figure 2-8).

across space and over time) that are somewhat below  $12.0 \mu\text{g}/\text{m}^3$  but still higher than the overall means reported by a number of key epidemiologic studies reporting  $\text{PM}_{2.5}$  health effect associations. This indicates that the current standards are likely to allow the distributions of short- and long-term  $\text{PM}_{2.5}$  exposures that are associated with health effects in some key studies.

Another approach to examine the potential implications of key epidemiologic studies for the primary  $\text{PM}_{2.5}$  standards is to consider analyses of  $\text{PM}_{2.5}$  pseudo-design values in locations of those studies, thereby focusing on a study-related air quality metric that is more directly comparable to the levels of the primary  $\text{PM}_{2.5}$  standards. As illustrated in Figure 3-9, and in Figure B-9 in Appendix B, for several key studies with available pseudo-design values (9 of the studies evaluated), most of the study area populations lived in locations with air quality likely to have met both the annual and 24-hour  $\text{PM}_{2.5}$  standards over study periods (or most of health events occurred in such areas). For the U.S. studies in this group, annual pseudo-design values from  $9.9$  to  $11.7 \mu\text{g}/\text{m}^3$  correspond to 50<sup>th</sup> percentiles of study area populations (or health events). That is, 50% of the study area populations lived in locations with pseudo-design values below these concentrations, or 50% of the health events occurred in such locations. For the U.S. study reporting the lowest annual average concentrations (Shi et al., 2016), 75% of the study area population lived in locations with annual pseudo-design values below  $11.0 \mu\text{g}/\text{m}^3$ . For the Canadian studies with the lowest ambient  $\text{PM}_{2.5}$  concentrations, annual pseudo-design values of about  $7.3$  to  $7.4 \mu\text{g}/\text{m}^3$  correspond to 50<sup>th</sup> percentiles of study area populations (or health events), and annual pseudo-design values from  $8.4$  to  $8.6 \mu\text{g}/\text{m}^3$  correspond to 75<sup>th</sup> percentiles.

When the information summarized above is taken together, along with the uncertainties discussed in section 3.2.3.2 above, we reach the conclusion that a number of key epidemiologic studies report positive and statistically significant  $\text{PM}_{2.5}$  health effect associations based largely, or entirely, on air quality that is likely to be allowed by the current primary  $\text{PM}_{2.5}$  standards. Our consideration of the evidence and air quality information to inform conclusions on the primary  $\text{PM}_{2.5}$  standards is discussed further in section 3.4 below.

### **3.3 RISK-BASED CONSIDERATIONS**

To inform conclusions regarding the primary  $\text{PM}_{2.5}$  standards that are “requisite” to protect the public health (i.e., neither more nor less stringent than necessary; section 1.2), it is important to consider the health risks that would be allowed under those standards. For the current standards, this means evaluating  $\text{PM}_{2.5}$ -related health risks in locations with three-year annual  $\text{PM}_{2.5}$  design values of  $12.0 \mu\text{g}/\text{m}^3$  and/or three-year 24-hour design values of  $35 \mu\text{g}/\text{m}^3$  (i.e., neither above nor below the levels of the current standards). Therefore, in addition to our evaluation of  $\text{PM}_{2.5}$  concentrations in locations of key epidemiologic studies (which are based on existing air quality; section 3.2.3.2), we use information from those studies in a risk assessment

that estimates population-level health risks associated with PM<sub>2.5</sub> air quality that has been adjusted to simulate “just meeting” the current standards (i.e., design values equal to 12.0 µg/m<sup>3</sup> and/or 35 µg/m<sup>3</sup>). Given our conclusions based on the evidence (section 3.2.3.3), we also estimate risks associated with PM<sub>2.5</sub> air quality adjusted to simulate “just meeting” alternative annual and 24-hour standards with lower levels. These risk estimates, when considered alongside analyses of the evidence discussed above in section 3.2.3, are meant to inform conclusions on the primary standards that would be requisite to protect the public health against long- and short-term PM<sub>2.5</sub> exposures. Our consideration of estimated risks focuses on addressing the following policy-relevant questions:

- **What are the estimated PM<sub>2.5</sub>-associated health risks for air quality just meeting the current primary PM<sub>2.5</sub> standards?**
- **To what extent are risks estimated to decline when air quality is adjusted to just meet potential alternative standards with lower levels?**
- **What are the uncertainties and limitations in these risk estimates?**

The sections below summarize our approach to estimating risks (section 3.3.1) and the results of the risk assessment (section 3.3.2). Additional detail on the risk assessment is provided in Appendix C.

### **3.3.1 Overview of Approach to Estimating Risks**

Our general approach to estimating PM<sub>2.5</sub>-associated health risks combines concentration-response functions from epidemiologic studies with ambient PM<sub>2.5</sub> concentrations corresponding to air quality scenarios of interest, baseline health incidence data, and population demographics for locations included in the risk assessment. Below we summarize key aspects of the risk modeling approach. Additional detail on the approach is provided in Appendix C (section C.1).

- **Study area selection:** In selecting U.S. study areas for inclusion in the risk assessment, we focus on the following characteristics:
  - *Available ambient monitors:* We focus on areas with relatively dense ambient monitoring networks, where we have greater confidence in adjustments to modeled air quality concentrations in order to simulate “just meeting” the current and alternative primary PM<sub>2.5</sub> standards (air quality adjustments are described in detail in Appendix C, section C.1.4).
  - *Geographical Diversity:* We focus on areas that represent a variety of regions across the U.S. and that include a substantial portion of the U.S. population.
  - *PM<sub>2.5</sub> air quality concentrations:* We balance the value of including a broad array of study areas from across the U.S. against the larger uncertainty associated with air quality adjustments in certain areas. For example, many areas have recent air quality that meets the current primary PM<sub>2.5</sub> standards. Inclusion of such areas in

the risk assessment necessitates an upward adjustment to PM<sub>2.5</sub> air quality concentrations in order to simulate just meeting the current standards. Given uncertainty in how such increases could potentially occur, we select areas (i.e., CBSAs<sup>54</sup>) requiring either a downward adjustment to air quality or a relatively modest upward adjustment (i.e., no more than 2.0 µg/m<sup>3</sup> for the annual standard and 5 µg/m<sup>3</sup> for the 24-hour standard, based on the 2014-2016 design-value period). In addition, as discussed further in Appendix C (section C.1.4), we excluded several areas that appeared to be strongly influenced by exceptional events. Forty-seven urban study areas met these criteria (Figure 3-10 and Appendix C, section C.1.3), including 30 study areas where just meeting the current standards is controlled by the annual standard,<sup>55</sup> 11 study areas where just meeting the current standards is controlled by the daily standard,<sup>56</sup> and 6 areas where the controlling standard differed depending on the air quality adjustment approach (Figure 3-10).<sup>57</sup>

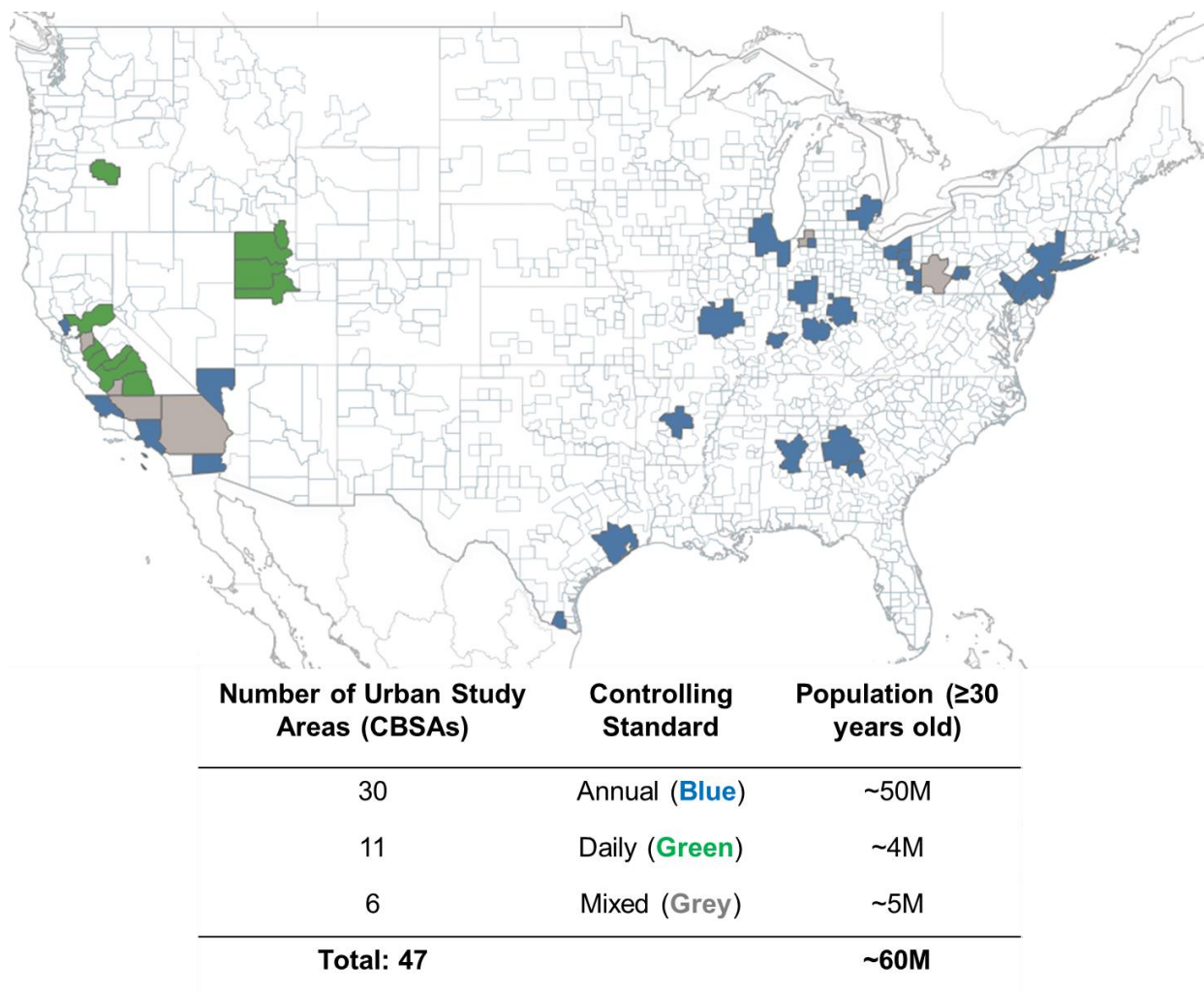
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<sup>54</sup> CBSAs (core-based statistical areas) can include one or more counties. Each CBSA selected included at least one monitor with valid design values and several CBSAs had more than 10 monitors. See Table C-3 in Appendix C.

<sup>55</sup> For these areas, the annual standard is the “controlling standard” because when air quality is adjusted to simulate just meeting the current or potential alternative annual standards, that air quality also would meet the 24-hour standard being evaluated.

<sup>56</sup> For these areas, the 24-hour standard is the controlling standard because when air quality is adjusted to simulate just meeting the current or potential alternative 24-hour standards, that air quality also would meet the annual standard being evaluated. Some areas classified as being controlled by the 24-hour standard also violate the annual standard.

<sup>57</sup> In these 6 areas, the controlling standard depended on the air quality adjustment method used and/or the standard scenarios evaluated.



**Figure 3-10. Map of 47 urban study areas included in risk modeling.**

- Health outcomes:** The health outcomes evaluated in the risk assessment are (a) total mortality (all-cause and non-accidental), ischemic heart disease mortality, and lung cancer mortality associated with long-term PM<sub>2.5</sub> exposures and (b) total mortality associated with short-term PM<sub>2.5</sub> exposures (Table 3-4 below and Appendix C, section C.1.1). Evidence for these outcomes supports “causal” or “likely to be causal” determinations in the ISA (U.S. EPA, 2019).
- Concentration-response functions:** Concentration-response functions used in this risk assessment are from large, multicity U.S. epidemiologic studies that evaluate PM<sub>2.5</sub> health effect associations (drawn from those identified above in Figures 3-3 to 3-6). The selection of specific epidemiologic studies and concentration-response functions for use in modeling risk is based on criteria that take into account factors such as study design, geographic coverage, demographic groups evaluated, and health endpoints examined. Information from these studies is summarized in Table 3-4. Additional detail regarding the selection of epidemiologic studies and specification of concentration-response functions can be found in Appendix C (section C.1.1).

**Table 3-4. Epidemiologic studies used to estimate PM<sub>2.5</sub>-associated risk.**

Epidemiology Study	Study Population <sup>a</sup>	Age Range (years)	Mortality Categories Covered
<i>Long-term mortality studies</i>			
Jerrett et al., 2016	ACS	30+	IHD
Pope et al., 2015	ACS	30+	All-cause, IHD
Turner et al., 2016	ACS	30+	Lung cancer
Thurston et al., 2016	AARP	55-85	All-cause
Di et al., 2017b	Medicare	65+	All-cause
<i>Short-term mortality</i>			
Baxter et al., 2017	77 cities	All ages	Non-accidental
Ito et al., 2013	NPACT	All ages	All cause
Zanobetti et al., 2014	121 communities	65+	All cause
<sup>a</sup> ACS (American Cancer Survey), AARP (American Association of Retired Persons), NPACT (National Particle Components Toxicity). See Appendix C Table C-1 for additional study details.			

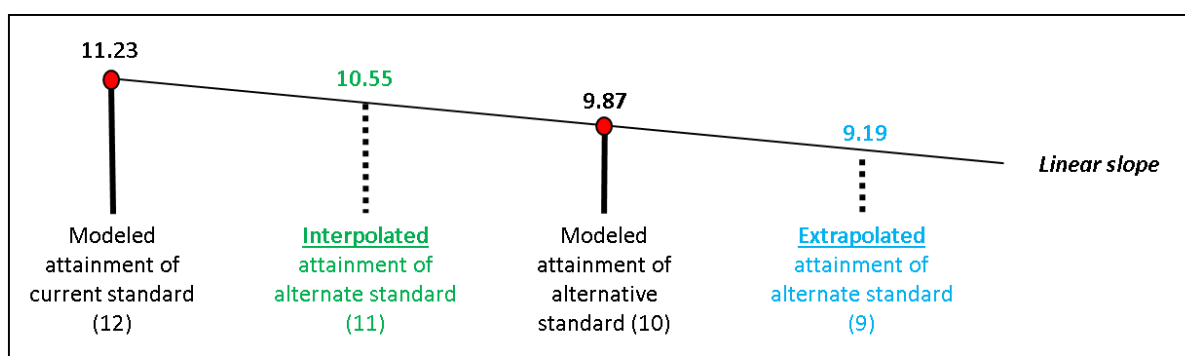
- PM<sub>2.5</sub> air quality scenarios evaluated:** We first estimate health risks associated with air quality adjusted to simulate “just meeting” the current primary PM<sub>2.5</sub> standards (i.e., the annual standard with its level of 12.0 µg/m<sup>3</sup> and the 24-hour standard with its level of 35 µg/m<sup>3</sup>). We additionally evaluate the potential for alternative annual standards with levels of 9.0, 10.0 and 11.0 µg/m<sup>3</sup> to reduce estimated risk, relative to the current standards. As discussed above (section 3.1.2), there is greater uncertainty regarding whether a revised 24-hour standard (i.e., with a lower level) would appropriately limit PM<sub>2.5</sub>-associated health risks by limiting the PM<sub>2.5</sub> concentrations that make up the middle portion of the air quality distribution (i.e., where epidemiologic studies provide the strongest support for reported associations). However, we recognize the potential for considering a revised 24-hour standard in this review (discussed below in section 3.5.2.4.2). Therefore, to provide insight into the possible public health implications of a revised 24-hour standard, we also examine an alternative 24-hour standard with a level of 30 µg/m<sup>3</sup>.<sup>58</sup>
- Model-based approach to adjusting air quality:** Air quality modeling is used to simulate just meeting the current standards and alternative standards with levels of 10.0 µg/m<sup>3</sup> (annual) and 30 µg/m<sup>3</sup> (24-hour). The air quality modeling employs a hybrid approach that combines CMAQ-modeled surfaces<sup>59</sup> and ambient monitoring data to generate ambient PM<sub>2.5</sub> estimates for 2015 on a national grid with 12-km horizontal resolution (downscaler). The modeled 2015 PM<sub>2.5</sub> concentrations were then adjusted using one of two approaches<sup>60</sup> for each air quality scenario (discussed in detail in Appendix C, section C.1.4):

<sup>58</sup> We also estimate population risks for recent (i.e., unadjusted) ambient PM<sub>2.5</sub> concentrations (Appendix C).

<sup>59</sup> <https://www.epa.gov/cmaq>

<sup>60</sup> These two modeling approaches provided sensitivity analyses on key aspects of the HHRA and are not additive.

- *Reductions in primarily-emitted PM<sub>2.5</sub> (Pri-PM)*: This approach simulates air quality scenarios of interest by preferentially adjusting modeled directly emitted PM.<sup>61</sup>
- *Reductions in secondarily produced PM<sub>2.5</sub> (Sec-PM)*: This approach simulates air quality scenarios of interest by preferentially adjusting modeled SO<sub>2</sub> and NO<sub>x</sub> precursor emissions to simulate changes in secondarily formed PM<sub>2.5</sub>.<sup>62</sup>
- **Linear interpolation/extrapolation to additional annual standard levels:** In addition to the hybrid modeling approach described above, we also employ linear interpolation and extrapolation to simulate just meeting alternative annual standards with levels of 11.0 (i.e., interpolated between 12.0 and 10.0 µg/m<sup>3</sup>) and 9.0 µg/m<sup>3</sup> (i.e., extrapolated from 12.0 and 10.0 µg/m<sup>3</sup>), respectively (illustrated in Figure 3-11). This interpolation/extrapolation was only performed for the subset of 30 urban study areas where the annual standard was controlling in all air quality scenarios evaluated.



**Figure 3-11. Illustration of approach to adjusting air quality to simulate just meeting annual standards with levels of 11.0 and 9.0 µg/m<sup>3</sup>.**

- **Characterization of variability and uncertainty in the risk estimates:** Both quantitative and qualitative methods have been used to characterize variability and uncertainty in the risk estimates (Appendix C, section C.3), including:
  - *Inclusion of 95 percent confidence intervals for risk estimates:* When modeling risk, we generate confidence intervals for each risk estimate. The confidence intervals reflect the standard error associated with the effect estimate reported in the epidemiologic study that is used to estimate risk.
  - *Sensitivity analyses:* For several of the mortality endpoints, we include a range of risk estimates reflecting epidemiology studies conducted in various populations and using a variety of study designs (e.g., differing in the methods used to estimate exposures and to control for potential confounders). We also estimate risk using two approaches to adjust air quality to simulate just meeting the current and alternative standards (i.e., Pri-PM and Sec-PM adjustment approaches).

<sup>61</sup> In locations for which air quality scenarios cannot be simulated by adjusting modeled directly emitted PM alone, modeled SO<sub>2</sub> and NO<sub>x</sub> precursor emissions are additionally adjusted to simulate changes in secondarily formed PM<sub>2.5</sub> (Appendix C, section C.1.4).

<sup>62</sup> In locations for which air quality scenarios cannot be simulated by adjusting modeled precursor emissions alone, a proportional adjustment of air quality is subsequently applied (Appendix C, section C.1.4).



- *Qualitative uncertainty assessment:* We additionally perform qualitative evaluations of the potential for key sources of uncertainty to impact the magnitude and direction of risk estimates (Appendix C, section C.3.2).

### 3.3.2 Results of the Risk Assessment

This section presents estimates of PM<sub>2.5</sub>-associated mortality risks for urban study areas (additional results are available in Appendix C, section C.2). These results are shown as point estimates with 95 percent confidence intervals for air quality adjusted to simulate just meeting the current, and potential alternative, standards. For alternative standards, we provide tables that include the total or *absolute risk*, the change in or *delta risk*, and the *percent risk reduction*.<sup>63</sup> We also quantify the *percent of baseline incidence*, which estimates the percent of total incidence (i.e., the total public health burden associated with that health effect) that is associated with ambient PM<sub>2.5</sub> exposure.<sup>64</sup> In addition to tables, we also provide figures to illustrate how risks are distributed across annual average ambient PM<sub>2.5</sub> concentrations. Figures present results for IHD mortality associated with long-term PM<sub>2.5</sub> exposures, based on the study by Jerrett et al. (2016). Additional results are presented in Appendix C (section C.2).

The sections below present risk estimates for the full set of 47 modeled urban study areas (section 3.3.2.1), the subset of 30 areas for which the annual PM<sub>2.5</sub> standard is controlling (section 3.3.2.2), and the subset of 11 areas for which the 24-hour PM<sub>2.5</sub> standard is controlling (section 3.3.2.3). Uncertainties in the risk assessment are summarized in section 3.3.2.4.

#### 3.3.2.1 Summary of Risk Estimates for 47 Urban Study Areas

Risk estimates for the 47 urban study areas are presented in Table 3-5 and Table 3-6. Table 3-5 presents absolute risk estimates for air quality just meeting the current primary PM<sub>2.5</sub> standards and alternative standards. Table 3-6 presents differences in estimated risk between air quality just meeting the current standards and air quality just meeting alternative standards. More specifically, the risk estimates presented in the column labeled “Alternative Annual Standard (10 ug/m<sup>3</sup>)” reflect the reductions estimated (compared to the current standards) in the subset of study areas for which the alternative annual standard, with a level of 10.0 ug/m<sup>3</sup>, is controlling. Risk estimates presented in the column labeled “Alternative 24-hour Standard (30 ug/m<sup>3</sup>)” reflect the reductions estimated in the subset of study areas for which the alternative 24-hour standard,

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<sup>63</sup> *Absolute risk* refers to risk associated with the full increment of exposure associated with either the current or alternative standard. Both *delta risk* and *percent risk reduction* reflect the change in risk in going from the current standard to a specific alternative standard, with delta risk referring to the change in incidence (i.e., premature PM<sub>2.5</sub>-attributable mortality) and percent risk reduction referring to the percent change when comparing risk under the current standard to risk under simulation of an alternative standard.

<sup>64</sup> In other words, the percent of the effect associated with PM<sub>2.5</sub> exposure. For example, risk results estimate that 13-14% of all IHD mortality in 2015 was associated with PM<sub>2.5</sub> exposure (Table 3-5).

with a level of 30 µg/m<sup>3</sup>, is controlling. The smaller reductions estimated for the alternative 24-hour standard reflect the smaller number of study areas controlled by the 24-hour standard and the relatively small population in those areas. Key observations from these results are summarized below.

**Table 3-5. Estimates of PM<sub>2.5</sub>-associated mortality for air quality adjusted to just meet the current or alternative standards (47 urban study areas).**

Endpoint	Study	Air quality simulation approach*	Current Standad Absolute Risk (12/35 µg/m³)	CS (12/35) % of baseline**	Alternative Standard Absolute Risk	
					Alternative Annual (10 µg/m³)	Alternative 24-hr (30 µg/m³)
Long-term exposure related mortality						
IHD	Jerrett 2016	Pri-PM	16,500 (12,600-20,300)	14.1	14,400 (11,000-17,700)	16,400 (12,500-20,000)
		Sec-PM	16,800 (12,800-20,500)	14.3	14,200 (10,900-17,500)	16,500 (12,600-20,200)
	Pope 2015	Pri-PM	15,600 (11,600-19,400)	13.3	13,600 (10,100-17,000)	15,400 (11,500-19,200)
		Sec-PM	15,800 (11,800-19,600)	13.4	13,400 (9,970-16,700)	15,600 (11,600-19,400)
All-cause	Di 2017	Pri-PM	46,200 (45,000-47,500)	8.4	40,300 (39,200-41,400)	45,700 (44,500-47,000)
		Sec-PM	46,900 (45,600-48,200)	8.5	39,700 (38,600-40,800)	46,200 (44,900-47,500)
	Pope 2015	Pri-PM	51,300 (41,000-61,400)	7.1	44,700 (35,700-53,500)	50,700 (40,500-60,700)
		Sec-PM	52,100 (41,600-62,300)	7.2	44,000 (35,100-52,700)	51,300 (41,000-61,400)
	Thurston 2015	Pri-PM	13,500 (2,360-24,200)	3.2	11,700 (2,050-21,100)	13,300 (2,330-24,000)
		Sec-PM	13,700 (2,400-24,600)	3.2	11,500 (2,010-20,700)	13,500 (2,360-24,200)
Lung cancer	Turner 2016	Pri-PM	3,890 (1,240-6,360)	8.9	3,390 (1,080-5,560)	3,850 (1,230-6,300)
		Sec-PM	3,950 (1,260-6,460)	9.1	3,330 (1,060-5,470)	3,890 (1,240-6,370)
Short-term exposure related mortality						
All cause	Baxter 2017	Pri-PM	2,490 (983-4,000)	0.4	2,160 (850-3,460)	2,460 (970-3,950)
		Sec-PM	2,530 (998-4,060)	0.4	2,120 (837-3,400)	2,490 (982-3,990)
	Ito 2013	Pri-PM	1,180 (-16-2,370)	0.2	1,020 (-14-2,050)	1,160 (-16-2,340)
		Sec-PM	1,200 (-16-2,400)	0.2	1,000 (-14-2,020)	1,180 (-16-2,370)
	Zanobetti 2014	Pri-PM	3,810 (2,530-5,080)	0.7	3,300 (2,190-4,400)	3,760 (2,500-5,020)
		Sec-PM	3,870 (2,570-5,160)	0.7	3,250 (2,160-4,330)	3,810 (2,530-5,070)

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

\*\* CS denotes the current standard.

**Table 3-6. Estimated reduction in PM<sub>2.5</sub>-associated mortality for alternative annual and 24-hour standards (47 urban study areas).**

Endpoint	Study	Air quality simulation approach*	Delta Risk		% Risk Reduction	
			CS-AS Annual Standard (10 µg/m <sup>3</sup> )**	CS-AS 24-hr Standard (30 µg/m <sup>3</sup> )**	Annual Standard (12-10)	24-hr Standard (35-30)
Long-term exposure related mortality						
IHD	Jerrett 2016	Pri-PM	2,390 (1,800-2,970)	200 (150-249)	12.6	1.1
		Sec-PM	2,870 (2,160-3,570)	266 (200-331)	15.0	1.4
	Pope 2015	Pri-PM	2,240 (1,640-2,830)	187 (137-237)	12.7	1.1
		Sec-PM	2,690 (1,970-3,400)	250 (183-315)	15.1	1.4
All-cause	Di 2017	Pri-PM	6,440 (6,260-6,630)	573 (557-589)	12.9	1.2
		Sec-PM	7,800 (7,580-8,020)	772 (750-793)	15.4	1.5
	Pope 2015	Pri-PM	7,100 (5,640-8,550)	644 (511-776)	13.0	1.2
		Sec-PM	8,630 (6,860-10,400)	828 (658-997)	15.6	1.5
	Thurston 2015	Pri-PM	1,830 (316-3,320)	168 (29-305)	13.2	1.2
		Sec-PM	2,230 (387-4,060)	209 (36-381)	15.9	1.5
Lung cancer	Turner 2016	Pri-PM	548 (170-921)	42 (13-70)	13.0	1.0
		Sec-PM	670 (208-1,120)	61 (19-102)	15.6	1.4
Short-term exposure related mortality						
All cause	Baxter 2017	Pri-PM	335 (132-537)	30 (12-48)	13.5	1.3
		Sec-PM	408 (160-654)	39 (15-62)	16.1	1.6
	Ito 2013	Pri-PM	158 (-2-317)	14 (0-29)	13.4	1.2
		Sec-PM	192 (-3-386)	18 (0-37)	16.1	1.5
	Zanobetti 2014	Pri-PM	513 (341-684)	46 (30-61)	13.4	1.2
		Sec-PM	622 (413-830)	62 (41-82)	16.0	1.6

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

\*\* CS denotes the current standard and AS denotes the alternative standard.

Drawing from the information in Table 3-5 and Table 3-6, we make the following key observations:

- Air quality adjusted to simulate just meeting the current PM<sub>2.5</sub> standards
  - Long-term PM<sub>2.5</sub> exposures are estimated to be associated with as many as 52,100 premature deaths (all-cause), including 16,800 IHD deaths and 3,950 lung cancer deaths, annually across the 47 study areas (and approximately 54 million people over the age of 30). These estimates account for approximately 3-9% of all-cause, 13-14% of IHD, and 9% of lung cancer mortality in these areas, respectively.<sup>65</sup>
  - Short-term PM<sub>2.5</sub> exposures are estimated to be associated with up to 3,870 deaths annually across the 47 study areas.
  - The approach used to adjust air quality (i.e., Pri-PM and Sec-PM) did not have a substantial impact on overall risk estimates (also see Appendix C, section C.1.4)
- Air quality adjusted to just meet potential alternative standards

<sup>65</sup> Mortality risk estimates for specific endpoints (e.g., IHD and lung cancer) are distinct subsets of total mortality.

- Compared to the current standards, risks are estimated to decrease when air quality is adjusted to just meet an alternative annual standard with a level of 10.0  $\mu\text{g}/\text{m}^3$  or an alternative 24-hour standard with a level of 30  $\mu\text{g}/\text{m}^3$  (Table 3-6).<sup>66</sup>
- Substantially larger risk reductions are estimated in the urban study areas for which the annual standard is controlling than in the study areas for which the 24-hour standard is controlling, reflecting the larger population in the study areas controlled by the annual standard.
- The approach used to adjust air quality did not have a substantial impact on estimated reductions in  $\text{PM}_{2.5}$ -associated mortality.

### 3.3.2.2 Summary of Risk Estimates for a Broader Range of Alternative Annual Standards

This section explores the potential impacts of a range of alternative annual standard levels using interpolation and extrapolation of the modeled  $\text{PM}_{2.5}$  concentrations. Table 3-7 and Table 3-8 below present mortality risk estimates for potential alternative annual standards with levels of 11.0, 10.0, and 9.0  $\mu\text{g}/\text{m}^3$ , based on the subset of 30 urban study areas for which the annual standard is controlling under all air quality scenarios evaluated. Figure 3-12 and Figure 3-13 present distributions of absolute (total) risk associated with air quality adjusted to just meet the current and alternative annual standards and the risk reductions estimated for each alternative annual standard (relative to the current standard), respectively.<sup>67</sup>

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<sup>66</sup> In most study areas, the risk reductions presented for an annual standard with a level of 10.0  $\mu\text{g}/\text{m}^3$  reflect the difference between air quality with a maximum three-year design value of 12.0  $\mu\text{g}/\text{m}^3$  and air quality with a maximum three-year design value of 10.0  $\mu\text{g}/\text{m}^3$ . Similarly, in most study areas, the risk reduction presented for a 24-hour standard with a level of 30  $\mu\text{g}/\text{m}^3$  reflects the difference between air quality with a maximum three-year design value of 35  $\mu\text{g}/\text{m}^3$  and air quality with a maximum three-year design value of 30  $\mu\text{g}/\text{m}^3$ . However, in a small number of study areas, the “starting concentration” for the annual standard are below 12.0  $\mu\text{g}/\text{m}^3$  (four study areas: Riverside-San Bernardino-Ontario, CA; Stockton-Lodi, CA; Bakersfield, CA; and Hanford-Corcoran, CA) or the starting concentration for the 24-hr standard are below 35  $\mu\text{g}/\text{m}^3$  (two study areas: Pittsburgh, PA and South Bend-Mishawaka, IN-MI:). This is because, in these areas, the controlling standard for air quality adjusted to just meet the current standards is different from the controlling standard for air quality adjusted to simulate just meeting the alternatives evaluated.

<sup>67</sup> As noted above, Figure 3-12 and Figure 3-13 present estimates of IHD mortality associated with long-term  $\text{PM}_{2.5}$  exposures, based on the study by Jerrett et al. (2016).

**Table 3-7. Estimates of PM<sub>2.5</sub>-associated mortality for the current and potential alternative annual standards in the 30 study areas where the annual standard is controlling.**

Endpoint	Study	Air quality simulation approach*	Current Standand Absolute Risk (12/35 µg/m³)	CS (12/35 µg/m3) % of baseline**	Alternative Annual Standard (absolute risk)		
					11 µg/m³	10 µg/m³	9 µg/m³
Long-term exposure related mortality							
IHD	Jerrett 2016	Pri-PM	14,300 (10,900-17,500)	14.1	13,300 (10,200-16,300)	12,300 (9,400-15,100)	11,300 (8,610-13,900)
		Sec-PM	14,600 (11,100-17,800)	14.3	13,300 (10,200-16,400)	12,100 (9,240-14,900)	10,900 (8,280-13,400)
	Pope 2015	Pri-PM	13,500 (10,100-16,800)	13.3	12,500 (9,340-15,600)	11,600 (8,620-14,500)	10,600 (7,900-13,300)
		Sec-PM	13,700 (10,200-17,000)	13.4	12,600 (9,360-15,600)	11,400 (8,480-14,200)	10,200 (7,590-12,800)
All-cause	Di 2017	Pri-PM	39,800 (38,700-40,900)	8.4	36,900 (35,900-38,000)	34,100 (33,200-35,000)	31,200 (30,400-32,100)
		Sec-PM	40,500 (39,400-41,600)	8.5	37,000 (36,000-38,000)	33,500 (32,600-34,400)	29,900 (29,100-30,800)
	Pope 2015	Pri-PM	44,200 (35,300-52,800)	7.1	41,000 (32,800-49,100)	37,800 (30,200-45,300)	34,600 (27,600-41,500)
		Sec-PM	45,000 (35,900-53,800)	7.2	41,000 (32,800-49,100)	37,100 (29,600-44,500)	33,200 (26,500-39,700)
	Thurston 2015	Pri-PM	11,600 (2,030-20,800)	3.2	10,700 (1,880-19,300)	9,900 (1,730-17,800)	9,050 (1,580-16,300)
		Sec-PM	11,800 (2,070-21,200)	3.2	10,800 (1,880-19,400)	9,710 (1,700-17,500)	8,650 (1,510-15,600)
Lung cancer	Turner 2016	Pri-PM	3,400 (1,080-5,550)	8.9	3,160 (1,010-5,170)	2,920 (927-4,790)	2,670 (847-4,400)
		Sec-PM	3,460 (1,110-5,650)	9.1	3,160 (1,010-5,180)	2,860 (908-4,700)	2,560 (809-4,210)
Short-term exposure related mortality							
All cause	Baxter 2017	Pri-PM	2,150 (846-3,440)	0.4	1,990 (784-3,190)	1,830 (721-2,930)	1,670 (658-2,680)
		Sec-PM	2,190 (862-3,510)	0.4	1,990 (785-3,190)	1,790 (707-2,880)	1,600 (630-2,560)
	Ito 2013	Pri-PM	1,010 (-14-2,040)	0.2	939 (-13-1,880)	864 (-12-1,730)	789 (-11-1,580)
		Sec-PM	1,030 (-14-2,070)	0.2	940 (-13-1,890)	847 (-11-1,700)	754 (-10-1,510)
	Zanobetti 2014	Pri-PM	3,280 (2,180-4,370)	0.7	3,040 (2,020-4,050)	2,790 (1,860-3,730)	2,550 (1,700-3,400)
		Sec-PM	3,340 (2,220-4,450)	0.7	3,040 (2,020-4,050)	2,740 (1,820-3,650)	2,440 (1,620-3,260)

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

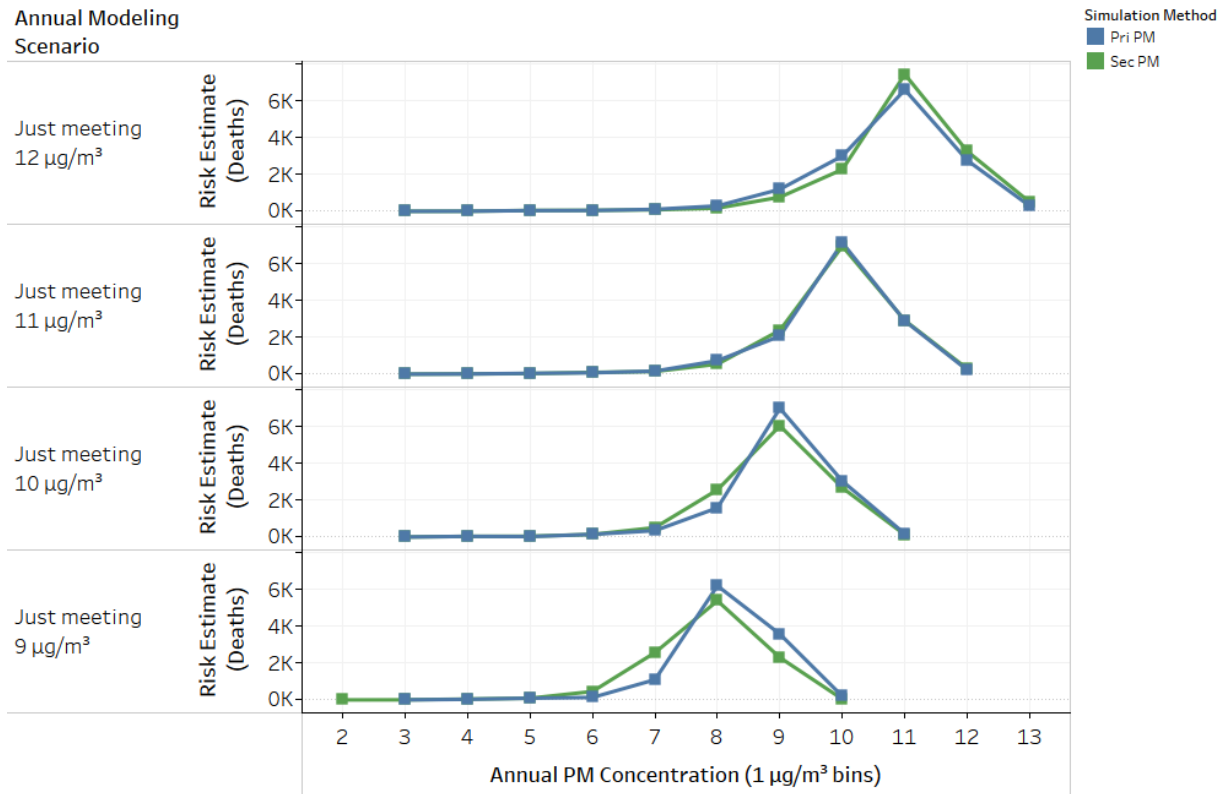
\*\* CS denotes the current standard.

**Table 3-8. Estimated delta and percent reduction in PM<sub>2.5</sub>-associated mortality for the current and potential alternative annual standards in the 30 study areas where the annual standard is controlling.**

Endpoint	Study	Air quality simulation approach*	Delta Risk (CS-AS)**			% Risk Reduction (CS-AS)**		
			12-11 µg/m³	12-10 µg/m³	12-9 µg/m³	12-11 µg/m³	12-10 µg/m³	12-9 µg/m³
Long-term exposure related mortality								
IHD	Jerrett 2016	Pri-PM	1,140 (859-1,420)	2,270 (1,710-2,830)	3,390 (2,550-4,210)	7%	14%	21%
		Sec-PM	1,400 (1,050-1,740)	2,770 (2,090-3,450)	4,130 (3,110-5,130)	8%	17%	25%
	Pope 2015	Pri-PM	1,070 (785-1,360)	2,130 (1,560-2,690)	3,180 (2,340-4,010)	7%	14%	21%
		Sec-PM	1,310 (960-1,660)	2,600 (1,910-3,280)	3,880 (2,850-4,890)	8%	17%	25%
All-cause	Di 2017	Pri-PM	3,070 (2,980-3,160)	6,120 (5,950-6,300)	9,150 (8,890-9,410)	7%	14%	21%
		Sec-PM	3,800 (3,690-3,900)	7,560 (7,340-7,770)	11,300 (11,000-11,600)	9%	17%	26%
	Pope 2015	Pri-PM	3,390 (2,690-4,080)	6,760 (5,370-8,140)	10,100 (8,030-12,200)	7%	14%	22%
		Sec-PM	4,190 (3,330-5,050)	8,350 (6,640-10,100)	12,500 (9,930-15,000)	9%	17%	26%
	Thurston 2015	Pri-PM	871 (151-1,590)	1,740 (301-3,170)	2,610 (452-4,740)	7%	15%	22%
		Sec-PM	1,080 (187-1,970)	2,160 (374-3,930)	3,230 (561-5,870)	9%	18%	27%
Lung cancer	Turner 2016	Pri-PM	262 (81-441)	522 (162-877)	780 (243-1,310)	7%	14%	21%
		Sec-PM	327 (101-550)	651 (202-1,090)	972 (303-1,630)	9%	17%	26%
Short-term exposure related mortality								
All cause	Baxter 2017	Pri-PM	160 (63-256)	319 (126-512)	478 (188-767)	7%	15%	22%
		Sec-PM	197 (78-316)	394 (155-632)	592 (233-948)	9%	18%	27%
	Ito 2013	Pri-PM	75 (-1-151)	150 (-2-302)	226 (-3-453)	7%	15%	22%
		Sec-PM	93 (-1-187)	186 (-2-374)	279 (-4-561)	9%	18%	27%
	Zanobetti 2014	Pri-PM	244 (162-325)	487 (324-650)	731 (486-975)	7%	15%	22%
		Sec-PM	301 (200-402)	603 (400-804)	904 (600-1,210)	9%	18%	27%

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

\*\* CS denotes the current standard and AS denotes the alternative standard.



**Figure 3-12. Distribution of absolute risk estimates (PM<sub>2.5</sub>-associated mortality) for the current and alternative annual standards for the subset of 30 urban study areas where the annual standard is controlling (blue and green lines represent the Pri-PM<sub>2.5</sub> and Sec-PM<sub>2.5</sub> estimates, respectively).<sup>68</sup>**

<sup>68</sup> In Figure 3-12, risk estimates are rounded toward zero into whole PM<sub>2.5</sub> concentration values (e.g., risk estimate at 10 µg/m³ includes risk occurring at 10.0-10.9 µg/m³). Risk is estimated in this figure using Jerrett et al., 2016. For each standard, a small amount of risk is estimated at concentrations higher than the level of the annual standard (e.g., some risk is estimated at an average concentration of 13 µg/m³ when air quality is adjusted to just meet the current standard). This can result because risk estimates are for a single year (i.e., 2015) within the 3-year design value period (i.e., 2014 to 2016). While the three-year average design value is 12.0 µg/m³, a single year can have grid cells with annual average concentrations above or below 12.0 µg/m³.

Annual Standard Change	Simulation Method	Annual PM Concentration (1 $\mu\text{g}/\text{m}^3$ bins)												Total
		2	3	4	5	6	7	8	9	10	11	12	13	
12-11 $\mu\text{g}/\text{m}^3$	Pri-PM	0	0	1	4	6	14	52	160	621	267	20	0	1,140
	Sec-PM	0	0	1	3	9	14	54	258	731	295	30	0	1,400
12-10 $\mu\text{g}/\text{m}^3$	Pri-PM	0	0	6	4	27	53	257	1,300	596	33	0	0	2,270
	Sec-PM	0	0	8	9	30	121	639	1,350	583	28	0	0	2,770
12-9 $\mu\text{g}/\text{m}^3$	Pri-PM	0	1	9	27	37	281	1,860	1,110	60	0	0	0	3,390
	Sec-PM	0	1	15	34	199	1,090	1,970	810	16	0	0	0	4,130

**Figure 3-13. Distribution of the difference in risk estimates between the current annual standard (level of 12.0  $\mu\text{g}/\text{m}^3$ ) and alternative annual standards with levels of 11.0, 10.0, and 9.0  $\mu\text{g}/\text{m}^3$  for the subset of 30 urban study areas where the annual standard is controlling.<sup>69</sup>**

Drawing from the information in Table 3-7, Table 3-8, Figure 3-12, and Figure 3-13, we note the following key observations:

- For air quality just meeting the current annual standard, in the subset of 30 study areas in which the annual standard is controlling, long-term  $\text{PM}_{2.5}$  exposures are estimated to be associated with as many as 45,000 total deaths and 14,600 IHD deaths annually, accounting for approximately 3-9% and 13-14% of baseline mortality, respectively. The majority of this estimated risk is associated with annual average  $\text{PM}_{2.5}$  concentrations from 10 to 12  $\mu\text{g}/\text{m}^3$  (Figure 3-12).
- Compared to the current annual standards, air quality adjusted to meet alternative annual standards with lower levels is associated with reductions in estimated IHD mortality risk across the 30 study areas (i.e., 7 to 9% reduction for a level of 11.0  $\mu\text{g}/\text{m}^3$ ; 14 to 18% reduction for a level of 10.0  $\mu\text{g}/\text{m}^3$ ; 21 to 27% reduction for a level of 9.0  $\mu\text{g}/\text{m}^3$ ) (Table 3-8 and Figure 3-12).
- The magnitude of estimated risk reduction increases as alternative annual standards with lower levels are simulated, and these estimated risk reductions are associated with lower ambient  $\text{PM}_{2.5}$  concentrations. Specifically, for air quality adjusted to simulate just meeting an annual standard with a level of 11.0  $\mu\text{g}/\text{m}^3$ , the majority of risk reduction occurs in grid cells with ambient  $\text{PM}_{2.5}$  concentrations between 9 and 11  $\mu\text{g}/\text{m}^3$ ; for air quality adjusted to simulate just meeting an annual standard with a level of 10.0  $\mu\text{g}/\text{m}^3$ , the majority of risk reduction occurs in grid cells with ambient  $\text{PM}_{2.5}$  concentrations between 8 and 10  $\mu\text{g}/\text{m}^3$ ; and for air quality adjusted to simulate just meeting an annual standard with a level of 9.0  $\mu\text{g}/\text{m}^3$ , the majority of risk reduction occurs in grid cells with ambient  $\text{PM}_{2.5}$  concentrations between 7 and 9  $\mu\text{g}/\text{m}^3$ <sup>70</sup> (Figure 3-13).

<sup>69</sup> Risks are presented as integers rounded to three significant digits and aggregated into 1  $\mu\text{g}/\text{m}^3$  bins. Bins begin at the whole number value indicated and include values up to, but not including, the next whole number (e.g., risk occurring at PM concentrations of 6.00 to 6.99 are shown in the bin at 6). Risk is estimated in this figure using Jerrett et al. (2016).

<sup>70</sup> Compared to adjusting primary  $\text{PM}_{2.5}$  emissions, adjustment of PM precursor emissions resulted in substantially larger estimated risk reductions at 7  $\mu\text{g}/\text{m}^3$ .



### 3.3.2.3 Summary of Risk Estimates for a Potential Alternative 24-Hour Standard

Table 3-9 presents risk estimates and key observations for the subset of 11 urban study areas in which the 24-hour standard controls the simulated attainment of all modeled standard levels. For air quality just meeting the current 24-hour standard, long-term PM<sub>2.5</sub> exposures are estimated to be associated with as many as 2,970 total deaths and 870 IHD deaths annually, accounting for approximately 3-8% and 12-13% of baseline mortality, respectively. Compared to the current standard, air quality just meeting an alternative 24-hour standard with a level of 30 µg/m<sup>3</sup> is associated with reductions in estimated risk of 14 to 18%.

**Table 3-9. Estimates of PM<sub>2.5</sub>-associated mortality for the current 24-hour standard, and an alternative, in the 11 study areas where the 24-hour standard is controlling.**

Endpoint	Study	Air quality simulation approach*	Current Standad Absolute Risk (12/35 µg/m³)	CS (12/35 µg/m³) % of baseline**	Alternative Standard Absolute Risk (30 µg/m³)	Delta Risk: CS-AS (daily 30 µg/m³)**	% Risk Reduction (CS-AS)*
Long-term exposure related mortality							
IHD	Jerrett 2016	Pri-PM	870 (665-1,070)	13.3	769 (586-945)	115 (87-144)	14%
		Sec-PM	862 (658-1,060)	13.1	786 (599-965)	87 (65-108)	17%
	Pope 2015	Pri-PM	820 (610-1,020)	12.5	724 (538-903)	108 (79-137)	14%
		Sec-PM	811 (604-1,010)	12.4	739 (550-922)	82 (60-103)	17%
All-cause	Di 2017	Pri-PM	2,650 (2,570-2,720)	7.7	2,320 (2,260-2,390)	348 (338-358)	14%
		Sec-PM	2,630 (2,550-2,700)	7.6	2,390 (2,330-2,460)	249 (242-256)	17%
	Pope 2015	Pri-PM	2,970 (2,370-3,560)	6.5	2,600 (2,080-3,120)	388 (308-467)	14%
		Sec-PM	2,950 (2,350-3,530)	6.4	2,680 (2,140-3,220)	279 (222-336)	17%
	Thurston 2015	Pri-PM	778 (136-1,400)	2.9	681 (119-1,230)	99 (17-181)	15%
		Sec-PM	771 (135-1,390)	2.9	701 (123-1,260)	72 (13-131)	18%
Lung cancer	Turner 2016	Pri-PM	183 (58-300)	8.4	161 (51-265)	24 (7-40)	14%
		Sec-PM	181 (58-297)	8.3	165 (52-270)	18 (6-30)	17%
Short-term exposure related mortality							
All cause	Baxter 2017	Pri-PM	142 (56-228)	0.3	124 (49-199)	18 (7-29)	15%
		Sec-PM	141 (56-226)	0.3	128 (51-206)	13 (5-21)	18%
	Ito 2013	Pri-PM	69 (-1-138)	0.1	60 (-1-120)	9 (0-18)	15%
		Sec-PM	68 (-1-137)	0.1	62 (-1-124)	6 (0-13)	18%
	Zanobetti 2014	Pri-PM	217 (145-290)	0.6	190 (126-253)	28 (18-37)	15%
		Sec-PM	216 (143-287)	0.6	196 (130-261)	20 (13-26)	18%

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

\*\* CS denotes the current standard and AS denotes the alternative standard.

### 3.3.2.4 Variability and Uncertainty in Risk Estimates

We characterize variability and uncertainty associated with risk estimates using several quantitative and qualitative approaches, as described in detail in Appendix C (section C.3). Approaches to addressing key uncertainties include the following:

- **Evaluating various effect estimates for the same health endpoint:** In some instances, the effect estimate used has only a small impact on risk estimates (i.e., IHD mortality using effect estimates from Jerrett et al., 2016) versus Pope et al., 2015), see Table 3-5). By contrast, for other mortality endpoints, such as all-cause mortality associated with long-term exposures (e.g., Di et al., 2017b) and Pope et al. (2015) versus Thurston et al., 2016)), the use of different effect estimates can have a larger impact (Table 3-5). The degree to which

different concentration-response functions result in different risk estimates could reflect differences in study design and/or study populations evaluated, as well as other factors.

- **Evaluating multiple methods for simulating air quality scenarios:** The approach used to adjust air quality (i.e., Pri-PM and Sec-PM adjustments) has little impact on overall estimates of risk (e.g., see Table 3-5). However, the adjustment approach has a larger impact on the distribution of risk reductions, particularly for the level of 9.0  $\mu\text{g}/\text{m}^3$  (Figure 3-13).
- **Characterizing the 95 percent confidence intervals associated with risk estimates:** There is considerable variation in the range of confidence intervals associated with the point estimates generated for this analysis (see Table 3-5), with some health endpoint/study combinations displaying substantially greater variability than others (e.g., short-term PM<sub>2.5</sub> exposure and all-cause mortality based on effect estimates from Ito et al. (2013) versus long-term PM<sub>2.5</sub> exposure IHD mortality estimates based on Jerrett et al. (2016)). There are a number of factors potentially responsible for the varying degrees of statistical precision in effect estimates, including sample size, exposure measurement error, degree of control for confounders/effect modifiers, and variability in PM<sub>2.5</sub> concentrations.
- **Qualitative assessment of additional sources of uncertainty:** Based in part on WHO (2008) guidance and on guidance documents developed by the EPA (U.S. EPA, 2001, U.S. EPA, 2004), we have also completed a qualitative characterization of sources of uncertainty including an assessment of both the magnitude and direction of impact of those uncertainties on risk estimates. The classification of the magnitude of impact for sources of uncertainty includes three levels: (a) low (unlikely to produce a sufficient impact on risk estimates to affect their interpretation), (b) medium (potential to have a sufficient impact to affect interpretation), and (c) high (likely to have an impact sufficient to affect interpretation). For several of the sources, we provide a classification between these levels (e.g., low-medium, medium-high).<sup>71</sup> Sources of uncertainty given at least a medium classification include the following (from Appendix C, Table C-32):<sup>72</sup>
  - **Use of air quality modeling to adjust PM<sub>2.5</sub> concentrations:** The baseline and adjusted air quality concentration fields were developed using modeling to fill spatial and temporal gaps in monitoring and explore “what if” scenarios. State-of-the-science modeling methods were used, but modeling-related biases and errors introduce uncertainty into the PM<sub>2.5</sub> concentration estimates. In addition, due to the national scale of the assessment, scenarios are based on changing modeled emissions of primary PM<sub>2.5</sub> or NO<sub>x</sub> and SO<sub>2</sub> from all anthropogenic sources throughout the U.S. by fixed percentages. Although this approach tends to target the key sources in each area, it does not tailor

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<sup>71</sup> Additional information is available in Appendix C, section C.3.

<sup>72</sup> We also identified several additional factors judged to have less than a medium classification of impact on the risk estimates generate, including: (a) the temporal mismatch between ambient air quality data characterizing exposure and mortality in long-term exposure-related epidemiology studies, (b) compositional and source differences in PM, (c) exposure measurement error in epidemiology studies assessing the relationship between mortality and exposure to ambient PM<sub>2.5</sub>, (d) lag structure in short-term exposure-related mortality epidemiology studies, and (e) assumed causal association between PM and mortality that supports modeling changes in risk associated with future changes in ambient PM<sub>2.5</sub>. See Table C-32 in Appendix C for additional discussion of these sources of uncertainty.

emission changes to specific periods or sources. The two adjustment cases span a wide range of emission conditions, but these cases are necessarily a subset of the full set of possible emission scenarios that could be used to adjust PM<sub>2.5</sub> concentrations to simulate “just meeting” standards.

- **Use of linear interpolation/extrapolation to adjust air quality:** The use of interpolation and extrapolation to simulate just meeting annual standards with levels of 11.0 and 9.0 µg/m<sup>3</sup>, respectively, does not fully capture potential non-linearities associated with real-world changes in air quality.
- **Potential confounding of the PM<sub>2.5</sub>-mortality effect:** Factors are considered potential confounders if demonstrated in the scientific literature to be related to health effects and correlated with PM<sub>2.5</sub>. Omitting potential confounders from analyses could either increase or decrease the magnitude of PM<sub>2.5</sub> effect estimates (e.g., Di et al., 2017b, Figure S2 in Supplementary Materials). Thus, not accounting for confounders can introduce uncertainty into effect estimates and, consequently, into the risk estimates generated using those effect estimates. Confounders vary according to study design, exposure duration, and health effect. For studies of short-term exposures, confounders may include meteorology (e.g., temperature, humidity), day of week, season, medication use, allergen exposure, and long-term temporal trends. For studies of long-term exposures, confounders may include socioeconomic status, race, age, medication use, smoking status, stress, noise, and occupational exposures. While various approaches to control for potential confounders have been adopted across the studies used in the risk assessment, and across the broader body of PM<sub>2.5</sub> epidemiologic studies assessed in the ISA, no individual study adjusts for all potential confounders.
- **Potential for exposure error:** Epidemiologic studies have employed a variety of approaches to estimate population-level PM<sub>2.5</sub> exposures (e.g., stationary monitors, hybrid modeling approaches). These approaches are based on using measured or predicted ambient PM<sub>2.5</sub> concentrations as surrogates for population exposures. As such, exposure estimates in epidemiologic studies are subject to exposure error. The ISA notes that, while bias in either direction can occur, exposure error tends to result in underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study by Hart et al. (2015) reports that correction for PM<sub>2.5</sub> exposure error using personal exposure information results in a moderately larger effect estimate for long-term PM<sub>2.5</sub> exposure and mortality (though with wider confidence intervals). This error in the underlying epidemiologic studies contributes to uncertainty in the risk estimates that are based on concentration-response relationships in those studies. Beyond the exposure error in epidemiologic studies themselves, the use of a different approach to represent exposures in the risk assessment (i.e., 12 x 12 km gridded surface based on modeling) could introduce additional error into risk estimates.
- **Shape of the concentration-response relationship at low ambient PM concentrations:** Interpreting the shapes of concentration-response

relationships, particularly at PM<sub>2.5</sub> concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and “linearize” population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015, section 6.c).

### **3.3.3 Conclusions from the risk assessment**

The risk assessment estimates that the current primary PM<sub>2.5</sub> standards could allow a substantial number of PM<sub>2.5</sub>-associated deaths in the U.S. For example, when air quality in the 47 study areas is adjusted to simulate just meeting the current standards, the risk assessment estimates from about 16,000 to 17,000 long-term PM<sub>2.5</sub> exposure-related deaths from ischemic heart disease in a single year (i.e., confidence intervals range from about 12,000 to 21,000 deaths). The absolute numbers of estimated PM<sub>2.5</sub>-associated deaths vary widely across exposure durations, endpoints, populations, and concentration-response functions. In addition, limitations in the underlying data and approaches (summarized above) lead to uncertainty regarding absolute estimates of PM<sub>2.5</sub>-associated risk for any given air quality scenario. However, the general magnitude of risk estimates supports the potential for significant public health impacts in locations meeting the current primary PM<sub>2.5</sub> standards. This is particularly the case given that the large majority of PM<sub>2.5</sub>-associated deaths for air quality just meeting the current standards are estimated at annual average PM<sub>2.5</sub> concentrations from about 10 to 12 µg/m<sup>3</sup>. These annual average PM<sub>2.5</sub> concentrations fall well-within the range of long-term average concentrations over which key epidemiologic studies provide strong support for reported positive and statistically significant PM<sub>2.5</sub> health effect associations.

Compared to the current annual standard, meeting a revised annual standard with a lower level is estimated to reduce PM<sub>2.5</sub>-associated health risks by about 7 to 9% for a level of 11.0 µg/m<sup>3</sup>, 14 to 18% for a level of 10.0 µg/m<sup>3</sup>, and 21 to 27% for a level of 9.0 µg/m<sup>3</sup>. As the magnitude of estimated risk reductions increases at lower levels, these estimated risk reductions are associated with lower ambient PM<sub>2.5</sub> concentrations. Specifically, for air quality adjusted to simulate just meeting an annual standard with a level of 11.0 µg/m<sup>3</sup>, the majority of risk reduction occurs at annual average PM<sub>2.5</sub> concentrations between 9 and 11 µg/m<sup>3</sup>; for air quality adjusted to simulate just meeting an annual standard with a level of 10.0 µg/m<sup>3</sup>, the majority of risk reduction occurs at PM<sub>2.5</sub> concentrations between 8 and 10 µg/m<sup>3</sup>; and for air quality adjusted to simulate just meeting an annual standard with a level of 9.0 µg/m<sup>3</sup>, the majority of risk reduction occurs at PM<sub>2.5</sub> concentrations between 7 and 9 µg/m<sup>3</sup>. Compared to a lower annual standard level, revising the level of the 24-hour standard to 30 µg/m<sup>3</sup> is estimated to

lower PM<sub>2.5</sub>-associated risks across a more limited range of areas, largely confined to areas located in the western U.S. (several of which are also likely to experience risk reductions upon meeting a revised annual standard).

### **3.4 CASAC ADVICE AND PUBLIC COMMENTS**

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards. Its advice is documented in a letter sent to the EPA Administrator (Cox, 2019). In this letter, the committee does not reach consensus on whether the scientific and technical information support retaining or revising the current annual PM<sub>2.5</sub> standard.<sup>73</sup> In particular, though the CASAC agrees that there is a long-standing body of health evidence supporting relationships between PM<sub>2.5</sub> exposures and various health outcomes, including mortality and serious morbidity effects, individual CASAC members “differ in their assessments of the causal and policy significance of these associations” (Cox, 2019, p. 8 of consensus responses). Drawing from this evidence, “some CASAC members” express support for retaining the current annual standard while “other members” express support for revising that standard in order to increase public health protection (Cox, 2019, p.1 of letter). These views are summarized below.

The CASAC members who support retaining the current annual standard express the view that substantial uncertainty remains in the evidence for associations between PM<sub>2.5</sub> exposures and mortality or serious morbidity effects. These committee members assert that “such associations can reasonably be explained in light of uncontrolled confounding and other potential sources of error and bias” (Cox, 2019, p. 8 of consensus responses). They note that associations do not necessarily reflect causal effects, and they cite recent reviews (i.e., Henneman et al., 2017; Burns et al., 2019) to support their position that in intervention studies, “reductions of PM<sub>2.5</sub> concentrations have not clearly reduced mortality risks” (Cox, 2019, p. 8 of consensus responses). These members of the CASAC additionally contend that recent epidemiologic studies reporting positive associations at lower estimated exposure concentrations mainly confirm what was anticipated or already assumed in setting the 2012 NAAQS, and that such studies do not provide new information calling into question the existing standard. Thus, they advise that, “while the data on associations should certainly be carefully considered, this data should not be interpreted more strongly than warranted based on its methodological limitations” (Cox, 2019, p. 8 of consensus responses).

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<sup>73</sup> In contrast, the CASAC reaches the consensus conclusion that the recent scientific evidence does not call into question the adequacy of the 24-hour PM<sub>2.5</sub> standard (Cox 2019, p. 11 of consensus responses).

These members of the CASAC further conclude that the PM<sub>2.5</sub> risk assessment does not provide a valid basis for revising the current standards. This conclusion is based on concerns that 1) "the risk assessment treats regression coefficients as causal coefficients with no justification or validation provided for this decision;" 2) the estimated regression concentration-response functions "have not been adequately adjusted to correct for confounding, errors in exposure estimates and other covariates, model uncertainty, and heterogeneity in individual biological (causal) [concentration-response] functions;" 3) the estimated concentration-response functions "do not contain quantitative uncertainty bands that reflect model uncertainty or effects of exposure and covariate estimation errors;" and 4) "no regression diagnostics are provided justifying the use of proportional hazards...and other modeling assumptions" (Cox, 2019, p. 9 of consensus responses). These committee members also contend that details regarding the derivation of concentration-response functions, including specification of the beta values and functional forms, are not well-documented, hampering the ability of readers to evaluate these design details. Thus, these members "think that the risk characterization does not provide useful information about whether the current standard is protective" (Cox, 2019, p. 11 of consensus responses).

Drawing from their evaluation of the evidence and the risk assessment, these committee members conclude that "the Draft PM PA does not establish that new scientific evidence and data reasonably call into question the public health protection afforded by the current 2012 PM<sub>2.5</sub> annual standard" (Cox, 2019, p.1 of letter).

In contrast, "[o]ther members of CASAC conclude that the weight of the evidence, particularly reflecting recent epidemiology studies showing positive associations between PM<sub>2.5</sub> and health effects at estimated annual average PM<sub>2.5</sub> concentrations below the current standard, does reasonably call into question the adequacy of the 2012 annual PM<sub>2.5</sub> [standard] to protect public health with an adequate margin of safety" (Cox, 2019, p.1 of letter). The committee members who support this conclusion note that the body of health evidence for PM<sub>2.5</sub> includes not only the repeated demonstration of associations in epidemiologic studies, but also includes support for biological plausibility established by human clinical and animal toxicology studies. They point to recent studies demonstrating that the associations between PM<sub>2.5</sub> and health effects occur in a diversity of locations, in different time periods, with different populations, and using different exposure estimation and statistical methods. They conclude that "the entire body of evidence for PM health effects justifies the causality determinations made in the Draft PM ISA" (Cox, 2019, p. 8 of consensus responses).

The members of the CASAC who support revising the current annual standard particularly emphasize recent findings of associations with PM<sub>2.5</sub> in areas with average long-term PM<sub>2.5</sub> concentrations below the level of the annual standard and studies that show positive associations even when estimated exposures above 12 µg/m<sup>3</sup> are excluded from analyses. They

find it “highly unlikely” that the extensive body of evidence indicating positive associations at low estimated exposures could be fully explained by confounding or by other non-causal explanations (Cox, 2019, p. 8 of consensus responses). They additionally conclude that “the risk characterization does provide a useful attempt to understand the potential impacts of alternate standards on public health risks” (Cox, 2019, p. 11 of consensus responses). These committee members conclude that the evidence available in this review reasonably calls into question the protection provided by the current primary PM<sub>2.5</sub> standards and supports revising the annual standard to increase that protection (Cox, 2019).

We also received a number of public comments on the adequacy of the current primary PM<sub>2.5</sub> standards. Some of these commenters, including several representing industry groups and states, agree with the CASAC members who conclude that the evidence supports retaining the current standards. These public commenters often cite the same types of uncertainties that are highlighted by members of the CASAC who support retaining (e.g., potential for confounding, exposure error, etc.). Other public commenters, including those representing environmental and public health organizations and several members of the academic research community, conclude that the current primary PM<sub>2.5</sub> standards should be revised in order to increase public health protection. These commenters generally cite the large body of evidence supporting relationships between PM<sub>2.5</sub> exposures and mortality or serious morbidity-related outcomes, including studies reporting such outcomes for PM<sub>2.5</sub> air quality likely to be allowed in locations meeting the current standards. They conclude that the existing body of epidemiologic studies appropriately considers potential confounders and sources of error, and that this evidence provides robust support for revising the current standards.

### **3.5 CONCLUSIONS ON THE PRIMARY PM<sub>2.5</sub> STANDARDS**

This section describes our conclusions regarding the adequacy of the current primary PM<sub>2.5</sub> standards (section 3.5.1) and regarding potential alternatives for consideration (section 3.5.2). As described more fully in section 3.1.2, our approach to reaching conclusions is based on considering the EPA’s assessment of the current scientific evidence for health effects attributable to PM<sub>2.5</sub> exposures (discussed in detail in the ISA; U.S. EPA, 2019), quantitative assessments of PM<sub>2.5</sub>-associated health risks, and analyses of PM<sub>2.5</sub> air quality. We also consider the range of advice received from the CASAC (Cox, 2019) and comments from the members of the public. These considerations and conclusions are intended to inform the Administrator’s judgments regarding primary standards for fine particles that are requisite to protect public health with an adequate margin of safety. We seek to provide as broad an array of policy options as is supportable by the available science, recognizing that the selection of a specific approach to

reaching final decisions on the primary PM<sub>2.5</sub> standards will reflect the judgments of the Administrator as to what weight to place on the various types of information.

### 3.5.1 Current Standards

We initially consider the adequacy of the current primary PM<sub>2.5</sub> standards. As discussed more fully in section 3.1.2, our approach recognizes that the current annual standard (based on arithmetic mean concentrations) and 24-hour standard (based on 98<sup>th</sup> percentile concentrations), together, are intended to protect the public health against the full distribution of short- and long-term PM<sub>2.5</sub> exposures. In considering the combined effects of these standards, we recognize that changes in PM<sub>2.5</sub> air quality designed to meet an annual standard would likely result not only in lower short- and long-term PM<sub>2.5</sub> concentrations near the middle of the air quality distribution, but also in fewer and lower short-term peak PM<sub>2.5</sub> concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98<sup>th</sup> percentile form, would result not only in fewer and lower peak 24-hour PM<sub>2.5</sub> concentrations, but also in lower annual average PM<sub>2.5</sub> concentrations. Thus, our focus in evaluating the current primary standards is on the protection provided by the combination of the annual and 24-hour standards against the distribution of both short- and long-term PM<sub>2.5</sub> exposures.

Our consideration of the adequacy of the current annual and 24-hour PM<sub>2.5</sub> standards is framed by the first overarching policy-relevant question posed at the beginning of this chapter:

- **Does the currently available scientific evidence and risk-based information support or call into question the adequacy of the public health protection afforded by the current annual and 24-hour PM<sub>2.5</sub> standards?**

In answering this question, we consider the nature of the health effects reported to occur following short- or long-term PM<sub>2.5</sub> exposures, the strength of the evidence supporting those effects, and the evidence that certain populations may be at increased risk (discussed in more detail in sections 3.2.1 and 3.2.2); the PM<sub>2.5</sub> exposures shown to cause effects and the ambient concentrations in locations where PM<sub>2.5</sub> health effect associations have been reported (section 3.2.3); estimates of PM<sub>2.5</sub>-associated health risks for air quality adjusted to simulate just meeting the current annual and 24-hour primary PM<sub>2.5</sub> standards (section 3.3); and advice from the CASAC, based on its review of the draft PA (Cox, 2019). These considerations, and our conclusions on the current primary PM<sub>2.5</sub> standards, are summarized below.

As an initial matter, we note the longstanding body of health evidence supporting relationships between PM<sub>2.5</sub> exposures (short- and long-term) and mortality or serious morbidity effects. The evidence available in this review (i.e., assessed in U.S. EPA, 2019 and summarized above in section 3.2.1) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of PM<sub>2.5</sub> exposures (U.S. EPA, 2009). Much of this evidence



comes from epidemiologic studies conducted in North America, Europe, or Asia that demonstrate generally positive, and often statistically significant, PM<sub>2.5</sub> health effect associations. Such studies report associations between estimated PM<sub>2.5</sub> exposures and non-accidental, cardiovascular, or respiratory mortality; cardiovascular or respiratory hospitalizations or emergency room visits; and other mortality/morbidity outcomes (e.g., lung cancer mortality or incidence, asthma development). Recent experimental evidence, as well as evidence from panel studies, strengthens support for potential biological pathways through which PM<sub>2.5</sub> exposures could lead to the serious effects reported in many population-level epidemiologic studies. This includes support for pathways that could lead to cardiovascular, respiratory, nervous system, and cancer-related effects.

Epidemiologic studies report PM<sub>2.5</sub> health effect associations with mortality and/or morbidity across multiple U.S. cities and in diverse populations, including in studies examining populations and lifestages that may be at comparatively higher risk of experiencing a PM<sub>2.5</sub>-related health effect (e.g., older adults, children). Such studies employ various designs and examine a variety of health outcomes, geographic areas, and approaches to controlling for confounding variables. With regard to controlling for potential confounders in particular, key studies use a wide array of approaches. Time-series studies control for potential confounders that vary over short time intervals (e.g., including temperature, humidity, dew point temperature, and day of the week) while cohort studies control for community- and/or individual-level confounders that vary spatially (e.g., including income, race, age, socioeconomic status, smoking, body mass index, and annual weather variables such as temperature and humidity) (Appendix B, Table B-12). Sensitivity analyses indicate that adding covariates to control for potential confounders can either increase or decrease the magnitude of PM<sub>2.5</sub> effect estimates, depending on the covariate, and that none of the covariates examined can fully explain the association with mortality (e.g., Di et al., 2017b, Figure S2 in Supplementary Materials). Thus, while no individual study adjusts for all potential confounders, a broad range of approaches have been adopted across studies to examine confounding, supporting the robustness of reported associations.

Available studies additionally indicate that PM<sub>2.5</sub> health effect associations are robust across various approaches to estimating PM<sub>2.5</sub> exposures and across exposure windows. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates the potential for exposure error in epidemiologic studies, such error does not call into question the fundamental findings of the broad body of PM<sub>2.5</sub> epidemiologic evidence. In fact, the ISA notes that while bias in either direction can occur, exposure error tends to lead to underestimation of health

effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study reports that correction for PM<sub>2.5</sub> exposure error using personal exposure information results in a moderately larger effect estimate for long-term PM<sub>2.5</sub> exposure and mortality (Hart et al., 2015). While most PM<sub>2.5</sub> epidemiologic studies have not employed similar corrections for exposure error, several studies report that restricting analyses to populations in close proximity to a monitor (i.e., in order to reduce exposure error) result in larger PM<sub>2.5</sub> effect estimates (e.g., Willis et al., 2003; Kloog et al., 2013). The consistent reporting of PM<sub>2.5</sub> health effect associations across exposure estimation approaches, even in the face of exposure error, together with the larger effect estimates reported in some studies that have attempted to reduce exposure error, provides further support for the robustness of associations between PM<sub>2.5</sub> exposures and mortality and morbidity.

Consistent findings from the broad body of epidemiologic studies are also supported by an emerging body of studies employing “causal inference” or quasi-experimental statistical approaches to further inform the causal nature of the relationship between long- or short-term term PM<sub>2.5</sub> exposure and mortality (U.S. EPA, 2019, sections 11.1.2.1, 11.2.2.4). These studies are summarized above in section 3.2.1.1, including a recent accountability study that reports a reduction in mortality following reductions in ambient PM<sub>2.5</sub> due to the introduction of diesel emission controls (Yorifuji et al., 2016).<sup>74</sup> Other recent studies additionally report that declines in ambient PM<sub>2.5</sub> concentrations over a period of years have been associated with decreases in mortality rates and increases in life expectancy, improvements in respiratory development, and decreased incidence of respiratory disease in children, further supporting the robustness of PM<sub>2.5</sub> health effect associations reported in the epidemiologic evidence (summarized in sections 3.2.1 to 3.2.3).

In addition to broadening our understanding of the health effects that can result from exposures to PM<sub>2.5</sub> and strengthening support for some key effects (e.g., nervous system effects, cancer), recent epidemiologic studies strengthen support for health effect associations at relatively low ambient PM<sub>2.5</sub> concentrations. Studies that examine the shapes of concentration-response functions over the full distribution of ambient PM<sub>2.5</sub> concentrations have not identified a threshold concentration, below which associations no longer exist (U.S. EPA, 2019, section 1.5.3). While such analyses are complicated by the relatively sparse data available at the lower

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<sup>74</sup> Air pollution accountability studies have reported mixed results overall (e.g., as reviewed in Burns et al., 2019 and Henneman et al., 2017). However, many of the available studies have not focused on PM<sub>2.5</sub>, were not able to attribute changes in ambient PM<sub>2.5</sub> concentrations to the interventions under evaluation, and/or were not able to disentangle health impacts of the intervention from background trends in health. The study by Yorifuji et al. (2016), included in the review by Burns et al. (2019), is an example of a study that was able to link a particular policy intervention to a decline in ambient PM<sub>2.5</sub> concentrations, and that did include a control population to correct for background trends in mortality.

end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), several studies report positive and statistically significant associations in additional analyses restricted to annual average PM<sub>2.5</sub> exposures below 12 µg/m<sup>3</sup> (Lee et al., 2015; Di et al., 2017b) and 10 µg/m<sup>3</sup> (Shi et al., 2016), or to daily exposures below 25 µg/m<sup>3</sup> (Di et al., 2017a), 30 µg/m<sup>3</sup> (Shi et al., 2016), and 35 µg/m<sup>3</sup> (Lee et al., 2015).

These and other recent studies provide support for health effect associations at lower ambient PM<sub>2.5</sub> concentrations than in previous reviews. For example, in the last review key epidemiologic studies that were conducted in the U.S. or Canada, and that supported “causal” or “likely to be causal” determinations in the ISA, reported generally positive and statistically significant associations with mortality or morbidity for PM<sub>2.5</sub> air quality distributions with overall mean concentrations at or above 12.8 µg/m<sup>3</sup> (U.S. EPA, 2011, Figure 2-8). In the current review, a large number of key studies report positive and statistically significant associations for air quality distributions with lower overall mean PM<sub>2.5</sub> concentrations (i.e., Figure 3-7 and Figure 3-8). These key studies indicate such associations consistently for distributions with long-term mean PM<sub>2.5</sub> concentrations at or above 8.1 µg/m<sup>3</sup> (8.2 µg/m<sup>3</sup> based on studies that use monitors alone to estimate PM<sub>2.5</sub> exposures), with the large majority (and all but one key U.S. study) reporting overall mean PM<sub>2.5</sub> concentrations at or above 9.6 µg/m<sup>3</sup> (10.7 µg/m<sup>3</sup> based on studies that use monitors alone). Air quality distributions with such low mean concentrations are likely to be allowed by the current PM<sub>2.5</sub> standards, based on analyses of the relationships between maximum annual PM<sub>2.5</sub> design values and annual average concentrations (i.e., averaged across multiple monitors in the same area) (section 3.2.3.2.1; Appendix B, section B.7).<sup>75</sup>

In assessing the adequacy of the current standard, we also consider what key epidemiologic studies may indicate for the current standards by calculating values similar to PM<sub>2.5</sub> design values, based on monitored air quality from the locations and time periods evaluated by those studies (i.e., section 3.2.3.2.2). This approach identifies study-relevant PM<sub>2.5</sub> air quality metrics similar to those used by the EPA to determine whether areas meet or violate the PM NAAQS. Compared to study-reported mean PM<sub>2.5</sub> concentrations, such “pseudo-design values” also have the advantage of being consistently calculated across key studies, regardless of how the studies themselves estimate PM<sub>2.5</sub> exposures (e.g., averaging across monitors, predictions from hybrid modeling approaches).

For some key studies that report positive and statistically significant PM<sub>2.5</sub> health effect associations, substantial portions of study area populations (e.g., > 50% or 75%) lived in

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<sup>75</sup> Given that the annual standard is the controlling standard across much of the U.S. (e.g., see section 3.3), the PM<sub>2.5</sub> air quality distributions that occur in most locations meeting the current annual PM<sub>2.5</sub> standard are also likely to meet the current 24-hour standard (i.e., illustrated in Chapter 2, Figure 2-11).

locations with air quality likely to have met both the current annual and 24-hour PM<sub>2.5</sub> standards over study periods (or substantial portions of health events occurred in such areas) (section 3.2.3.2.2). While there is uncertainty in interpreting analyses of PM<sub>2.5</sub> pseudo-design values (e.g., some study locations and time periods would have met the current standards while others would have violated those standards, unmonitored areas are excluded from analyses; section 3.2.3.2.2), the importance of these uncertainties is lessened for studies with the large majority of the study area population in locations with pseudo-design values well-below current standard levels (e.g., Pinault et al., 2016; Shi et al., 2016; Weichenthal et al., 2016c). This uncertainty is also lessened for key studies reporting that positive and statistically significant associations persist in analyses restricted to relatively low annual average PM<sub>2.5</sub> exposure estimates (e.g., below 12 µg/m<sup>3</sup> in Di et al., 2017b; below 10 µg/m<sup>3</sup> in Shi et al., 2016), particularly given that the excluded exposure estimates account for about half of the deaths in the entire cohort.<sup>76</sup> Thus, analyses of PM<sub>2.5</sub> pseudo-design values support the occurrence of positive and statistically significant PM<sub>2.5</sub> health effect associations based largely on air quality likely to have met the current primary standards.

In addition to the evidence, we also consider what the risk assessment indicates with regard to the adequacy of the current primary PM<sub>2.5</sub> standards. The risk assessment estimates that the current primary PM<sub>2.5</sub> standards could allow a substantial number of deaths in the U.S., with the large majority of those deaths associated with long-term PM<sub>2.5</sub> exposures. For example, when air quality in the 47 study areas is adjusted to simulate just meeting the current standards, the risk assessment estimates from about 16,000 to 17,000 PM<sub>2.5</sub>-related deaths from ischemic heart disease in a single year (i.e., for long-term exposures; confidence intervals range from about 12,000 to 21,000 deaths). While the absolute numbers of estimated PM<sub>2.5</sub>-associated deaths vary widely across exposure durations, endpoints, populations, and concentration-response functions, the general magnitude of risk estimates supports the potential for significant public health impacts in locations meeting the current primary PM<sub>2.5</sub> standards. This is particularly the case given that the large majority of PM<sub>2.5</sub>-associated deaths for air quality just meeting the current standards are estimated at annual average PM<sub>2.5</sub> concentrations from about 10 to 12 µg/m<sup>3</sup>. These annual average PM<sub>2.5</sub> concentrations fall well-within the range of long-term average concentrations over which key epidemiologic studies provide strong support for reported positive and statistically significant PM<sub>2.5</sub> health effect associations.

Based on the information summarized above, and discussed in more detail in sections 3.2 and 3.3 of this PA, we particularly note the following in reaching conclusions on the current primary PM<sub>2.5</sub> standards:

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<sup>76</sup> PM<sub>2.5</sub> effect estimates in these restricted analyses are slightly larger than in those based on the entire cohort.

- There is a long-standing body of strong health evidence demonstrating relationships between long- or short-term PM<sub>2.5</sub> exposures and a variety of outcomes, including mortality and serious morbidity effects. Studies published since the last review have reduced key uncertainties and broadened our understanding of the health effects that can result from exposures to PM<sub>2.5</sub>.
- Recent U.S. and Canadian epidemiologic studies provide support for generally positive and statistically significant health effect associations across a broad range of ambient PM<sub>2.5</sub> concentrations, including for air quality distributions with overall mean concentrations lower than in the last review and for distributions likely to be allowed by the current primary PM<sub>2.5</sub> standards.
- Analyses of PM<sub>2.5</sub> pseudo-design values additionally support the occurrence of positive and statistically significant health effect associations based largely on air quality likely to have met the current annual and 24-hour primary standards.
- The risk assessment estimates that the current primary PM<sub>2.5</sub> standards could allow a substantial number of PM<sub>2.5</sub>-associated deaths in the U.S. The large majority of these estimated deaths are associated with the annual average PM<sub>2.5</sub> concentrations near (and above in some cases) the average concentrations in key epidemiologic studies reporting positive and statistically significant health effect associations.

When taken together, we reach the conclusion that the available scientific evidence, air quality analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the combination of the current annual and 24-hour primary PM<sub>2.5</sub> standards.

In contrast to this conclusion, a conclusion that the current primary PM<sub>2.5</sub> standards do provide adequate public health protection would place little weight on the broad body of epidemiologic evidence reporting generally positive and statistically significant health effect associations, particularly for PM<sub>2.5</sub> air quality distributions likely to have been allowed by the current primary standards, or on the PM<sub>2.5</sub> risk assessment. Rather, such a conclusion would place greater weight on uncertainties and limitations in the evidence and analyses (i.e., discussed in sections 3.2.3 and 3.3.2 above), including the following:

- Uncertainty in the biological pathways through which PM<sub>2.5</sub> exposures could cause serious health effects increases as the ambient concentrations being considered fall farther below the PM<sub>2.5</sub> exposure concentrations shown to cause effects in experimental studies. In the current review, such studies generally examine the occurrence of PM<sub>2.5</sub>-attributable effects following exposures to PM<sub>2.5</sub> concentrations well-above those likely to occur in the ambient air in areas meeting the current primary PM<sub>2.5</sub> standards (i.e., discussed in section 3.2.3.1).
- Uncertainty in the potential public health impacts of air quality improvements increases as the ambient concentrations being considered fall farther below those present in studies that report improved health with reductions in PM<sub>2.5</sub> concentrations. In the current review, such

studies evaluate air quality improvements with “starting” mean PM<sub>2.5</sub> concentrations (i.e., prior to the reductions being evaluated) from about 13 to > 20 µg/m<sup>3</sup> (i.e., Table 3-3).<sup>77</sup>

- Uncertainty in the risk assessment results from uncertainties in the underlying epidemiologic studies, in the air quality adjustments, and in the application of study and air quality information to develop quantitative estimates of PM<sub>2.5</sub>-associated mortality risks (section 3.3.2.4).

The considerations and conclusions discussed above are intended to inform the Administrator’s judgments regarding the current primary PM<sub>2.5</sub> standards. In presenting these considerations and conclusions, we seek to provide information on a range of policy options, and on the potential approaches to viewing the scientific evidence and technical information that could potentially support various options. We recognize that the selection of a particular approach to reaching final decisions on the primary PM<sub>2.5</sub> standards will reflect the judgments of the Administrator as to what weight to place on the various types of evidence and information, including associated uncertainties. Given that this PA seeks to provide information on the range of policy options that could be supported by the scientific information, and given our conclusion (noted above) that the evidence and information can reasonably be viewed as calling into question the adequacy of the current primary PM<sub>2.5</sub> standards, in the next section we additionally consider support for potential alternative standards.

### 3.5.2 Potential Alternative Standards

In this section, we consider the potential alternative primary PM<sub>2.5</sub> standards that could be supported by the evidence and quantitative information available in this review. These considerations are framed by the following overarching policy-relevant question, posed at the beginning of this chapter:

- **What is the range of potential alternative standards that could be supported by the available scientific evidence and risk-based information to increase public health protection against short- and long-term fine particle exposures?**

In answering this question, we consider each of the elements of the annual and 24-hour PM<sub>2.5</sub> standards: indicator, averaging time, form, and level. The sections below discuss our consideration of these elements, and our conclusions that (1) it is appropriate to consider revising the level of the current annual standard, in conjunction with retaining the current indicator, averaging time, and form of that standard, to increase public health protection against fine

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<sup>77</sup> As noted above, these retrospective studies tend to include data from earlier time periods where ambient PM<sub>2.5</sub> concentrations in the U.S. were considerably higher than they are at present.

particle exposures and (2) depending on the decision made on the annual standard, consideration could be given to either retaining or revising the level of the 24-hour PM<sub>2.5</sub> standard.

### **3.5.2.1 Indicator**

In initially setting standards for fine particles in 1997, the EPA concluded it was appropriate to control fine particles as a group, rather than singling out any particular component or class of fine particles. The Agency noted that community health studies had found significant health effect associations using various indicators of fine particles, and that health effects in a large number of areas had significant mass contributions from differing components or sources of fine particles. In addition, a number of toxicological and controlled human exposure studies had reported health effects following exposures to high concentrations of numerous fine particle components (62 FR 38667, July 18, 1997). In establishing a size-based indicator in 1997 to distinguish fine particles from particles in the coarse mode, the EPA noted that the available epidemiologic studies of fine particles were based largely on PM<sub>2.5</sub> mass. The selection of a 2.5 µm size cut additionally reflected the regulatory importance of defining an indicator that would more completely capture fine particles under all conditions likely to be encountered across the U.S. and the monitoring technology that was generally available (62 FR 38666 to 38668, July 18, 1997).

Since the 1997 review, studies that evaluate fine particle-related health effects continue to provide strong support for such effects using PM<sub>2.5</sub> mass as the metric for fine particle exposures. Subsequent reviews have recognized the strength of this evidence, concluding that it has continued to support a PM<sub>2.5</sub> mass-based indicator for a standard meant to protect against fine particle exposures. In the last review, some studies had additionally examined health effects of exposures to particular sources or components of fine particles, or to the ultrafine fraction of fine particles. Based on limitations in such studies, together with the continued strong support for effects of PM<sub>2.5</sub> exposures, the Agency retained PM<sub>2.5</sub> mass as the indicator for fine particles and did not supplement the PM<sub>2.5</sub> standards with standards based on particle composition or on the ultrafine fraction (78 FR 3123, January 15, 2013).

As in the last review, studies available in the current review continue to provide strong support for health effects following long- and short-term PM<sub>2.5</sub> exposures (U.S. EPA, 2019). While some studies evaluate the health effects of particular sources of fine particles, or of particular fine particle components, evidence from these studies does not identify any one source or component that is a better predictor of health effects than PM<sub>2.5</sub> mass (U.S. EPA, 2019, section 1.5.4). The ISA specifically notes that the results of recent studies confirm and further support the conclusion of the 2009 ISA that many PM<sub>2.5</sub> components and sources are associated with health effects, and the evidence does not indicate that any one source or component is

consistently more strongly related with health effects than PM<sub>2.5</sub> mass (U.S. EPA, 2019, section 1.5.4). In addition, the evidence for health effects following exposures specifically to the ultrafine fraction of fine particles continues to be far more limited than the evidence for PM<sub>2.5</sub> mass as a whole. As discussed in the ISA, the lack of a consistent UFP definition in health studies and across disciplines, together with the variety of approaches to administering and measuring UFP in those studies, contribute to such limitations (U.S. EPA, 2019, section 1.4.3). Thus, for reasons similar to those discussed in the last review (78 FR 3121 to 3123, January 15, 2013), we conclude that the available information continues to support the PM<sub>2.5</sub> mass-based indicator and remains too limited to support a distinct standard for any specific PM<sub>2.5</sub> component or group of components, and too limited to support a distinct standard for the ultrafine fraction.

### **3.5.2.2 Averaging Time**

In 1997, the EPA initially set an annual PM<sub>2.5</sub> standard to protect against health effects associated with both long- and short-term PM<sub>2.5</sub> exposures, and a 24-hour standard to supplement the protection afforded by the annual standard (62 FR 38667 to 38668, July 18, 1997). In subsequent reviews, the EPA retained both annual and 24-hour averaging times, largely reflecting the strong evidence for health effects associated with annual and daily PM<sub>2.5</sub> exposure estimates (71 FR 61164, October 17, 2006; 78 FR 3123 to 3124, January 15, 2013).

In the current review, epidemiologic and controlled human exposure studies have examined a variety of PM<sub>2.5</sub> exposure durations. Epidemiologic studies continue to provide strong support for health effects associated with both long- and short-term PM<sub>2.5</sub> exposures based on annual (or multiyear) and 24-hour PM<sub>2.5</sub> averaging periods, respectively.

With regard to short-term exposures in particular, a smaller number of epidemiologic studies examine associations between sub-daily PM<sub>2.5</sub> exposures and respiratory effects, cardiovascular effects, or mortality. Compared to 24-hour PM<sub>2.5</sub> exposure estimates, associations with sub-daily estimates are less consistent and, in some cases, smaller in magnitude (U.S. EPA, 2019, section 1.5.2.1). In addition, studies of sub-daily exposures typically examine subclinical effects, rather than the more serious population-level effects that have been reported to be associated with 24-hour exposures (e.g., mortality, hospitalizations). Taken together, the ISA concludes that epidemiologic studies do not indicate sub-daily averaging periods are more closely associated with health effects than the 24-hour average exposure metric (U.S. EPA, 2019, section 1.5.2.1).

Additionally, while recent controlled human exposure studies provide consistent evidence for cardiovascular effects following PM<sub>2.5</sub> exposures for less than 24 hours (i.e., < 30 minutes to 5 hours), exposure concentrations in these studies are well-above the ambient concentrations typically measured in locations meeting the current standards (section 3.2.3.1). Thus, these



studies also do not suggest the need for additional protection against sub-daily PM<sub>2.5</sub> exposures, beyond that provided by the current primary standards.

Drawing from the evidence assessed in the ISA, and the observations noted above, we reach the conclusion that the available evidence continues to provide strong support for consideration of retaining the current annual and 24-hour averaging times. The available evidence suggests that PM<sub>2.5</sub> standards with these averaging times, when coupled with appropriate forms and levels, can protect against the range of long- and short-term PM<sub>2.5</sub> exposures that have been associated with health effects. Thus, as in the last review, the currently available evidence does not support considering alternatives to the annual and 24-hour averaging times for standards meant to protect against long- and short-term PM<sub>2.5</sub> exposures.

### **3.5.2.3 Form**

The form of a standard defines the air quality statistic that is to be compared to the level in determining whether an area attains that standard. As in other recent reviews, our foremost consideration in reaching conclusions on form is the adequacy of the public health protection provided by the combination of the form and the other elements of the standard.

As noted above, in 1997 the EPA initially set an annual PM<sub>2.5</sub> standard to protect against health effects associated with both long- and short-term PM<sub>2.5</sub> exposures and a 24-hour standard to provide supplemental protection, particularly against the short-term exposures to “peak” PM<sub>2.5</sub> concentrations that can occur in some areas (62 FR 38667 to 38668, July 18, 1997). The EPA established the form of the annual PM<sub>2.5</sub> standard as an annual arithmetic mean, averaged over 3 years, from single or multiple community-oriented monitors. That is, the level of the annual standard was to be compared to measurements made at each community-oriented monitoring site or, if specific criteria were met, measurements from multiple community-oriented monitoring sites could be averaged together (i.e., spatial averaging) (62 FR 38671 to 38672, July 18, 1997). In the 1997 review, the EPA also established the form of the 24-hour PM<sub>2.5</sub> standard as the 98<sup>th</sup> percentile of 24-hour concentrations at each monitor within an area (i.e., no spatial averaging), averaged over three years (62 FR at 38671 to 38674, July 18, 1997). In the 2006 review, the EPA retained these standard forms but tightened the criteria for using spatial averaging with the annual standard (78 FR 3124, January 15, 2013).<sup>78</sup>

In the last review, the EPA’s consideration of the form of the annual PM<sub>2.5</sub> standard again included a focus on the issue of spatial averaging. An analysis of air quality and population demographic information indicated that the highest PM<sub>2.5</sub> concentrations in a given area tended

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<sup>78</sup> Specifically, the Administrator revised spatial averaging criteria such that “(1) [t]he annual mean concentration at each site shall be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.9 for each calendar quarter (71 FR 61167, October 17, 2006).

to be measured at monitors in locations where the surrounding populations were more likely to live below the poverty line and to include larger percentages of racial and ethnic minorities (U.S. EPA, 2011, p. 2-60). Based on this analysis, the PA concluded that spatial averaging could result in disproportionate impacts in minority populations and populations with lower SES. The Administrator concluded that public health would not be protected with an adequate margin of safety in all locations, as required by law, if disproportionately higher PM<sub>2.5</sub> concentrations in low income and minority communities were averaged together with lower concentrations measured at other sites in a large urban area. Therefore, she concluded that the form of the annual PM<sub>2.5</sub> standard should be revised to eliminate spatial averaging provisions (78 FR 3124, January 15, 2013).

In the last review, the EPA also considered the form of the 24-hour PM<sub>2.5</sub> standard. The Agency recognized that the existing 98<sup>th</sup> percentile form for the 24-hour standard was originally selected to provide a balance between limiting the occurrence of peak 24-hour PM<sub>2.5</sub> concentrations and identifying a stable target for risk management programs. Updated air quality analyses in the last review provided additional support for the increased stability of the 98<sup>th</sup> percentile PM<sub>2.5</sub> concentration, compared to the 99<sup>th</sup> percentile (U.S. EPA, 2011, Figure 2-2, p. 2-62). Thus, the Administrator concluded that it was appropriate to retain the 98<sup>th</sup> percentile form for the 24-hour PM<sub>2.5</sub> standard (78 FR 3127, January 15, 2013).

Nothing in the evidence that has become available since the last review calls into question the current forms of the annual and 24-hour PM<sub>2.5</sub> standards. As discussed above (section 3.2.3.2), epidemiologic studies continue to provide strong support for health effect associations with both long-term (e.g., annual or multi-year) and short-term (e.g., mostly 24-hour) PM<sub>2.5</sub> exposures. These studies provide the strongest support for such associations for the part of the air quality distribution corresponding to the bulk of the underlying data, typically around the overall mean concentrations reported (section 3.2.3.2.1). The form of the current annual standard (i.e., arithmetic mean, averaged over three years) remains appropriate for targeting protection against the annual and daily PM<sub>2.5</sub> exposures around these means of the PM<sub>2.5</sub> air quality distribution. In addition, controlled human exposure studies provide evidence for health effects following single short-term PM<sub>2.5</sub> exposures near the peak concentrations measured in the ambient air (section 3.2.3.1). Thus, the evidence also supports retaining a standard focused on providing supplemental protection against short-term peak exposures. Nothing in the evidence that has become available since the last review calls into question the decision to use a 98<sup>th</sup> percentile form for a 24-hour standard that is meant to provide a balance between limiting the occurrence of such peak 24-hour PM<sub>2.5</sub> concentrations and identifying a stable target for risk management programs. Thus, when the information summarized above is taken together, we reach the conclusion that it is appropriate in the current review to consider

retaining the forms of the current annual and 24-hour PM<sub>2.5</sub> standards, in conjunction with a revised level as discussed below.

#### 3.5.2.4 Level

With regard to level, we specifically address the following policy-relevant question:

- **For primary PM<sub>2.5</sub> standards defined in terms of the current averaging times and forms, what potential alternative levels are appropriate to consider in order to increase public health protection against long- and short-term exposures to PM<sub>2.5</sub> in ambient air?**

In answering this question, we consider key epidemiologic studies that evaluate associations between PM<sub>2.5</sub> air quality distributions and mortality or morbidity, controlled human exposure studies examining effects following short-term PM<sub>2.5</sub> exposures, air quality analyses that help to place these studies into a policy-relevant context, and the risk assessment estimates of PM<sub>2.5</sub>-associated mortality under various alternative standard scenarios.

As discussed above in section 3.1.2, consideration of the evidence and analyses, as summarized in this chapter, informs our evaluation of the public health protection that could be provided by alternative annual and 24-hour standards with revised levels. There are various ways to combine an annual standard (based on arithmetic mean concentrations) and a 24-hour standard (based on 98<sup>th</sup> percentile concentrations), to achieve an appropriate degree of public health protection. In particular, as noted in section 3.1.2, we recognize that changes in PM<sub>2.5</sub> air quality designed to meet an annual standard would likely result not only in lower short- and long-term PM<sub>2.5</sub> concentrations near the middle of the air quality distribution (i.e., around the mean of the distribution), but also in fewer and lower short-term peak PM<sub>2.5</sub> concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98<sup>th</sup> percentile form, would result not only in fewer and lower peak 24-hour PM<sub>2.5</sub> concentrations, but also in lower average PM<sub>2.5</sub> concentrations.

However, while either standard could be viewed as providing some measure of protection against both average exposures and peak exposures, the 24-hour and annual standards are not expected to be equally effective at limiting both types of exposures. Specifically, the 24-hour standard (with its 98<sup>th</sup> percentile form) is more directly tied to short-term peak PM<sub>2.5</sub> concentrations, and thus more likely to appropriately limit exposures to such concentrations, than to the more typical concentrations that make up the middle portion of the air quality distribution. Therefore, compared to a standard that is directly tied to the middle of the air quality distribution, the 24-hour standard is less likely to appropriately limit the “typical” daily and annual exposures that are most strongly associated with the health effects observed in epidemiologic studies. In contrast, the annual standard, with its form based on the arithmetic mean concentration, is more likely to effectively limit the PM<sub>2.5</sub> concentrations that comprise the middle portion of the air

quality distribution, affording protection against the daily and annual PM<sub>2.5</sub> exposures that strongly support associations with the most serious PM<sub>2.5</sub>-related effects in epidemiologic studies (e.g., mortality, hospitalizations).

For these reasons, as discussed in section 3.1.2, we focus on alternative levels of the annual PM<sub>2.5</sub> standard as the principle means of providing increased public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures, and thus protecting against the exposures that provide strong support for associations with mortality and morbidity in key epidemiologic studies. We additionally consider the 24-hour standard, with its 98<sup>th</sup> percentile form, primarily as a means of providing supplemental protection against the short-term exposures to peak PM<sub>2.5</sub> concentrations that can occur in some areas (e.g., those with strong contributions from local or seasonal sources), even when overall mean PM<sub>2.5</sub> concentrations remain relatively low.

To inform our consideration of potential alternative annual and 24-hour standard levels, we specifically note the following key observations regarding (1) the overall mean PM<sub>2.5</sub> concentrations reported in U.S. or Canadian epidemiologic studies, (2) the relationships between long-term mean PM<sub>2.5</sub> concentrations and annual design values in U.S. CBSAs, (3) the PM<sub>2.5</sub> pseudo-design values in study locations, (4) the PM<sub>2.5</sub> exposures shown to cause effects in controlled human exposure studies, and (5) estimated PM<sub>2.5</sub>-associated risks.

***(1) Long-Term Mean PM<sub>2.5</sub> Concentrations in Key Epidemiologic Studies (section 3.2.3.2)***

- Key epidemiologic studies indicate consistently positive and statistically significant health effect associations based on air quality distributions with overall long-term mean PM<sub>2.5</sub> concentrations at and above 8.1 µg/m<sup>3</sup> (8.2 µg/m<sup>3</sup> based on studies that use monitors alone to estimate PM<sub>2.5</sub> exposures), with mean concentrations at or above 9.6 µg/m<sup>3</sup> in most key studies (10.7 µg/m<sup>3</sup> based on studies that use monitors alone to estimate PM<sub>2.5</sub> exposures). The ranges of ambient PM<sub>2.5</sub> concentrations accounting for the bulk of exposures and health data in these studies are expected to extend at least somewhat below the overall long-term mean concentrations reported.
- Epidemiologic studies provide more limited support for health effect associations based on air quality distributions with lower overall mean PM<sub>2.5</sub> concentrations. Specifically, two key studies report positive associations between short-term PM<sub>2.5</sub> exposures and emergency room visits based on cities in Ontario, Canada (Weichenthal et al., 2016b and Weichenthal et al., 2016c and), with overall mean PM<sub>2.5</sub> concentrations around 7.0 µg/m<sup>3</sup> (one of these studies reports an association that is statistically significant). Additionally, a U.S. study (Shi et al. (2016) reports positive and statistically significant associations in analyses restricted to relatively low annual or 24-hour PM<sub>2.5</sub> exposure estimates. This study does not report the overall mean PM<sub>2.5</sub> concentrations in restricted analyses, though such means are presumably somewhat below those based on the overall cohort (i.e., 8.1 and 8.2 µg/m<sup>3</sup>).

**(2) *Relationships between long-term mean PM<sub>2.5</sub> concentrations and annual design values (section 3.2.3.3; Appendix B, section B.7)***

- Areas meeting a particular annual PM<sub>2.5</sub> standard would be expected to have average PM<sub>2.5</sub> concentrations (i.e., averaged across the area and over time) somewhat below the level of that standard. This is supported by analyses of monitoring data in CBSAs across the U.S., which show that maximum annual PM<sub>2.5</sub> design values are often 10% to 20% higher than long-term mean PM<sub>2.5</sub> concentrations (Appendix B, Figure B-7; Table B-9).

**(3) *PM<sub>2.5</sub> Pseudo-Design Values in Study Locations (section 3.2.3.2.2 and Appendix B, Figure B-9)***

- For most key epidemiologic studies with PM<sub>2.5</sub> pseudo-design values available, about 25% or more of study area populations lived in locations likely to have met the current primary PM<sub>2.5</sub> standards over study periods (or about 25% or more of health events occurred in such locations). For the U.S. studies in this group, annual pseudo-design values as low as 8.7 µg/m<sup>3</sup> correspond to 25<sup>th</sup> percentiles of study area population (or health events). For the smaller number of Canadian studies included in this group, annual pseudo-design values as low as 6.0 µg/m<sup>3</sup> correspond to the 25<sup>th</sup> percentiles of study area population (or health events).
- For several key epidemiologic studies, most of the study area populations (i.e., >50% of those living in areas with pseudo-design values) lived in locations with air quality likely to have met both standards over study periods (or >50% of health events occurred in locations with such air quality). For the U.S. studies in this group, annual pseudo-design values from 9.9 to 11.7 µg/m<sup>3</sup> correspond to 50<sup>th</sup> percentiles of study area populations (or health events). For the smaller number of Canadian studies included in this group, annual pseudo-design values from 7.3 to 7.4 µg/m<sup>3</sup> correspond to 50<sup>th</sup> percentiles of study area populations (or health events).
- For the U.S. study reporting the lowest annual average concentrations (Shi et al., 2016), an annual pseudo-design value of 11.0 µg/m<sup>3</sup> corresponds to the 75<sup>th</sup> percentile of the study area population (i.e., 75% of the study area population lives in locations with pseudo-design values < 11.0 µg/m<sup>3</sup>). For the Canadian studies with the lowest ambient PM<sub>2.5</sub> concentrations, annual pseudo-design values from 8.4 to 8.6 µg/m<sup>3</sup> correspond to 75<sup>th</sup> percentiles of the study area populations (or health events).

**(4) *PM<sub>2.5</sub> exposures shown to cause effects in controlled human exposure studies (section 3.2.3.1)***

- While controlled human exposure studies support the plausibility of the serious cardiovascular effects that have been linked with ambient PM<sub>2.5</sub> exposures (U.S. EPA, 2019, Chapter 6), the PM<sub>2.5</sub> exposure concentrations evaluated in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards (and thus well-above those likely to be measured in locations that would meet revised standards with lower annual or 24-hour levels).

**(5) *PM<sub>2.5</sub>-Associated Risk Estimates (section 3.3)***

- The risk assessment estimates that, compared to the current standards, potential alternative annual standards with levels from 11.0 down to 9.0  $\mu\text{g}/\text{m}^3$  could reduce  $\text{PM}_{2.5}$ -associated mortality broadly across the U.S., including in most of the 47 urban study areas evaluated. In such locations, estimated risk reductions range from about 7 to 9% for a level of 11.0  $\mu\text{g}/\text{m}^3$ , 14 to 18% for a level of 10.0  $\mu\text{g}/\text{m}^3$ , and 21 to 27% for a level of 9.0  $\mu\text{g}/\text{m}^3$ . For each of these standards, most of the risk remaining is estimated at annual average  $\text{PM}_{2.5}$  concentrations that fall somewhat below the standard level.
- Risk reductions estimated for an alternative 24-hour standard with a level of 30  $\mu\text{g}/\text{m}^3$  are concentrated in only a few study areas in the western U.S. (several of which could also experience risk reductions in response to a revised annual standard with a level below 12.0  $\mu\text{g}/\text{m}^3$ ). In those few study areas for which risk reductions are estimated upon just meeting an alternative 24-hour standard with a level of 30  $\mu\text{g}/\text{m}^3$ , reductions range from about 14 to 18%.

The information summarized in these key observations could support various decisions on the levels of the annual and 24-hour  $\text{PM}_{2.5}$  standards, depending on the weight given to different aspects of the evidence, air quality and risk information, including its uncertainties. As noted above (section 3.1.2), in this PA we seek to provide as broad an array of policy options as is supportable by the available evidence and quantitative information, recognizing that the selection of a specific approach to reaching final decisions on the primary  $\text{PM}_{2.5}$  standards will reflect the judgments of the Administrator as to what weight to place on the various types of evidence and information, and on associated uncertainties. Potential approaches to considering support for particular alternative annual and 24-hour standard levels are discussed below.

#### **3.5.2.4.1 Alternative Annual Standard Levels**

As discussed above, the degree to which particular alternative annual standard levels below 12.0  $\mu\text{g}/\text{m}^3$  are supported will depend on the weight placed on various aspects of the scientific evidence, air quality and risk information, and its associated uncertainties. For example, a level as low as about 10.0  $\mu\text{g}/\text{m}^3$  could be supported to the extent weight is placed on the following:

- Setting a standard expected to maintain the  $\text{PM}_{2.5}$  air quality distribution below those present in most key epidemiologic studies, recognizing that (1) the large majority of key studies reporting positive and statistically significant health effect associations (and all but one key U.S. study) examine distributions of ambient  $\text{PM}_{2.5}$  with overall mean concentrations at or above 9.6  $\mu\text{g}/\text{m}^3$ , while a few studies reporting such associations examine distributions with overall mean concentrations just above 8.0  $\mu\text{g}/\text{m}^3$  (section 3.2.3.2.1) and (2) analyses of  $\text{PM}_{2.5}$  air quality in CBSAs indicate that maximum annual  $\text{PM}_{2.5}$  design values are often 10% to 20% higher than average  $\text{PM}_{2.5}$  concentrations (i.e., averaged across space and over several years) suggesting that areas meeting a particular annual  $\text{PM}_{2.5}$  standard would be expected to have average  $\text{PM}_{2.5}$  concentrations somewhat below the level of that standard (section 3.2.3.2.2; Appendix B, section B.7);

- Setting the standard level at or below the pseudo-design values corresponding to about the 50<sup>th</sup> percentiles of study area populations (or health events) in most key studies (particularly key U.S. studies), recognizing that a revised annual standard with a level as low as 10.0  $\mu\text{g}/\text{m}^3$  would be expected to maintain ambient  $\text{PM}_{2.5}$  concentrations below the concentrations present during study periods for most of those populations (or below the concentrations in locations accounting for most health events) (section 3.2.3.2.2);
- Setting a standard estimated to reduce  $\text{PM}_{2.5}$ -associated health risks, such that a substantial portion of the risk reduction is estimated at annual average  $\text{PM}_{2.5}$  concentrations  $\geq$  about 8  $\mu\text{g}/\text{m}^3$  and recognizing that these concentrations are within the range of overall means for which key epidemiologic studies indicate consistently positive and statistically significant health effect associations (section 3.3.2).

In selecting a particular level from 10.0  $\mu\text{g}/\text{m}^3$  to < 12.0  $\mu\text{g}/\text{m}^3$ , consideration of the evidence could take into account individual study characteristics such as study design and statistical approaches, precision of reported associations, study size and location, and uncertainties in the study itself or in our analyses of study area air quality. For example, if less weight is placed on the small number of studies reporting overall mean concentrations below 9.6  $\mu\text{g}/\text{m}^3$  and on the small number of studies with 50<sup>th</sup> percentile pseudo-design values below 10.0  $\mu\text{g}/\text{m}^3$ , a standard higher than 10  $\mu\text{g}/\text{m}^3$  (but still below 12.0  $\mu\text{g}/\text{m}^3$ ) might be considered. Similarly, consideration of the risk assessment could take into account the magnitude of estimated risk reductions, compared to the current standards; the annual average  $\text{PM}_{2.5}$  concentrations at which those reductions are estimated to occur; and the uncertainties in the underlying epidemiologic studies, in the air quality adjustments, or in other information that was used to model risks. For example, concern about the uncertainty in the potential public health importance of risk reductions estimated for a level as low as 10.0  $\mu\text{g}/\text{m}^3$ , much of which is estimated at annual average  $\text{PM}_{2.5}$  concentrations around 8  $\mu\text{g}/\text{m}^3$ , might focus consideration on a standard level above 10  $\mu\text{g}/\text{m}^3$ , where estimated risk reductions would occur at slightly higher concentrations.

A decision to not consider annual standard levels below 10.0  $\mu\text{g}/\text{m}^3$  might take into account the increasing uncertainty in the degree to which lower levels would result in additional public health improvements, due in part to the more limited amount of data available. Such a decision could note the following regarding the increasing uncertainty at lower ambient concentrations:

- Few key epidemiologic studies (and only one key U.S. study) report positive and statistically significant health effect associations for  $\text{PM}_{2.5}$  air quality distributions with overall mean concentrations below 9.6  $\mu\text{g}/\text{m}^3$ , and areas meeting a standard with a level of 10.0  $\mu\text{g}/\text{m}^3$  would generally be expected to have lower long-term mean  $\text{PM}_{2.5}$  concentrations (and potentially around 8.0  $\mu\text{g}/\text{m}^3$  in some areas) (section 3.2.3.2.1; Appendix B, section B.7).

- There is increasing uncertainty in PM<sub>2.5</sub> exposure estimates in some of the largest key studies at lower ambient concentrations (i.e., those that use hybrid model predictions to estimate exposures), given the more limited information available to develop and validate model predictions (sections 2.3.3 and 3.2.3.2.1).
- Pseudo-design values corresponding to the 50<sup>th</sup> percentiles of study area populations (or health events) are  $\geq$  about 10.0  $\mu\text{g}/\text{m}^3$  for almost all key studies, particularly those conducted in the U.S. (section 3.2.3.2.2).
- There is increasing uncertainty in quantitative estimates of PM<sub>2.5</sub>-associated mortality risk for standard levels below 10.0  $\mu\text{g}/\text{m}^3$ , given that a substantial proportion of the risk reductions estimated for lower standard levels occur at annual average PM<sub>2.5</sub> concentrations below 8  $\mu\text{g}/\text{m}^3$ , and thus below the lower end of the range of overall mean PM<sub>2.5</sub> concentrations in key epidemiologic studies that consistently report positive and statistically significant associations (section 3.3.2).

In contrast, an annual standard with a level below 10.0  $\mu\text{g}/\text{m}^3$ , and potentially as low as 8.0  $\mu\text{g}/\text{m}^3$ , could be supported to the extent greater weight is placed on the potential public health improvements that could result from additional reductions in ambient PM<sub>2.5</sub> concentrations (i.e., beyond those achieved by a standard with a level of 10.0  $\mu\text{g}/\text{m}^3$ ) and less weight is placed on the limitations in the evidence that contribute to greater uncertainty at lower concentrations. For example, a level below 10.0  $\mu\text{g}/\text{m}^3$  could be supported to the extent greater weight is placed on the following:

- The two key studies in Canada with overall mean PM<sub>2.5</sub> concentrations below 8.0  $\mu\text{g}/\text{m}^3$  and the potential for overall mean concentrations below 8.0  $\mu\text{g}/\text{m}^3$  in restricted analyses in a key U.S. study (section 3.2.3.2.1);
- The ambient PM<sub>2.5</sub> concentrations somewhat below overall means (e.g., corresponding the lower quartile of underlying data), which contribute to the bulk of the data informing reported associations (section 3.2.3.2.1);
- Annual pseudo-design values corresponding to 25<sup>th</sup> percentiles of study area populations or health events for most studies, recognizing that the revised standard would be expected to maintain ambient PM<sub>2.5</sub> concentrations below the concentrations present during study periods for  $> \sim 75\%$  of those populations (or below the concentrations in locations accounting for  $> 75\%$  of health events) (section 3.2.3.2.2);
- Annual pseudo-design values for the smaller number of key studies conducted in Canada, which tend to be somewhat lower than those in the U.S. (section 3.2.3.2.2);
- The potential public health importance of the additional reductions in PM<sub>2.5</sub>-associated health risks estimated for a level of 9.0  $\mu\text{g}/\text{m}^3$  and the potential for continued reductions at lower standard levels (i.e., below the lowest level examined in the risk assessment) (section 3.3).

As above, various levels from 8.0  $\mu\text{g}/\text{m}^3$  to  $< 10.0 \mu\text{g}/\text{m}^3$  could be supported, depending on the weight placed on specific aspects of the evidence and analyses. For example, compared to a level of 8.0  $\mu\text{g}/\text{m}^3$ , a higher level could be supported to the extent less weight is placed on the



two key Canadian studies reporting overall mean concentrations below  $8.0 \mu\text{g}/\text{m}^3$ , on the potential for overall mean concentrations below  $8.0 \mu\text{g}/\text{m}^3$  in a U.S. study that reports associations in restricted analyses, and on the three Canadian studies with the lowest pseudo-design values. Such a judgment could also be informed by increasing uncertainty in the potential public health importance of risks estimated for a level as low as  $8.0 \mu\text{g}/\text{m}^3$ , given that such risks, which were not quantified in the risk assessment, are likely to occur at annual average  $\text{PM}_{2.5}$  concentrations largely below  $8 \mu\text{g}/\text{m}^3$  (i.e., below the mean concentrations in almost all key epidemiologic studies).

#### **3.5.2.4.2 Alternative 24-Hour Standard Levels**

We additionally evaluate the degree to which the evidence supports considering potential alternative levels for the 24-hour  $\text{PM}_{2.5}$  standard, in conjunction with the current 98<sup>th</sup> percentile form of that standard. As discussed above (section 3.1.1), in the last review, the EPA recognized that the annual standard would generally be the controlling standard across much of the U.S., except for certain areas in the western U.S. “where annual mean  $\text{PM}_{2.5}$  concentrations have historically been low but where relatively high 24-hour concentrations occur, often related to seasonal wood smoke emissions” (78 FR 3163, January 15, 2013). In such areas, the 24-hour standard is the generally controlling standard. Thus, the EPA’s approach in the last review was to focus on the annual standard as the principle means of limiting both long- and short-term  $\text{PM}_{2.5}$  concentrations, recognizing that the 24-hour standard, with its 98<sup>th</sup> percentile form, would provide supplemental protection against short-term peak exposures, particularly for areas with high peak-to-mean ratios (e.g., areas with strong seasonal sources).

As discussed above (section 3.1.2), in the current review we again view the 24-hour standard (with its 98<sup>th</sup> percentile form) largely within the context of limiting short-term exposures to peak  $\text{PM}_{2.5}$  concentrations. Compared to the annual standard, we recognize that the 24-hour standard is less likely to appropriately limit the more typical  $\text{PM}_{2.5}$  exposures (i.e., corresponding to the middle portion of the air quality distribution) that are most strongly associated with the health effects observed in epidemiologic studies. Thus, as in the last review (78 FR 3161-3162, January 15, 2013), we focus on the annual  $\text{PM}_{2.5}$  standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term  $\text{PM}_{2.5}$  exposures, and the 24-hour standard as a means of providing supplemental protection against the short-term exposures to “peak”  $\text{PM}_{2.5}$  concentrations, such as can occur in areas with strong contributions from local or seasonal sources.

Results of the risk assessment and of recent air quality analyses are consistent with our reliance on the 24-hour standard to provide supplemental protection in areas with relatively low long-term mean  $\text{PM}_{2.5}$  concentrations. In particular, the risk assessment indicates that the annual

standard is the controlling standard across most of the urban study areas evaluated and revising the level of the 24-hour standard to  $30 \mu\text{g}/\text{m}^3$  would be estimated to lower  $\text{PM}_{2.5}$ -associated risks, compared to the current standards, largely in a few study areas located in the western U.S. (several of which are also likely to experience risk reductions upon meeting a revised annual standard). Additionally, recent air quality analyses indicate that almost all CBSAs with maximum annual  $\text{PM}_{2.5}$  design values at or below  $12.0 \mu\text{g}/\text{m}^3$  also have maximum 24-hour design values below  $35 \mu\text{g}/\text{m}^3$  (and below  $30 \mu\text{g}/\text{m}^3$  in most areas) (Chapter 2, Figure 2-11). The exceptions are a few CBSAs in the western U.S.

Thus, taking into account the approach described above, an important consideration is whether additional protection is needed against short-term exposures to peak  $\text{PM}_{2.5}$  concentrations in areas meeting both the current 24-hour standard and the current, or a revised, annual standard. To the extent the evidence indicates that such exposures can lead to adverse health effects, it would be appropriate to consider alternative levels for the 24-hour standard. In considering this issue, we evaluate the evidence from key health studies. With regard to these studies, we particularly note the following:

- To the extent a revised annual standard is determined to provide adequate protection against the 24-hour and annual  $\text{PM}_{2.5}$  exposures associated with health effects in key epidemiologic studies, those studies do not indicate the need for additional protection against short-term exposures to peak  $\text{PM}_{2.5}$  concentrations. As discussed in detail above (section 3.2.3.2.1), epidemiologic studies provide the strongest support for reported health effect associations for the part of the air quality distribution corresponding to the bulk of the underlying data (i.e., estimated exposures and/or health events), often around the overall mean concentrations evaluated rather than near the upper end of the distribution. Consistent with this, analyses that exclude the upper end of the distribution of estimated exposures still find positive and statistically significant associations with mortality. The magnitudes of the associations in restricted analyses are similar to (Shi et al., 2016) or larger than (Di et al., 2017a) the magnitudes of the associations based on the full cohorts, suggesting that, at a minimum, short-term exposures to peak  $\text{PM}_{2.5}$  concentrations are not disproportionately responsible for reported health effect associations.
- Controlled human exposure studies do provide evidence for health effects following single, short-term  $\text{PM}_{2.5}$  exposures to concentrations that typically correspond to upper end of the  $\text{PM}_{2.5}$  air quality distribution in the U.S. (i.e., “peak” concentrations). However, most of these studies examine exposure concentrations considerably higher than are typically measured in areas meeting the current standards (section 3.2.3.1). In particular, while controlled human exposure studies often report statistically significant effects on one or more indicators of cardiovascular function following 2-hour exposures to  $\text{PM}_{2.5}$  concentrations at and above  $120 \mu\text{g}/\text{m}^3$  (at and above  $149 \mu\text{g}/\text{m}^3$  for vascular impairment, the effect shown to be most consistent across studies), 2-hour ambient concentrations of  $\text{PM}_{2.5}$  at monitoring sites meeting the current standards almost never exceed  $32 \mu\text{g}/\text{m}^3$ . In fact, even the extreme upper end of the distribution of 2-hour  $\text{PM}_{2.5}$  concentrations at sites meeting the current standards remains well-below the  $\text{PM}_{2.5}$  exposure concentrations consistently shown to elicit effects

(i.e., 99.9<sup>th</sup> percentile of 2-hour concentrations at these sites is 68  $\mu\text{g}/\text{m}^3$  during the warm season). Thus, available  $\text{PM}_{2.5}$  controlled human exposure studies do not indicate the need for additional protection against exposures to peak  $\text{PM}_{2.5}$  concentrations, beyond the protection provided by the combination of the current 24-hour standard and the current or a revised annual standard (section 3.2.3.1).

When the information summarized above is considered in the context of the 24-hour standard, we reach the conclusion that, in conjunction with a lower annual standard level intended to increase protection against short- and long-term  $\text{PM}_{2.5}$  exposures broadly across the U.S., the evidence does not support the need for additional protection against short-term exposures to peak  $\text{PM}_{2.5}$  concentrations. In particular, while epidemiologic studies do support the need to consider increasing protection against the typical 24-hour and annual  $\text{PM}_{2.5}$  exposures that provide strong support for reported health effect associations, these studies do not indicate that such associations are strongly influenced by exposures to the peak concentrations in the air quality distribution. Also, while controlled human exposure studies support the occurrence of effects following single short-term exposures to  $\text{PM}_{2.5}$  concentrations that correspond to the peak of the air quality distribution, these concentrations are well above those typically measured in areas meeting the current standards. Thus, in the context of a 24-hour standard that is meant to provide supplemental protection (i.e., beyond that provided by the annual standard alone) against short-term exposures to peak  $\text{PM}_{2.5}$  concentrations, the available evidence supports consideration of retaining the current 24-hour standard with its level of 35  $\mu\text{g}/\text{m}^3$ .

However, we also recognize that a different policy approach than that described above could be applied to considering the level of the 24-hour standard. For example, consideration could be given to lower 24-hour standard levels in order to increase protection across the U.S. against the broader  $\text{PM}_{2.5}$  air quality distribution. If such an approach is evaluated in the current review, consideration of 24-hour standard levels at least as low as 30  $\mu\text{g}/\text{m}^3$  could be supported (either alone or in conjunction with a lower annual standard level). The risk assessment estimates that a level of 30  $\mu\text{g}/\text{m}^3$  would increase protection compared to the current standards, though only in a small number of study areas largely confined to the western U.S. (section 3.3.2). Analyses of air quality in locations of some key epidemiologic studies indicate that substantial portions of study area populations lived in locations with 24-hour  $\text{PM}_{2.5}$  pseudo-design values at or below about 30  $\mu\text{g}/\text{m}^3$  (or that substantial portions of study health events occurred in such locations), providing additional support for considering lower levels.

If this alternative approach to revising the primary  $\text{PM}_{2.5}$  standards is adopted, the uncertainty inherent in using the 24-hour standard to increase protection against the broad distribution of  $\text{PM}_{2.5}$  air quality should be carefully considered. Specifically, the degree of

protection provided by any particular 24-hour standard against the typical short- and long-term PM<sub>2.5</sub> exposures corresponding to the middle portion of the air quality distribution will vary across locations and over time, depending on the relationship between those typical concentrations and the short-term peak PM<sub>2.5</sub> concentrations that are directly targeted by the 24-hour standard (i.e., with its 98<sup>th</sup> percentile form). Thus, lowering the level of the 24-hour standard is likely to have a more variable impact on public health than lowering the level of the annual standard. Depending on the 24-hour standard level set, some areas could experience reductions that are greater than warranted, based on the evidence, while others could experience reductions that are less than warranted. Therefore, the rationale supporting this approach would need to recognize and account for the uncertainty inherent in using 24-hour standard, with a 98<sup>th</sup> percentile form, to increase protection against the broad distribution of PM<sub>2.5</sub> air quality.

### **3.6 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

In this section, we identify key areas for additional research and data collection for fine particles, based on the uncertainties and limitations that remain in the evidence and technical information. Additional research in these areas could reduce uncertainties and limitations in future reviews of the primary PM<sub>2.5</sub> standards. Important areas for future research include the following:

- Further elucidating the physiological pathways through which exposures to the PM<sub>2.5</sub> concentrations present in the ambient air across much of the U.S. could be causing mortality and the morbidity effects shown in many epidemiologic studies. This could include the following:
  - Controlled human exposure studies that examine longer exposure periods (e.g., 24-hour as in Bräuner et al. (2008); 5-hour as in Hemmingsen et al. (2015b)), or repeated exposures, to concentrations typically measured in the ambient air across the U.S.
  - Studies that evaluate the health impacts of decreasing PM<sub>2.5</sub> exposures (e.g., due to changes in policies or behavior, shifts in important emissions sources, or targeted interventions).
  - Additional animal toxicological studies that evaluate exposures to low PM<sub>2.5</sub> concentrations.
- Additional research into “causal inference” methods in epidemiologic studies to evaluate the causal nature of relationships between PM<sub>2.5</sub> exposure and mortality or morbidity.
- Improving our understanding of the PM<sub>2.5</sub> concentration-response relationships near the lower end of the PM<sub>2.5</sub> air quality distribution, including the shapes of concentration-response functions and the uncertainties around estimated functions for various health outcomes and populations (e.g., older adults, people with pre-existing diseases, children).

- Understanding of the potential for particle characteristics, other than size-fractionated mass, to influence PM toxicity (e.g., composition, oxidative potential, etc.) and the PM health effect associations observed in epidemiologic studies.
- Improving our understanding of the uncertainties inherent in the various approaches used to estimate PM<sub>2.5</sub> exposures in epidemiologic studies, including how those uncertainties may vary across space and time, and over the PM<sub>2.5</sub> air quality distribution. Approaches to incorporating these uncertainties into quantitative estimates of PM<sub>2.5</sub> concentration-response relationships should also be explored.
- Additional health research on ultrafine particles, with a focus on consistently defining UFPs across studies and across disciplines (i.e., animal, controlled human exposure, and epidemiologic studies), on using consistent exposure approaches in experimental studies, and on improving exposure characterizations in epidemiologic studies. Also, further examine the potential for translocation of ultrafine particles from the respiratory tract into other compartments (i.e., blood) and organs (e.g., heart, brain), with particular emphasis on studies conducted in humans.
- Additional work to measure ultrafine particle emissions, using comparable methods to measure emissions from various types of sources (e.g., mobile sources, fires, etc.).
- Further evaluate the potential for some groups to be at higher risk of PM<sub>2.5</sub>-related effects than the general population and the potential for PM<sub>2.5</sub> exposures to contribute to the development of underlying conditions that may then confer higher risk of PM<sub>2.5</sub>-related effects. For example, research to address this latter need could include efforts to understand the potential for long-term PM exposures to contribute to the development and progression of atherosclerosis in adults and/or asthma in children. It could also include research to understand the potential role of PM exposures in developmental outcomes (e.g., neurodevelopmental effects, reproductive and birth outcomes).
- Research to further evaluate the combination of factors that contribute to differences in risk estimates between cities, potentially including differences in exposures, demographics, particle characteristics.
- Research to improve our understanding of variability in PM<sub>2.5</sub> exposures within and across various populations (e.g., defined by life stage, pre-existing condition, etc.), the most health-relevant exposure durations, as well as the temporal and spatial variability in ambient PM<sub>2.5</sub> that is not captured by existing ambient monitors.

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## 4 REVIEW OF THE PRIMARY STANDARD FOR PM<sub>10</sub>

This chapter presents key policy-relevant considerations and conclusions regarding the public health protection provided by the current primary PM<sub>10</sub> standard. These considerations and conclusions are framed by a series of policy-relevant questions, including the following overarching policy-relevant question:

- **Does the currently available scientific evidence support or call into question the adequacy of the protection afforded by the current 24-hour primary PM<sub>10</sub> standard against health effects associated with exposures to PM<sub>10-2.5</sub>?**

The answer to this question is intended to inform decisions by the Administrator on whether, and if so, how to revise the primary standard for PM<sub>10</sub>.

Section 4.1 summarizes the EPA's approach to reviewing the primary PM<sub>10</sub> standard in the last review and our general approach to considering the updated scientific evidence in the current review. Section 4.2 presents our consideration of the available evidence as assessed in the ISA. Section 4.3 summarizes CASAC advice and public comments. Drawing from that consideration of the evidence, section 4.4 summarizes our conclusions regarding the adequacy of the current primary PM<sub>10</sub> standard. Section 4.5 discusses areas for future research and data collection to improve our understanding of potential PM<sub>10-2.5</sub>-related health effects in future reviews.

### 4.1 APPROACH

#### 4.1.1 Approach Used in the Last review

The last review of the PM NAAQS was completed in 2012 (78 FR 3086, January 15, 2013). In that review the EPA retained the existing 24-hour primary PM<sub>10</sub> standard, with its level of 150 µg/m<sup>3</sup> and its one-expected-exceedance form on average over three years, to continue to provide public health protection against exposures to PM<sub>10-2.5</sub>. In support of this decision, the Administrator emphasized her consideration of three issues: the extent to which it was appropriate to maintain a standard that provides some measure of protection against all PM<sub>10-2.5</sub> (regardless of composition or source or origin), the extent to which a standard with a PM<sub>10</sub> indicator can provide protection against exposures to PM<sub>10-2.5</sub>, and the degree of public health protection provided by the existing PM<sub>10</sub> standard. Her consideration of each of these issues is summarized below.

First, the Administrator judged that the evidence provided “ample support for a standard that protects against exposures to all thoracic coarse particles, regardless of their location or source of origin” (78 FR 3176, January 15, 2013). In support of this, she noted that

epidemiologic studies had reported positive associations between PM<sub>10-2.5</sub> and mortality or morbidity in a large number of cities across North America, Europe, and Asia, encompassing a variety of environments where PM<sub>10-2.5</sub> sources and composition are expected to vary widely. Though most of the available studies examined associations in urban areas, the Administrator noted that some studies had also linked mortality and morbidity with relatively high ambient concentrations of particles of non-urban crustal origin. In light of this body of available evidence, and consistent with the CASAC's advice, the Administrator concluded that it was appropriate to maintain a standard that provides some measure of protection against exposures to all thoracic coarse particles, regardless of their location, source of origin, or composition (78 FR 3176, January 15, 2013).

In next reaching the conclusion that it was appropriate to retain a PM<sub>10</sub> indicator for a standard meant to protect against exposures to ambient PM<sub>10-2.5</sub>, the Administrator noted that PM<sub>10</sub> mass includes both coarse PM (PM<sub>10-2.5</sub>) and fine PM (PM<sub>2.5</sub>). As a result, the concentration of PM<sub>10-2.5</sub> allowed by a PM<sub>10</sub> standard set at a single level declines as the concentration of PM<sub>2.5</sub> increases. Because PM<sub>2.5</sub> concentrations tend to be higher in urban areas than rural areas (e.g., Chan et al., 2018), the Administrator observed that a PM<sub>10</sub> standard would generally allow lower PM<sub>10-2.5</sub> concentrations in urban areas than in rural areas. She judged it appropriate to maintain such a standard given that much of the evidence for PM<sub>10-2.5</sub> toxicity, particularly at relatively low particle concentrations, came from study locations where thoracic coarse particles were of urban origin, and given the possibility that PM<sub>10-2.5</sub> contaminants in urban areas could increase particle toxicity. Thus, in the last review the Administrator concluded that it remained appropriate to maintain a standard that allows lower ambient concentrations of PM<sub>10-2.5</sub> in urban areas, where the evidence was strongest that exposure to thoracic coarse particles was associated with morbidity and mortality, and higher concentrations in non-urban areas, where the public health concerns were less certain. The Administrator concluded that the varying concentrations of coarse particles that would be permitted in urban versus non-urban areas under the 24-hour PM<sub>10</sub> standard, based on the varying levels of PM<sub>2.5</sub> present, appropriately reflected the differences in the strength of evidence regarding coarse particle health effects.

Finally, in specifically evaluating the degree of public health protection provided by the primary PM<sub>10</sub> standard, with its level of 150 µg/m<sup>3</sup> and its one-expected-exceedance form on average over three years, the Administrator recognized that the available health evidence and air quality information was much more limited for PM<sub>10-2.5</sub> than for PM<sub>2.5</sub>. In particular, the strongest evidence for health effects attributable to PM<sub>10-2.5</sub> exposure was for cardiovascular effects, respiratory effects, and/or premature mortality following short-term exposures. For each of these categories of effects, the 2009 ISA concluded that the evidence was “suggestive of a

causal relationship” (U.S. EPA, 2009, section 2.3.3). These determinations contrasted with those for PM<sub>2.5</sub>, as described in Chapter 3 above, which were determined in the ISA to be either “causal” or “likely to be causal” for mortality, cardiovascular effects, and respiratory effects (U.S. EPA, 2009, Tables 2-1 and 2-2).

The Administrator judged that the important uncertainties and limitations associated with the PM<sub>10-2.5</sub> evidence and information raised questions as to whether additional public health improvements would be achieved by revising the existing PM<sub>10</sub> standard. She specifically noted several uncertainties, including the following:

- (1) The number of epidemiologic studies that have employed copollutant models to address the potential for confounding, particularly by PM<sub>2.5</sub>, was limited. Therefore, the extent to which PM<sub>10-2.5</sub> itself, rather than one or more copollutants, contributes to reported health effects remained uncertain.
- (2) Only a limited number of experimental studies provided support for the associations reported in epidemiologic studies, resulting in further uncertainty regarding the plausibility of the associations between PM<sub>10-2.5</sub> and mortality and morbidity reported in epidemiologic studies.
- (3) Limitations in PM<sub>10-2.5</sub> monitoring data (i.e., limited data available from FRM/FEM sampling methods) and the different approaches used to estimate PM<sub>10-2.5</sub> concentrations across epidemiologic studies resulted in uncertainty in the ambient PM<sub>10-2.5</sub> concentrations at which the reported effects occur, increasing uncertainty in estimates of the extent to which changes in ambient PM<sub>10-2.5</sub> concentrations would likely impact public health.
- (4) While PM<sub>10-2.5</sub> effect estimates reported for mortality and morbidity were generally positive, most were not statistically significant, even in single-pollutant models. This included effect estimates reported in some study locations with PM<sub>10</sub> concentrations above those allowed by the current 24-hour PM<sub>10</sub> standard.
- (5) The composition of PM<sub>10-2.5</sub>, and the effects associated with various components, were also key uncertainties in the available evidence. Without more information on the chemical speciation of PM<sub>10-2.5</sub>, the apparent variability in associations across locations was difficult to characterize.

In considering these uncertainties, the Administrator particularly emphasized the considerable degree of uncertainty in the extent to which health effects reported in epidemiologic studies are due to PM<sub>10-2.5</sub> itself, as opposed to one or more co-occurring pollutants. This uncertainty reflected the relatively small number of PM<sub>10-2.5</sub> studies that had evaluated copollutant models, particularly copollutant models that included PM<sub>2.5</sub>, and the very limited body of controlled human exposure evidence supporting the plausibility of PM<sub>10-2.5</sub>-attributable adverse effects at ambient concentrations.

When considering the evidence as a whole, the Administrator concluded that the degree of public health protection provided by the current PM<sub>10</sub> standard against exposures to PM<sub>10-2.5</sub> should be maintained (i.e., neither increased nor decreased). The Administrator’s judgment that protection did not need to be increased was supported by her consideration of uncertainties in the overall body of evidence. Her judgment that the degree of public health protection provided by the current standard is not greater than warranted was supported by the observation that positive and statistically significant associations with mortality were reported in some single-city U.S. study locations likely to have violated the current PM<sub>10</sub> standard. Thus, the Administrator concluded that the existing 24-hour PM<sub>10</sub> standard, with its one-expected exceedance form on average over three years and a level of 150 µg/m<sup>3</sup>, was requisite to protect public health with an adequate margin of safety against effects that have been associated with PM<sub>10-2.5</sub>. In light of this conclusion, the EPA retained the existing PM<sub>10</sub> standard.

#### **4.1.2 Approach in the Current Review**

As discussed above for PM<sub>2.5</sub> (section 3.2.1), in this PA we place the greatest emphasis on effects for which the evidence has been determined to demonstrate a “causal” or a “likely to be causal” relationship with PM exposures (U.S. EPA, 2019). This approach focuses policy considerations and conclusions on health outcomes for which the evidence is strongest. Unlike for PM<sub>2.5</sub>, the ISA does not identify any PM<sub>10-2.5</sub>-related health outcomes for which the evidence supports either a “causal” or a “likely to be causal” relationship. Thus, for PM<sub>10-2.5</sub> this PA considers the evidence determined to be “suggestive of, but not sufficient to infer, a causal relationship,” recognizing the greater uncertainty in such evidence.

The preamble to the ISA states that “suggestive” evidence is “limited, and chance, confounding, and other biases cannot be ruled out” (U.S. EPA, 2015, Table II). In light of the additional uncertainty in the evidence for PM<sub>10-2.5</sub>-related health outcomes, compared to the evidence supporting “causal” or “likely to be causal” relationships for PM<sub>2.5</sub>, our approach to evaluating the primary PM<sub>10</sub> standard in this review is more limited than our approach to evaluating the primary PM<sub>2.5</sub> standards (discussed in Chapter 3). Specifically, our approach for PM<sub>10</sub> does not include evaluations of air quality distributions in locations of individual epidemiologic studies, comparisons of experimental exposures with ambient air quality, or the quantitative assessment of PM<sub>10-2.5</sub> health risks. The substantial uncertainty in such analyses, if they were to be conducted based on the currently available PM<sub>10-2.5</sub> health studies, would limit their utility for informing conclusions on the primary PM<sub>10</sub> standard. Therefore, as discussed further below, we focus our evaluation of the primary PM<sub>10</sub> standard on the overall body of evidence for PM<sub>10-2.5</sub>-related health effects. This includes consideration of the degree to which uncertainties in the evidence from the last review have been reduced and the degree to which

new uncertainties have been identified. In adopting this approach, we recognize that the Administrator's decisions as to whether to retain or revise the primary PM<sub>10</sub> standard will largely be public health policy judgments that will draw upon the scientific evidence for PM<sub>10-2.5</sub>-related health effects and judgments about how to consider the uncertainties and limitations inherent in that evidence.

## 4.2 EVIDENCE-BASED CONSIDERATIONS

This section draws from the EPA's synthesis and assessment of the scientific evidence presented in the ISA (U.S. EPA, 2019) to consider the following policy-relevant questions:

- **To what extent does the currently available scientific evidence strengthen, or otherwise alter, our conclusions from the last review regarding health effects attributable to long- or short-term PM<sub>10-2.5</sub> exposures? Have previously identified uncertainties been reduced? What important uncertainties remain and have new uncertainties been identified?**

Answers to these questions will inform our answer to the overarching question on the adequacy of the current primary PM<sub>10</sub> standard, posed at the beginning of this chapter. In section 4.2.1 below, we consider the nature of the effects attributable to long-term and short-term PM<sub>10-2.5</sub> exposures.

### 4.2.1 Nature of Effects

As noted above, for the health outcome categories and exposure duration combinations evaluated, the ISA concludes that the evidence supports causality determinations for PM<sub>10-2.5</sub> no stronger than "suggestive of, but not sufficient to infer, a causal relationship." These outcomes, along with their corresponding causality determinations from the 2009 ISA, are highlighted below in Table 4-1 (adapted from U.S. EPA, 2019, Table 1-4).



**Table 4-1. Key Causality Determinations for PM<sub>10-2.5</sub> Exposures**

Health Outcome	Exposure Duration	2009 PM ISA	2019 PM ISA
Mortality	Long-term	Inadequate	Suggestive of, but not sufficient to infer
	Short-term	Suggestive of, but not sufficient to infer	
Cardiovascular effects	Long-term	Inadequate	
	Short-term	Suggestive of, but not sufficient to infer	
Respiratory effects	Short-term	Suggestive of, but not sufficient to infer	
Cancer	Long-term	Inadequate	
Nervous System effects	Long-term	---	
Metabolic effects	Long-term	---	

While the evidence for some of the health outcomes listed in Table 4-1 has strengthened since the last review, the ISA concludes that overall “the uncertainties in the evidence identified in the 2009 PM ISA have, to date, still not been addressed” (U.S. EPA, 2019, section 1.4.2, p. 1-41). For example, epidemiologic studies available in the last review relied on various methods to estimate PM<sub>10-2.5</sub> exposures, and these methods had not been systematically compared to evaluate spatial and temporal correlations in exposure estimates. Methods included (1) calculating the difference between PM<sub>10</sub> and PM<sub>2.5</sub> concentrations at co-located monitors, (2) calculating the difference between county-wide averages of monitored PM<sub>10</sub> and PM<sub>2.5</sub> based on monitors that are not necessarily co-located, and (3) direct measurement of PM<sub>10-2.5</sub> using a dichotomous sampler (U.S. EPA, 2019, section 1.4.2). In the current review, more recent epidemiologic studies continue to use these approaches to estimate PM<sub>10-2.5</sub> concentrations. Additionally, some recent studies estimate long-term PM<sub>10-2.5</sub> exposures as the difference between PM<sub>10</sub> and PM<sub>2.5</sub> concentrations based on information from spatiotemporal or land use regression (LUR) models, in addition to monitors. As in the last review, the various methods used to estimate PM<sub>10-2.5</sub> concentrations have not been systematically evaluated (U.S. EPA, 2019, section 3.3.1.1), contributing to uncertainty regarding the spatial and temporal correlations in PM<sub>10-2.5</sub> concentrations across methods and in the PM<sub>10-2.5</sub> exposure estimates used in epidemiologic studies (U.S. EPA, 2019, section 2.5.1.2.3). Given the greater spatial and temporal variability of PM<sub>10-2.5</sub> and fewer PM<sub>10-2.5</sub> monitoring sites, compared to PM<sub>2.5</sub>, this uncertainty is particularly important for the coarse size fraction.

Beyond uncertainty associated with PM<sub>10-2.5</sub> exposure estimates in epidemiologic studies, the limited information on the potential for confounding by copollutants and the limited support available for the biological plausibility of serious effects following PM<sub>10-2.5</sub> exposures also continue to contribute broadly to uncertainty in the PM<sub>10-2.5</sub> health evidence. Uncertainty related to potential confounding stems from the relatively small number of epidemiologic studies that have evaluated PM<sub>10-2.5</sub> health effect associations in copollutants models with both gaseous pollutants and other PM size fractions. Uncertainty related to the biological plausibility of serious effects caused by PM<sub>10-2.5</sub> exposures results from the small number of controlled human exposure and animal toxicology<sup>1</sup> studies that have evaluated the health effects of experimental PM<sub>10-2.5</sub> inhalation exposures. The evidence supporting the ISA's "suggestive" causality determinations for PM<sub>10-2.5</sub>, including uncertainties in this evidence, is summarized in sections 4.2.1.1 to 4.2.1.6 below.

#### **4.2.1.1 Mortality**

##### Long-term exposures

Due to the dearth of studies examining the association between long-term PM<sub>10-2.5</sub> exposure and mortality, the 2009 PM ISA concluded that the evidence was "inadequate to determine if a causal relationship exists" (U.S. EPA, 2009). Since the completion of the 2009 ISA, some recent cohort studies conducted in the U.S. and Europe report positive associations between long-term PM<sub>10-2.5</sub> exposure and total (nonaccidental) mortality, though results are inconsistent across studies (U.S. EPA, 2019, Table 11-11). The examination of copollutant models in these studies remains limited and, when included, PM<sub>10-2.5</sub> effect estimates are often attenuated after adjusting for PM<sub>2.5</sub> (U.S. EPA, 2019, Table 11-11). Across studies, PM<sub>10-2.5</sub> exposure concentrations are estimated using a variety of approaches, including direct measurements from dichotomous samplers, calculating the difference between PM<sub>10</sub> and PM<sub>2.5</sub> concentrations measured at collocated monitors, and calculating difference of area-wide concentrations of PM<sub>10</sub> and PM<sub>2.5</sub>. As discussed above, temporal and spatial correlations between these approaches have not been evaluated, contributing to uncertainty regarding the potential for exposure measurement error (U.S. EPA, 2019, section 3.3.1.1 and Table 11-11). The ISA concludes that this uncertainty "reduces the confidence in the associations observed across studies" (U.S. EPA, 2019, p. 11-125). The ISA additionally concludes that the evidence for long-term PM<sub>10-2.5</sub> exposures and cardiovascular effects, respiratory morbidity, and metabolic disease provide limited biological plausibility for PM<sub>10-2.5</sub>-related mortality (U.S. EPA, 2019, sections 11.4.1 and 11.4). Taken together, the ISA concludes that, "this body of evidence is

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<sup>1</sup> Compared to humans, smaller fractions of inhaled PM<sub>10-2.5</sub> penetrate into the thoracic regions of rats and mice (U.S. EPA, 2018, section 4.1.6), contributing to the relatively limited evaluation of PM<sub>10-2.5</sub> exposures in animal studies.

suggestive, but not sufficient to infer, that a causal relationship exists between long-term PM<sub>10-2.5</sub> exposure and total mortality” (U.S. EPA, 2019, p. 11-125).

#### Short-term exposures

The 2009 ISA concluded that the evidence is "suggestive of a causal relationship between short-term exposure to PM<sub>10-2.5</sub> and mortality” (U.S. EPA, 2009). Since the completion of the 2009 ISA, multicity epidemiologic studies conducted primarily in Europe and Asia continue to provide consistent evidence of positive associations between short-term PM<sub>10-2.5</sub> exposure and total (nonaccidental) mortality (U.S. EPA, 2019, Table 11-9). Although these studies contribute to increasing confidence in the PM<sub>10-2.5</sub>-mortality relationship, the use of a variety of approaches to estimate PM<sub>10-2.5</sub> exposures continues to contribute uncertainty to the associations observed. In addition, the ISA notes that an analysis by Adar et al. (2014) indicates “possible evidence of publication bias, which was not observed for PM<sub>2.5</sub>” (U.S. EPA, 2019, section 11.3.2, p. 11-106). Recent studies expand the assessment of potential copollutant confounding of the PM<sub>10-2.5</sub>-mortality relationship and provide evidence that PM<sub>10-2.5</sub> associations generally remain positive in copollutant models, though associations are attenuated in some instances (U.S. EPA, 2019, section 11.3.4.1, Figure 11-28, Table 11-10). The ISA concludes that, overall, the assessment of potential copollutant confounding is limited due to the lack of information on the correlation between PM<sub>10-2.5</sub> and gaseous pollutants and the small number of locations in which copollutant analyses have been conducted. Associations with cause-specific mortality provide some support for associations with total (nonaccidental) mortality, though associations with cause-specific mortality, particularly respiratory mortality, are more uncertain (i.e., wider confidence intervals) and less consistent (U.S. EPA, 2019, section 11.3.7). The ISA concludes that the evidence for PM<sub>10-2.5</sub>-related cardiovascular and respiratory effects provides only limited support for the biological plausibility of a relationship between short-term PM<sub>10-2.5</sub> exposure and cardiovascular mortality (U.S. EPA, 2019, Section 11.3.7). Based on the overall evidence, the ISA concludes that, “this body of evidence is suggestive, but not sufficient to infer, that a causal relationship exists between short-term PM<sub>10-2.5</sub> exposure and total mortality” (U.S. EPA, 2019, p. 11-120).

#### **4.2.1.2 Cardiovascular Effects**

##### Long-term exposures

In the 2009 PM ISA, the evidence describing the relationship between long-term exposure to PM<sub>10-2.5</sub> and cardiovascular effects was characterized as “inadequate to infer the presence or absence of a causal relationship.” The limited number of epidemiologic studies reported contradictory results and experimental evidence demonstrating an effect of PM<sub>10-2.5</sub> on the cardiovascular system was lacking (U.S. EPA, 2019, section 6.4).

The evidence relating long-term PM<sub>10-2.5</sub> exposures to cardiovascular mortality remains limited, with no consistent pattern of associations across studies and, as discussed above, uncertainty stemming from the use of various approaches to estimate PM<sub>10-2.5</sub> concentrations (U.S. EPA, 2019, Table 6-70). The evidence for associations with cardiovascular morbidity has grown and, while results across studies are not entirely consistent, some epidemiologic studies report positive associations with ischemic heart disease (IHD) and myocardial infarction (MI) (U.S. EPA, 2019, Figure 6-34); stroke (U.S. EPA, 2019, Figure 6-35); atherosclerosis; venous thromboembolism (VTE); and blood pressure and hypertension (U.S. EPA, 2019, Section 6.4.6). PM<sub>10-2.5</sub> cardiovascular mortality effect estimates are often attenuated, but remain positive, in copollutants models that adjust for PM<sub>2.5</sub>. For morbidity outcomes, associations are inconsistent in copollutant models that adjust for PM<sub>2.5</sub>, NO<sub>2</sub>, and chronic noise pollution (U.S. EPA, 2019, p. 6-276). The lack of toxicological evidence for long-term PM<sub>10-2.5</sub> exposures represents a substantial data gap (U.S. EPA, 2019, section 6.4.10), resulting in the ISA conclusion that “evidence from experimental animal studies is of insufficient quantity to establish biological plausibility” (U.S. EPA, 2019, p. 6-277). Based largely on the observation of positive associations in some high-quality epidemiologic studies, the ISA concludes that “evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term PM<sub>10-2.5</sub> exposure and cardiovascular effects” (U.S. EPA, 2019, p. 6-277).

#### Short-term exposures

The 2009 ISA found that the available evidence for short-term PM<sub>10-2.5</sub> exposure and cardiovascular effects was “suggestive of a causal relationship.” This conclusion was based on several epidemiologic studies reporting associations between short-term PM<sub>10-2.5</sub> exposure and cardiovascular effects, including IHD hospitalizations, supraventricular ectopy, and changes in heart rate variability (HRV). In addition, dust storm events resulting in high concentrations of crustal material were linked to increases in total cardiovascular disease emergency department visits and hospital admissions. However, the 2009 ISA noted the potential for exposure measurement error and copollutant confounding in these epidemiologic studies. In addition, there was only limited evidence of cardiovascular effects from a small number of experimental studies (e.g. animal toxicological studies and controlled human exposure studies) that examined short-term PM<sub>10-2.5</sub> exposures (U.S. EPA, 2009, section 6.2.12.2). In the last review, key uncertainties included the potential for exposure measurement error, copollutant confounding, and limited evidence of biological plausibility for cardiovascular effects following inhalation exposure (U.S. EPA, 2019, section 6.3.13).

The evidence for short-term PM<sub>10-2.5</sub> exposure and cardiovascular outcomes has expanded since the last review, though important uncertainties remain. The ISA notes that there are a small number of epidemiologic studies reporting positive associations between short-term exposure to

PM<sub>10-2.5</sub> and cardiovascular-related morbidity outcomes. However, there is limited evidence to suggest that these associations are biologically plausible, or independent of copollutant confounding. The ISA also concludes that it remains unclear how the approaches used to estimate PM<sub>10-2.5</sub> concentrations in epidemiologic studies may impact exposure measurement error. Taken together, the ISA concludes that “the evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term PM<sub>10-2.5</sub> exposures and cardiovascular effects” (U.S. EPA, 2019, p.6-254).

#### **4.2.1.3 Respiratory Effects**

##### Short-term exposures

Based on a small number of epidemiologic studies observing associations with some respiratory effects and limited evidence from experimental studies to support biological plausibility, the 2009 ISA (U.S. EPA, 2009) concluded that the relationship between short-term exposure to PM<sub>10-2.5</sub> and respiratory effects is “suggestive of a causal relationship.” Epidemiologic findings were consistent for respiratory infection and combined respiratory-related diseases, but not for COPD. Studies were characterized by overall uncertainty in the exposure assignment approach and limited information regarding potential copollutant confounding. Controlled human exposure studies of short-term PM<sub>10-2.5</sub> exposures found no lung function decrements and inconsistent evidence for pulmonary inflammation. Animal toxicological studies were limited to those using non-inhalation (e.g., intra-tracheal instillation) routes of PM<sub>10-2.5</sub> exposure.

Recent epidemiologic findings consistently link PM<sub>10-2.5</sub> exposure to asthma exacerbation and respiratory mortality, with some evidence that associations remain positive (though attenuated in some studies of mortality) in copollutant models that include PM<sub>2.5</sub> or gaseous pollutants. Studies provide limited evidence for positive associations with other respiratory outcomes, including COPD exacerbation, respiratory infection, and combined respiratory-related diseases (U.S. EPA, 2019, Table 5-36). As noted above for other endpoints, an uncertainty in these epidemiologic studies is the lack of a systematic evaluation of the various methods used to estimate PM<sub>10-2.5</sub> concentrations and the resulting uncertainty in the spatial and temporal variability in PM<sub>10-2.5</sub> concentrations compared to PM<sub>2.5</sub> (U.S. EPA, 2019, sections 2.5.1.2.3 and 3.3.1.1). Taken together, the ISA concludes that “the collective evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term PM<sub>10-2.5</sub> exposure and respiratory effects” (U.S. EPA, 2019, p. 5-270).

#### **4.2.1.4 Cancer**

##### Long-term exposures

In the last review, little information was available from studies of cancer following inhalation exposures to PM<sub>10-2.5</sub>. Thus, the 2009 ISA determined the evidence was “inadequate to assess the relationship between long-term PM<sub>10-2.5</sub> exposures and cancer” (U.S. EPA, 2009). Since the 2009 ISA, the assessment of long-term PM<sub>10-2.5</sub> exposure and cancer remains limited, with a few recent epidemiologic studies reporting positive, but imprecise, associations with lung cancer incidence. Uncertainty remains in these studies with respect to exposure measurement error due to the use of PM<sub>10-2.5</sub> predictions that have not been validated by monitored PM<sub>10-2.5</sub> concentrations (U.S. EPA, 2019, sections 3.3.2.3 and 10.3.4). Relatively few experimental studies of PM<sub>10-2.5</sub> have been conducted, though available studies indicate that PM<sub>10-2.5</sub> exhibits two key characteristics of carcinogens: genotoxicity and oxidative stress. While limited, such experimental studies provide some evidence of biological plausibility for the findings in a small number of epidemiologic studies (U.S. EPA, 2019, section 10.3.4).

Taken together, the small number of epidemiologic and experimental studies, along with uncertainty with respect to exposure measurement error, contribute to the determination in the ISA that, “the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term PM<sub>10-2.5</sub> exposure and cancer” (U.S. EPA, 2019, p. 10-87).

#### **4.2.1.5 Metabolic Effects**

##### Long-term exposures

The 2009 ISA did not make a causality determination for PM<sub>10-2.5</sub>-related metabolic effects. Since the last review, one epidemiologic study shows an association between long-term PM<sub>10-2.5</sub> exposure and incident diabetes, while additional cross-sectional studies report associations with effects on glucose or insulin homeostasis (U.S. EPA, 2019, section 7.4). As discussed above for other outcomes, uncertainties with the epidemiologic evidence include the potential for copollutant confounding and exposure measurement error (U.S. EPA, 2019, Tables 7-14 and 7-15). The evidence base to support the biological plausibility of metabolic effects following PM<sub>10-2.5</sub> exposures is limited, but a cross-sectional study that investigated biomarkers of insulin resistance and systemic and peripheral inflammation may support a pathway leading to type 2 diabetes (U.S. EPA, 2019, sections 7.4.1 and 7.4.3). Based on the expanded, though still limited evidence base, the ISA concludes that, “[o]verall, the evidence is suggestive of, but not sufficient to infer, a causal relationship between [long]-term PM<sub>10-2.5</sub> exposure and metabolic effects” (U.S. EPA, 2019, p. 7-56).

#### **4.2.1.6 Nervous system effects**

##### Long-term exposures

The 2009 ISA did not make a causality determination for PM<sub>10-2.5</sub>-related nervous system effects. In the current review, newly available epidemiologic studies report associations between

PM<sub>10-2.5</sub> and impaired cognition and anxiety in adults in longitudinal analyses (U.S. EPA, 2019, Table 8-25, section 8.4.5). Associations of long-term exposure with neurodevelopmental effects are not consistently reported in children (U.S. EPA, 2019, sections 8.4.4 and 8.4.5). Uncertainties in these studies include the potential for copollutant confounding, as no studies examined copollutants models (U.S. EPA, 2019, section 8.4.5), and for exposure measurement error, given the use of various model-based subtraction methods to estimate PM<sub>10-2.5</sub> concentrations (U.S. EPA, 2019, Table 8-25). In addition, there is only limited animal toxicological evidence supporting the biological plausibility of nervous system effects (U.S. EPA, 2019, sections 8.4.1 and 8.4.5). Overall, the ISA concludes that, “the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term PM<sub>10-2.5</sub> exposure and nervous system effects (U.S. EPA, 2019, p. 8-75).

#### **4.2.1.7 Conclusions Drawn from the Evidence**

Based on the evidence available in the current review, as assessed in the ISA (U.S. EPA, 2019) and summarized in 4.2.1.1 to 4.2.1.6 above, we revisit the policy-relevant questions posed at the beginning of this section:

- **To what extent does the currently available scientific evidence strengthen, or otherwise alter, our conclusions from the last review regarding health effects attributable to long- or short-term PM<sub>10-2.5</sub> exposures? Have previously identified uncertainties been reduced? What important uncertainties remain and have new uncertainties been identified?**

In the last review, the strongest evidence for PM<sub>10-2.5</sub>-related health effects was for cardiovascular effects, respiratory effects, and premature mortality following short-term exposures. For each of these categories of effects, the ISA concluded that the evidence was “suggestive of a causal relationship” (U.S. EPA, 2009, section 2.3.3). As summarized in the sections above, key uncertainties in the evidence resulted from limitations in the approaches used to estimate ambient PM<sub>10-2.5</sub> concentrations in epidemiologic studies, limited examination of the potential for confounding by co-occurring pollutants, and limited support for the biological plausibility of the serious effects reported in many epidemiologic studies. Since 2009, the evidence base for several PM<sub>10-2.5</sub>-related health effects has expanded, broadening our understanding of the range of health effects linked to PM<sub>10-2.5</sub> exposures. This includes expanded evidence for the relationships between long-term exposures and cardiovascular effects, metabolic effects, nervous system effects, cancer, and mortality. However, key limitations in the evidence that were identified in the 2009 ISA persist in studies that have become available since the last review. These limitations include the following:

- The use of a variety of methods to estimate PM<sub>10-2.5</sub> exposures in epidemiologic studies and the lack of systematic evaluation of these methods, together with the relatively high

spatial and temporal variability in ambient PM<sub>10-2.5</sub> concentrations and the small number of monitoring sites, results in uncertainty in exposure estimates;

- The limited number of studies that evaluate PM<sub>10-2.5</sub> health effect associations in copollutant models, together with evidence from some studies for attenuation of associations in such models, results in uncertainty in the independence of PM<sub>10-2.5</sub> health effect associations from co-occurring pollutants;
- The limited number of controlled human exposure and animal toxicology studies of PM<sub>10-2.5</sub> inhalation contributes to uncertainty in the biological plausibility of the PM<sub>10-2.5</sub>-related effects reported in epidemiologic studies.

Thus, while new evidence is available for a broader range of health outcomes in the current review, that evidence is subject to the same types of uncertainties that were identified in the last review of the PM NAAQS. As in the last review, these uncertainties contribute to the conclusions in the ISA that the evidence for the PM<sub>10-2.5</sub>-related health effects discussed in this section is “suggestive of, but not sufficient to infer” causal relationships.

### **4.3 CASAC ADVICE AND PUBLIC COMMENTS**

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard. As for PM<sub>2.5</sub> (section 3.4), the CASAC’s advice is documented in a letter sent to the EPA Administrator (Cox, 2019).

In its comments on the draft PA, the CASAC concurs with the draft PA’s overall preliminary conclusions that it is appropriate to consider retaining the current primary PM<sub>10</sub> standard without revision, stating that “[t]he CASAC agrees with the EPA conclusion that ‘...the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports considering of retaining the current standard in this review’” (Cox, 2019, p.3 of letter). The CASAC finds the more limited approach taken for PM<sub>10</sub>, compared to PM<sub>2.5</sub>, to be “reasonable and appropriate” given the less certain evidence and the conclusion that “key uncertainties identified in the last review remain” (Cox, 2019, p. 13 of consensus responses). To reduce these uncertainties in future reviews, the CASAC recommends improvements to PM<sub>10-2.5</sub> exposure assessment, including a more extensive network for direct monitoring of the PM<sub>10-2.5</sub> fraction (Cox, 2019, p. 13 of consensus responses). The CASAC also recommends additional human clinical and animal toxicology studies of the PM<sub>10-2.5</sub> fraction to improve the understanding of biological causal mechanisms and pathways (Cox, 2019, p. 13 of consensus responses).

We also received a limited number of public comments on the adequacy of the primary PM<sub>10</sub> standard. Of those who provided comments on the PM<sub>10</sub> standard, most commenters support the preliminary conclusion that it is appropriate to consider retaining the current PM<sub>10</sub> standard, without revision. One group that includes members of the academic research



community (i.e., the Independent PM Review Panel), however, supports lowering the level of the primary PM<sub>10</sub> standard, consistent with their recommendation to also lower the level of the 24-hour primary PM<sub>2.5</sub> standard.

#### **4.4 CONCLUSIONS ON THE ADEQUACY OF THE CURRENT STANDARD**

This section describes our conclusions regarding the adequacy of the current primary PM<sub>10</sub> standard. Our approach to reaching conclusions considers the EPA's assessment of the current scientific evidence for PM<sub>10-2.5</sub>-related health effects in the ISA and takes into account the advice received from the CASAC (Cox, 2019) and comments from the members of the public. We revisit the overarching question for this chapter:

- **Does the currently available scientific evidence support or call into question the adequacy of the protection afforded by the current primary PM<sub>10</sub> standard against health effects associated with exposures to PM<sub>10-2.5</sub>?**

In answering this question, we consider the currently available evidence within the context of the rationale supporting the decision in the last review to retain the primary PM<sub>10</sub> standard. We recognize that a final decision on the primary PM<sub>10</sub> standard in the current review will be largely a public health policy judgement in which the Administrator weighs the evidence, including its associated uncertainties.

As discussed in section 4.1.1 above, the decision to retain the primary PM<sub>10</sub> standard in the last review recognized the importance of maintaining some degree of protection against PM<sub>10-2.5</sub> exposures, given the evidence for PM<sub>10-2.5</sub>-related health effects, but noted uncertainties in the potential public health implications of revising the existing PM<sub>10</sub> standard. Regarding evidence for PM<sub>10-2.5</sub>-related health effects, the decision noted that epidemiologic studies had reported positive associations between PM<sub>10-2.5</sub> and mortality or morbidity in cities across North America, Europe, and Asia, encompassing a variety of environments where PM<sub>10-2.5</sub> sources and composition are expected to vary widely. Although most of these studies examined PM<sub>10-2.5</sub> health effect associations in urban areas, some studies had also linked mortality and morbidity with relatively high ambient concentrations of particles of non-urban crustal origin. Drawing from this evidence, it was judged appropriate to maintain a standard that provides some measure of protection against exposures to PM<sub>10-2.5</sub>, regardless of location, source of origin, or particle composition (78 FR 3176, January 15, 2013). As discussed above in section 4.1.1, it was further judged appropriate to retain the PM<sub>10</sub> indicator given that the varying concentrations of PM<sub>10-2.5</sub> permitted in urban versus non-urban areas under a PM<sub>10</sub> standard, based on the varying levels of PM<sub>2.5</sub> present (i.e., lower PM<sub>10-2.5</sub> concentrations allowed in urban areas, where PM<sub>2.5</sub> concentrations tend to be higher), appropriately reflected differences in the strength of PM<sub>10-2.5</sub>

health effects evidence. With regard to uncertainties, limitations in the estimates of ambient  $PM_{10-2.5}$  used in epidemiologic studies, the limited evaluation of copollutant models to address the potential for confounding, and the limited number of experimental studies supporting biologically plausible pathways for  $PM_{10-2.5}$ -related effects were all highlighted. These and other limitations in the  $PM_{10-2.5}$  evidence raised questions as to whether additional public health improvements would be achieved by revising the existing  $PM_{10}$  standard.

Since the last review, the evidence for several  $PM_{10-2.5}$ -related health effects has expanded, particularly for long-term exposures, broadening our understanding of the range of effects linked to  $PM_{10-2.5}$  exposures. As in the last review, epidemiologic studies continue to report positive associations with mortality or morbidity in cities across North America, Europe, and Asia, where  $PM_{10-2.5}$  sources and composition are expected to vary widely. Such studies provide an important part of the body of evidence supporting the strengthened causality determinations (and new determinations) for long-term  $PM_{10-2.5}$  exposures and mortality, cardiovascular effects, metabolic effects, nervous system effects and cancer (U.S. EPA, 2019). Thus, the scientific evidence that has become available since the last review does not call into question the decision in that review to maintain a primary standard that provides some measure of public health protection against  $PM_{10-2.5}$  exposures, regardless of location, source of origin, or particle composition. In addition, recent epidemiologic studies do not call into question the judgment in the last review that it is appropriate to retain the  $PM_{10}$  indicator, given that the varying concentrations of coarse particles permitted in urban versus non-urban areas under a  $PM_{10}$  standard (i.e., based on the varying concentrations of  $PM_{2.5}$  present) appropriately reflect the differences in the strength of evidence regarding coarse particle health effects.

As in the last review, important uncertainties remain in the evidence base for  $PM_{10-2.5}$ -related health effects. As summarized in section 4.2.1 above, these include uncertainties in the  $PM_{10-2.5}$  exposure estimates used in epidemiologic studies, in the independence of  $PM_{10-2.5}$  health effect associations, and in the biological plausibility of the  $PM_{10-2.5}$ -related effects. Thus, the evidence available in the current review is subject to the same broad uncertainties as were present in the last review. Consistent with the assessment of the evidence in the 2009 ISA (U.S. EPA, 2009), these uncertainties contribute to the determinations in the current ISA that the evidence for key  $PM_{10-2.5}$ -related health effects is “suggestive of, but not sufficient to infer” causal relationships (U.S. EPA, 2019). Drawing from this information, we reach the conclusion that, as in the last review, such uncertainties raise questions regarding the degree to which additional public health improvements would be achieved by revising the existing  $PM_{10}$  standard.

When the above information is taken together, we reach the conclusion that the available evidence does not call into question the scientific judgments that informed the decision in the last

review to retain the current primary PM<sub>10</sub> standard in order to protect against PM<sub>10-2.5</sub> exposures. Specifically, while the available evidence supports maintaining a PM<sub>10</sub> standard to provide some measure of protection against PM<sub>10-2.5</sub> exposures, uncertainties in the evidence lead to questions regarding the potential public health implications of revising the existing PM<sub>10</sub> standard. Thus, consistent with the approach taken in the last review and with the advice from the CASAC in this review, we reach the conclusions that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports consideration of retaining the current standard in this review. As such, we have not evaluated alternative standards in this PA.

## **4.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

As discussed above, a number of key uncertainties and limitations in the health evidence have been considered in this review. In this section, we highlight areas for future health-related research and data collection activities to address these uncertainties and limitations in the current body of evidence. These efforts, if undertaken, could provide important evidence for informing future reviews of the PM NAAQS. Key areas for future research efforts are summarized below.

- The body of experimental inhalation studies of exposure to PM<sub>10-2.5</sub> (e.g., controlled human exposure and animal toxicology studies) is currently relatively sparse. While coarse PM inhalation studies in rats and mice are complicated by substantial differences in dosimetry (i.e., compared to humans), additional experimental studies of short- or long-term PM<sub>10-2.5</sub> exposures could play an important role in weight of evidence judgments in future ISAs. Experimental evaluation of effects that are plausibly related to the serious health outcomes documented in epidemiologic studies could be particularly informative. Such effects could include changes in markers of cardiovascular or respiratory function, similar to the effects that have been evaluated following PM<sub>2.5</sub> exposures (e.g., vascular function, blood pressure, heart rate and heart rate variability, markers of potential for coagulation, systemic and respiratory inflammation, respiratory function, etc.).
- The potential for exposure error is of particular concern for PM<sub>10-2.5</sub>, given its less homogeneous atmospheric distribution compared to fine particles (U.S. EPA, 2019, section 1.2.1.5) and the relatively sparse PM<sub>10-2.5</sub> monitoring network. Therefore, efforts to develop and validate new exposure estimation approaches, or to further validate existing approaches, would be informative.
- Existing epidemiologic studies have rarely examined associations with PM<sub>10-2.5</sub> in copollutant models, contributing to uncertainty in the degree to which reported health effect associations are independent of potential confounding variables. Additional epidemiologic studies that evaluate copollutants models would be informative.
- Epidemiologic studies currently use a variety of approaches to measure/estimate PM<sub>10-2.5</sub> concentrations, including: (1) difference method with co-located monitors, (2) difference method with area-wide averages of monitored PM<sub>10</sub> and PM<sub>2.5</sub>, (3) difference method

with area-wide averages of modeled PM<sub>10</sub> and PM<sub>2.5</sub> or (4) direct measurement of PM<sub>10-2.5</sub> using a dichotomous sampler. It is important that we better understand how these methods compare to one another, both in terms of absolute estimated concentrations and in terms of the spatial and temporal correlations in those estimated concentrations between methods.

- Measurement capabilities and the availability of PM<sub>10-2.5</sub> ambient concentration data have greatly increased since the 2009 ISA (U.S. EPA, 2019, section 2.5.1.1.3). Starting in 2011, PM<sub>10-2.5</sub> has been monitored at NCore stations, IMPROVE stations, and several sites run by State and local agencies. To date, epidemiologic studies have used a variety of approaches to measure/estimate PM<sub>10-2.5</sub> concentrations but have not used direct measurements from NCore or IMPROVE stations to evaluate health effects associations with PM<sub>10-2.5</sub> exposure. A body of epidemiologic studies that evaluate health effect associations using monitoring data from these stations could allow more direct comparisons of results across studies.
- Evaluate and expand the PM<sub>10-2.5</sub> network, along with speciation of PM<sub>10-2.5</sub> including multi-elements, major ions, carbon (including carbonate carbon), and bioaerosols
- Characterize PM<sub>10-2.5</sub> in different health-relevant exposure environments (e.g., city center, suburban, roadside, agricultural, and rural areas) for mass, elements (including potential toxic species), carbonaceous materials (including selected organic compounds and carbonate), water-soluble ions, and bioaerosols (including endotoxins, 1,3 beta glucan, and total protein).
- Additional areas of interest for future research include:
  - Further evaluation of the potential for particular PM<sub>10-2.5</sub> components, groups of components, or other particle characteristics to contribute to exposure-related health effects.
  - Research to improve our understanding of concentration-response relationships and the confidence bounds around these relationships, especially at lower ambient PM<sub>10-2.5</sub> concentrations.
  - Identifying novel populations that could be at-risk of PM<sub>10-2.5</sub>-related health effects.
  - Modeling to estimate PM<sub>10-2.5</sub> mass and composition in areas with sparse or less-than-daily monitoring.

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## 5 REVIEW OF THE SECONDARY STANDARDS

This chapter presents key policy-relevant considerations and summary conclusions regarding the public welfare protection provided by the current secondary PM standards to protect against PM-related visibility impairment, climate effects, and materials effects. These considerations and conclusions are framed by a series of policy-relevant questions, including the following overarching question:

- **Does the currently available scientific evidence and quantitative information support or call into question the adequacy of the protection afforded by the current secondary PM standards?**

The answer to this question is informed by evaluation of a series of more specific policy-relevant questions, which expand upon those presented at the outset of this review in the IRP (U.S. EPA, 2016). Answers to these questions are intended to inform decisions by the Administrator on whether, and if so how, to revise the secondary PM standards.

Section 5.1 presents our approach for reviewing the secondary standards for PM. Section 5.2.1 presents our consideration of the available scientific evidence and our consideration of quantitative information for visibility effects, while section 5.2.2 considers the available scientific evidence for each of the non-visibility welfare effects (climate effects and materials effects) separately.<sup>1</sup> Section 5.3 summarizes the advice and recommendations received from the CASAC during its review of the draft PA, and by public comments received on the draft document. Conclusions regarding the public welfare protection provided by the current secondary PM standards are summarized in section 5.4. Section 5.5 discusses areas for future research and data collection to improve our understanding of PM-related welfare effects in future reviews.

### 5.1 APPROACH

In the last review of the PM NAAQS, completed in 2012, the EPA retained the secondary 24-hour PM<sub>2.5</sub> standard, with its level of 35 µg/m<sup>3</sup>, and the 24-hour PM<sub>10</sub> standard, with its level of 150 µg/m<sup>3</sup> (78 FR 3228, January 15, 2013). The EPA also retained the level, set at 15 µg/m<sup>3</sup>, and averaging time of the annual PM<sub>2.5</sub> standard, while revising the form. With regard to the

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<sup>1</sup> Other welfare effects of PM, such as ecological effects, are being considered in the separate, on-going review of the secondary NAAQS for oxides of nitrogen and oxides of sulfur. Accordingly, the public welfare protection provided by the secondary PM standards against ecological effects such as those related to deposition of nitrogen- and sulfur-containing compounds in vulnerable ecosystems is being considered in that separate review. Thus, the Administrator's conclusion in this review will be focused only and specifically on the adequacy of public welfare protection provided by the secondary PM standards from effects related to visibility, climate, and materials.

form of the annual PM<sub>2.5</sub> standard, the EPA removed the option for spatial averaging (78 FR 3228, January 15, 2013). Key aspects of the Administrator’s decisions on the secondary PM standards for non-visibility effects and visibility effects are described below in section 5.1.1.

### **5.1.1 Approach Used in the Last Review**

The 2012 decision on the adequacy of the secondary PM standards was based on consideration of the protection provided by those standards for visibility and for the non-visibility effects of materials damage, climate effects and ecological effects. As noted earlier, the current review of the public welfare protection provided by the secondary PM standards against ecological effects is occurring in the separate, on-going review of the secondary NAAQS for oxides of nitrogen and oxides of sulfur. Thus, the consideration of ecological effects in the 2012 review is not discussed here. Rather, the sections below focus on the Administrator’s consideration of climate and materials effects (section 5.1.1.1) and visibility effects (section 5.1.1.2).

#### **5.1.1.1 Non-Visibility Effects**

With regard to the role of PM in climate, the Administrator considered whether it was appropriate to establish any distinct secondary PM standards to address welfare effects associated with climate impacts. In considering the scientific evidence, she noted the 2009 ISA conclusion “that a causal relationship exists between PM and effects on climate” and that aerosols<sup>2</sup> alter climate processes directly through radiative forcing and by indirect effects on cloud brightness, changes in precipitation, and possible changes in cloud lifetimes (U.S. EPA, 2009, section 9.3.10). Additionally, the major aerosol components with the potential to affect climate processes (i.e., black carbon (BC), organic carbon (OC), sulfates, nitrates and mineral dusts) vary in their reflectivity, forcing efficiencies, and direction of climate forcing (U.S. EPA, 2009, section 9.3.10).

Noting the strong evidence indicating that aerosols affect climate, the Administrator further considered what the available information indicated regarding the adequacy of protection provided by the secondary PM standards. She noted that a number of uncertainties in the scientific information affected our ability to quantitatively evaluate the standards in this regard.

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<sup>2</sup> In the climate sciences research community, PM is encompassed by what is typically referred to as aerosol. An aerosol is defined as a solid or liquid suspended in a gas, but PM refers to the solid or liquid phase of an aerosol. In this review of the secondary PM NAAQS the discussion on climate effects of PM uses the term PM throughout for consistency with the ISA (U.S. EPA, 2019) as well as to emphasize that the climate processes altered by aerosols are generally altered by the PM portion of the aerosol. Exceptions to this practice include the discussion of climate effects in the last review, when aerosol was used when discussing suspending aerosol particles, and for certain acronyms that are widely used by the climate community that include the term aerosol (e.g., aerosol optical depth, or AOD).

For example, the ISA and PA noted the spatial and temporal heterogeneity of PM components that contribute to climate forcing, uncertainties in the measurement of aerosol components, inadequate consideration of aerosol impacts in climate modeling, insufficient data on local and regional microclimate variations and heterogeneity of cloud formations. In light of these uncertainties and the lack of sufficient data, the 2011 PA concluded that it was not feasible in the last review “to conduct a quantitative analysis for the purpose of informing revisions [to the secondary PM NAAQS] based on climate” (U.S. EPA, 2011, pp. 5-11 to 5-12) and that there was insufficient information available to base a national ambient air quality standard on climate impacts associated with ambient air concentrations of PM or its constituents (U.S. EPA, 2011, section 5.2.3). The Administrator agreed with this conclusion (78 FR 3225-3226, January 15, 2013).

With regard to materials effects, the Administrator also considered effects associated with the deposition of PM (i.e., dry and wet deposition), including both physical damage (materials effects) and aesthetic qualities (soiling effects). The deposition of PM can physically affect materials, adding to the effects of natural weathering processes, by promoting or accelerating the corrosion of metals; by degrading paints; and by deteriorating building materials such as stone, concrete, and marble (U.S. EPA, 2009, section 9.5). Additionally, the deposition of PM from ambient air can reduce the aesthetic appeal of buildings and objects through soiling. The ISA concluded that evidence was “sufficient to conclude that a causal relationship exists between PM and effects on materials” (U.S. EPA, 2009, sections 2.5.4 and 9.5.4). However, the 2011 PA noted that quantitative relationships were lacking between particle size, concentrations, and frequency of repainting and repair of surfaces and that considerable uncertainty exists in the contributions of co-occurring pollutants to materials damage and soiling processes (U.S. EPA, 2011, p. 5-29). The 2011 PA concluded that none of the evidence available in the last review called into question the adequacy of the existing secondary PM standards to protect against material effects (U.S. EPA, 2011, p. 5-29). The Administrator agreed with this conclusion (78 FR 3225-3226, January 15, 2013).

In considering non-visibility welfare effects in the last review, as discussed above, the Administrator concluded that, while it is important to maintain an appropriate degree of control of fine and coarse particles to address non-visibility welfare effects, “[i]n the absence of information that would support any different standards...it is appropriate to retain the existing suite of secondary standards” (78 FR 3225-3226, January 15, 2013). Her decision was consistent with the CASAC advice related to non-visibility effects. Specifically, the CASAC agreed with the 2011 PA conclusions that, while these effects are important, “there is not currently a strong technical basis to support revisions of the current standards to protect against these other welfare effects” (Samet, 2010, p. 5). Thus, the Administrator concluded that it was appropriate to retain



all aspects of the existing 24-hour PM<sub>2.5</sub> and PM<sub>10</sub> secondary standards. With regard to the secondary annual PM<sub>2.5</sub> standard, the Administrator concluded that it was appropriate to retain a level of 15.0 µg/m<sup>3</sup> while revising only the form of the standard to remove the option for spatial averaging (78 FR 3225-3226, January 15, 2013).

#### **5.1.1.2 Visibility Effects**

Having reached the conclusion to retain the existing secondary PM standards to protect against non-visibility welfare effects, the Administrator next considered the level of protection that would be requisite to protect public welfare against PM-related visibility impairment and whether to adopt a distinct secondary standard to achieve this level of protection. In reaching her final decision that the existing 24-hour PM<sub>2.5</sub> standard provides sufficient protection against PM-related visibility impairment (78 FR 3228, January 15, 2013), the Administrator considered the evidence assessed in the 2009 ISA (U.S. EPA, 2009) and the analyses included in the Urban-Focused Visibility Assessment (2010 UFVA; U.S. EPA, 2010) and the 2011 PA (U.S. EPA, 2011). She also considered the degree of protection for visibility that would be provided by the existing secondary standard, focusing specifically on the secondary 24-hour PM<sub>2.5</sub> standard with its level of 35 µg/m<sup>3</sup>. These considerations, and the Administrator's conclusions regarding visibility are discussed in more detail below.

In the last review, the ISA concluded that, “collectively, the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment” (U.S. EPA, 2009, p. 2-28). Visibility impairment is caused by light scattering and absorption by suspended particles and gases, including water content of aerosols.<sup>3</sup> The available evidence in the last review indicated that specific components of PM have been shown to contribute to visibility impairment. For example, at sufficiently high relative humidity values, sulfate and nitrate are the PM components that scatter more light and thus contribute most efficiently to visibility impairment. Elemental carbon (EC) and OC are also important contributors, especially in the northwestern U.S. where their contribution to PM<sub>2.5</sub> mass is higher. Crustal materials can be significant contributors to visibility impairment, particularly for remote areas in the arid southwestern U.S. (U.S. EPA, 2009, section 2.5.1).

Visibility impairment can have implications for people's enjoyment of daily activities and for their overall sense of well-being (U.S. EPA, 2009, section 9.2). In consideration of the potential public welfare implication of various degrees of PM-related visibility impairment, the

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<sup>3</sup> All particles scatter light and, although a larger particle scatters more light than a similarly shaped smaller particle of the same composition, the light scattered per unit of mass is greatest for particles with diameters from ~0.3-1.0 µm (U.S. EPA, 2009, section 2.5.1). Particles with hygroscopic components (e.g., particulate sulfate and nitrate) contribute more to light extinction at higher relative humidity than at lower relative humidity because they change size in the atmosphere in response to relative humidity.

Administrator considered the available visibility preference studies that were part of the overall body of evidence in the 2009 ISA and reviewed as a part of the 2010 UFVA. These preference studies provided information about the potential public welfare implications of visibility impairment from surveys in which participants were asked questions about their preferences or the values they placed on various visibility conditions, as displayed to them in scenic photographs or in images with a range of known light extinction levels.<sup>4</sup>

In noting the relationship between PM concentrations and PM-related light extinction, the Administrator focused on identifying an adequate level of protection against visibility-related welfare effects. She first concluded that a standard in terms of a PM<sub>2.5</sub> visibility index would provide a measure of protection against PM-related light extinction that directly takes into account the factors (i.e., species composition and relative humidity) that influence the relationship between PM<sub>2.5</sub> in ambient air and PM-related visibility impairment. A PM<sub>2.5</sub> visibility index standard would afford a relatively high degree of uniformity of visual air quality protection in areas across the country by directly incorporating the effects of differences of PM<sub>2.5</sub> composition and relative humidity. In defining a target level of protection in terms of a PM<sub>2.5</sub> visibility index, as discussed below, the Administrator considered specific elements of the index, including the basis for its derivation, as well as an appropriate averaging time, level, and form.

With regard to the basis for derivation of a visibility index, the Administrator concluded that it was appropriate to use an adjusted version of the original IMPROVE algorithm,<sup>5</sup> in conjunction with monthly average relative humidity data based on long-term climatological means. In so concluding, the Administrator noted the CASAC conclusion on the reasonableness of reliance on a PM<sub>2.5</sub> light extinction indicator calculated from PM<sub>2.5</sub> chemical composition and relative humidity. In considering alternative approaches for a focus on visibility, the Administrator recognized that the available mass monitoring methods did not include measurement of the full water content of ambient PM<sub>2.5</sub>, nor did they provide information on the composition of PM<sub>2.5</sub>, both of which contribute to visibility impacts (77 FR 38980, June 29, 2012). In addition, at the time of the proposal, the Administrator recognized that suitable equipment and performance-based verification procedures did not then exist for direct

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<sup>4</sup> Preference studies were available in four urban areas in the last review. Three western preference studies were available, including one in Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009). More details about these studies are available in Appendix D.

<sup>5</sup> The revised IMPROVE algorithm (Pitchford et al., 2007) uses major PM chemical composition measurements and relative humidity estimates to calculate light extinction. For more information about the derivation of and input data required for the original and revised IMPROVE algorithms, see 78 FR 3168-3177, January 15, 2013.

measurement of light extinction and could not be developed within the time frame of the review (77 FR 38980-38981, June 29, 2012).

With regard to the averaging time of the index, the Administrator concluded that a 24-hour averaging time would be appropriate for a visibility index (78 FR 3226, January 15, 2013). Although she recognized that hourly or sub-daily (4- to 6-hour) averaging times, within daylight hours and excluding hours with relatively high humidity, are more directly related to the short-term nature of the perception of PM-related visibility impairment and relevant exposure periods for segments of the viewing public than a 24-hour averaging time, she also noted that there were data quality uncertainties associated with the instruments used to provide the hourly PM<sub>2.5</sub> mass measurements required for an averaging time shorter than 24 hours. The Administrator also considered the results of analyses that compared 24-hour and 4-hour averaging times for calculating the index. These analyses showed good correlation between 24-hour and 4-hour average PM<sub>2.5</sub> light extinction, as evidenced by reasonably high city-specific and pooled R-squared values, generally in the range of over 0.6 to over 0.8. Based on these analyses and the 2011 PA conclusions regarding them, the Administrator concluded that a 24-hour averaging time would be a reasonable and appropriate surrogate for a sub-daily averaging time.

With regard to the statistical form of the index, the Administrator settled on a 3-year average of annual 90<sup>th</sup> percentile values. In so doing, she noted that a 3-year average form provided stability from the occasional effect of inter-annual meteorological variability that can result in unusually high pollution levels for a particular year (78 FR 3198, January 15, 2013; U.S. EPA, 2011, p. 4-58). Regarding the annual statistic to be averaged, the 2010 UFVA evaluated three different statistics: 90<sup>th</sup>, 95<sup>th</sup>, and 98<sup>th</sup> percentiles (U.S. EPA, 2010, chapter 4). In considering these alternative percentiles, the 2011 PA noted that the Regional Haze Program targets the 20 percent most impaired days for improvements in visual air quality in Federal Class I areas and that the median of the distribution of these 20 percent worst days would be the 90<sup>th</sup> percentile. The 2011 PA further noted that strategies that are implemented so that 90 percent of days would have visual air quality that is at or below the level of the standard would reasonably be expected to lead to improvements in visual air quality for the 20 percent most impaired days. Lastly, the 2011 PA recognized that the available studies on people's preferences did not address frequency of occurrence of different levels of visibility and did not identify a basis for a different target for urban areas than that for Class I areas (U.S. EPA, 2011, p. 4-59). These considerations led the Administrator to conclude that 90<sup>th</sup> percentile form was the most appropriate annual statistic to be averaged across three years (78 FR 3226, January 15, 2013).

With regard to the level of the index, the Administrator considered the visibility preferences studies conducted in four urban areas (U.S. EPA, 2011, p. 4-61). Based on these

studies, the PA identified a range of levels from 20 to 30 deciviews (dv)<sup>6</sup> as being a reasonable range of “candidate protection levels” (CPLs).<sup>7</sup> In considering this range of CPLs, the Administrator noted the uncertainties and limitations in public preference studies, including the small number of stated preference studies available; the relatively small number of study participants and the extent to which the study participants may not be representative of the broader study area population in some of the studies; and the variations in the specific materials and methods used in each study. She concluded that the substantial degrees of variability and uncertainty in the public preference studies should be reflected in a target protection level at the upper end of the range of CPLs than if the information were more consistent and certain. Therefore, the Administrator concluded that it was appropriate to set a target level of protection in terms of a 24-hour PM<sub>2.5</sub> visibility index at 30 dv (78 FR 3226-3227, January 15, 2013).

Based on her considerations and conclusions summarized above, the Administrator concluded that the protection provided by a secondary standard based on a 3-year visibility metric, defined in terms of a PM<sub>2.5</sub> visibility index with a 24-hour averaging time, a 90<sup>th</sup> percentile form averaged over 3 years, and a level of 30 dv, would be requisite to protect public welfare with regard to visual air quality (78 FR 3227, January 15, 2013). Having reached this conclusion, she next determined whether an additional distinct secondary standard in terms of a visibility index was needed given the degree of protection from visibility impairment afforded by the existing secondary standards. Specifically, she noted that the air quality analyses showed that all areas meeting the existing 24-hour PM<sub>2.5</sub> standard, with its level of 35 µg/m<sup>3</sup>, had visual air quality at least as good as 30 dv, based on the visibility index defined above (Kelly et al., 2012b, Kelly et al., 2012a). Thus, the secondary 24-hour PM<sub>2.5</sub> standard would likely be controlling relative to a 24-hour visibility index set at a level of 30 dv. Additionally, areas would be unlikely to exceed the target level of protection for visibility of 30 dv without also exceeding the existing secondary 24-hour standard. Thus, the Administrator judged that the 24-hour PM<sub>2.5</sub> standard “provides sufficient protection in all areas against the effects of visibility impairment – i.e., that the existing 24-hour PM<sub>2.5</sub> standard would provide *at least* the target level of protection for visual air quality of 30 dv which the Administrator judges appropriate” (78 FR 3227, January 15, 2013). She further judged that “[s]ince sufficient protection from visibility impairment would be provided for all areas of the country without adoption of a distinct secondary standard, and adoption of a distinct secondary standard will not change the degree of over-protection for some areas of the country...adoption of such a distinct secondary standard is not needed to provide

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<sup>6</sup> Deciview (dv) refers to a scale for characterizing visibility that is defined directly in terms of light extinction. The deciview scale is frequently used in the scientific and regulatory literature on visibility.

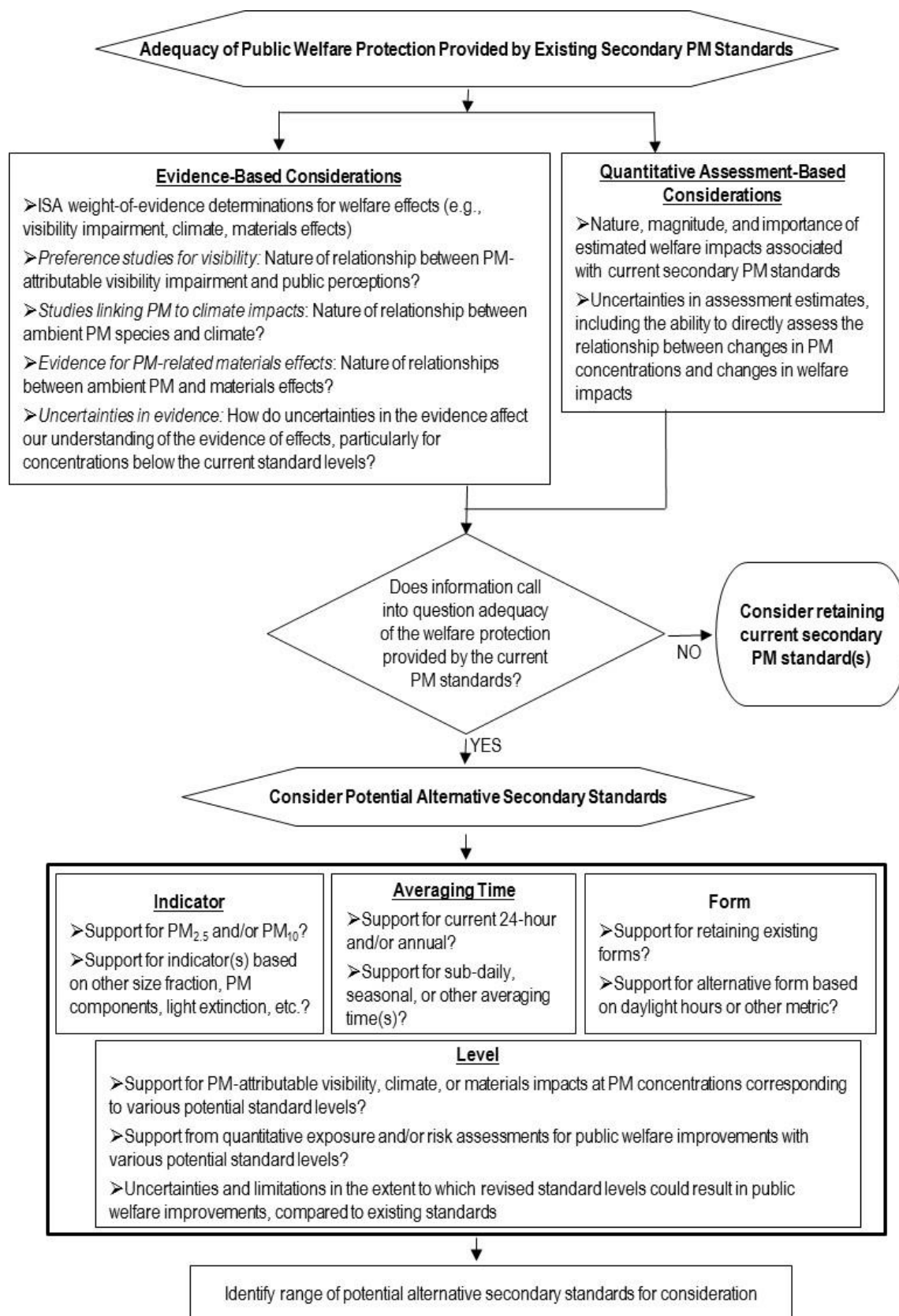
<sup>7</sup> For comparison, 20 dv, 25 dv, and 30 dv are equivalent to 64, 112, and 191 megameters (Mm<sup>-1</sup>), respectively.

requisite protection for both visibility and nonvisibility related welfare effects” (78 FR 3228, January 15, 2013).

### **5.1.2 General Approach Used in the Current Review**

To evaluate whether it is appropriate to consider retaining the current suite of secondary PM standards, or whether consideration of revision is appropriate, we have adopted an approach in this review that builds on the general approach used in the last review and reflects the body of evidence and information now available. As summarized above, past approaches have been based most fundamentally on using information from PM visibility studies and quantitative analyses of PM-related visibility impairment to inform the selection of secondary PM standards that, in the Administrator’s judgment, protect the public welfare from any known or anticipated effects. These fundamental considerations are again the basis for our approach in this review.

In conducting this assessment, we draw on the current evidence and quantitative assessments of visibility impairment associated with PM in ambient air. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since the last review, including the evidence assessed in the ISA and updated air quality-based analyses (Appendix D). Figure 5-1 below illustrates our general approach in developing conclusions regarding the adequacy of the current secondary standards and, as appropriate, potential alternative standards. In the boxes in Figure 5-1, the range of questions that we consider in sections 5.2.1 and 5.2.2 below are represented by a summary of policy-relevant questions that frame our consideration of the scientific evidence and quantitative analyses.



**Figure 5-1. Overview of general approach for review of secondary PM standards.**

## 5.2 ADEQUACY OF THE CURRENT SECONDARY PM STANDARDS

In considering the available evidence for welfare effects attributable to PM as presented in the ISA, this section poses the following policy-relevant questions:

- **Does the currently available scientific evidence and quantitative information support or call into question the adequacy of the welfare protection afforded by the current secondary PM standards?**

In answering this question, we have posed a series of more specific questions to aid in considering the currently available scientific evidence and quantitative information, as discussed below. In considering the scientific and technical information, we reflect upon both the information available in the last review and information that is newly available since the last review as assessed and presented in the ISA (U.S. EPA, 2019), focusing on welfare effects for which the evidence supports either a “causal” or a “likely to be causal” relationship as described in the Preamble to the ISA (U.S. EPA, 2015). Table 5-1 lists such causality determinations from the ISA for welfare effects. As in the last review, the evidence is sufficient to support a causal relationship between PM and visibility effects (section 5.2.1), climate effects (section 5.2.2) and materials effects (section 5.2.2).

**Table 5-1. Key causality determinations for PM-related welfare effects.**

Effect	2009 PM ISA	2019 PM ISA
Visibility effects	Causal	Causal
Climate effects	Causal	Causal
Materials effects	Causal	Causal

### 5.2.1 Visibility Effects

In the sections below, we consider the nature of visibility-related effects attributable to PM (section 5.2.1.1) and the quantitative information currently available (section 5.2.1.2).

#### 5.2.1.1 Evidence-Based Considerations

In considering the available evidence of visibility welfare effects attributable to PM as presented in the ISA, this section addresses the following policy-relevant questions:

- **Does the current evidence alter our conclusions from the last review regarding the nature of visibility effects attributable to PM in ambient air?**

Visibility refers to the visual quality of a human’s view with respect to color rendition and contrast definition. It is the ability to perceive landscape form, colors, and textures. Visibility involves optical and psychophysical properties involving human perception, judgment, and interpretation. Light between the observer and the object can be scattered into or out of the sight

path and absorbed by PM or gases in the sight path. As recognized above, the conclusion of the ISA that “the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment” is consistent with conclusions of causality in the last review (U.S. EPA, 2019, section 13.2.6). These conclusions are based on strong and consistent evidence that ambient PM can impair visibility in both urban and remote areas (U.S. EPA, 2009, section 9.2.5).

These subsequent questions consider the characterization and quantification of light extinction and preferences associated with varying degrees of visibility impairment.

- **To what extent is new information available that changes or enhances our understanding of the physics of light extinction and/or its quantification (e.g., through light extinction or other monitoring methods or through algorithms such as IMPROVE)?**

Our understanding of the relationship between light extinction and PM mass has changed little since the 2009 ISA (U.S. EPA, 2009). The combined effect of light scattering and absorption by particles and gases is characterized as light extinction, i.e., the fraction of light that is scattered or absorbed per unit of distance in the atmosphere. Light extinction is measured in units of 1/distance, which is often expressed in the technical literature as visibility per megameter (abbreviated  $\text{Mm}^{-1}$ ). Higher values of light extinction (usually given in terms of  $\text{Mm}^{-1}$  or  $\text{dv}$ ) correspond to lower visibility. When PM is present in the air, its contribution to light extinction is typically much greater than that of gases (U.S. EPA, 2019, section 13.2.1). The impact of PM on light scattering depends on particle size and composition, as well as relative humidity. All particles scatter light, as described by the Mie theory, which relates light scattering to particle size, shape and index of refraction (U.S. EPA, 2019, section 13.2.3; Van de Hulst, 1981; Mie, 1908). Fine particles scatter more light than coarse particles on a per unit mass basis and include sulfates, nitrates, organics, light-absorbing carbon, and soil (Malm et al., 1994). Hygroscopic particles like ammonium sulfate, ammonium nitrate, and sea salt increase in size as relative humidity increases, leading to increased light scattering (U.S. EPA, 2019, section 13.2.3).

Direct measurements of PM light extinction, scattering, and absorption are considered more accurate for quantifying visibility impairment than PM mass-based estimates because they do not depend on assumptions about particle characteristics (e.g., size, shape, density, component mixture, etc.). Measurements of light extinction can be made with high time resolution, allowing for characterization of subdaily temporal patterns of visibility impairment. Measurement methods include transmissometers for measurement of light extinction and the determination of visual range and integrating nephelometers for measurement of light scattering, as well as teleradiometers and telephotometers, and photography and photographic modeling (U.S. EPA, 2009; U.S. EPA, 2004). While some recent research confirms and adds to the body of knowledge



available regarding direct measurements as is described in the ISA, no major new developments have been made with these measurement methods since the last review (U.S. EPA, 2019, section 13.2.2.2).

A theoretical relationship between light extinction and PM characteristics has been derived from Mie theory (U.S. EPA, 2019, Equation 13-5) and can be used to estimate light extinction by combining mass scattering efficiencies of particles with particle concentrations (U.S. EPA, 2019, section 13.2.3; U.S. EPA, 2009, sections 9.2.2.2 and 9.2.3.1). However, routine ambient air monitoring rarely includes measurements of particle size and composition information with sufficient detail for these calculations. Accordingly, a much simpler algorithm has been developed to make estimating light extinction more practical.

This algorithm, known as the IMPROVE algorithm,<sup>8</sup> provides for the estimation of light extinction ( $b_{ext}$ ), in units of  $Mm^{-1}$ , using routinely monitored components of fine ( $PM_{2.5}$ ) and coarse ( $PM_{10-2.5}$ ) PM. Relative humidity data are also needed to estimate the contribution by liquid water that is in solution with the hygroscopic components of PM. To estimate each component's contribution to light extinction, their concentrations are multiplied by extinction coefficients and are additionally multiplied by a water growth factor that accounts for their expansion with moisture. Both the extinction efficiency coefficients and water growth factors of the IMPROVE algorithm have been developed by a combination of empirical assessment and theoretical calculation using particle size distributions associated with each of the major aerosol components (U.S. EPA, 2019, section 13.2.3.1, section 13.2.3.3).

The *original IMPROVE algorithm* (Equation D-1 in Appendix D), so referenced here to distinguish it from subsequent variations developed later, was found to underestimate the highest light scattering values and overestimate the lowest values at IMPROVE monitors throughout the U.S. (Malm and Hand, 2007; Ryan et al., 2005; Lowenthal and Kumar, 2004) and at sites in China (U.S. EPA, 2019, section 13.2.3.3). To resolve these biases, a *revised IMPROVE equation*, shown in Equation D-2 in Appendix D, was developed (Pitchford et al., 2007) that divides PM components into smaller and larger sizes of particles in  $PM_{2.5}$ , with separate mass scattering efficiencies and hygroscopic growth functions for each size category. The revised IMPROVE equation was described in detail in the 2009 ISA (U.S. EPA, 2009) and it both reduced bias at the lowest and highest scattering values and improved the accuracy of the calculated light  $b_{ext}$ .

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<sup>8</sup> The algorithm is referred to as the IMPROVE algorithm as it was developed specifically to use monitoring data generated at IMPROVE network sites and with equipment specifically designed to support the IMPROVE program and was evaluated using IMPROVE optical measurements at the subset of monitoring sites that make those measurements (Malm et al., 1994).

However, poorer precision was observed with the revised IMPROVE equation compared to the original IMPROVE equation (U.S. EPA, 2009).<sup>9</sup>

Since the time of the last review, Lowenthal and Kumar (2016) have tested and evaluated a number of modifications to the revised IMPROVE equation based on evaluations of monitoring data from remote IMPROVE sites. In these locations, they observed that the multiplier to estimate the concentration of organic matter, [OM], from the concentration of organic carbon, [OC], was closer to 2.1 than the value of 1.8 used in the revised IMPROVE equation.<sup>10</sup> They also observed that water soluble organic matter absorbs water as a function of relative humidity, which is not accounted for in either the original or revised IMPROVE equations and was therefore underestimated in these equations. They further suggested that light scattering by sulfate was overestimated because the assumption that all sulfate is fully neutralized ammonium sulfate is not always true (U.S. EPA, 2019, section 13.2.3.3). Modifications based on these points are reflected in Equation D-3 in Appendix D.

In summary, rather than altering our understanding from the previous review, we continue to recognize that direct measurements are better at characterizing light extinction than estimating light extinction with an algorithm. However, in the absence of advances in the monitoring methods and/or network for directly measuring light extinction, the use of the IMPROVE equation for estimating light extinction continues to be supported by the evidence, with some new refinements to the inputs of the IMPROVE equation. Accordingly, as in the last review, the current review focuses on calculated light extinction when quantifying visibility impairment resulting from recent concentrations of PM in ambient air.

- **What does the available information indicate with regard to factors that influence light extinction and visibility, as well as variation in these factors and resulting light extinction across the U.S.?**

The ISA provides a comprehensive discussion of the spatial and temporal patterns of PM<sub>2.5</sub> composition and its contribution to light extinction from IMPROVE and CSN monitoring

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<sup>9</sup> In the most recent IMPROVE report, a combination of the original and revised IMPROVE equations (the *modified original IMPROVE equation*) was used (Hand et al., 2011). This equation uses the sea salt term of the revised equation but does not subdivide the components into two size classes. Further, it uses a factor of 1.8 to estimate organic matter from organic carbon concentrations and also replaces the constant value of 10 Mm<sup>-1</sup> used for Rayleigh scattering in the original and revised equations with a site-specific term based on elevation and mean temperature.

<sup>10</sup> In areas near sources, PM is often less oxygenated, and therefore, in these locations, much of the organic PM mass is present as OC (Jimenez et al., 2009). In areas further away from PM sources, organic PM mass is often more oxygenated as a result of photochemical activity and interactions with other PM and gaseous components in the atmosphere (Jimenez et al., 2009). Under these conditions, the multiplier to convert OC to OM may be higher than in locations with less aged organic PM.

sites, which are mostly rural and urban, respectively.<sup>11</sup> The data from these sites for the periods of 2005-2008 and 2011-2014 were used in the ISA to identify differences in species contributing to light extinction in urban and rural areas by region and season. This is an expansion over the analysis in the 2009 ISA, in that the measurements at that time were primarily based measurements from monitors located in rural areas and at remote sites (U.S. EPA, 2019, section 13.2.4.1, Figures 13-1 through 13-14).

Focusing on the more recent time period of 2011-2014, some major differences in estimated light extinction are apparent among regions of the U.S. Annual average calculated  $b_{ext}$  was considerably greater in the East and Midwest than in the Southwest. Based on IMPROVE data, annual average  $b_{ext}$  was greater than  $40 \text{ Mm}^{-1}$  in the Southeast, East Coast, Mid-South, Central Great Plains, and Appalachian regions, with the highest annual average  $b_{ext}$  (greater than  $50 \text{ Mm}^{-1}$ ) in the Ohio River Valley,<sup>12</sup> while annual average  $b_{ext}$  was below  $40 \text{ Mm}^{-1}$  for all Western IMPROVE regions. Annual average  $b_{ext}$  values were also generally higher in the East than the West based on CSN data, although the highest annual average  $b_{ext}$  was in the Sacramento/San Joaquin Valley and Los Angeles areas (U.S. EPA, 2019, section 13.2.4.1, Figure 13-1, Figure 13-3, Figure 13-5).

Components of  $\text{PM}_{2.5}$  contributing to light extinction vary regionally. For example, in the Eastern regions, ammonium sulfate accounted for approximately 35 to 60% of the annual average  $b_{ext}$ , with the greatest contributions typically occurring in the summer. The second greatest contribution to light extinction came from particulate organic matter (POM), ranging from about 20 to 30% of annual average  $b_{ext}$  with less seasonal variation than ammonium sulfate. Ammonium nitrate also contributed approximately 10% to 35% of annual average  $b_{ext}$ , with much higher concentrations in the winter than in the summer (U.S. EPA, 2019, section 13.2.4.1). In the Northwest, POM was the largest contributor to annual average  $b_{ext}$ , up to 70%, in most urban and rural regions with the greatest contributions in the fall. This seasonal contribution of POM may be related to wildfires. A few exceptions included Boise and sites in North Dakota, where ammonium nitrate was the greatest contributor, and sites in the Alaska IMPROVE region, where ammonium sulfate was the greatest contributor (U.S. EPA, 2019, section 13.2.4.1). In the Southwest, based on IMPROVE data, ammonium sulfate or POM were generally the greatest contributors to annual average  $b_{ext}$ , with nearly equivalent contributions in several regions. Based on CSN data, ammonium nitrate was often the greatest contributor, with especially high  $b_{ext}$  contributions in the winter. While  $\text{PM}_{10-2.5}$  mass scattering was relatively small in the eastern and

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<sup>11</sup> Monitors were grouped into 28 IMPROVE regions and 31 CSN regions based on site location and PM concentrations for major species. For comparison purposes, and where possible, CSN regions were defined similarly to those for the IMPROVE network (Hand et al., 2011; U.S. EPA, 2019, section 13.2.4.1).

<sup>12</sup> A  $b_{ext}$  value of  $40 \text{ Mm}^{-1}$  corresponds to a visual range of about 100 km.

northwestern U.S., in the Southwest, PM<sub>10-2.5</sub> mass scattering contributed to more than 20% of light extinction (U.S. EPA, 2019, section 13.2.4.1).

Differences also exist between the urban CSN and the mainly rural IMPROVE data. Light extinction is generally higher in CSN regions than the geographically corresponding IMPROVE regions. Annual average  $b_{ext}$  was greater than 50 Mm<sup>-1</sup> in 11 CSN regions, compared to only one IMPROVE region, and was greater than 20 Mm<sup>-1</sup> in all CSN regions, compared to just over half of the IMPROVE regions. Light absorbing carbon was the greatest contributor to light extinction in several Western CSN regions but was not a large contributor in any of the IMPROVE regions (U.S. EPA, 2019, Figure 13-11). Ammonium nitrate also accounted for more light extinction in the CSN regions, while it was only a top contributor to  $b_{ext}$  in one IMPROVE region (U.S. EPA, 2019, section 13.2.4.1).

From the 2005-2008 time period to the 2011-2014 time period, the annual average  $b_{ext}$  in most CSN regions in the Eastern U.S. decreased by more than 20 Mm<sup>-1</sup>. This corresponds to an improvement in average visual range in most Eastern U.S. regions of more than 6 Mm<sup>-1</sup> (or 15 km) from 2005-2008 to 2011-2014. Additionally, the contribution of ammonium sulfate to light extinction has also changed over this period. Due to decreased atmospheric sulfate concentrations, the impact on visibility impairment is evident with a smaller fraction of the total  $b_{ext}$  accounted for by ammonium sulfate in 2011-2014 compared to 2005-2008 (U.S. EPA, 2019, section 13.2.4.1).

In summary, the spatial and temporal analysis of PM monitoring network data in the ISA emphasizes that the extent of light extinction by PM<sub>2.5</sub> depends on PM<sub>2.5</sub> composition and relative humidity. Regional differences in PM<sub>2.5</sub> composition greatly influence light extinction. Changes in PM<sub>2.5</sub> composition over time can also affect light extinction based on concentrations of specific PM components in ambient air.

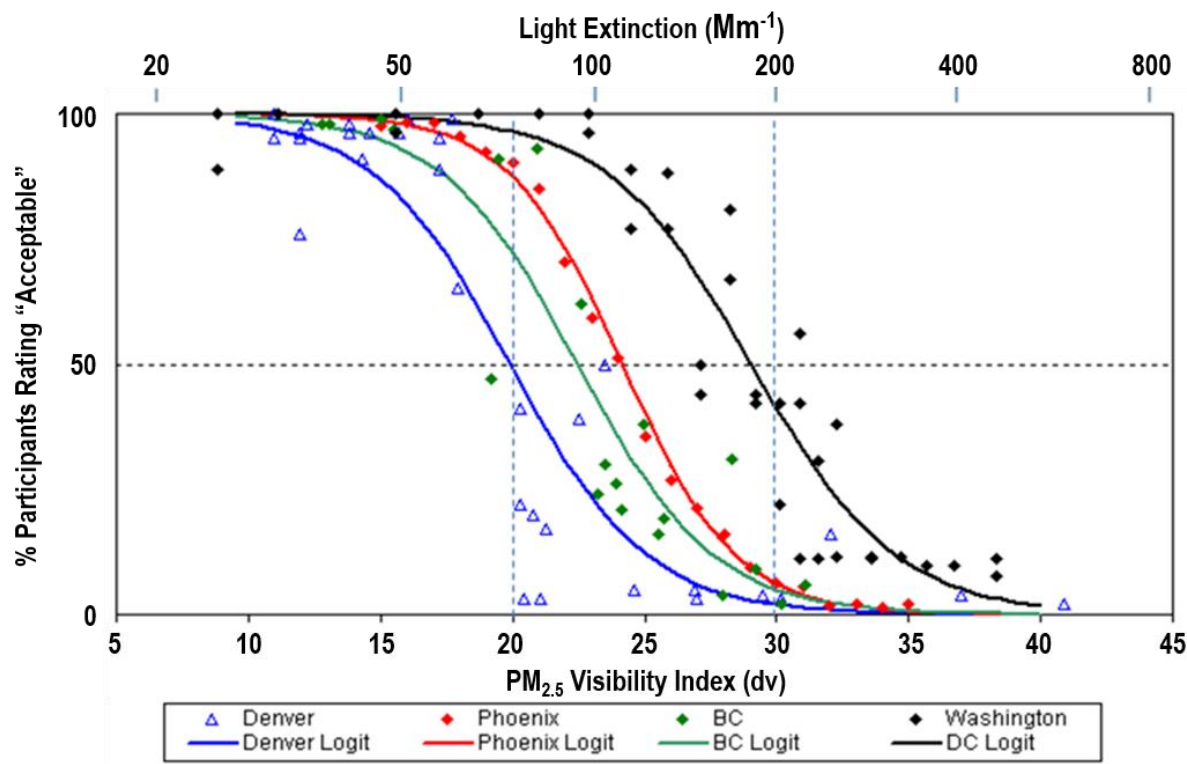
- **To what extent are new studies available that might inform judgments about the potential adversity to public welfare of PM-attributable visibility impairment and the nature of the relationship between PM-attributable visibility impairment and public perceptions of such impairment?**

In the last review, visibility preference studies were available from four areas in North America,<sup>13</sup> as described in section 5.1.1 above. Study participants were queried regarding multiple images that, depending on the study, were either photographs of the same location and scenery that had been taken on different days on which measured extinction data were available

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<sup>13</sup> As noted above, preference studies were available in four urban areas in the last review: Denver, Colorado (Ely et al., 1991, Pryor, 1996), Vancouver, British Columbia, Canada (Pryor, 1996), Phoenix, Arizona (BBC Research & Consulting, 2003), and Washington, DC (Abt Associates, 2001; Smith and Howell, 2009). More details about these studies are available in Appendix D.

or digitized photographs onto which a uniform “haze” had been superimposed. Results of these studies indicated a wide range of judgments on what study participants considered to be acceptable visibility across the different study areas, depending on the setting depicted in each photograph. As a part of the 2010 UFVA, each study was evaluated separately, and figures were developed to display the percentage of participants that rated the visual air quality depicted as “acceptable” (U.S. EPA, 2010). Figure 5-2 represents a graphical summary of the results of the studies in the four cities and identifies a range encompassing the PM<sub>2.5</sub> visibility index values from images that were judged to be acceptable by at least 50% of study participants across all four of the urban preference studies (U.S. EPA, 2010, p. 4-24).<sup>14</sup> As shown in Figure 5-2, much lower visibility (considerably more haze resulting in higher values of light extinction) was considered acceptable in Washington, D.C. than was in Denver. The median judgment for the study groups in the two areas differed by 9.2 dv (which roughly corresponds to about 30 µg/m<sup>3</sup> of PM) (U.S. EPA, 2010).



**Figure 5-2. Relationship of viewer acceptability ratings to light extinction.** (Source: U.S. EPA, 2011, Figure 4-2; U.S. EPA, 2010, Figure 2-16)

<sup>14</sup> Figure 5-2 shows the results of a logistical regression analysis using a logit model of the acceptable or unacceptable ratings from participants of the studies. The logit model is a generalized linear model used for binomial regression analysis which fits explanatory data about binary outcomes (in this case, a person rating an image as acceptable or unacceptable) to a logistic function curve. A detailed description is available in Appendix J of the 2010 UFVA (U.S. EPA, 2010).

Since the time of the last review, no new visibility preference studies have been conducted in the U.S. Outside of the U.S., a visibility preference study was carried out in Beijing, China (Fajardo et al., 2013). This study found a higher range of acceptable visibility impairment among participants than was found in preference studies previously conducted in the U.S. This finding may be related to the common occurrence of higher PM<sub>2.5</sub> concentrations in Beijing (with associated visibility impairment) than is typical in the U.S. (U.S. EPA, 2019, section 13.2.5). Similarly, there is little newly available information regarding acceptable levels of visibility impairment in the U.S.

- **To what extent have important uncertainties in the evidence from the last review been addressed, and have new uncertainties emerged?**

While some refinements have been made to the IMPROVE equation to better estimate light extinction since the last review, there has been no expansion of monitoring efforts for direct measurement of light extinction. At the time of the last review, it was noted that a PM<sub>2.5</sub> light extinction monitoring program could help with characterizing visibility conditions and the relationships between PM component concentrations and light extinction.

Little to no new research is available that helps to expand our understanding of visibility preferences or our characterization of visibility conditions. Uncertainties and limitations consistent with those identified in the last review persist in this review.

- Given the potential for people to have different preferences based on the visibility they are used to based on conditions that they commonly encounter, and the potential for them to also have different preferences for different types of scenes, the currently available preference studies may not capture the range of preferences of people in the U.S.
- The available preference studies were conducted 15 to 30 years ago and may not reflect the visibility preferences of the U.S. population today. Given that air quality has improved over the last several decades, the available studies may not reflect current preferences of people in the U.S.
- The available preference studies have used different methods to evaluate what level of visibility impairment is acceptable. Variability in study methodology may influence an individual's response as to what level of visibility impairment is deemed acceptable, and thereby influence the results of the study.
- Many factors that are not captured by the methods used in the currently available preference studies may influence people's judgments on acceptable visibility. For example, an individual's perception of an acceptable level of visibility impairment could be influenced by the duration of visibility impairment experienced, the time of day during which light extinction is greatest, and the frequency of episodes of visibility impairment, as well as the intensity of the visibility impairment (i.e., the focus of the available studies).

Overall, the body of evidence regarding visibility effects remains largely unchanged since the time of the last review. While one new study provides refinements to the methods for estimating light extinction, uncertainties and limitations in the scientific evidence during the last review remain.

#### **5.2.1.2 Quantitative Assessment-Based Considerations**

Beyond our consideration of the scientific evidence, discussed in section 5.2.1.1 above, we have also considered quantitative analyses of PM air quality and visibility impairment with regard to the extent they could inform conclusions on the adequacy of the public welfare protection provided by the current secondary PM standards. In the last review, quantitative analyses focused on daily visibility impairment, given the short-term nature of PM-related visibility effects. Such quantitative analyses conducted as part of the last review informed the decision on the secondary standards in that review (U.S. EPA, 2010, U.S. EPA, 2011; 78 FR 3189-3192, January 15, 2013). The information newly available in this review includes an updated equation for estimating light extinction, summarized in section 5.2.1.1 above, as well as more recent air monitoring data, that together allow for development of an updated assessment with the potential to substantially add to our understanding of PM-related visibility impairment. Thus, we have conducted updated analyses for this review based on the currently available technical information, tools, and methods.

- **How much visibility impairment is estimated to occur in areas that meet the current secondary PM standards? What are the factors contributing to the estimates in areas with higher values?**

Consistent with the analyses conducted in the last review, we have conducted analyses examining the relationship between PM mass concentrations and calculated light extinction using the 3-year design values<sup>15</sup> for the current secondary standards and a 3-year average visibility metric based on light extinction estimated using IMPROVE equations.<sup>16</sup> These analyses are intended to inform our understanding of visibility impairment in the U.S. under recent air quality conditions, particularly those conditions that meet the current standards, and the relative influence of various factors on light extinction. Given the relationship of visibility with short-term PM, we focus particularly on the short-term PM standards.

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<sup>15</sup> A design value is a statistic that summarizes the air quality data for a given area in terms of the indicator, averaging time, and form of the standard. Design values can be compared to the level of the standard and are typically used to designate areas as meeting or not meeting the standard and assess progress towards meeting the NAAQS.

<sup>16</sup> This is the 3-year visibility metric that was used to evaluate visibility impairment in the last review. Given that there has been almost no new research since the time of the last review to better inform our understanding of visibility preferences in the U.S., there is no new information available to inform selection of a visibility metric for evaluating visibility impairment in the current review different from the one identified in the last review.

Given that visibility-related effects are often associated with short-term PM concentrations, and recognizing the relatively larger role of PM<sub>2.5</sub> and its components in light extinction and as inputs to the IMPROVE equation, we have given somewhat more attention to consideration of the 24-hour PM<sub>2.5</sub> standard. Analyses were conducted using three versions of the IMPROVE equation (Equations D-1 through D-3 in Appendix D) to estimate light extinction to better understand the influence of variability in inputs across the three equations. This analysis included 67 monitoring sites that are geographically distributed across the U.S. in both urban and rural areas (see Figure D-1 in Appendix D). These sites are those that have a valid 24-hour PM<sub>2.5</sub> design value for the 2015-2017 period and met strict criteria for PM species for this analysis.<sup>17</sup> We first present results for these 67 sites using the original IMPROVE equation, with modifications to the equation consistent with those made in evaluating light extinction in the last review (described in detail in section D.1 of Appendix D). We then present results for these 67 sites with light extinction calculated using the Lowenthal and Kumar (2016) IMPROVE equation described in section 5.2.1.1 above. For a subset of 20 of the 67 monitoring sites where PM<sub>10</sub> data were available and met completeness criteria for this analysis, we then present results of a second analysis that included the coarse fraction as an input to the IMPROVE equations for calculating light extinction to better characterize the influence of coarse PM on light extinction.

In considering the relationship between the 24-hour PM<sub>2.5</sub> mass-based design value and the 3-year visibility metric using recent air quality data, we first examine the relationship using the original IMPROVE equation, consistent with the methods used in the last review (Kelly et al., 2012b; 78 FR 3201, January 15, 2013; Appendix D). In those areas that meet the current 24-hour PM<sub>2.5</sub> standard, all sites have light extinction estimates at or below 27 dv (Figure 5-3; 78 FR 3218, January 15, 2013). This is also true for the one location that exceeds the current 24-hour PM<sub>2.5</sub> standard (Figure 5-3). These findings are consistent with the findings of the analysis in the last review that used the same IMPROVE equation with data from 102 sites with data from 2008-2010. This indicates similar findings from this analysis as was the case with the similar analysis in the last review, i.e., the updated quantitative analysis shows that the 3-year visibility metric was no higher than 30 dv<sup>18</sup> at sites meeting the current secondary PM standards, and at

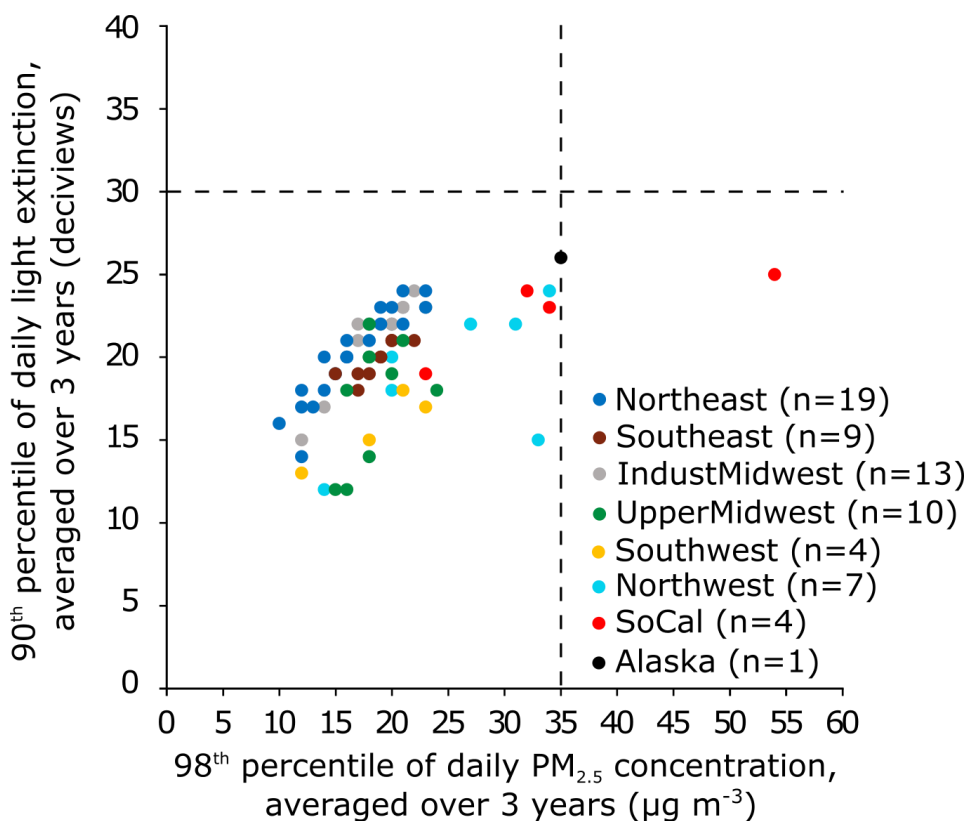
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<sup>17</sup> For this analysis, completeness criteria for speciated PM data at these sites included having all 12 quarters in the 2015-2017 period with at least 11 days in each quarter with a valid PM<sub>2.5</sub> mass, sulfate, nitrate, organic carbon, elemental carbon, sea salt (chlorine or chloride), and fine soil (aluminum, silica, calcium, iron, and titanium) measurement.

<sup>18</sup> For comparison purposes in these air quality analyses, we use a 3-year visibility metric with a level of 30 dv, which is the highest level of visibility impairment judged to be acceptable by at least 50 percent of the participants in the preference studies that were available at the time of the last review (78 FR 3191, January 15, 2013).



most such sites the 3-year visibility index values are much lower (e.g., an average of 20 dv across the 67 sites).

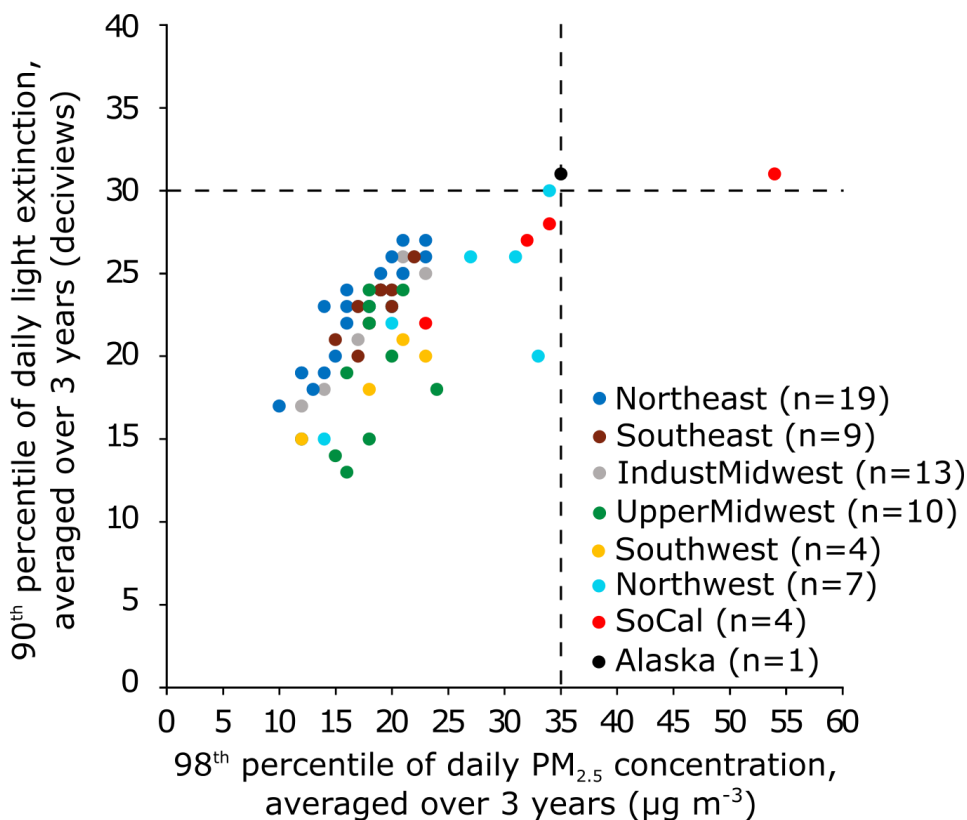


**Figure 5-3. Comparison of 90<sup>th</sup> percentile of daily light extinction, averaged over three years, and 98<sup>th</sup> percentile of daily PM<sub>2.5</sub> concentrations, averaged over three years, for 2015-2017 using the original IMPROVE equation.** (Note: Dashed lines indicate the level of current 24-hour PM<sub>2.5</sub> standard (35 µg/m<sup>3</sup>) and the target level of protection identified for the 3-year visibility metric (30 dv).)

When light extinction was calculated using the refined equation from Lowenthal and Kumar (2016), the resulting 3-year visibility metrics are slightly higher at all sites compared to light extinction estimates calculated using the original IMPROVE equation (Figure 5-4). As noted in section 5.2.1.1, this version of the IMPROVE equation uses a multiplier of 2.1 to convert the measured OC to OM for input into the equation and also accounts for water absorption by water soluble organic matter as a function of relative humidity, likely contributing to the slightly higher estimates of light extinction. As noted in section 5.2.1.1, the Lowenthal and Kumar (2016) refinements to the IMPROVE equation are based on evaluations of monitoring data from remote IMPROVE sites. More remote areas tend to have more aged organic particles than urban areas, and these adjustments to the IMPROVE equation account for the higher concentration of organic matter as a result of more aged organic particles at these sites. It is

important to note that, since the Lowenthal and Kumar (2016) refinements to the IMPROVE equation likely result in one of the higher estimates of light extinction, this equation may overestimate light extinction in non-remote areas, including those urban areas in our analyses.

For those sites that meet the current 24-hour  $\text{PM}_{2.5}$  standard, the 3-year visibility metric is at or below 30 dv when light extinction is calculated using the Lowenthal and Kumar (2016) equation, with the exception of one site in Fairbanks, Alaska. This site just meets the current 24-hour  $\text{PM}_{2.5}$  standard and has a 3-year visibility index value of 31 dv (compared to 27 dv when light extinction is calculated with the original IMPROVE equation) (see Table D-3 in Appendix D). The conditions at this site, however, may differ considerably from those under which the Lowenthal and Kumar (2016) IMPROVE equation, with 2.1 as the multiplier to estimate OM from OC, has been evaluated. Some of these differences, which include higher OC concentrations, with OC as a much higher fraction of OM, much lower temperatures, and the complete lack of sunlight for long periods, may affect the quantitative relationships of OC and OM with visibility (e.g., Hand et al., 2012; Hand et al., 2013).



**Figure 5-4. Comparison of 90<sup>th</sup> percentile of daily light extinction, averaged over three years, and 98<sup>th</sup> percentile of daily  $\text{PM}_{2.5}$  concentrations, averaged over three years, for 2015-2017 using the Lowenthal and Kumar equation.** (Note: Dashed lines indicate the level of current 24-hour  $\text{PM}_{2.5}$  standard ( $35 \mu\text{g/m}^3$ ) and the target level of protection identified for the 3-year visibility metric (30 dv).)

In considering visibility impairment under recent air quality conditions, we recognize that the differences in the inputs to equations estimating light extinction can influence the resulting values. For example, given the varying chemical composition of emissions from different sources, the 2.1 multiplier in the Lowenthal and Kumar (2016) equation may not be appropriate for all source types. At the time of the last review, the EPA judged that a 1.6 multiplier for converting OC to OM was more appropriate, for the purposes of estimating visibility index at sites across the U.S., than the 1.4 or 1.8 multipliers used in the original and revised IMPROVE equations, respectively. A multiplier of 1.8 or 2.1 would account for the more aged and oxygenated organic PM that tends to be found in more remote regions than in urban regions, whereas a multiplier of 1.4 may underestimate the contribution of organic PM found in remote regions when estimating light extinction (78 FR 3206, January 15, 2013; U.S. EPA, 2012b, p. IV-5). The information and analyses available in the current review indicate that it may be appropriate to select inputs to the IMPROVE equation (e.g., the multiplier for OC to OM) on a regional basis rather than a national basis when calculating light extinction. This is especially true when comparing sites with localized PM sources (such as sites in urban or industrial areas) to sites with PM derived largely from biogenic precursor emissions (that contribute to widespread secondary organic aerosol formation), such as those in the southeastern U.S. We note, however, that conditions involving PM from such different sources have not been well studied in the context of applying a multiplier to estimate light extinction, contributing uncertainty to estimates of light extinction for such conditions.

At the time of the last review, the EPA noted that  $PM_{2.5}$  is the size fraction of PM responsible for most of the visibility impairment in urban areas (77 FR 38980, June 29, 2012). Data available at the time of the last review suggested that, generally,  $PM_{10-2.5}$  was a minor contributor to visibility impairment most of the time (U.S. EPA, 2010) although the coarse fraction may be a major contributor in some areas in the desert southwestern region of the U.S. Moreover, at the time of the last review, there were few data available from  $PM_{10-2.5}$  monitors to quantify the contribution of coarse PM to calculated light extinction. Since that time, an expansion in  $PM_{10-2.5}$  monitoring efforts has increased the availability of data for use in estimating light extinction with both  $PM_{2.5}$  and  $PM_{10-2.5}$  concentrations included as inputs in the equations. Collocated  $PM_{10-2.5}$  monitoring data were available at 20 of the 67  $PM_{2.5}$  sites (see Appendix D) for 2015-2017. Thus, the analysis in this review addressed light extinction estimated with coarse and fine PM at sites where feasible. All 20 of these sites met the 24-hour  $PM_{2.5}$  standard and 24-hour  $PM_{10}$  standard, and they all had 3-year visibility metrics at or below 30 dv when light extinction was calculated with and without the coarse fraction for any of the three versions of the IMPROVE equation. Generally, the contribution of the coarse fraction to

light extinction at these sites is minimal, contributing less than 1 dv to the 3-year visibility metric. However, we note that in our analysis, none of the locations included areas that would be expected to have greater concentrations of coarse PM, such as the southwest. In such locations, if PM<sub>10</sub> and PM<sub>10-2.5</sub> data were available, the coarse fraction may be a more important contributor to light extinction and visibility impairment than in those locations with lower concentrations of coarse PM. These results are consistent with those in the analyses in the ISA, which found that mass scattering from PM<sub>10-2.5</sub> was relatively small (less than 10%) in the eastern and northwestern U.S., whereas mass scattering was much larger in the Southwest (more than 20%) particularly in southern Arizona and New Mexico (U.S. EPA, 2019, section 13.2.4.1, p. 13-36).

In summary, the findings of these updated quantitative analyses are generally consistent with those in the last review. The 3-year visibility metric was generally below 25 dv in most areas that meet the current 24-hour PM<sub>2.5</sub> standard, with one location slightly above 30 dv, rounding to 31 dv. Small differences in the 3-year visibility metric were observed between the variations of the IMPROVE equation, which may suggest that it may be more appropriate to use one version over another in different regions of the U.S. based on PM characteristics such as particle size and composition to more accurately estimate light extinction. There was also very little difference in estimates of light extinction when the coarse fraction was included in the equation, although this may be more important in areas that have a higher concentration of coarse PM than those included in this analysis.

## 5.2.2 Non-Visibility Effects

### 5.2.2.1 Evidence-Based Considerations

In considering the available evidence for non-visibility welfare effects attributable to PM as presented in the ISA, this section poses the following policy-relevant questions:

- **To what extent has new scientific evidence improved our understanding of the nature and magnitude of non-visibility welfare effects of PM in ambient air, including the variability associated with such effects? To what extent have important uncertainties in the evidence from the last review been addressed, and have new uncertainties emerged?**

We address these questions for PM and climate effects (section 5.2.2.1.1) and materials effects (section 5.2.2.1.2) below.

#### 5.2.2.1.1 Climate Effects

In considering the available evidence of climate effects attributable to PM, this section poses the following policy-relevant question:

- **To what extent is new information available that changes or enhances our understanding of the climate impacts of PM-related aerosols, particularly regarding**

**a quantitative relationship between PM concentrations and effects on climate (e.g., through radiative forcing)?**

In the last review, the 2009 PM ISA concluded that there was “sufficient evidence to determine a causal relationship between PM and climate effects – specifically on the radiative forcing of the climate system, including both direct effects of PM on radiative forcing and indirect effects that involve cloud feedbacks that influence precipitation formation and cloud lifetimes” (U.S. EPA, 2009, section 9.3.10).<sup>19</sup> Since the last review, climate impacts have been extensively studied and the ISA concludes that “overall the evidence is sufficient to conclude that a causal relationship exists between PM and climate effects” (U.S. EPA, 2019, section 13.3.9). Recent research reinforces and strengthens the evidence evaluated in the 2009 ISA. New evidence provides greater specificity about the details of these radiative forcing effects and increased understanding of additional climate impacts driven by PM radiative effects. The Intergovernmental Panel on Climate Change (IPCC) assesses the role of anthropogenic activity in past and future climate change. In the last review, the 2009 ISA relied heavily on the Fourth IPCC Assessment Report (AR4); since that time the IPCC has issued an updated report. The Fifth IPCC Assessment Report (AR5; IPCC, 2013) reports on the key scientific advances in understanding the climate effects of PM since AR4. The ISA draws substantially upon AR5 in summarizing these effects.

Atmospheric PM has the potential to affect climate in multiple ways, including absorbing and scattering of incoming solar radiation, alterations in terrestrial radiation, effects on the hydrological cycle, and changes in cloud properties (U.S. EPA, 2019, section 13.3.1). Atmospheric PM interacts with incoming solar radiation. Many species of PM (e.g., sulfate and nitrate) efficiently scatter solar energy. By enhancing reflection of solar energy back to space, scattering PM exerts a cooling effect on the surface below. Certain species of PM such as black carbon (BC), brown carbon (BrC), or dust can also absorb incoming sunlight. A recent study found that whether absorbing PM warms or cools the underlying surface depends on several factors, including the altitude of the PM layer relative to cloud cover and the albedo of the surface (Ban-Weiss et al., 2014). PM also perturbs incoming solar energy by influencing cloud cover and cloud lifetime. For example, PM provides nuclei upon which water vapor condenses, forming cloud droplets. Finally, absorbing PM deposited on snow and ice can diminish surface albedo and lead to regional warming (U.S. EPA, 2019, section 13.3.2).

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<sup>19</sup> Radiative forcing (RF) for a given atmospheric constituent is defined as the perturbation in net radiative flux, at the tropopause (or the top of the atmosphere) caused by that constituent, in watts per square meter ( $\text{Wm}^{-2}$ ), after allowing for temperatures in the stratosphere to adjust to the perturbation but holding all other climate responses constant, including surface and tropospheric temperatures (Fiore et al., 2015, Myhre et al., 2013). A positive forcing indicates net energy trapped in the Earth system and suggests warming of the Earth’s surface, whereas a negative forcing indicates net loss of energy and suggests cooling (U.S. EPA, 2019, section 13.3.2.2).

PM has direct and indirect effects on climate processes. PM interactions with solar radiation through scattering and absorption, collectively referred to as aerosol-radiation interactions (ARI), are also known as the direct effects of PM on climate, as opposed to the indirect effects that involve aerosol-cloud interactions (ACI). The direct effects of PM on climate result primarily from particles scattering light away from Earth and sending a fraction of solar energy back into space, decreasing the transmission of visible radiation to the surface of the Earth and resulting in a decrease in the heating rate of the surface and the lower atmosphere. The IPCC AR5, taking into account both model simulations and satellite observations, reports a radiative forcing from aerosol-radiation interactions (RFari) from anthropogenic PM of  $-0.35 \pm 0.5$  watts per square meter ( $\text{Wm}^{-2}$ ) (Boucher, 2013), which is slightly reduced compared to AR4. Estimates of effective radiative forcing<sup>20</sup> from aerosol-radiation interactions (ERFari), which include the rapid feedback effects of temperature and cloud cover, rely mainly on model simulations, as this forcing is complex and difficult to observe (U.S. EPA, 2019, section 13.3.4.1). The IPCC AR5 best estimate for ERFari is  $-0.45 \pm 0.5 \text{ Wm}^{-2}$ , which reflects this uncertainty (Boucher, 2013).

By providing cloud condensation nuclei, PM increases cloud droplet number, thereby increasing cloud droplet surface area and albedo (Twomey, 1977). The climate effects of these perturbations are more difficult to quantify than the direct effects of aerosols with RF but likely enhance the cooling influence of clouds by increasing cloud reflectivity (traditionally referred to as the first indirect effect) and lengthening cloud lifetime (the second indirect effect). These effects are reported as the radiative forcing from aerosol-cloud interactions (RFaci) and the effective radiative forcing from aerosol-cloud interactions (ERFaci) (U.S. EPA, 2019, section 13.3.3.2). IPCC AR5 estimates ERFaci at  $-0.45 \text{ Wm}^{-2}$ , with a 90% confidence interval of  $-1.2$  to  $0 \text{ Wm}^{-2}$  (U.S. EPA, 2019, section 13.3.4.2).<sup>21</sup> Studies have also calculated the combined effective radiative forcing from aerosol-radiation and aerosol-cloud interactions (ERFari+aci) (U.S. EPA, 2019, section 13.3.4.3). IPCC AR5 reports a best estimate of ERFari+aci of  $-0.90$  ( $-1.9$  to  $-0.1$ )  $\text{Wm}^{-2}$ , consistent with these estimates (Boucher, 2013).

PM can also strongly reflect incoming solar radiation in areas of high albedo, such as snow- and ice-covered surfaces. The transport and subsequent deposition of absorbing PM such as BC to snow- and ice-covered regions can decrease the local surface albedo, leading to surface

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<sup>20</sup> Effective radiative forcing (ERF), new in the IPCC AR5, takes into account not just the instantaneous forcing but also a set of climate feedbacks, involving atmospheric temperature, cloud cover, and water vapor, that occur naturally in response to the initial radiative perturbation (U.S. EPA, 2019, section 13.3.2.2).

<sup>21</sup> While the ISA includes estimates of RFaci and ERFaci from a number of studies (U.S. EPA, 2019, sections 13.3.4.2, 13.3.4.3, 13.3.3.3), this PA focuses on the single best estimate with a range of uncertainty, as reported in IPCC AR5 (Boucher, 2013).

heating. The absorbed energy can then melt the snow and ice cover and further depress the albedo, resulting in a positive feedback loop (U.S. EPA, 2019, section 13.3.3.3; Bond et al., 2013; U.S. EPA, 2012a). Deposition of absorbing PM, such as BC, may also affect surface temperatures over glacial regions (U.S. EPA, 2019, section 13.3.3.3). The IPCC AR5 best estimate of RF from the albedo effect is  $+0.04 \text{ Wm}^{-2}$ , with an uncertainty range of  $+0.02$  to  $+0.09 \text{ Wm}^{-2}$  (Boucher, 2013).

While research on PM-related effects on climate has expanded since the last review, there are still significant uncertainties associated with the accurate measurement of PM contributions to the direct and indirect effects of PM on climate.

- **To what extent does the currently available information provide evidence of a quantitative relationship between specific PM constituents (i.e., BC, OC, sulfate) and climate-related effects?**

Since the last review, a number of new studies have examined the individual climate effects associated with key PM components, including sulfate, nitrate, OC, BC, and dust, along with updated quantitative estimates of the radiative forcing associated with the individual species.

Sulfate particles form through oxidation of  $\text{SO}_2$  by OH in the gas phase and in the aqueous phase by a number of pathways, including in particular those involving ozone and  $\text{H}_2\text{O}_2$  (U.S. EPA, 2019, section 13.3.5.1). The main source of anthropogenic sulfate is from coal-fired power plants, and global trends in the anthropogenic  $\text{SO}_2$  emissions are estimated to have increased dramatically during the 20<sup>th</sup> and early 21<sup>st</sup> centuries, although the recent implementation of more stringent air pollution controls on sources has led to a reversal in such trends in many places (U.S. EPA, 2019, section 13.3.5.1). Sulfate particles are highly reflective. Consistent with other recent estimates, on a global scale, the IPCC AR5 estimates that sulfate contributes more than other PM types to RF, with  $\text{RF}_{\text{ari}}$  of  $-0.4$  ( $-0.6$  to  $-0.2$ )  $\text{Wm}^{-2}$ , where the 5% and 95% uncertainty range is represented by the numbers in the parentheses (Myhre et al., 2013). This uncertainty range indicates the challenges associated with estimating  $\text{SO}_2$  from sources in developing regions and estimating the lifetime of sulfate against wet deposition. Sulfate is also a major contributor to the influence of PM on clouds (Takemura, 2012). A total effective radiative forcing ( $\text{ERF}_{\text{ari+aci}}$ ) for anthropogenic sulfate has been estimated to be nearly  $-1.0 \text{ Wm}^{-2}$  (Zelinka et al., 2014, Adams et al., 2001).

Nitrate particles form through the oxidation of nitrogen oxides and occur mainly in the form of ammonium nitrate. Ammonium preferentially associates with sulfate rather than nitrate, leading to formation of ammonium sulfate at the expense of ammonium nitrate (Adams et al., 2001). As anthropogenic emissions of  $\text{SO}_2$  decline, more ammonium will be available to react with nitrate, potentially leading to future increases in ammonium nitrate particles in the

atmosphere (U.S. EPA, 2019, section 13.3.5.2; Hauglustaine et al., 2014; Lee et al., 2013; Shindell et al., 2013). Warmer global temperatures, however, may decrease nitrate abundance given that it is highly volatile at higher temperatures (Tai et al., 2010). The IPCC AR5 estimates RFari of nitrate of  $-0.11$  ( $-0.3$  to  $-0.03$ )  $\text{Wm}^{-2}$  (Boucher, 2013), which is one-fourth of the RFari of sulfate.

Primary organic carbonaceous PM, including BrC, are emitted from wildfires, agricultural fires, and fossil fuel and biofuel combustion. Secondary organic aerosols (SOA) form when anthropogenic or biogenic nonmethane hydrocarbons are oxidized in the atmosphere, leading to less volatile products that may partition into PM (U.S. EPA, 2019, section 13.3.5.3). Organic particles are generally reflective, but in the case of BrC, a portion is significantly absorbing at shorter wavelengths ( $<400$  nm). The IPCC AR5 estimates an RFari for primary organic PM from fossil fuel combustion and biofuel use of  $-0.09$  ( $-0.16$  to  $-0.03$ )  $\text{Wm}^{-2}$  and an RFari estimate for SOA from these sources of  $-0.03$  ( $-0.27$  to  $+0.20$ )  $\text{Wm}^{-2}$  (Myhre et al., 2013). The wide range in these estimates, including inconsistent signs for forcing, reflect uncertainties in the optical properties of organic PM and its atmospheric budgets, including the production pathways of anthropogenic SOA (Scott et al., 2014; Myhre et al., 2013; McNeill et al., 2012; Heald et al., 2010). The IPCC AR5 also estimates an RFari of  $-0.2$   $\text{Wm}^{-2}$  for primary organic PM arising from biomass burning (Boucher, 2013).

Black carbon (BC) particles occur as a result of inefficient combustion of carbon-containing fuels. Like directly emitted organic PM, BC is emitted from biofuel and fossil fuel combustion and by biomass burning. BC is absorbing at all wavelengths and likely has a large impact on the Earth's energy budget (Bond et al., 2013). The IPCC AR5 estimates a RFari from anthropogenic fossil fuel and biofuel use of  $+0.4$  ( $+0.5$  to  $+0.8$ )  $\text{Wm}^{-2}$  (Myhre et al., 2013). Biomass burning contributes an additional  $+0.2$  ( $+0.03$  to  $+0.4$ )  $\text{Wm}^{-2}$  to BC RFari, while the albedo effect of BC on snow and ice adds another  $+0.04$  ( $+0.02$  to  $+0.09$ )  $\text{Wm}^{-2}$  (Myhre et al., 2013; U.S. EPA, 2019, section 13.3.5.4, section 13.3.4.4).

Dust, or mineral dust, is mobilized from dry or disturbed soils as a result of both meteorological and anthropogenic activities. Dust has traditionally been classified as scattering, but a recent study found that dust may be substantially coarser than currently represented in climate models, and thus more light-absorbing (Kok et al., 2017). The IPCC AR5 estimates RFari as  $-0.1 \pm 0.2$   $\text{Wm}^{-2}$  (Boucher, 2013), although the results of the study by Kok et al. (2017) would suggest that in some regions dust may have led to warming, not cooling (U.S. EPA, 2019, section 13.3.5.5).

The new research available in this review expands upon the evidence available at the time of the last review. Consistent with the evidence available in the last review, the key PM



components, including sulfate, nitrate, OC, BC, and dust, that contribute to climate processes vary in their reflectivity, forcing efficiencies, and direction of forcing.

- **To what extent does newly available evidence change or improve our understanding of the spatial and temporal variation in climate responses to PM?**

Radiative forcing due to PM elicits a number of responses in the climate system that can lead to significant effects on weather and climate over a range of spatial and temporal scales, mediated by a number of feedbacks that link PM and climate. Since the last review, the evidence base has expanded with respect to the mechanisms of climate responses and feedbacks to PM radiative forcing, described below, although considerable uncertainties continue to exist. We focus our discussion primarily on the climate impacts in the U.S.

Unlike well-mixed, long-lived greenhouse gases in the atmosphere, PM has a very heterogeneous distribution across the Earth. As such, patterns of  $RF_{air}$  and  $RF_{surf}$  tend to correlate with PM loading, with the greatest forcings centralized over continental regions. The climate response is more complicated since the perturbation to one climate variable (e.g., temperature, cloud cover, precipitation) can lead to a cascade of effects on other variables. While the initial PM radiative forcing may be concentrated regionally, the eventual climate response can be much broader spatially or be concentrated in remote regions (U.S. EPA, 2019, section 13.3.6). The complex climate system interactions lead to variation among climate models, with some studies showing relatively close correlation between forcing and surface response temperatures (e.g., Leibenberger et al., 2012), while other studies show much less correlation (e.g., Levy et al., 2013). Many studies have examined observed trends in PM and temperature in the U.S. Climate models have suggested a range of factors which can influence large-scale meteorological processes and may affect temperature, including local feedback effects involving soil moisture and cloud cover, changes in the hygroscopicity of the PM, and interactions with clouds alone (U.S. EPA, 2019, section 13.3.7). While evidence in this review suggests that PM influenced temperature trends across the southern and eastern U.S. in the 20<sup>th</sup> century, uncertainties continue to exist and further research is needed to better characterize the effects of PM on regional climate in the U.S.

- **To what extent have important uncertainties identified in the last review been reduced and/or have new uncertainties emerged?**

Since 2009, significant progress has been made in evaluating PM-related climate effects and uncertainties. The IPCC AR5 states that “climate-relevant aerosol processes are better understood, and climate-relevant aerosol properties are better observed, than at the time of the AR4” (Boucher, 2013). However, significant uncertainties remain that make it difficult to

quantify the climate effects of PM. Such uncertainties include those related to our understanding of:

- The magnitude of PM radiative forcing and the portion of that associated with anthropogenic emissions;
- The contribution of regional differences in PM concentrations, and of individual components, to radiative forcing;
- The mechanisms of climate responses and feedbacks resulting from PM-related radiative forcing; and,
- The process by which PM interacts with clouds and how to represent such interactions in climate models.

While research has progressed significantly since the last review, substantial uncertainties still remain with respect to key processes linking PM and climate, because of the small scale of PM-relevant atmospheric processes compared to the resolution of state-of-the-art models, and because of the complex cascade of indirect impacts and feedbacks in the climate system that result from an initial PM-related radiative perturbation (U.S. EPA, 2019, section 13.3.9).

#### **5.2.2.1.2 Materials Effects**

In considering the available evidence on materials effects attributable to PM, this section poses the following policy-relevant question:

- **To what extent is new information available to link PM to materials effects, including degradation of surfaces, and deterioration of materials such as metal, stone, concrete and marble?**

In the last review, the 2009 ISA concluded that there was “a causal relationship between PM and effects on materials” (U.S. EPA, 2009, sections 2.5.4 and 9.5.4). Rather than altering our conclusions from the last review, the current evidence continues to support our prior conclusion regarding materials effects associated with PM deposition. Effects of deposited PM, particularly sulfates and nitrates,<sup>22</sup> to materials include both physical damage and impaired aesthetic qualities. Because of their electrolytic, hygroscopic, and acidic properties and their ability to sorb corrosive gases, particles contribute to materials damage by adding to the effects of natural weathering processes, by potentially promoting or accelerating the corrosion of metals, degradation of painted surfaces, deterioration of building materials, and weakening of material components. The majority of the newly available evidence on materials effects of PM are from

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<sup>22</sup> In the case of materials effects, it is difficult to isolate the effects of gaseous and particulate N and S wet deposition so both will be considered along with other PM-related deposition effects on materials in this review of the PM NAAQS.

outside the U.S. on buildings and other items of cultural heritage; however, they provide limited new data for consideration in this review (U.S. EPA, 2019, section 13.4).

Materials damage from PM generally involves one or both of two processes: soiling and corrosion (U.S. EPA, 2019, section 13.4.2). Soiling and corrosion are complex, interdependent processes, typically beginning with deposition of atmospheric PM or SO<sub>2</sub> to exposed surfaces. Constituents of deposited PM can interact directly with materials or undergo further chemical and/or physical transformation to cause soiling, corrosion, and physical damage. Weathering, including exposure to moisture, ultraviolet (UV) radiation and temperature fluctuations, affects the rate and degree of damage (U.S. EPA, 2019, section 13.4.2).

Soiling is the result of PM accumulation on an object that alters its optical characteristics or appearance. These soiling effects can impact the aesthetic value of a structure or result in reversible or irreversible damage to the surface. The presence of air pollution can increase the frequency and duration of cleaning and can enhance biodeterioration processes on the surface of materials. For example, deposition of carbonaceous components of PM can lead to the formation of black crusts on surfaces, and the buildup of microbial biofilms<sup>23</sup> can discolor surfaces by trapping PM more efficiently (U.S. EPA, 2009, p. 9-195; U.S. EPA, 2019, section 13.4.2). The presence of PM may alter light transmission or change the reflectivity of a surface. Additionally, the organic or nutrient content of deposited PM may enhance microbial growth on surfaces.

Since the last review, very little new evidence has become available related to deposition of SO<sub>2</sub> to materials such as limestone, granite, and metal. Deposition of SO<sub>2</sub> onto limestone can transform the limestone into gypsum, resulting in a rougher surface, which allows for increased surface area for accumulation of deposited PM (Camuffo and Bernardi, 1993; U.S. EPA, 2019, section 13.4.2). Oxidation of deposited SO<sub>2</sub> that contributes to the transformation of limestone to gypsum can be enhanced by the formation of surface coatings from deposited carbonaceous PM (both elemental and organic carbon) (McAlister et al., 2008, Grossi et al., 2007). Ozga et al. (2011) characterized damage to two concrete buildings in Poland and Italy. Gypsum was the main damage product on surfaces of these buildings that were sheltered from rain runoff, while PM embedded in the concrete, particularly carbonaceous particles, were responsible for darkening of the building walls (Ozga et al., 2011).

Building on the evidence available in the 2009 ISA, research has progressed on the theoretical understanding of soiling of cultural heritage in a number of studies. Barca et al. (2010) developed and tested a new methodological approach for characterizing trace elements and heavy metals in black crusts on stone monuments to identify the origin of the chemicals and

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<sup>23</sup> Microbial biofilms are communities of microorganisms, which may include bacteria, algae, fungi and lichens, that colonize an inert surface. Microbial biofilms can contribute to biodeterioration of materials via modification of the chemical environment.

the relationship between the concentrations of elements in the black crusts and local environmental conditions. Recent research has also used isotope tracers to distinguish between contributions from local sources versus atmospheric pollution to black crusts on historical monuments in France (Kloppmann et al., 2011). A study in Portugal found that biological activity played a major role in soiling, specifically in the development of colored layers and in the detachment process (de Oliveira et al., 2011). Another study found damage to cement renders, often used for restoration, consolidation, and decorative purposes on buildings, following exposure to sulfuric acid, resulting in the formation of gypsum (Lanzon and Garcia-Ruiz, 2010).

Corrosion of stone and the decay of stone building materials by acid deposition and sulfate salts were described in the 2009 ISA (U.S. EPA, 2009, section 9.5.3). Since that time, advances have been made on the quantification of degradation rates and further characterization of the factors that influence damage of stone materials (U.S. EPA, 2019, section 13.4.2). Decay rates of marble grave stones were found to be greater in heavily polluted areas compared to a relatively pristine area (Mooers et al., 2016). The time of wetness and the number of dissolution/crystallization cycles were identified as hazard indicators for stone materials, with greater hazard during the spring and fall when these indicators are relatively high (Casati et al., 2015).

A study examining the corrosion of steel as a function of PM composition and particle size found that changes in the composition of resulting rust gradually changed with particle size (Lau et al., 2008). In a study of damage to metal materials under in Hong Kong, which generally has much higher PM concentrations than those observed in the U.S., Liu et al. (2015) found that iron and steel were corroded by both PM and gaseous pollutants ( $\text{SO}_2$  and  $\text{NO}_2$ ), while copper and copper alloys were mainly corroded by gaseous pollutants ( $\text{SO}_2$  and  $\text{O}_3$ ) and aluminum and aluminum alloy corrosion was mainly attributed to PM and  $\text{NO}_2$ .

A number of studies have also found materials damage from PM components besides sulfate and black carbon and atmospheric gases besides  $\text{SO}_2$ . Studies have characterized impacts of nitrates,  $\text{NO}_x$ , and organic compounds on direct materials damage or on chemical reactions that enhance materials damage (U.S. EPA, 2019, section 13.4.2). Other studies have found that soiling of building materials can be attributed to enhanced biological processes and colonization, including the development and thickening of biofilms, resulting from the deposition of PM components and atmospheric gases (U.S. EPA, 2019, section 13.4.2).

Since the last review, other materials have been studied for damage attributable to PM, including glass and photovoltaic panels. Soiling of glass can impact its optical and thermal

properties, and can lead to increased cleaning costs and frequency. The development of haze<sup>24</sup> on modern glass has been measured and modeled, with a strong correlation between the size distribution of particles and the evolution of the mass deposited on the surface of the glass. Measurements showed that, under sheltered conditions, mass deposition accelerated regularly with time in areas closest to sources of PM (i.e., near roadways) and coarse mineral particles were more prevalent compared to other sites (Alfaro et al., 2012). Model predictions were found to correctly simulate the development of haze at site locations when compared with measurements (Alfaro et al., 2012).

Soiling of photovoltaic panels can lead to decreased energy efficiency. For example, soiling by carbonaceous PM decreased solar efficiency by nearly 38%, while soil particles reduced efficiency by almost 70% (Radonjic et al., 2017). The rate of photovoltaic power output can also be degraded by soiling and has been found to be related to the rate of dust accumulation. In five sites in the U.S. representing different meteorological and climatological conditions,<sup>25</sup> photovoltaic module power transmission was reduced by approximately 3% for every g/m<sup>2</sup> of PM deposited on the cover plate of the photovoltaic panel, independent of geographical location (Boyle et al., 2017). Another study found that photovoltaic module power output was reduced by 40% after 10 months of exposure without cleaning, although a number of anti-reflective coatings can generally mitigate power reduction resulting from dust deposition (Walwil et al., 2017). Energy efficiency can also be impacted by the soiling of building materials, such as light-colored marble panels on building exteriors, that are used to reflect a large portion of solar radiation for passive cooling and to counter the urban heat island effect. Exposure to acidic pollutants in urban environments have been found to reduce the solar reflectance of marble, decreasing the cooling effect (Rosso et al., 2016). Highly reflective roofs, or cool roofs, have been designed and constructed to increase reflectance from buildings in urban areas, to both decrease air conditioning needs and urban heat island effects, but these efforts can be impeded by soiling of materials used for constructing cool roofs. Methods have been developed for accelerating the aging process of roofing materials to better characterize the impact of soiling and natural weather on materials used in constructing cool roofs (Sleiman et al., 2014).

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<sup>24</sup> In this discussion of non-visibility welfare effects (section 5.2.2), haze is used as it has been defined in the scientific literature on soiling of glass, i.e., the ratio of diffuse transmitted light to direct transmitted light (Lombardo et al., 2010). This differs from the definition of haze as used in the discussion of visibility welfare effects in section 5.2.1, where it is used as a qualitative description of the blockage of sunlight by dust, smoke, and pollution.

<sup>25</sup> Of the five sites studied, three were in rural, suburban, and urban areas representing a semi-arid environment (Front Range of Colorado), one site represented a hot and humid environment (Cocoa, Florida), and one represented a hot and arid environment (Albuquerque, New Mexico) (U.S. EPA, 2019, section 13.4.2; Boyle et al., 2017).

- **To what extent has new information emerged for quantifying material damage attributable to PM through dose-response relationships or damage functions? Are there studies linking perceptions of reduced aesthetic appeal of buildings and other objects to PM or wet deposition of N and S species?**

Some progress has been made since the last review in the development of dose-response relationships for soiling of building materials, although some key relationships remain poorly characterized. The first general dose-response relationships for soiling of materials were generated by measuring contrast reflectance of a soiled surface to the reflectance of the unsoiled substrate for different materials, including acrylic house paint, cedar siding, concrete, brick, limestone, asphalt shingles, and window glass with varying total suspended particulate (TSP) concentrations (Beloin and Haynie, 1975; U.S. EPA, 2019, section 13.4.3). Continued efforts to develop dose-response curves for soiling have led to some advancements for modern materials, but these relationships remain poorly characterized for limestone. A recent study quantified the dose-response relationships between  $PM_{10}$  and soiling for painted steel, white plastic, and polycarbonate filter material, but there was too much scatter in the data to produce a dose-response relationship for limestone (Watt et al., 2008). A dose-response relationship for silica-soda-lime window glass soiling by  $PM_{10}$ ,  $NO_2$ , and  $SO_2$  was quantified based on 31 different locations (Lombardo et al., 2010; U.S. EPA, 2019, section 13.4.3, Figure 13-32, Equation 13-8). The development of this dose-response relationship required several years of observation time and had inconsistent data reporting across the locations.

Since the last review, there has also been progress in developing methods to more rapidly evaluate soiling of different materials by PM mixtures. Modern buildings typically have simpler lines, less detailed surfaces, and a greater use of glass, tile, and metal, which are easier to clean than stone. There have also been major changes in the types of materials used for buildings, including a variety of polymers available for use as coatings and sealants. New economic and environmental considerations beyond aesthetic appeal and structural damage are emerging (U.S. EPA, 2019, section 13.4.3). Changes in building materials and design, coupled with new approaches in quantifying the dose-response relationship between PM and materials effects, may reduce the amount of time needed for observations to support the development of material-specific dose-response relationships.

In addition to dose-response functions, damage functions have also been used to quantify material decay as a function of pollutant type and load. Damage can be determined from sample surveys or inspection of actual damage and a damage function can be developed to link the rate of material damage to time of replacement or maintenance. A cost function can then link the time for replacement and maintenance to a monetary cost, and an economic function links cost to the dose of pollution based on the dose-response relationship (U.S. EPA, 2019, section 13.4.3).

Damage functions are difficult to assess because it depends on human perception of the level of soiling deemed to be acceptable and evidence in this area remains limited in the current review. Since the last review, damage functions for a wide range of building materials (i.e., stone, aluminum, zinc, copper, plastic, paint, rubber, stone) have been developed and reviewed (Brimblecombe and Grossi, 2010). One study estimated long-term deterioration of building materials and found that damage to durable building material (such as limestone, iron, copper, and discoloration of stone) is no longer controlled by pollution as was historically documented but rather that natural weathering is a more important influence on these materials in modern times (Brimblecombe and Grossi, 2009). Even as PM-attributable damage to stone and metals has decreased over time, it has been predicted that there will be potentially higher degradation rates for polymeric materials, plastic, paint, and rubber due to increased oxidant concentrations and solar radiation (Brimblecombe and Grossi, 2009).

- **To what extent have important uncertainties identified in the last review been reduced and/or have new uncertainties emerged?**

While there are a number of new studies in the ISA that investigate the effect of PM on newly studied materials and further characterize the effects of PM on previously studied materials, there remains insufficient evidence to relate soiling or damage to specific PM levels or to establish a quantitative relationship between PM in ambient air and materials degradation. Uncertainties that were identified in the last review still largely remain with respect to quantitative relationships between particle size, concentration, chemical concentrations, and frequency of repainting and repair. No new studies are available that link perceptions of reduced aesthetic appeal of buildings and other objects to PM-related materials effects. Moreover, uncertainties about the deposition rates of airborne PM to surfaces and the interaction of co-pollutants still remain.

#### **5.2.2.2 Quantitative Assessment-Based Considerations**

Beyond our consideration of the scientific evidence, discussed above in section 5.2.2.1 above, we also consider the extent to which quantitative analyses of PM air quality and quantitative assessments for climate and materials effects could inform conclusions on the adequacy of the public welfare protection provided by the current secondary PM standards. We have evaluated the potential support for conducting new analyses of PM air quality concentrations and non-visibility welfare effects.

##### **5.2.2.2.1 Climate Effects**

While expanded since the last review, our current understanding of PM-related climate effects is still limited by significant uncertainties. Large spatial and temporal heterogeneities in direct and indirect PM climate forcing can occur for a number of reasons, including the

frequency and distribution of emissions of key PM components contributing to climate forcing, the chemical and microphysical processing that occurs in the atmosphere, and the atmospheric lifetime of PM relative to other pollutants contributing to climate forcing (U.S. EPA, 2019, section 13.3). These issues particularly introduce uncertainty at the local and regional scales in the U.S. that would likely be most relevant to a quantitative assessment of the potential effects of a national PM standard on climate in this review. Limitations and uncertainties in the evidence make it difficult to quantify the impact of PM on climate and in particular how changes in the level of PM mass in ambient air would result in changes to climate in the U.S. Thus, as in the last review, the data remain insufficient to conduct quantitative analyses for PM effects on climate in the current review.

#### **5.2.2.2.2 Materials Effects**

As at the time of the last review, sufficient evidence is not available to conduct a quantitative assessment of PM-related soiling and corrosion effects. While soiling associated with PM can lead to increased cleaning frequency and repainting of surfaces, no quantitative relationships have been established between characteristics of PM or the frequency of cleaning or repainting that would help inform our understanding of the public welfare implications of soiling (U.S. EPA, 2019, section 13.4). Similarly, while some information is available with regard to microbial deterioration of surfaces and the contribution of carbonaceous PM to the formation of black crusts that contribute to soiling, the available evidence does not support quantitative analyses (U.S. EPA, 2019, section 13.4). While some new evidence is available with respect to PM-attributable materials effects, the data are insufficient to conduct quantitative analyses for PM effects on materials in the current review.

### **5.3 CASAC ADVICE**

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the current PM secondary standards. In its comments on the draft PA, the CASAC concurs with staff's overall preliminary conclusions that it is appropriate to consider retaining the current secondary PM standards without revision (Cox, 2019). The CASAC "finds much of the information...on visibility and materials effects of PM<sub>2.5</sub> to be useful, while recognizing that uncertainties and controversies remain about the best ways to evaluate these effects" (Cox, 2019, p. 13 of consensus responses). Regarding climate, while the CASAC recommends that the EPA consider recent research evaluating the impacts of reducing PM<sub>2.5</sub> and suggests that the EPA include quantitative analyses to more thoroughly address these effects,<sup>26</sup> the committee also

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<sup>26</sup> While this final PA does consider research evaluating the impacts of PM on climate, we have not conducted analyses to quantify the impacts of changes in U.S. ambient PM concentrations on regional and national climate endpoints in the U.S. that would be of potential relevance for the NAAQS review. This approach to addressing



agrees with the EPA that “the available evidence does not call into question the protection afforded by the current secondary PM standards and concurs that they should be retained” (Cox, 2019, p. 3 of letter).

A number of public comments have been received in this review to date, including comments focused on the draft PA. A limited number of public comment submissions on the draft PA provide comments related to the adequacy of the secondary standards. Of those who provide comments on the secondary standards, the majority of commenters support the preliminary conclusion that it is appropriate to consider retaining the current secondary PM standards, without revision. These commenters generally cite a lack of newly available evidence and information that would inform quantitative assessments and consideration of alternate secondary standards to protect against PM-related effects on visibility, climate, and materials. One commenter (the Independent PM Review Panel), however, supports revision of the secondary PM standards to provide additional protection against PM-related visibility effects, citing inconsistencies between preliminary conclusions in the draft PA to consider retaining the current secondary PM standards and the currently available scientific evidence regarding public visibility preferences and indices for evaluating visibility impairment. This commenter also recognizes the regional heterogeneity in PM<sub>2.5</sub> mass and light extinction and that one single level may not be appropriate in all regions of the country.

## **5.4 CONCLUSIONS ON THE SECONDARY PM STANDARDS**

This section discusses staff conclusions for the Administrator’s consideration in judging the adequacy of the current secondary PM standards. These conclusions are based on consideration of the assessment and integrative synthesis of evidence presented in the ISA, as well as our analyses of recent air quality. Further, the staff conclusions have taken into account advice from the CASAC and public comments on the draft PA and the associated preliminary staff conclusions. Taking into consideration the responses to specific questions discussed above, we revisit the overarching policy question for this chapter:

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the CASAC’s comments on climate reflects our consideration of the timeline for this review as well as the uncertainties that would be inherent in such analyses and their likely impact on decision making. As discussed above (section 5.2.2.2.1), limitations in the evidence would result in considerable uncertainty in analyses that attempt to quantify the impact of changes in ambient PM in the US on climate in the U.S.

- **Does the currently available scientific evidence and quantitative information support or call into question the adequacy of the protection afforded by the current secondary PM standards?**

As provided in section 109(b)(2) of the CAA, the secondary standard is to “specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator...is requisite to protect public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.” Effects on welfare include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being” (CAA section 302(h)). The secondary standards are not meant to protect against all known or anticipated PM-related effects, but rather those that are judged to be adverse to the public welfare (78 FR 3212, January 15, 2013). Similarly, the extent to which secondary standards are concluded to provide adequate protection from such effects also depends on judgments by the Administrator.

Therefore, we recognize that, as is the case in NAAQS reviews in general, the extent to which the current secondary PM standards are judged to be adequate will depend on a variety of factors and judgments to be made by the Administrator. Such judgments include those concerning the extent or severity of welfare effects that may be considered adverse to the public welfare, and accordingly, what level of protection from such known or anticipated effects may be judged requisite. In general, the public welfare significance of PM-related effects for different air quality conditions and in different locations depend upon the type and severity of the effects, as well as the strength of the underlying information and associated uncertainties. Thus, in the discussion below, our intention is to focus on such aspects of the currently available evidence and quantitative analyses.

With regard to visibility, climate, and materials effects of PM, our response to the question above takes into consideration the discussions that address the specific policy-relevant questions in prior sections of this chapter (see sections 5.2.1 and 5.2.2) and the approach described in section 5.1 that builds on the approach from the last review. With respect to the evidence-based considerations, we note that the currently available evidence, while somewhat expanded since the last review, does not include evidence of effects at lower concentrations or other welfare effects of PM than those identified at the time of the last review. There continue to be significant uncertainties related to quantifying the relationships between PM mass concentrations in ambient air and welfare effects, including visibility impairment, climate effects, and materials effects.

With respect to the visibility effects of PM, the currently available evidence continues to support a causal relationship. With respect to evidence for visibility effects of PM, we note that the currently available evidence, while somewhat expanded since the last review, does not include evidence of effects at lower concentrations than those identified at the time of the last review. Consistent with the evidence available at the time of the last review, significant limitations remain in directly measuring light extinction. However, a number of small refinements have been made to the algorithm commonly used to estimate light extinction (U.S. EPA, 2019, section 13.2.3.3; section 5.2.1.1 above). Light extinction by PM<sub>2.5</sub> is dependent on PM<sub>2.5</sub> composition and relative humidity, which varies regionally, with component contributions to light extinction also changing over time with changes in emissions, as can be seen in analyses of recent air quality. We also note that no new research is available on methods of characterizing visibility or on how visibility is valued by the public, such as visibility preference studies. Thus, while limited new research has further informed our understanding of the influence of atmospheric components of PM<sub>2.5</sub> on light extinction, the available evidence to inform consideration of the public welfare implications of PM-related visibility impairment remains relatively unchanged.

With respect to quantitative-based considerations, analyses using recent air quality and considering updated and alternative methods for estimating visibility impairment provide results generally similar to those given a focus in the decision for the last review. We recognize that conclusions reached regarding visibility in the last review were based primarily on the quantitative analyses that considered the relationship of estimated visibility impairment (light extinction) with design values for the secondary 24-hour PM<sub>2.5</sub> standard. These analyses demonstrated that visibility index values were below 30 dv – the value identified as the target level of protection for visibility-related welfare effects – at all locations that met the daily standard. In our evaluation in this chapter, we have considered the currently available information regarding the equations to estimate light extinction and the inputs to the equations and regarding identification of the target level of protection. With regard to the equations, we have utilized both the most recently published equations as well as alternatives considered in the last review in recognition of the uncertainties inherent in the quantitative relationship between PM and light extinction and the variability in applicability to different locations. Further, we have considered key coefficients in estimating and adjusting concentrations of specific PM<sub>2.5</sub> components, a key example of which is the multiplier used to estimate the concentration of organic matter from the concentration of organic carbon. For consistency with the analysis on which the decision was based in the last review, we have focused on a 3-year average of the 90<sup>th</sup> percentile of daily light extinction (calculated using old and new algorithms) in considering visibility impairment at the analyzed locations.

In reaching a conclusion in the 2012 review with regard to the adequacy of visibility protection provided by the secondary PM standards, the Administrator identified 30 dv as an appropriate target level of protection. We have not identified new information in this review that would challenge this public policy. Thus, in our consideration of the current information and analyses in this document, we have compared the results of the updated analyses to the value of 30 dv, finding only one site that exceeds this target level of protection while meeting the current daily standards, albeit just marginally at 31 dv. In so finding, we additionally note the uncertainties recognized above regarding estimation of OM for use in the IMPROVE equations, and also the variability across sites in characteristics that affect the relationship between PM in ambient air and light extinction, and in characteristics that affect human visibility and preferences in that regard. Based on the findings of this comparison, in light of all of these considerations, we find it reasonable to conclude that the quantitative information available in this review does not call into question the adequacy of visibility-related public welfare protection provided by the current secondary PM standards. As a result, we have not conducted additional analyses to evaluate the level of visibility protection that might be afforded by potential alternative standards.

With respect to the non-visibility welfare effects of PM, the currently available evidence continues to support causal relationships between climate effects and PM and materials effects and PM. The currently available evidence related to climate effects and PM, while expanded since the last review, has not appreciably improved our understanding of the spatial and temporal heterogeneity of PM components that contribute to climate forcing. We note that, as at the time of the last review, the evidence describes differences among individual PM components in their reflective properties and direction of climate forcing. We also note that, while climate research has continued, there are still significant limitations in our ability to quantify contributions of PM, and of individual PM components, to the direct and indirect effects of PM on climate (e.g. changes to the pattern of rainfall, changes to wind patterns, effects on vertical mixing in the atmosphere). While climate models have been improved and refined since the last review, climate models simulating aerosol-climate interactions on regional scales (e.g., ~100 km) tend to have more variability in estimates of the PM-related climate effects than simulations at the global scale, and fewer studies are available that simulate specific regions (e.g., the U.S.) than that provide global-scale simulations. While new research has added to the understanding of climate forcing on a global scale, there remain significant limitations to quantifying potential adverse effects from PM on climate in the U.S. and how they would vary in response to changes in PM concentrations in the U.S. That is, the information currently available with regard to climate does not provide a clear understanding of a quantitative relationship between concentrations of PM mass in ambient air and associated climate-related effects, and consequently, precludes a

quantitative evaluation of the level of protection provided by a PM concentration-based secondary standard from adverse climate-related effects on the public welfare in the U.S. Thus, on the whole, we do not find the currently available information to provide support for different conclusions than were reached in the last review with regard to climate-related effects of PM in ambient air.

In considering the currently available evidence related to materials effects and PM, we note that there is newly available evidence that informs our understanding on the soiling process and types of materials affected, and provides limited information on dose-response relationships and damage functions, although most of the recent evidence comes from studies outside of the U.S. In particular, there is a growing body of research on PM and energy efficiency-related materials, such as solar panels and passive cooling building materials, affecting the optical and thermal properties, thereby impacting the intended energy efficiency of these materials. While new research has added to the understanding of PM-related materials effects, there remains a lack of research related to quantifying materials effects and understanding the public welfare implications of such effects.

In summary, with regard to the two main non-visibility effects – climate effects and materials effects – the available evidence, as in the last review, documents a causal role for PM in ambient air. This evidence, however, as in the last review, also includes substantial uncertainties with regard to quantitative relationships with PM concentrations and concentration patterns that limit our ability to quantitatively assess the public welfare protection provided by the standards from these effects. Thus, as a whole, the current information, which is not appreciably different from that available in the last review, does not call into question the adequacy of protection provided by the current standards for these effects.

Based on all of the above considerations and consistent with CASAC advice, we find that the available evidence does not call into question the protection afforded by the current secondary PM standards against PM-related welfare effects. Thus, our conclusion for the Administrator's consideration is that it is appropriate to consider retaining the current secondary PM standards, without revision. In so concluding, we recognize, as noted above, that the final decision on this review of the secondary PM standards to be made by the Administrator is largely a public welfare judgment, based on his judgment as to the requisite protection of the public welfare from any known or anticipated adverse effects. This final decision will draw upon the available scientific evidence and quantitative analyses on PM-attributable welfare effects, along with consideration of CASAC advice and public comments, and on judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses.

## 5.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

In this section, we highlight key uncertainties in the available information related to the effects of PM on public welfare. Such key uncertainties and areas for future research, model development, and data gathering are outlined below. We note, however, that a full set of research recommendations is beyond the scope of this discussion. Rather, listed below are key uncertainties, research questions and data gaps that have been thus far highlighted in this review of the secondary PM standards.

- A critical aspect of our consideration of the evidence and quantitative information for visibility impairment is our understanding of human perception of visibility impairment in the preference studies. This is essential to the Administrator's consideration of the public welfare implications of visibility effects and to decisions on the adequacy of protection provided by the secondary PM standards from them. Additional information related to several areas would reduce uncertainty in our interpretation of the available information for purposes of characterizing visibility impairment. These areas include the following:
  - Expanding the number and geographic coverage of preference studies in urban, rural and Class I areas to account for the potential for people to have different preferences based on the conditions that they commonly encounter and potential differences in preferences based on the scene types;
  - Evaluating visibility preferences of the U.S. population today, given that the currently available preference studies were conducted more than 15 years ago, during which time air quality in the U.S. has improved;
  - Accounting for the influence that varying study methods may have on an individual's response as to what level of visibility impairment is acceptable; and
  - Providing insights regarding people's judgments on acceptable visibility based on those factors that can influence an individual's perception of visibility impairment, including the duration of visibility impairment experiences, the time of day during which light extinction is greatest, and the frequency of episodes of visibility impairment, as well as the intensity of the visibility impairment.
- Direct monitoring of PM<sub>2.5</sub> light extinction would help to characterize visibility and the relationships between PM component concentrations and light extinction and to evaluate and refine light extinction calculation algorithms for use in areas near anthropogenic sources, and would provide measurements for future visibility effects assessments.
- Substantial uncertainties still remain with respect to key processes linking PM and climate, because of the small scale of PM-relevant atmospheric processes compared to the resolution of state-of-the-art models, and because of the complex cascade of indirect impacts and feedbacks in the climate system that result from an initial PM-related radiative perturbation. Such uncertainties include those related to our understanding of:
  - The magnitude of PM radiative forcing and the portion of that associated with anthropogenic emissions;

- The contribution of regional differences in PM concentrations, and of individual components, to radiative forcing; and,
  - The process by which PM interacts with clouds and how to represent such interactions in climate models.
- Research on more accurate U.S. and global emission inventories would provide source-specific data on PM and PM component contributions to climate effects, particularly those effects resulting from climate forcing.
- While CASAC highlighted a number of studies as providing quantitative information regarding the impact of reductions in PM<sub>2.5</sub> on direct and indirect climate effects, these studies largely are conducted at a global scale and assume a zeroing out or near-zeroing out of global PM emissions. Research is needed regarding the impacts of incremental changes in PM mass on direct and indirect climate effects on a regional scale, thereby limiting our ability to quantify the impact of these changes at this time.
- Insufficient evidence is available to relate soiling or damage to specific PM concentrations or to establish a quantitative relationship between PM concentrations in ambient air and materials degradation. Additional information would reduce uncertainty in in our interpretation of the available information, including in the following areas:
  - Identifying quantitative relationships between particle size, PM concentration, chemical concentrations, and frequency of repainting and repair;
  - Understanding human perceptions of reduced aesthetic appeal of buildings, and other objects to PM-related materials effects; and
  - Characterizing deposition rates of airborne PM to surfaces and the interaction of co-pollutants.

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# APPENDIX A. SUPPLEMENTAL INFORMATION ON PM AIR QUALITY ANALYSES

This appendix provides supplemental information on the data sources and methods used to generate the figures and table presented in Chapter 2 of this PA. Sections A.1 to A.4 describe the data sources and methods used to generate figures and tables in section 2.3.2. Section A.5 describes the data sources and methods used to generate figures and tables in section 2.3.3. Section A.6 describes the data sources and methods used to generate figures and tables in section 2.4.

## A.1 DATA SOURCES AND METHODS FOR GENERATING NATIONAL PM<sub>2.5</sub>, PM<sub>10</sub>, PM<sub>10-2.5</sub>, AND PM<sub>2.5</sub> SPECIATION FIGURES

- PM<sub>2.5</sub> annual average and 98<sup>th</sup> percentile mass concentrations: calculated from regulatory-quality (Federal Reference Method or Federal Equivalent Method) 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98<sup>th</sup> percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2017 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM<sub>10</sub> annual average and 98<sup>th</sup> percentile mass concentrations: calculated from both regulatory and non-regulatory methods using 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98<sup>th</sup> percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2017 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM<sub>10-2.5</sub> annual average and 98<sup>th</sup> percentile mass concentrations: calculated from both regulatory and non-regulatory methods using 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98<sup>th</sup> percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2017 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM<sub>2.5</sub> speciated annual average mass concentrations: calculated from filter-based, 24-hour averages from monitors with at least 75% completeness for each year. We downloaded data from monitors that are part of the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, Chemical Speciation Network (CSN), and the NCore Multipollutant Monitoring Network for 2015-2017.

- The 2000-2017 trends are calculated from the Pearson correlation coefficient for monitors having at least 75% of the available years with 75% completeness within each year. When a single site has multiple monitors, the average of the annual averages and 98<sup>th</sup> percentiles from each monitor at the site is taken prior to calculation of the Pearson correlation coefficient.

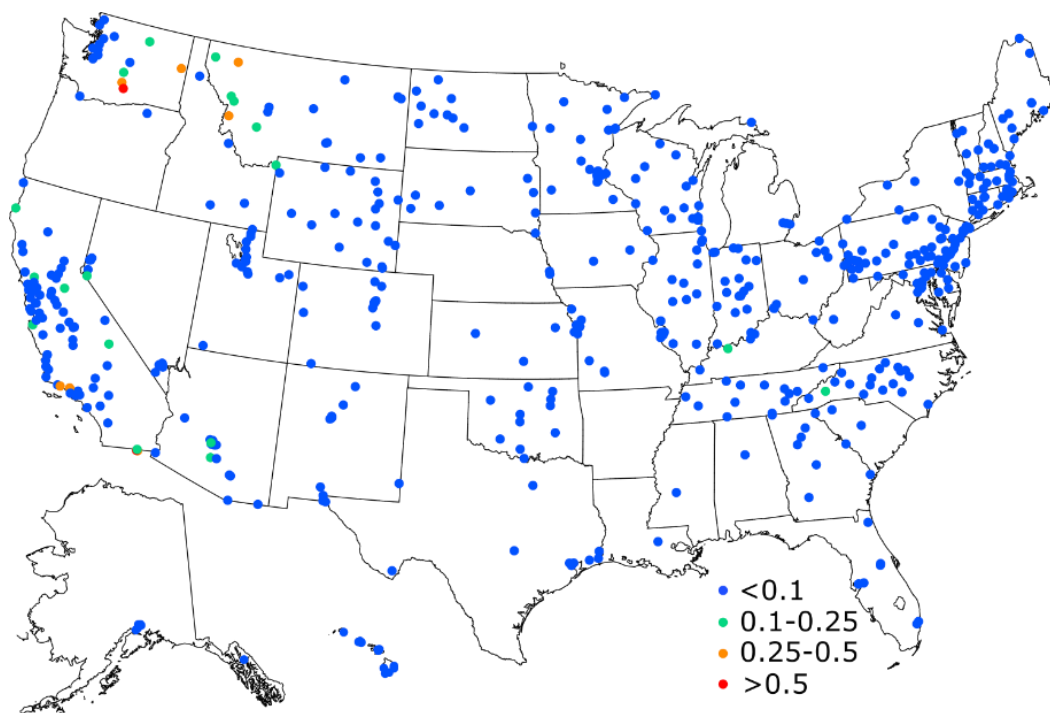
## **A.2 DATA SOURCES AND METHODS FOR GENERATING NEAR-ROAD PM<sub>2.5</sub> DESIGN VALUE TABLE AND INCREMENT FIGURES**

- PM<sub>2.5</sub> design values: calculated using the data handling described by 40 CFR Appendix N to Part 50 - Interpretation of the National Ambient Air Quality Standards for PM<sub>2.5</sub>. We downloaded the design values for all sites in the United States for all available days (including potential exceptional events) for 2015-2017 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM<sub>2.5</sub> hourly, daily, and annual average mass concentrations: calculated from regulatory-quality (Federal Reference Method or Federal Equivalent Method) monitors. When a single site has multiple monitors, the figures shows the average from all monitors at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2017 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- Near-road sites: designated from the list of near-road sites found at <https://www3.epa.gov/ttnamti1/files/nearroad/Near-road%20Monitoring%20Network%20Site%20List%20-%20May%202017.xlsx>.
- The near-road PM<sub>2.5</sub> increment is calculated by excluding the near-road site within a CBSA, predict the interpolated concentration at the near-road site location using Inverse Distance Weighting (IDW), and subtract the predicted concentration from the actual concentration at the near-road site for each daily or hourly average. Only CBSAs with at least one non-near-road site within 5km of the near-road site are considered. For the Elizabeth, NJ figure, the Elizabeth Lab site was considered a near-road site for the IDW calculation.

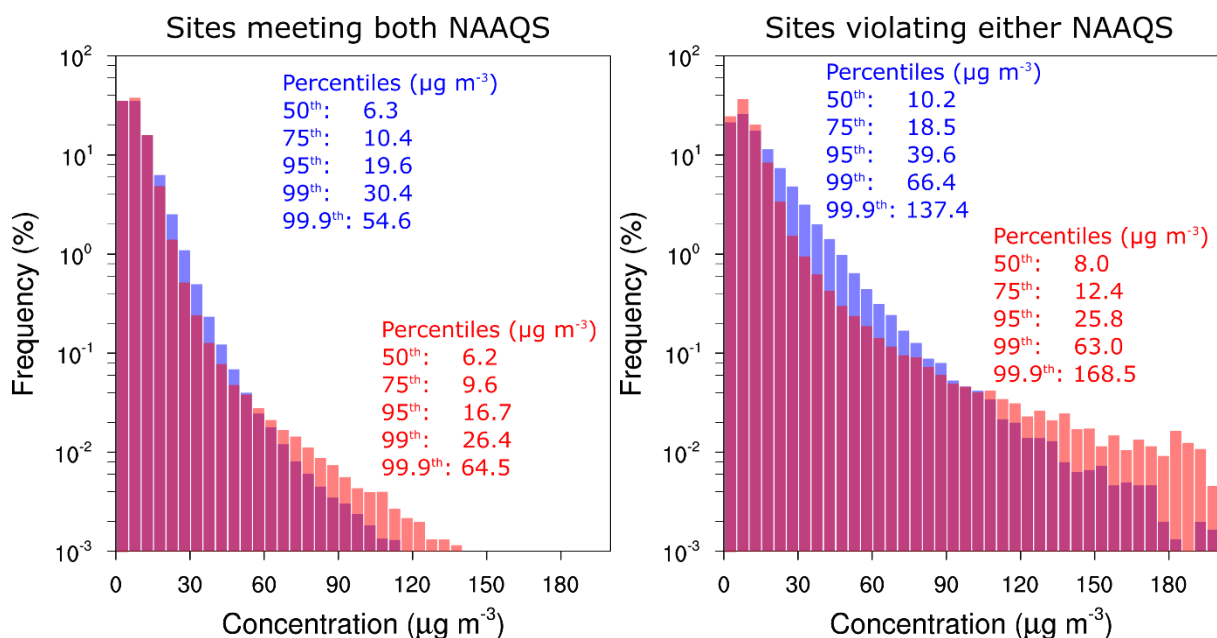
## **A.3 DATA SOURCES FOR SUB-DAILY PM<sub>2.5</sub> CONCENTRATION FIGURE**

- PM<sub>2.5</sub> hourly average mass concentrations: calculated from regulatory-quality Federal Equivalent Method monitors. The 2-hour and 5-hour averages were calculated for periods with each hourly average available. Only sites with a valid annual or 24-hour design value for 2015-2017 are shown in the figure. The percentages of 2-hour average PM<sub>2.5</sub> mass concentrations above 140 µg/m<sup>3</sup> at individual sites are illustrated in Figure A-1. Frequency distributions of 5-hour averages are presented in Figure A-2.





**Figure A-1. Percentages of 2015-2017 2-hour average PM<sub>2.5</sub> mass concentrations above 140 µg/m<sup>3</sup>.**



**Figure A-2. Frequency distribution of 2015-2017 5-hour averages for sites meeting both or violating either PM<sub>2.5</sub> NAAQS for October to March (blue) and April to September (red).**

## A.4 DATA SOURCES FOR ULTRAFINE FRACTION OF PM<sub>2.5</sub> MASS FIGURE

- Annual average particle number and mass concentrations for Bondville, IL: calculated from 24-hour average values for years with 66% data completion in 75% of the months of the year from 2000-2017. We downloaded the mass concentrations from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>) and particle number concentrations from NOAA's Earth System Research Laboratory's Global Monitoring Division (<https://www.esrl.noaa.gov/gmd> ).

## A.5 METHODS FOR PREDICTING AMBIENT PM<sub>2.5</sub> BASED ON HYBRID MODELING APPROACHES

### A.5.1 Data Sources for 2011 PM<sub>2.5</sub> Spatial Fields

- The “HU2017” fields were provided by Professor Yang Liu of Emory University in the form of comma-separated-values files (\*.csv) of daily average PM<sub>2.5</sub> on a national grid.
- The “DI2016” fields were provided by Dr. Qian Di of Harvard in the form of MATLAB files (\*.mat) of daily average PM<sub>2.5</sub> on a national grid.
- The “VD2019” fields were provided by Dr. Aaron van Donkelaar in the form of netCDF files (\*.nc) of annual average concentration. These files are also available at: [http://fizz.phys.dal.ca/~atmos/martin/?page\\_id=140](http://fizz.phys.dal.ca/~atmos/martin/?page_id=140).
- The “downscaler” files were developed in terms of daily average Downscaler predictions on a national grid following methods described in the risk assessment appendix.

### A.5.2 Data Averaging and Coefficient of Variation

- PM<sub>2.5</sub> concentration fields were loaded into R version 3.4.4, and daily fields were averaged to the annual period. Concentrations for each method at prediction points were then averaged to the corresponding CMAQ grid cells to enable consistent comparisons for Figure 2-24, 2-26, and Table 2-3.
- The coefficient of variation (CoV) was calculated for each grid cell using the following formula

$$CoV(\%) = \frac{100}{\bar{P}} \sqrt{\frac{\sum_{i=1}^N (P_i - \bar{P})^2}{N}}$$

where P is the prediction for each of the four methods (i.e., N=4).

## A.6 ANALYSES OF BACKGROUND PM

- Data sources for Figure 2-30: Smoke and fire detections observed by MODIS in August 2017
  - Image was produced using the NASA Worldview platform (<https://worldview.earthdata.nasa.gov/>). Layers selected were 1) Corrected

Reflectance and 2) Fires and Thermal Anomalies, both from Aqua/MODIS. Day selected was August 4, 2017.

- Data sources for Figure 2-31: Fine PM mass time series during 2017 from North Cascades IMPROVE site
  - Image was archived from the IMPROVE website ([http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF\\_PmHazeComp](http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_PmHazeComp); hosted by CIRA/CSU and sponsored by NPS and USFS) for the North Cascades (NOCA1) site in 2017.
- Data sources for Figure 2-32: Speciated annual average fine PM mass from IMPROVE at select remote monitors in 2004 and 2016
  - Speciated IMPROVE data from 2004 and 2016 ([http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF\\_PmHazeComp](http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_PmHazeComp)) were averaged annually for each monitor. Corresponding monitor locations are shown in Figure 2-32.

## **APPENDIX B. DATA INCLUSION CRITERIA AND SENSITIVITY ANALYSES**

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This appendix presents supplemental information on the methods used to conduct the analyses discussed in section 3.2.3.2 of this PA. It also presents information on additional sensitivity analyses. Section B.1 provides supplemental information on the forest plots presented in Figures 3-3 to 3-6. Sections B.2 and B.3 provide supplemental information on the study-reported PM<sub>2.5</sub> concentrations presented in Figure 3-7 and Figure 3-8. Sections B.4 to B.6, and sections B.8 to B.10, present supplemental information and sensitivity analyses related to the analyses of study area pseudo-design values in section 3.2.3.2.2. Section B.7 presents comparisons between annual and daily design values in CBSAs.

## **B.1 FOREST PLOTS**

Forest Plots exhibiting effect estimates and 95% confidence intervals from epidemiologic studies that have the potential to be most informative in reaching conclusions on the adequacy of the current primary PM<sub>2.5</sub> standards are shown in Figure 3-3 to Figure 3-6. Epidemiologic studies included in these figures support “causal” or “likely to be causal” relationships with PM exposures in the ISA U.S. EPA, 2019 and include mortality (all-cause mortality, CVD mortality, respiratory mortality, lung cancer mortality), and morbidity (asthma incidence, lung cancer incidence, lung function and lung development, CVD and respiratory emergency room visit or hospital admission) health endpoints. Further, studies included in Figure 3-3 to Figure 3-6 were restricted to multi-city studies in the United States or Canada. Multi-city studies within a single State were not included, with the exception of respiratory morbidity endpoints, where multi-city studies were limited. For some of the major cohort studies included in the previous ISA, like the American Cancer Society (ACS) cohort, we included new studies that reanalyze epidemiologic associations for multiple mortality endpoints (e.g. lung cancer mortality and IHD mortality) and an extension of follow-up periods (e.g., Pope et al. (2015b), Turner et al. (2016), Jerrett et al. (2016), and Thurston et al. (2016b)), as well as a reanalysis (Krewski et al. (2009) of the original ACS dataset, including an extended follow-up period, that was evaluated in the previous ISA (EPA, 2009). In total, 67 studies were included in Figure 3-3 to Figure 3-6.

## **B.2 MONITORED PM<sub>2.5</sub> CONCENTRATIONS IN KEY EPIDEMIOLOGIC STUDIES**

Of the 67 key studies identified in Figure 3-3 to Figure 3-6, Figure 3-7 includes key epidemiologic studies that report an overall study mean or median concentration of PM<sub>2.5</sub> (as opposed to a study mean/median range across study area locations) and based on ambient PM<sub>2.5</sub>

monitored data. The plot includes studies that report significant effect estimates (22 studies) and studies that only report non-significant effect estimates (5 studies). Further, to be included, only key studies for which the years of air quality data used to estimate exposures overlap entirely with the years during which health events are reported were included. The PM<sub>2.5</sub> concentrations reported by studies that estimate exposures from air quality corresponding to only part of the study period, often including only the later years of the health data (e.g., Miller et al., 2007; Hart et al., 2011; Thurston et al., 2013; Weichenthal et al., 2014; Weichenthal et al., 2016a; Pope et al., 2015a; Villeneuve et al., 2015; Turner et al., 2016), are not likely to reflect the full ranges of ambient PM<sub>2.5</sub> concentrations that contributed to reported associations.<sup>1</sup>

Some of the included studies also provide city-specific study mean concentrations and city-specific health events. Hence, PM<sub>2.5</sub> exposure estimates corresponding to the 10<sup>th</sup> and 25<sup>th</sup> percentiles of those events were determined in the following manner. City-specific cases and PM<sub>2.5</sub> concentrations were input in ascending order by PM<sub>2.5</sub> concentration. The city-specific percent of cases was calculated as a proportion of the total study cases and the cumulative percent of cases was determined. The PM<sub>2.5</sub> concentration associated with the cumulative percent closest to the 10<sup>th</sup> and 25<sup>th</sup> percentiles were input in Figure 3-7 and the cumulative percent values closest to the associated 10<sup>th</sup> and 25<sup>th</sup> percentile inputs are shown in Table B-1<sup>2</sup>. Data for Bell et al. (2008) and Zanobetti and Schwartz (2009) were previously provided by the study authors, as described in Rajan (2011).

**Table B-1. PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of estimated health events.**

Citation	10 <sup>th</sup> Percentile PM <sub>2.5</sub> (µg/m <sup>3</sup> ) (Cumulative percent value closest)	25 <sup>th</sup> Percentile PM <sub>2.5</sub> (µg/m <sup>3</sup> ) (Cumulative percent value closest)
Bell et al. (2008)	9.8	11.5
Franklin et al. (2007)	10.4 (11.1%)	12.9 (25.3%)
Stieb et al. (2009)	6.7 (16.5%)	6.8 (20.5%)
Szyszkowicz (2009)	6.4 (4.1%)	6.5 (18.6%)
Zanobetti and Schwartz (2009)	10.3	12.5

<sup>1</sup> This is an issue only for some studies of long-term PM<sub>2.5</sub> exposures. While this approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term PM<sub>2.5</sub> exposures, under the assumption that spatial patterns in PM<sub>2.5</sub> concentrations are not appreciably different during time periods for which air quality information is not available (e.g., Chen et al., 2016), our interest is in understanding the distribution of ambient PM<sub>2.5</sub> concentrations that could have contributed to reported health outcomes.

<sup>2</sup> That is, 25% of the total health events occurred in study locations with mean PM<sub>2.5</sub> concentrations (i.e., averaged over the study period) below the 25<sup>th</sup> percentiles identified in Figure 3-7 and 10% of the total health events occurred in study locations with mean PM<sub>2.5</sub> concentrations below the 10<sup>th</sup> percentiles identified.

### B.3 HYBRID MODEL PREDICTED PM<sub>2.5</sub> CONCENTRATIONS IN KEY EPIDEMIOLOGIC STUDIES

Figure 3-8 focuses on multicity studies that are part of the evidence supporting “causal” or “likely to be causal” determinations in the ISA and that use air quality data to estimate PM<sub>2.5</sub> exposures for the entire range of years during which health events occurred. In addition, as detailed in section 3.2.3.2.1, we also consider the approach used to validate model predictions, and the studies included in Figure 3-8 are those for which relatively robust model validation analyses are reported to have been conducted for the full range of years during which PM<sub>2.5</sub> exposures are estimated in the health study.<sup>3</sup> All studies that met the criteria for inclusion were conducted in the U.S.

Figure 3-8 presents overall means of hybrid model-predicted PM<sub>2.5</sub> concentrations for key studies, and the concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of estimated exposures or health events, when available. For Di et al. (2017b), we present 25<sup>th</sup> and 10<sup>th</sup> percentiles of annual PM<sub>2.5</sub> concentrations by zip code corresponding to long-term exposure estimates, while for Di et al. (2017a), we present daily air pollution concentrations (short-term exposure estimates) corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of deaths at the zip-code level. These values, along with other percentiles, are illustrated in Figure B-1 and Figure B-2 (Jenkins, 2019a, Jenkins, 2019b). The study authors for Di et al. (2017b) additionally provided information on population weighted percentile values corresponding to long-term PM<sub>2.5</sub> exposure (Chan, 2019). These are presented in Table B-2. For other studies included in Figure 3-8 [Kloog et al. (2012), Kloog et al. (2014), Shi et al. (2016), Wang et al. (2017)], 25<sup>th</sup> percentiles of exposure estimates were derived from study manuscripts of air quality descriptive statistics and can be found in Table B-3.

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<sup>3</sup> For example, due to lack of spatial field availability before 1998, Crouse et al. (2015) use median annual PM<sub>2.5</sub> concentrations for the 1998-2006 time period (van Donkelaar et al., 2010; van Donkelaar et al., 2015a; van Donkelaar et al., 2013) to predict exposures during the 1984-2006 period. Similarly, for Pinault et al. (2016), model validation is for 2004 to 2008 (van Donkelaar et al., 2015b) while exposures are estimated for 1998 to 2012. Paciorek et al. (2009), which presents the model validation results for Puett et al. (2009) and Puett et al. (2011), notes that PM<sub>2.5</sub> monitoring was sparse prior to 1999, with many of the available PM<sub>2.5</sub> monitors in rural and protected areas. Therefore, Paciorek et al. (2009) conclude that coverage in the validation set for most of the study period (1988-1998) is poor and that their model “strongly” underestimates uncertainty Paciorek et al. (2009), p. 392 in published manuscript). Hystad et al. (2013) used exposure fields developed by calibrating satellite-based PM<sub>2.5</sub> surfaces from a recent period (van Donkelaar et al., 2010) to estimate exposure for the 1975 to 1994 (Hystad et al., 2012). Hystad et al. (2012) noted that a random effect model was used to estimate PM<sub>2.5</sub> based on TSP measurements and metropolitan indicator variables because only small number of PM<sub>2.5</sub> measurements were available, and no measurements were made prior to 1984. Thus, these studies are not included in Figure 3-8.



# Percentiles of PM<sub>2.5</sub> By Zip Code

Thresholds defining percentiles of PM<sub>2.5</sub> exposure for each zip code.

Percentile of PM <sub>2.5</sub> , Based on ZIP code	PM <sub>2.5</sub> Value
0%	0.0209025
5%	6.1962803
10%	7.2742546
15%	8.0043245
20%	8.5892973
25%	9.0612931
30%	9.4644903
35%	9.8273901
40%	10.1797192
45%	10.5371831
50%	10.9015790
55%	11.2791073
60%	11.6666804
65%	12.0707952
70%	12.4916270
75%	12.9386305
80%	13.4294338
85%	13.9765291
90%	14.6375324
95%	15.6106067
100%	32.5759482

**Figure B-1. Percentiles of annual PM<sub>2.5</sub> concentrations by zip code corresponding to long-term exposure estimates in Di et al., 2017b.**

**Table B-2. Population weighted percentiles of annual PM<sub>2.5</sub> concentrations by zip code corresponding to long-term exposure estimates in Di et al., 2017b.**

Percentile	Population Weighted PM <sub>2.5</sub> (µg/m <sup>3</sup> )
0.0	0.0
5.0	7.1
10.0	7.9
15.0	8.6
20.0	9.1
25.0	9.5
30.0	9.9
35.0	10.3
40.0	10.6
45.0	11.0
50.0	11.4
55.0	11.7
60.0	12.1
65.0	12.5
70.0	12.9
75.0	13.4
80.0	13.9
85.0	14.4
90.0	15.1
95.0	16.1
100.0	32.6

# Percentiles of PM<sub>2.5</sub> By Zip Code

Thresholds defining percentiles of Daily PM<sub>2.5</sub> exposure for each zip code.

Percentile of Daily PM <sub>2.5</sub> , Based on ZIP code	PM <sub>2.5</sub> Value
0%	0.0006378
5%	3.8286960
10%	4.7224770
15%	5.4309290
20%	6.0727840
25%	6.6863868
30%	7.2922285
35%	7.9031599
40%	8.5292050
45%	9.1836408
50%	9.8740436
55%	10.6124979
60%	11.4111824
65%	12.2910351
70%	13.2835707
75%	14.4301324
80%	15.8159815
85%	17.5894591
90%	20.0959732
95%	24.4759063
100%	201.3071287

**Figure B-2. Daily air pollution concentrations (short-term exposure estimates) corresponding to various percentiles of deaths at the zip-county level in Di et al., 2017a.**

**Table B-3. PM<sub>2.5</sub> concentrations corresponding to the 25<sup>th</sup> and 10<sup>th</sup> percentiles of estimated exposures in Figure 3-8.**

Citation	10 <sup>th</sup> Percentile PM <sub>2.5</sub> (µg/m <sup>3</sup> )	25 <sup>th</sup> Percentile PM <sub>2.5</sub> (µg/m <sup>3</sup> )
Di et al. (2017a)	4.7	6.7
Di et al. (2017b)	7.3	9.1
Kloog et al. (2012)		6.4
Kloog et al. (2014)		7.9
Shi et al. (2016)		4.6
Shi et al. (2016)		6.2
Wang et al. (2017)		9.1

## B.4 DESIGN VALUE BOX PLOT INCLUSION CRITERIA

Studies selected from Figure 3-3 to Figure 3-6 for inclusion in Figure 3-9 and Figure B-9 (box plots of pseudo-design value distributions) are those studies that define the study area/s (city or county) and study-specific populations or study area health events. Studies that provide county/city-specific health counts across the study period include: Lepeule et al. (2012); Kioumourtzoglou et al. (2016); Franklin et al. (2008); Zanobetti et al. (2014); Yap et al. (2013); Ostro et al. (2016); and Weichenthal et al. (2016b). In U.S. studies for which health counts were not provided, county-specific population data derived from the 2015 American Community Survey data<sup>4</sup> was used. For Canadian studies, city-specific population from 2016 Statistics Canada<sup>5</sup> was used.

In constructing the plots in Figure 3-9 and Figure B-9, several assumptions were made. In studies that report mortality, hospital admissions data or emergency department visits, it was assumed that the number of cases is directly proportional to the population of the area. To test this assumption, census population data and case event data is used in a sensitivity analysis and discussed in Section B.6. It was assumed that the population of a county did not change substantially over time relative to other counties, and that the rank order is consistent over time since only U.S. 2015 Census data and 2016 data from Statistics Canada was used. In studies that state the study area is the entire U.S. (*i.e.* in Medicare studies), it was assumed that cases came from each county of the U.S. (*i.e.*, proportional to the county population 65 years or older for Medicare studies) and therefore, air quality was used from all U.S. counties with data.

<sup>4</sup>Available from: <https://data.census.gov/cedsci/>

<sup>5</sup> Available from: <https://www12.statcan.gc.ca/census-recensement/2016/dp-pd/prof/index.cfm?Lang=E&TABID=1>

Studies that had health data that started before 1999 in the U.S. and before 2000 in Canada were excluded since U.S. and Canadian PM<sub>2.5</sub> monitoring became more widespread starting around these times. 29 studies met these criteria and are found in Figure 3-9 and Figure B-9. Details on study-area assignment (Section B.4.1), population/health events assignment (Section B.4.2), and air quality linkages (Section B.4.3) for studies included in the pseudo-design value (DV) box plots are outlined below.

#### **B.4.1 Study area assignment**

The first step in developing Figure 3-9 and Figure B-9 was to identify the study area. The U.S. based analysis is at the county-level and each U.S. county within the study area was identified for each specific study. For the studies that provided city names, the U.S. cities were used to identify all counties from the metropolitan area of that city, unless the entire city is contained within a single county or unless otherwise noted. In cases of studies where the study authors state that data was used for the entire U.S., all U.S. counties were included in the study area assignment. For example, all counties were included in studies using Medicare or National Center for Health Statistics (NCHS) data, unless the study identified a subset of cities or counties included. For some studies, there are uncertainties related to how we chose counties to represent study areas. Many studies identify the counties or cities used for the study; however, some only said that they used HA or ED visit data from a specific state or region and didn't specify any counties or cities. In those instances, we operated under the assumption that every county that fell within the state or region identified contributed to the study population.<sup>6</sup>

For studies based in Canada, city was used as the geographic unit for the study area, since Canadian air quality data is available at the city-level. In cases where a study notes that the study is a national study, all cities for which air quality was available were included to define the study area.

Studies were excluded from Figure 3-9 and Figure B-9 if the counties included are unclear or not identified. Studies were also excluded in situations where the study population selection criteria was not random and not likely to be proportional to the underlying population, or the population selection criteria was not clearly specified (e.g., such as in cohort studies like the American Cancer Society cohort (ACS), Nurses' Health Study cohort (NHS), and the Health Professionals Follow-up Study (HPFS)).

#### **B.4.2 Study population assignment**

Based on the study areas identified in step 1, area-specific health events or populations were then assigned to U.S. counties and Canadian cities. If the study reported health events for

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<sup>6</sup> As discussed below (section B.4.3), not all counties have PM<sub>2.5</sub> monitor.

U.S. counties or Canadian cities, we assigned those events to the specific counties or cities identified. In the absence of reported health events at these geographic levels for studies where hospital admissions or emergency department visits data, Medicare data, NCHS data, or other national survey data was used, we assumed that study participants were randomly selected and that the number of health events reported in the study was directly proportional to the population of the area. For these studies, area-specific populations were assigned using U.S. 2015 American Community Survey population data or 2016 Canadian population data (Statistics Canada).<sup>7</sup> For the remaining studies (i.e., for which the number of study participants or health events in each location was not provided and for which the study population selection process appeared to not be random or proportional to underlying populations), area-specific populations were not assigned, and the studies were excluded from analysis.

In U.S. studies that evaluate cities, and for which some cities are associated with more than one county, 2016 “City-to-County finder” data from Stats America<sup>8</sup> was used to find the proportional distribution of city population within each county, and the same proportional distribution strategy was used to divide the reported health events between counties. An example of the proportional distribution of city populations within counties is illustrated in Table B-4, using a subset of cities reported in Zanobetti et al. (2014). Note, for cities not listed in Table B-4, the city population was associated with one county and as a result, the health events for the specific city were assigned to the corresponding county.

**Table B-4. Percent of population by county associated with each city reported in the study area.**

City	Counties (% of population)
Atlanta, GA	Dekalb (6.7%), Fulton (93.3%)
Austin, TX	Travis (95.5%), Williamson (4.5%)
Columbus, OH	Franklin (97.9%), Fairfield (1.2%)
Dallas, TX	Dallas (93.9%), Collin (3.9%), Denton (2.2%)
Fort Worth, TX	Tarrant (99%), Denton (1%)
Holland, MI	Ottawa (78.8%), Allegan (21.2%)
Houston, TX	Harris (98%), Fort Bend (2%)
Lansing, MI	Ingham (96%), Eaton (4%)
Middletown, OH	Butler (94.5%), Warren (5.5%)

<sup>7</sup> While this approach contributes uncertainty to our analyses of pseudo-design values, we do not expect the rank order of county population to substantially differ over the time periods of the studies and, therefore, we do not expect this uncertainty to systematically bias our results.

<sup>8</sup> Available from: <http://www.statsamerica.org/Default.aspx>

New York, NY	Kings (30.6%), Queens (27.3%), New York (19.4%), Bronx (16.9%), Richmond (5.7%)
Oklahoma City, OK	Oklahoma (81.3%), Cleveland (11%), Canadian (7.7%)
Tulsa, OK	Tulsa (98.4%), Osage (1.6%)
Charleston, SC	Charleston (93.3%), Berkeley (6.7%)

#### B.4.3 Air Quality data assignment by study area, by study period

The third step in developing Figure 3-9 and Figure B-9 was to assign air quality data by study area, by study period. Ambient air quality data for PM<sub>2.5</sub> in the United States and Canada became more widely available across a broad proportion of the United States and Canada in the late 1990s. To ensure a large proportion of air quality data points and subsequent 3-year design values were available, the studies selected were those that examine air quality data starting in 1999 for U.S. studies and 2000 for Canadian studies. Construction of pseudo-design value box plots (Figure 3-9 and Figure B-9) is described below. The air quality metric is termed a “pseudo-design value”, since both FRM/FEM monitors, as well as high quality non-FRM/FEM data, are used to expand the number of areas with air quality data.<sup>9</sup> Air quality data in the U.S. was obtained from the EPA Air Quality System (AQS)<sup>10</sup>. For regulatory monitors, design values were calculated using the data handling described by 40 CFR Appendix N to Part 50 - Interpretation of the National Ambient Air Quality Standards for PM<sub>2.5</sub>. For non-regulatory data, only monitors with 75% completeness for each of the 12 quarters in a 3-year design value period were included. For Canadian air quality data, only sites with 75% completeness for each year of the 3-year design value period were included.<sup>11</sup> These criteria are slightly different than that of actual design values, which have strict rounding conventions and substitution tests for sites with less than 75% completeness for each quarter. For each given study and each previously identified study area, each valid pseudo-DV was identified over each study period. For each county, or city, the maximum PM<sub>2.5</sub> pseudo-design value for each 3-year period of the study was identified. Next, by county/city, the study-period average of the maximum pseudo-design value was calculated (“average maximum pseudo-design value” or “average max pseudo-DV”). For each study, locations were ordered by increasing average max pseudo-DVs and the corresponding population or number of health events was used to calculate the cumulative percent of population

<sup>9</sup> As noted in section B.5, sensitivity analyses using only regulatory FRM/FEM monitors gave similar results.

<sup>10</sup> Available from: <https://www.epa.gov/aqs>

<sup>11</sup> Available from: <http://maps-cartes.ec.gc.ca/rnsps-naps/data.aspx?lang=en>

at or below each corresponding average max pseudo-DV. Next, the average max pseudo-DV associated with the cumulative population closest to the 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> percentiles were identified. The actual cumulative percents that are closest to the 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup>, and 95<sup>th</sup> percentiles, for all long- and short-term exposure studies and for annual and 24-hr PM<sub>2.5</sub> concentrations, are illustrated in Figure B-3 and Figure B-4. The average max pseudo-DVs associated with these percentiles in these studies are then presented in Table B-5 and Table B-6. Counties that had no air quality monitors or no valid design values did not contribute to the percentile calculation.



Citation	Population in the study area	Percentile				
		5	25	50	75	95
Di et al., 2017b*	2015 population 65 and older	• 5.01	• 25.14	• 50.02	75.02 •	94.35 •
Kiomourtzoglou et al., 2016*	2015 population 65 and older	• 5.25	• 25.05	• 49.70	75.02 •	97.07 •
	Kiomourtzoglou 2016	• 5.03	• 24.64	• 50.07	75.17 •	96.52 •
Lepule et al., 2012*	Lepule 2012	• 20.60	• 20.60	• 59.20	79.18 •	100.00 •
McConnell et al., 2010	2015 population 18 and under	• 1.99	• 30.82	• 30.82	76.34 •	100.00 •
Pinault et al., 2016*	Canada 2016	• 5.15	• 24.29	• 50.50	80.61 •	99.32 •
Shi et al., 2016*	2015 population 65 and older	• 6.02	• 25.91	• 51.49	75.44 •	92.48 •
Urman et al., 2014*	Urman 2014 Lung Function 5-7	• 14.63	• 25.12	63.22 •	75.70 •	100.00 •
Wang et al., 2017*	2015 population 65 and older	• 5.17	• 25.67	• 49.43	75.13 •	94.91 •
		0 50 100	0 50 100	0 50 100	0 50 100	0 50 100
Citation	Population in the study area	Percentile				
		5	25	50	75	95
Franklin et al., 2008*	2015 population	• 3.55	• 22.57	• 49.63	69.89 •	94.33 •
	Franklin 2008	• 5.01	• 24.82	• 49.61	72.68 •	96.52 •
Dai et al., 2014*	2015 population	• 4.59	• 24.80	• 49.90	75.84 •	97.41 •
Baxter et al., 2017*	2015 population	• 4.10	• 25.03	• 50.77	75.50 •	97.64 •
Zanobetti et al., 2014*	2015 population 65 and older	• 4.86	• 25.02	• 49.22	74.69 •	96.07 •
	Zanobetti 2014	• 5.85	• 25.25	• 51.10	74.93 •	94.41 •
Zanobetti and Schwartz, 2009*	2015 population	• 4.61	• 25.46	• 50.67	74.99 •	97.94 •
Lee et al., 2015b*	2015 population	• 5.32	• 23.95	• 50.72	77.78 •	94.85 •
Yap et al., 2013*	2015 population 18 and under	• 5.70	• 22.65	• 45.34	81.31 •	90.33 •
	Yap 2013 Asthma 1-9	• 5.03	• 28.11	• 36.95	82.59 •	92.58 •
Ostro et al., 2016*	2015 population	• 3.87	• 34.47	• 38.91	85.23 •	95.98 •
	Ostro Asthma 2016	• 4.00	• 34.10	• 43.89	77.50 •	96.82 •
	Ostro COPD 2016	• 4.50	• 35.62	• 42.60	71.35 •	94.38 •
Malig et al., 2013*	2015 population	• 5.11	• 25.44	• 52.06	62.03 •	92.28 •
Peng et al., 2009*	2015 population 65 and older	• 4.82	• 24.39	• 49.90	75.66 •	97.67 •
Zanobetti et al., 2009*	2015 population 65 and older	• 4.75	• 27.84	• 46.98	73.26 •	94.64 •
Domini et al., 2006*	2015 population 65 and older	• 5.14	• 25.12	• 50.17	74.75 •	97.29 •
Kloog et al., 2014*	2015 population 65 and older	• 5.67	• 24.74	• 49.43	74.51 •	94.99 •
Bell et al., 2008*	2015 population 65 and older	• 4.71	• 25.41	• 50.34	74.62 •	97.50 •
Bell et al., 2014*	2015 population 65 and older	• 29.08	• 29.08	• 57.18	72.05 •	100.00 •
Bravo et al., 2017*	2015 population 65 and older	• 4.93	• 25.05	• 50.21	75.83 •	94.84 •
Bell et al., 2015*	2015 population 65 and older	• 4.61	• 24.40	• 49.94	74.99 •	96.21 •
Kloog et al., 2012*	2015 population 65 and older	• 5.25	• 25.06	• 47.14	77.25 •	91.37 •
Weichenthal et al., 2016b	Canada 2016	• 5.71	• 30.50	• 40.98	89.25 •	98.73 •
	Weichenthal MI 2016	• 5.20	• 22.76	• 57.63	90.55 •	98.36 •
Weichenthal et al., 2016c*	Canada 2016	• 3.82	• 23.55	• 28.54	86.98 •	98.47 •
Shi et al., 2016*	2015 population 65 and older	• 6.02	• 25.91	• 51.49	75.44 •	92.48 •
		0 50 100	0 50 100	0 50 100	0 50 100	0 50 100

**Figure B-3. Cumulative population percentile closest to the 5<sup>th</sup>, 25<sup>th</sup>, 50, 75, and 95<sup>th</sup> percentile: studies of long-term exposure and annual PM<sub>2.5</sub> concentrations (top panel) and studies of short-term exposure and annual PM<sub>2.5</sub> concentrations (bottom panel).**

Citation	Population in the study area	Percentile				
		5	25	50	75	95
Di et al., 2017b*	2015 population 65 and older	• 4.90	• 25.07	• 49.93	74.99 •	96.14 •
Kiomourtzoglou et al., 2016*	2015 population 65 and older	• 4.87	• 25.03	• 50.06	76.14 •	95.08 •
	Kiomourtzoglou 2016	• 5.13	• 25.01	• 49.45	74.53 •	94.93 •
Lepeule et al., 2012*	Lepeule 2012	• 20.60	• 20.60	• 59.20	79.18 •	100.00 •
McConnell et al., 2010	2015 population 18 and under	• 1.99	• 30.82	• 30.82	76.34 •	100.00 •
Pinault et al., 2016*	Canada 2016	• 4.72	• 24.06	• 50.47	68.97 •	90.37 •
Shi et al., 2016*	2015 population 65 and older	• 6.02	• 26.19	• 51.13	75.83 •	92.48 •
Urman et al., 2014*	Urman 2014 Lung Function 5-7	• 14.63	• 25.12	63.22 •	75.70 •	100.00 •
Wang et al., 2017*	2015 population 65 and older	• 3.06	• 25.37	• 49.62	75.12 •	94.95 •
		0 50 100	0 50 100	0 50 100	0 50 100	0 50 100
Citation	Population in the study area	Percentile				
		5	25	50	75	95
Franklin et al., 2008*	2015 population	• 3.55	• 27.33	• 49.61	86.91 •	95.54 •
	Franklin 2008	• 5.01	• 23.87	• 49.73	71.01 •	93.52 •
Baxter et al., 2017*	2015 population	• 5.56	• 25.08	• 49.56	74.34 •	94.39 •
Zanobetti and Schwartz, 2009*	2015 population	• 4.18	• 26.01	• 49.51	76.42 •	95.24 •
Dai et al., 2014*	2015 population	• 6.14	• 26.92	• 49.82	74.62 •	95.38 •
Zanobetti et al., 2014*	2015 population 65 and older	• 4.86	• 24.97	• 49.12	75.49 •	95.36 •
	Zanobetti 2014	• 5.89	• 24.85	• 50.16	72.92 •	93.69 •
Lee et al., 2015b*	2015 population	• 5.34	• 26.43	• 51.02	74.81 •	95.69 •
Yap et al., 2013*	2015 population 18 and under	• 11.48	• 25.93	• 33.94	78.93 •	95.93 •
	Yap 2013 Asthma 1-9	• 9.51	• 26.25	• 28.11	73.75 •	93.18 •
Ostro et al., 2016*	2015 population	• 3.87	• 27.64	• 27.64	73.95 •	95.98 •
	Ostro Asthma 2016	• 4.00	• 19.83	• 53.44	72.76 •	96.82 •
	Ostro COPD 2016	• 4.50	• 20.02	• 48.76	71.80 •	94.38 •
Malig et al., 2013*	2015 population	• 6.17	• 24.65	• 45.67	74.74 •	94.83 •
Zanobetti et al., 2009*	2015 population 65 and older	• 4.17	• 25.43	• 52.92	69.90 •	96.63 •
Peng et al., 2009*	2015 population 65 and older	• 4.87	• 24.68	• 50.38	75.12 •	94.57 •
Bell et al., 2014*	2015 population 65 and older	• 29.08	• 29.08	• 57.18	72.05 •	100.00 •
Dominici et al., 2006*	2015 population 65 and older	• 5.42	• 25.29	• 50.67	75.36 •	95.93 •
Kloog et al., 2014*	2015 population 65 and older	• 4.91	• 24.52	• 50.47	74.90 •	94.04 •
Bell et al., 2008*	2015 population 65 and older	• 4.09	• 26.09	• 49.97	74.21 •	95.28 •
Bell et al., 2015*	2015 population 65 and older	• 5.02	• 24.83	• 49.95	75.01 •	95.42 •
Bravo et al., 2017*	2015 population 65 and older	• 4.43	• 25.12	• 50.71	74.88 •	95.06 •
Kloog et al., 2012*	2015 population 65 and older	• 5.40	• 25.52	• 50.23	77.51 •	91.14 •
Weichenenthal et al., 2016c*	Canada 2016	• 3.82	• 25.69	• 28.54	86.98 •	98.47 •
Weichenenthal et al., 2016b	Canada 2016	• 5.71	• 23.48	• 40.98	89.25 •	98.73 •
	Weichenenthal MI 2016	• 7.40	• 24.05	• 47.71	90.55 •	98.36 •
Shi et al., 2016*	2015 population 65 and older	• 6.02	• 26.19	• 51.13	75.83 •	92.48 •
		0 50 100	0 50 100	0 50 100	0 50 100	0 50 100

**Figure B-4. Cumulative population percentile closest to the 5th, 25th, 50, 75, and 95th percentile: studies of long-term exposure and 24-hr PM<sub>2.5</sub> concentrations (top panel) and studies of short-term exposure and 24-hr PM<sub>2.5</sub> concentrations (bottom panel).**

**Table B-5. Annual average maximum pseudo-DVs corresponding to population or health event percentiles in box-and-whisker plots in Figure 3-9.<sup>12</sup>**

Citation	Pseudo DVs by percentiles				
	5th percentile	25th percentile	50th percentile	75th percentile	95th percentile
Baxter et al., 2017	7.53	11.86	14.63	16.70	21.95
Bell et al., 2008	8.55	11.35	13.72	15.94	23.05
Bell et al., 2014	12.43	12.43	13.30	13.40	16.47
Bell et al., 2015	8.18	10.81	12.81	15.31	20.95
Bravo et al., 2017	8.17	11.20	13.03	14.93	17.40
Dai et al., 2014	10.13	12.43	14.94	16.96	21.96
Di et al., 2017b	6.63	9.98	11.70	13.88	19.38
Di et al., 2017a	6.63	9.98	11.70	13.88	19.38
Dominici et al., 2006	9.15	12.05	14.10	17.00	24.70
Franklin et al., 2008	11.30	14.13	15.79	19.97	22.56
Kioumourtoglou et al., 2016	8.49	10.86	13.36	15.70	20.50
Kloog et al., 2012	6.35	9.50	11.17	12.94	14.04
Kloog et al., 2014	11.10	12.44	13.77	15.22	16.96
Lee et al., 2015a	9.20	10.53	11.60	12.98	13.20
Lepeule et al., 2012	8.65	8.65	14.26	14.82	16.29
Malig et al., 2013	8.25	11.05	15.39	19.31	21.04
McConnell et al., 2010	10.50	16.30	16.30	20.56	24.11
Ostro et al., 2016	10.97	13.52	19.00	19.32	20.45
Peng et al., 2009	8.32	11.86	14.70	16.86	21.96
Pinault et al., 2016	4.33	6.00	7.31	8.62	10.57
Shi et al., 2016	6.11	8.70	9.93	10.95	13.63
Urman et al., 2014	9.85	16.70	21.59	22.87	25.58
Wang et al., 2017	7.27	9.03	11.09	13.13	14.94
Weichenthal et al., 2016b	4.20	6.67	7.39	8.42	8.44
Weichenthal et al., 2016c	4.22	7.22	7.39	8.42	8.44
Yap et al., 2013	12.68	17.67	21.05	22.56	23.93
Zanobetti et al., 2009	11.60	14.15	16.90	22.30	24.00

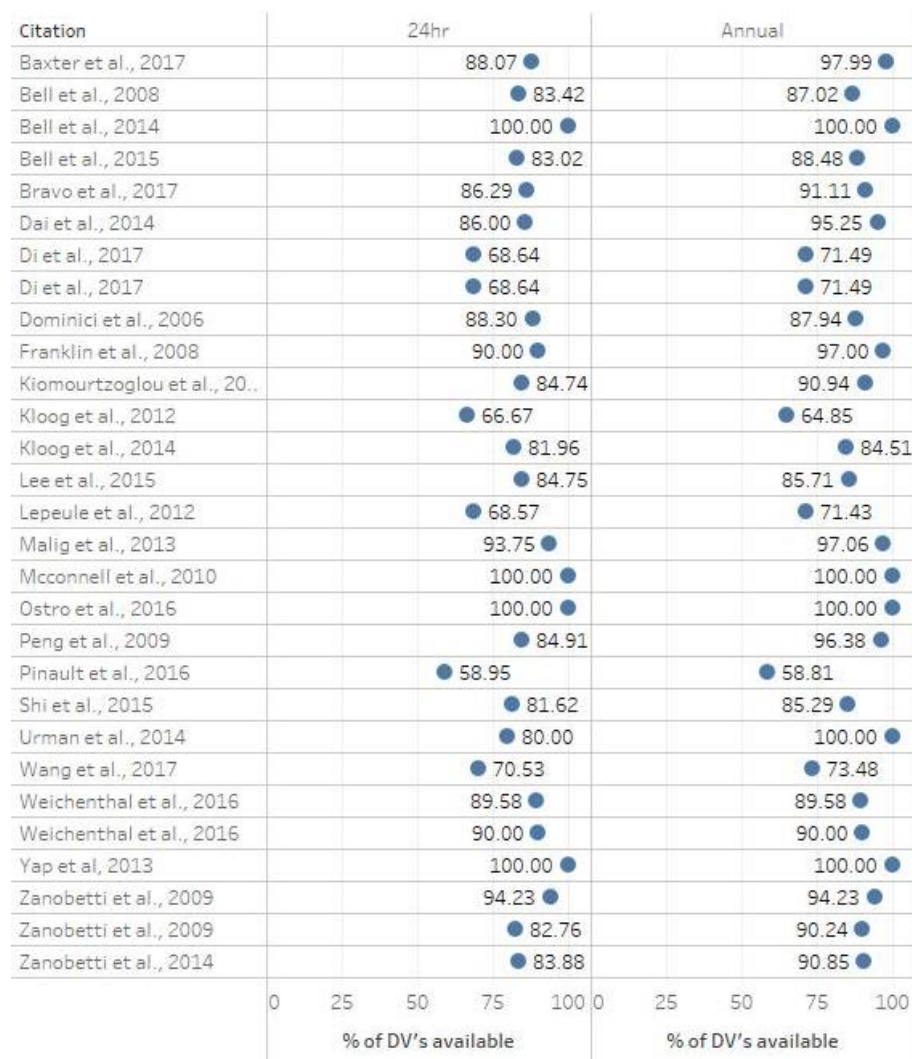
<sup>12</sup> As a sensitivity analysis, we also calculated study period averages of maximum design values using only regulatory FRM/FEM monitors for Di et al. (2017a) and Di et al. (2017b) and Shi et al. (2016). Results were similar to those based on the pseudo-design values using both regulatory and non-regulatory monitors. Using only regulatory monitors for the studies by Di et al. (2017a) and Di et al. (2017b), 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> percentiles of annual design values were 7.4, 9.7, 11.7, 13.9 and 17.6 µg/m<sup>3</sup>, respectively. For these studies, 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> percentiles of 24-hour design values were 19, 26, 30, 36 and 49 µg/m<sup>3</sup>, respectively. For Shi et al., 2016, 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> percentiles of annual design values were 7.7, 9.1, 10.4, 11.4 and 13.0 µg/m<sup>3</sup>, respectively while 5<sup>th</sup>, 25<sup>th</sup>, 50<sup>th</sup>, 75<sup>th</sup> and 95<sup>th</sup> percentiles of 24-hour design values were 21, 26, 29, 31 and 35 µg/m<sup>3</sup>, respectively.

Zanobetti and Schwartz, 2009	9.72	12.18	14.43	17.30	23.05
Zanobetti et al., 2014	8.82	11.92	14.59	16.43	20.95

**Table B-6. 24-hr average maximum pseudo-DVs corresponding to population or health event percentiles in box-and-whisker plots in Figure B-9.**

Citation	Pseudo DVs by percentiles				
	5th percentile	25th percentile	50th percentile	75th percentile	95th percentile
Baxter et al., 2017	22.00	31.00	38.67	45.50	58.33
Bell et al., 2008	19.20	30.34	36.40	42.67	62.20
Bell et al., 2014	34.67	34.67	37.67	40.00	40.33
Bell et al., 2015	21.23	28.10	33.56	39.57	55.78
Bravo et al., 2017	19.00	28.00	33.00	37.50	43.00
Dai et al., 2014	22.13	31.34	38.14	45.25	64.80
Di et al., 2017b	17.35	25.38	30.27	35.50	51.18
Di et al., 2017a	17.35	25.38	30.27	35.50	51.18
Dominici et al., 2006	22.00	31.00	37.50	44.50	68.00
Franklin et al., 2008	28.93	30.75	40.75	55.00	64.75
Kioumourtzoglou et al., 2016	20.22	29.72	34.38	40.07	54.05
Kloog et al., 2012	20.77	30.40	32.50	36.80	37.89
Kloog et al., 2014	30.00	34.00	37.20	39.50	45.60
Lee et al., 2015a	19.73	23.00	24.33	26.33	29.23
Lepeule et al., 2012	22.00	22.00	30.20	34.77	41.29
Malig et al., 2013	28.50	40.50	48.00	52.00	65.20
McConnell et al., 2010	23.00	47.00	47.00	56.00	65.00
Ostro et al., 2016	27.67	40.33	50.27	54.68	64.47
Peng et al., 2009	20.50	31.34	38.33	44.27	58.91
Pinault et al., 2016	12.44	20.67	24.20	28.04	33.07
Shi et al., 2016	18.84	25.00	29.23	31.00	35.25
Urman et al., 2014	20.00	48.00	57.78	61.92	67.52
Wang et al., 2017	17.63	21.85	25.00	29.05	33.33
Weichenthal et al., 2016b	16.13	22.44	23.83	26.39	27.06
Weichenthal et al., 2016c	14.33	23.83	25.06	26.39	27.06
Yap et al., 2013	41.50	55.00	58.75	61.00	71.00
Zanobetti et al., 2009	28.00	38.50	43.50	63.00	72.50
Zanobetti and Schwartz, 2009	21.59	30.34	37.53	44.60	62.20
Zanobetti et al., 2014	22.67	31.11	37.91	41.25	55.78

For each study in Figure 3-9 and Figure B-9, an assessment of the percent of 3-year average pseudo-DVs available for each study area and study period is presented in Figure B-5. For example, in a study with a study area of 5 counties that was completed for study a period from 2000-2004, 3 possible 3-year average pseudo-DVs exist per county (*i.e.* 2000-2002, 2001-2003, and 2002-2004), with a total of 15 possible pseudo-DVs. However, if one county only has one valid 3-year average pseudo-DV, then the study would have 13 out of a possible 15 pseudo-DVs. Figure B-5 displays a percent of 3-year average pseudo-DV data points available in each study.



**Figure B-5. Studies used in box-and-whisker plots (Figure 3-9 and Figure B-9) and the percent of pseudo-DVs available by study.**

There are important uncertainties to consider when assigning air quality to a study area. Pseudo-design values are based on individual monitors in each county included in study areas. Counties may or may not reflect actual non-attainment areas, which can include multiple counties or parts of counties. For studies conducted in Canada, this potential mismatch is of greater concern. Pseudo-design values are not actual design values. Our analyses considered all available monitoring data, even from monitors not meeting strict completeness requirements for determining non-attainment. While we conclude this is a reasonable approach, as it allows the consideration of ambient PM<sub>2.5</sub> concentrations in a greater proportion of study areas than if the analysis were restricted only to valid design values, it remains an uncertainty in our analyses. Additional uncertainties are discussed above in section 3.2.3.2.2.

## **B.5 PERCENT OF STUDY AREA POPULATION CAPTURED IN DESIGN VALUE PLOTS**

Figure 3-9 and Figure B-9 include annual (Figure 3-9) and 24-hour (Figure B-9) pseudo-design values corresponding to 5,25,50,75, and 95<sup>th</sup> percentiles of study populations or health events for U.S. and Canadian studies of long-term or short-term exposures, and for studies of mortality or morbidity outcomes. Further analyses were completed to determine the proportion of the study area populations captured in these analyses. Within each study, the cumulative population of counties with a valid 3-year average pseudo-DV was determined as a proportion of the total population in counties included in the study. For example, if valid air quality data was available in each county of the study area, then 100% of the study area population would be captured within the design value box plots. For most studies included in Figure 3-9 and Figure B-9, valid pseudo-DVs are available for counties accounting for at least about 70% of the total study area population (Table B-7 and Table B-8).

When design values are calculated using only the regulatory monitors, as discussed in section B.4.3 above, the total study area population captured in the calculation declines. For example, for Di et al. (2017b) and Di et al. (2017a), when calculation of design values was completed using air quality data only from regulatory monitors, the analyses captured 67.35% of population for annual design values (compared to 70.38% of population for annual pseudo-design values when data from all monitors was used). Similarly, analyses captured 67.43% of population for 24-hour design values from regulatory monitors alone, compared to 70.47% of population for pseudo-design values when data from all monitors was used. For Shi et al. (2016), calculation of annual and 24-hour design values from regulatory monitors captured 71.37% of population, compared to 77.22% of population when data from all the monitors was used.

**Table B-7. Percent population included in annual pseudo-DV boxplots (Figure 3-9).**

<b>Citation</b>	<b>Population Used</b>	<b>Study Area Counties</b>	<b>Total Population</b>	<b>Population with DV</b>	<b>Population with DV (%)</b>
Baxter et al., 2017	US 2015	113	113,053,365	100,129,153	88.57
Bell et al., 2008	US 2015 (65+yrs)	202	23,206,934	21,974,015	94.69
Bell et al., 2014	US 2015 (65+yrs)	4	490,357	490,357	100.00
Bell et al., 2015	US 2015 (65+yrs)	202	23,206,934	22,529,386	97.08
Bravo et al., 2017	US 2015 (65+yrs)	807	31,056,109	21,909,224	70.55
Dai et al., 2014	US 2015	95	95,890,830	91,262,160	95.17
Di et al., 2017b	US 2015 (65+yrs)	3220	48,387,814	34,057,020	70.38
Di et al., 2017a	US 2015 (65+yrs)	3220	48,387,814	34,057,020	70.38
Dominici et al., 2006	US 2015 (65+yrs)	202	23,206,934	20,272,093	87.35
Franklin et al., 2008	Franklin 2008	25	1,313,983	1,313,983	100.00
Kioumourtzoglou et al., 2016	Kiomourtzoglou 2016	222	11,391,912	11,050,835	97.01
Kloog et al., 2012	US 2015 (65+yrs)	67	2,361,375	1,588,345	67.26
Kloog et al., 2014	US 2015 (65+yrs)	366	9,099,500	6,471,367	71.12
Lee et al., 2015a	US 2015	305	25,153,808	14,033,573	55.79
Lepeule et al., 2012	Lepeule 2012	11	14,562	12,932	88.81
Malig et al., 2013	US 2015	35	36,607,640	36,533,148	99.80
McConnell et al., 2010	US 2015 (18 and under)	7	5,008,800	5,008,587	100.00
Ostro et al., 2016	Ostro Asthma 2016	8	43,904	43,904	100.00
Peng et al., 2009	US 2015 (65+yrs)	119	13,944,304	13,732,109	98.48
Pinault et al., 2016	Canada 2016	5162	35,151,728	18,242,308	51.90
Shi et al., 2016	US 2015 (65+yrs)	67	2,361,375	1,823,456	77.22
Urman et al., 2014	Urman 2014 5-7yrs	5	1,811	1,811	100.00
Wang et al., 2017	US 2015 (65+yrs)	616	9,779,426	6,336,200	64.79
Weichenthal et al., 2016b	Weichenthal MI 2016	16	30,101	30,101	100.00
Weichenthal et al., 2016c	Canada 2016	15	4,673,938	4,673,938	100.00
Yap et al., 2013	Yap 2013 Asthma 1-9yrs	12	146,224	146,224	100.00
Zanobetti et al., 2009	US 2015 (65+yrs)	35	6,630,577	5,974,387	90.10
Zanobetti and Schwartz, 2009	US 2015	156	126,026,116	114,529,073	90.88
Zanobetti et al., 2014	Zanobetti 2014	126	6,828,055	6,703,284	98.17

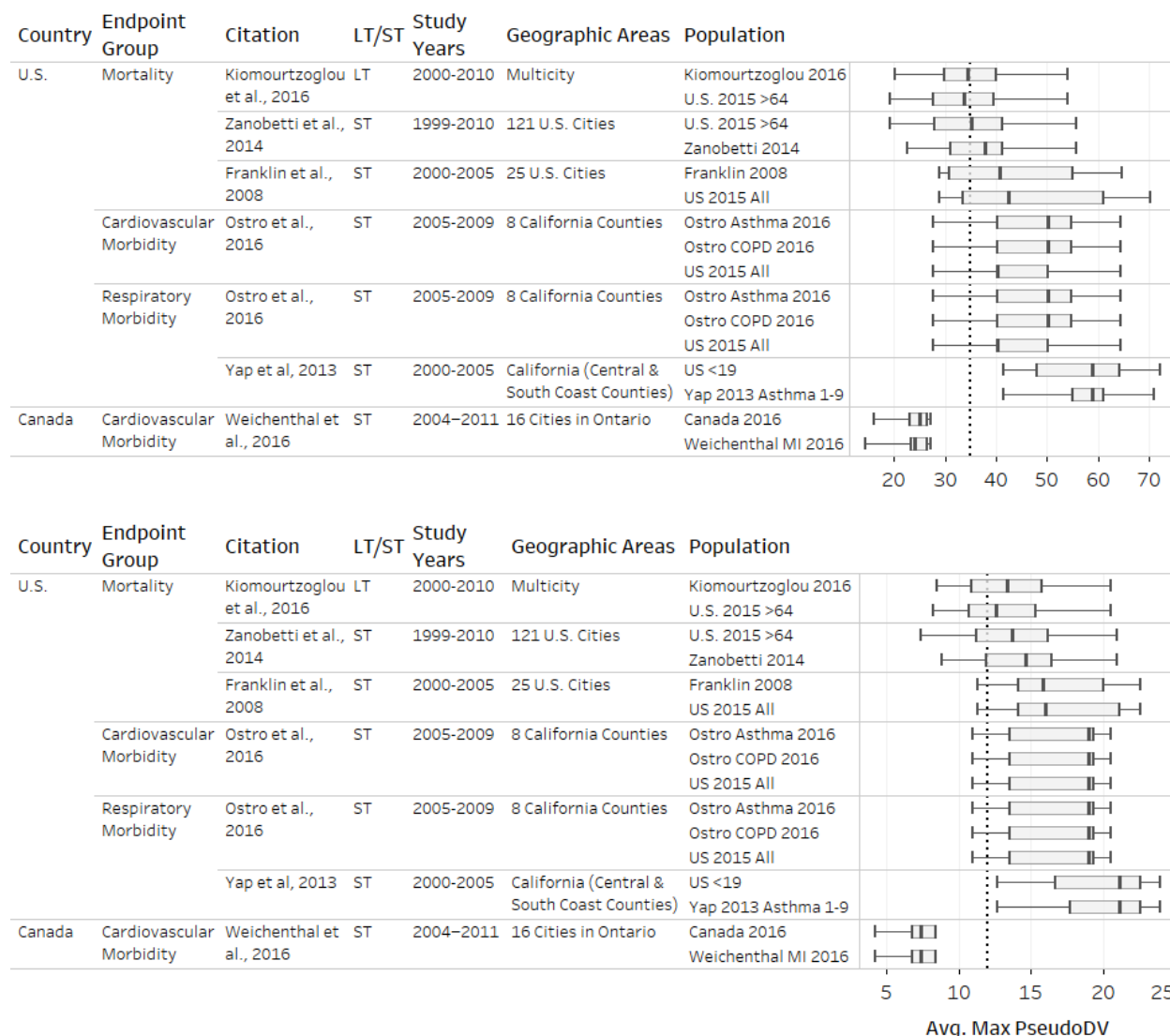
**Table B-8. Percent population included in 24-hr pseudo-DV boxplots (Figure B-9).**

Citation	Population Used	Study Area Counties	Total Population	Population with DV	Population with DV (%)
Baxter et al., 2017	US 2015	113	113,053,365	97,125,414	85.91
Bell et al., 2008	US 2015 (65+yrs)	202	23,206,934	21,903,002	94.38
Bell et al., 2014	US 2015 (65+yrs)	4	490,357	490,357	100.00
Bell et al., 2015	US 2015 (65+yrs)	202	23,206,934	22,564,564	97.23
Bravo et al., 2017	US 2015 (65+yrs)	807	31,056,109	21,083,502	67.89
Dai et al., 2014	US 2015	95	95,890,830	91,262,160	95.17
Di et al., 2017b	US 2015 (65+yrs)	3220	48,387,814	34,097,655	70.47
Di et al., 2017a	US 2015 (65+yrs)	3220	48,387,814	34,097,655	70.47
Dominici et al., 2006	US 2015 (65+yrs)	202	23,206,934	20,097,018	86.60
Franklin et al., 2008	Franklin 2008	25	1,313,983	1,313,983	100.00
Kioumourtzoglou et al., 2016	Kioumourtzoglou 2016	222	11,391,912	11,050,835	97.01
Kloog et al., 2012	US 2015 (65+yrs)	67	2,361,375	1,546,500	65.49
Kloog et al., 2014	US 2015 (65+yrs)	366	9,099,500	6,429,318	70.66
Lee et al., 2015a	US 2015	305	25,153,808	12,127,123	48.21
Lepeule et al., 2012	Lepeule 2012	11	14,562	12,932	88.81
Malig et al., 2013	US 2015	35	36,607,640	35,908,846	98.09
McConnell et al., 2010	US 2015 (18 and under)	7	5,008,800	5,008,587	100.00
Ostro et al., 2016	Ostro Asthma 2016	8	43,904	43,904	100.00
Peng et al., 2009	US 2015 (65+yrs)	119	13,944,304	13,596,370	97.50
Pinault et al., 2016	Canada 2016	5162	35,151,728	18,242,308	51.90
Shi et al., 2016	US 2015 (65+yrs)	67	2,361,375	1,823,456	77.22
Urman et al., 2014	Urman 2014 5-7yrs	5	1,811	1,811	100.00
Wang et al., 2017	US 2015 (65+yrs)	616	9,779,426	6,306,215	64.48
Weichenthal et al., 2016b	Weichenthal MI 2016	16	30,101	30,101	100.00
Weichenthal et al., 2016c	Canada 2016	15	4,673,938	4,673,938	100.00
Yap et al., 2013	Yap 2013 Asthma 1-9yrs	12	146,224	146,224	100.00
Zanobetti et al., 2009	US 2015 (65+yrs)	35	6,630,577	5,974,387	90.10
Zanobetti and Schwartz, 2009	US 2015	156	126,026,116	114,529,073	90.88
Zanobetti et al., 2014	Zanobetti 2014	126	6,828,055	6,703,284	98.17



## **B.6 SENSITIVITY ANALYSIS: BOX PLOTS USING COUNTS OF HEALTH EVENTS VERSUS STUDY AREA POPULATION**

As discussed in Section 3.2.3.2.2, Figure 3-9 and Figure B-9 present box-and-whisker plots reflecting the PM<sub>2.5</sub> 3-year average maximum pseudo-design values that correspond to various percentiles of the study area population or study area health events. When area-specific health events are available, Figure 3-9 and Figure B-9 present percentiles of air quality and study area health events. There is uncertainty regarding the extent to which the populations in counties included in key studies reflect the true distribution of cases in those studies. Many studies used registry data, or similar data sources that may be expected to capture the majority of cases within a study location; however, these studies often didn't report the exact number of cases per area. When the number of cases were not available, we instead used the underlying county-level population obtained using 2015 U.S. census data. While this approach contributes uncertainty to our analyses of pseudo-design values, for the limited number of studies with information on the number of cases per county, the distributions of pseudo-design values relative to the number of cases were similar to the distributions relative to the county population (particularly for annual pseudo-design values). Figure B-6 provides a comparison of studies where health event data are available, to assess the distribution of pseudo-design values when study area population is used versus study area health events.

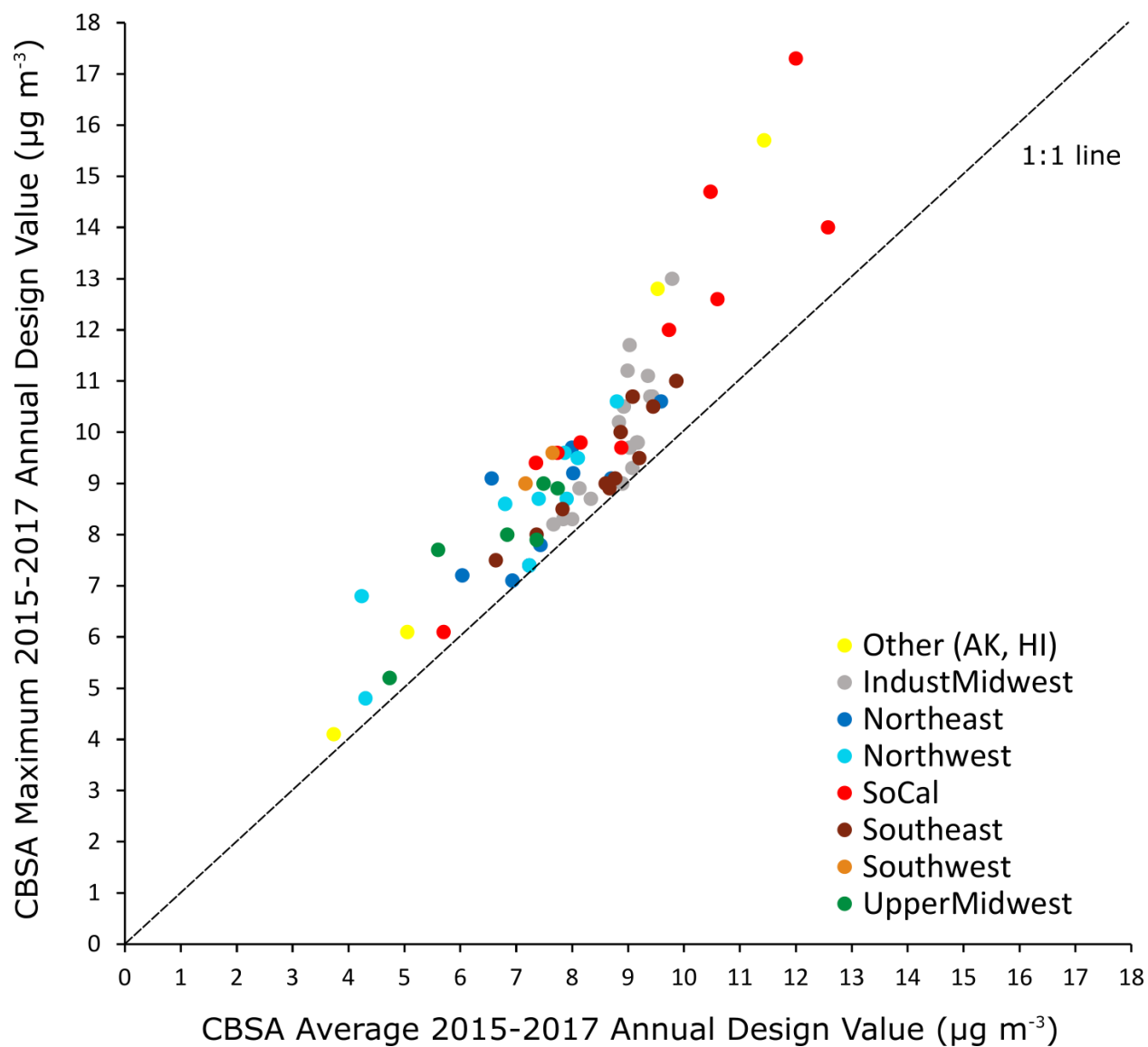


**Figure B-6. PM<sub>2.5</sub> pseudo-design values corresponding to various percentiles of study area populations and health events for studies of 24-hour PM<sub>2.5</sub> exposures and long-term studies (top panel) and annual PM<sub>2.5</sub> exposures and long-term studies (bottom panel).**

## B.7 COMPARISONS BETWEEN ANNUAL AND DAILY DESIGN VALUES

As discussed above in section 3.2.3.2, for an area to meet the NAAQS, all valid design values in that area, including the highest annual and 24-hour values, must be at or below the levels of the standards. Because monitors are often required in locations with high PM<sub>2.5</sub> concentrations (section 2.2.3), areas meeting an annual PM<sub>2.5</sub> standard with a particular level would be expected to have long-term average PM<sub>2.5</sub> concentrations (i.e., averaged across space and over time in the area) somewhat below that standard level. Figure B-7 and Table B-9

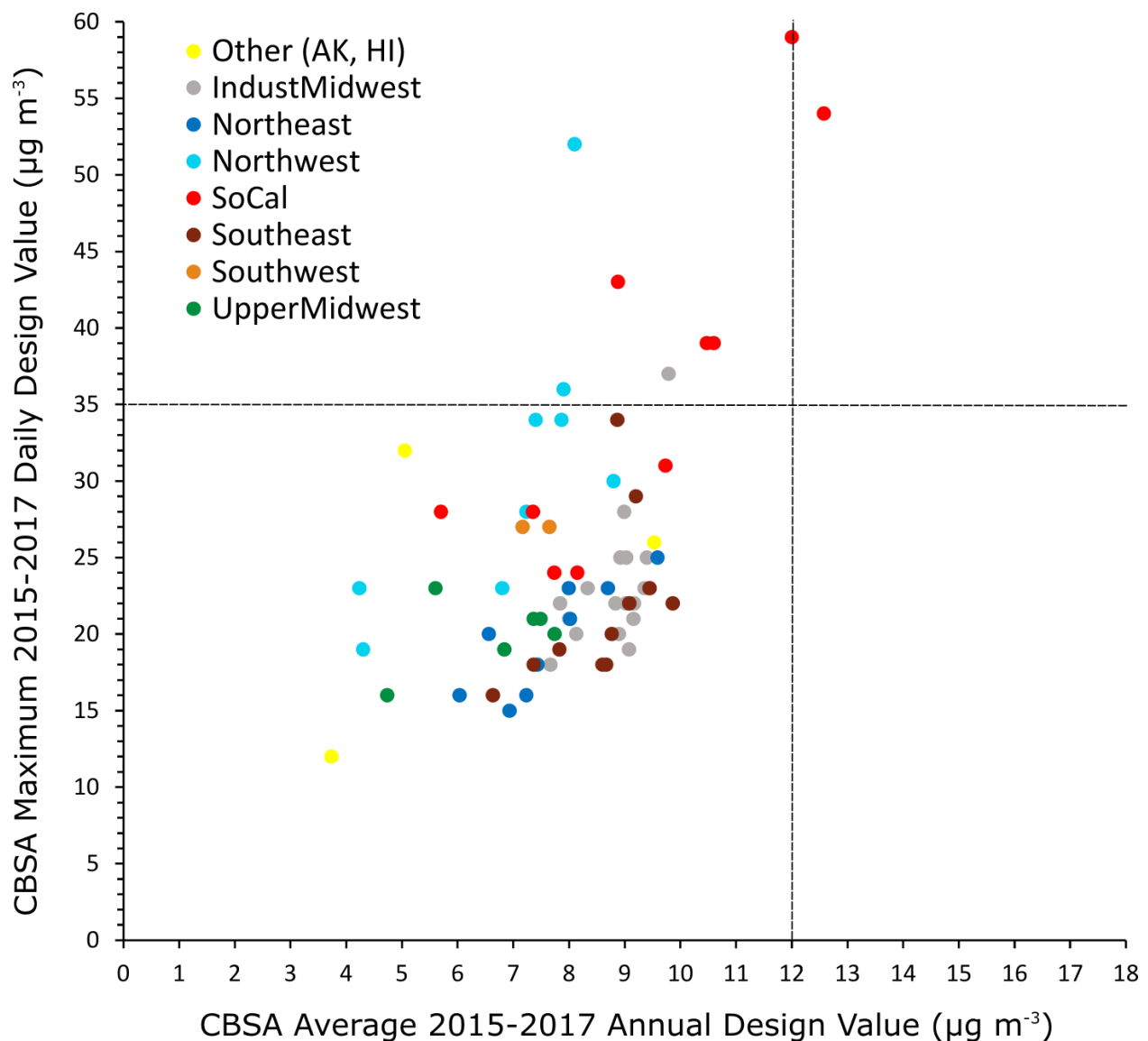
indicate that, based on recent air quality in U.S. CBSAs, maximum annual PM<sub>2.5</sub> design values are often 10% to 20% higher than annual average concentrations (i.e., averaged across multiple monitors in the same CBSA). The difference between the maximum annual design value and average concentration in an area can be smaller or larger than this range, likely depending on factors such as the number of monitors, monitor siting characteristics, and the distribution of ambient PM<sub>2.5</sub> concentrations. Given that higher PM<sub>2.5</sub> concentrations have been reported at some near-road monitoring sites, relative to the surrounding area (section 2.3.2.2.2), recent requirements for PM<sub>2.5</sub> monitoring at near-road locations in large urban areas (section 2.2.3.3) may increase the ratios of maximum annual design values to averaged concentrations in some areas. Such ratios may also depend on how the average concentrations are calculated (i.e., averaged across monitors versus across modeled grid cells). Compared to annual design values, Figure B-8 indicates a more variable relationship between maximum 24-hour PM<sub>2.5</sub> design values and annual average concentrations.



**Figure B-7. Comparison of CBSA average annual design values and CBSA maximum annual design values for 2015-2017.** (Note: Includes all CBSAs with at least 3 valid annual DVs.)

**Table B-9. National Averages of ratios of maximum annual design values to averaged concentrations.**

Year of monitoring data	Number of monitors per CBSA	Number of CBSAs	Ratio of max Annual DV to CBSA average	Ratio of max 24-hr DV to CBSA average
2009-2011	3 or more	67	1.12	1.13
	4 or more	33	1.14	1.16
	5 or more	18	1.17	1.19
2012-2014	3 or more	60	1.15	1.15
	4 or more	38	1.17	1.18
	5 or more	23	1.19	1.21
2015-2017	3 or more	65	1.16	1.19
	4 or more	38	1.19	1.21
	5 or more	30	1.20	1.24



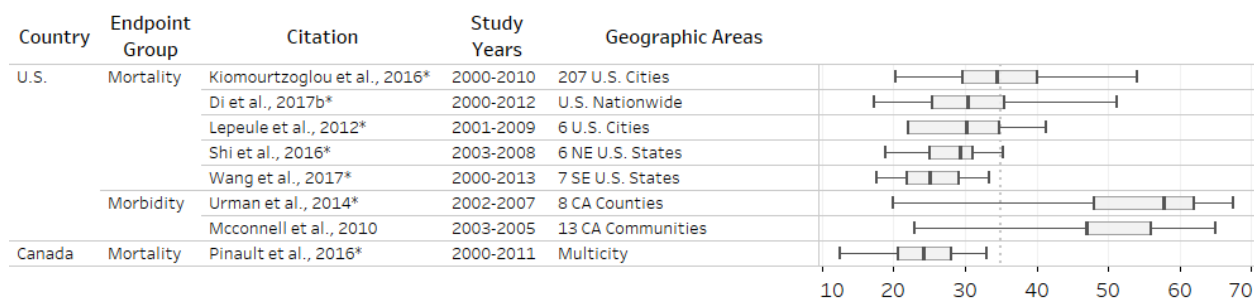
**Figure B-8. Comparison of CBSA average annual design values and CBSA maximum daily design values for 2015-2017.** (Note: Dashed lines indicate the level of the current 24-hour PM<sub>2.5</sub> standard (35  $\mu\text{g m}^{-3}$ ) and the current annual PM standard (12  $\mu\text{g m}^{-3}$ ). Includes all CBSAs with at least 3 valid daily and 3 valid annual DVs.)<sup>13</sup>.

<sup>13</sup> The CBSA maximum 2015-2017 daily design value (y-axis) was cut off at 60  $\mu\text{g m}^{-3}$ , to improve the visualization of data, but this removed the Fairbanks CBSA from the plot, which had a daily design value of 85  $\mu\text{g m}^{-3}$  and an annual design value of 15.7  $\mu\text{g m}^{-3}$ .

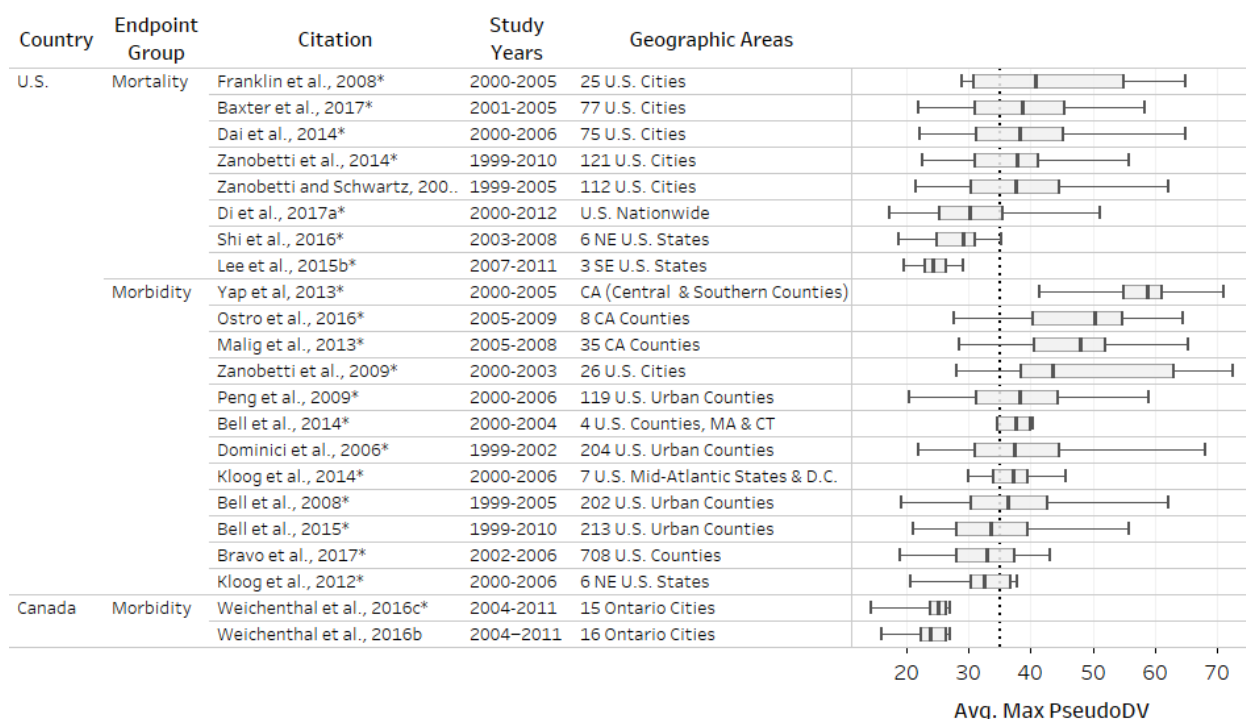
## **B.8 24-HOUR PSEUDO-DESIGN VALUES AND DISTRIBUTIONS ACROSS STUDY AREAS**

As described in section 3.2.3.2.2 of the PA, and section B.4 of this appendix, for locations evaluated in key epidemiologic studies we identify annual and 24-hour PM<sub>2.5</sub> pseudo-design values and the number of people (or health events). Figure 3-9 in the PA presents box-and-whisker plots summarizing those data for annual pseudo-design values. Figure B-9 (below) presents box-and-whisker plots summarizing those data for 24-hour pseudo-design values.

## Long-term exposure studies



## Short-term exposure studies



**Figure B-9. PM<sub>2.5</sub> 24-hour pseudo-design values corresponding to various percentiles<sup>[1]</sup> of study area populations or health events for studies of long-term and short-term PM<sub>2.5</sub> exposures.<sup>[2]</sup>**

<sup>[1]</sup> Whiskers reflect PM<sub>2.5</sub> pseudo-design values corresponding to 5<sup>th</sup> and 95<sup>th</sup> percentiles of study area populations (or health events), boxes correspond to the 25<sup>th</sup> and 75<sup>th</sup> percentiles, and the vertical lines inside the boxes correspond to 50<sup>th</sup> percentiles. Asterisks next to study citations denote statistically significant effect estimates.

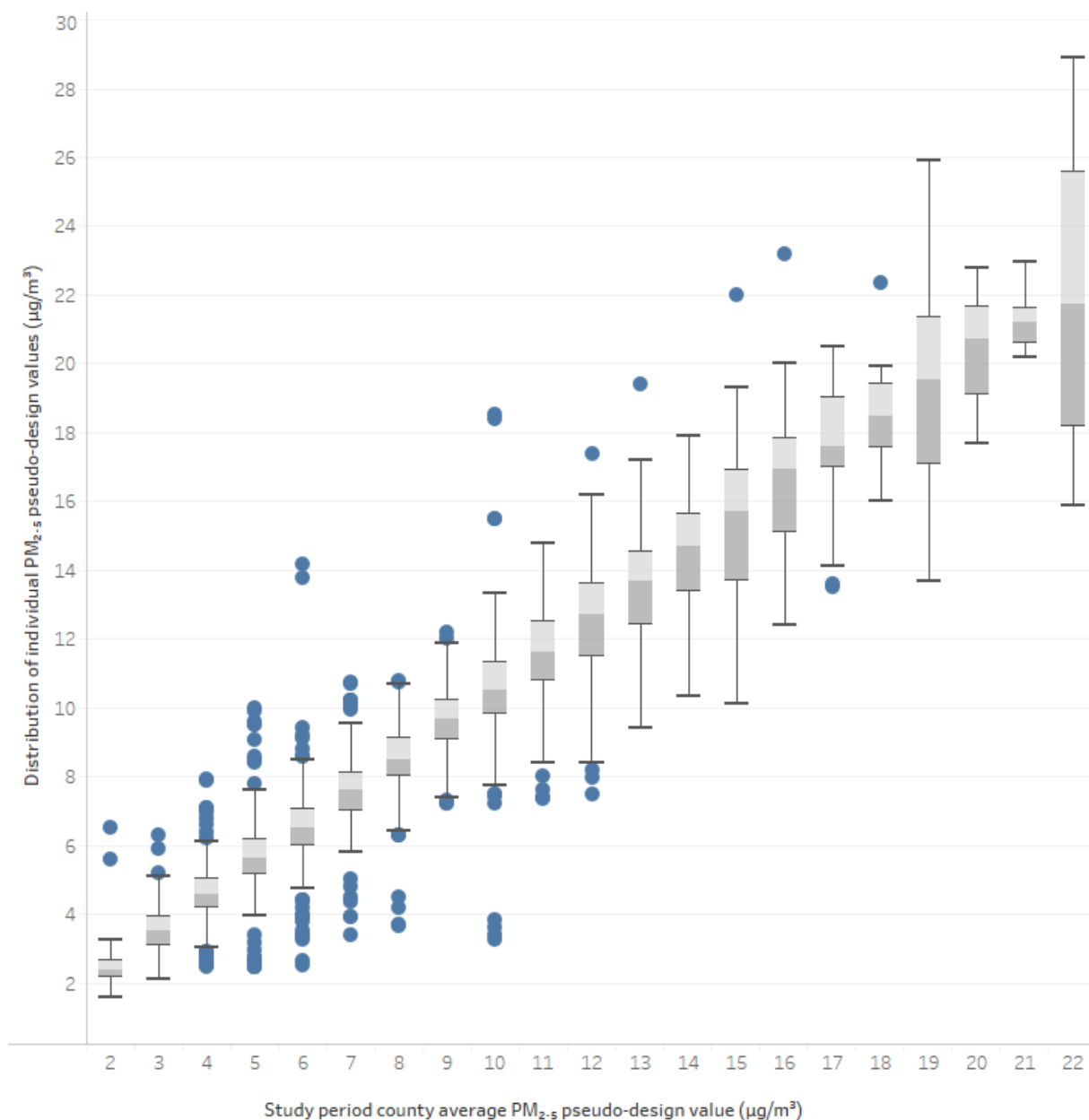
<sup>[2]</sup> For most of the studies included in Figure B-9, pseudo-design values are available for >70% of study area populations (or health events). Exceptions are Kloog et al. (2012), Lee et al. (2015b), Pinault et al. (2016), Wang et al. (2017), and Bravo et al. (2017), with pseudo-design values available for 65%, 48%, 51%, 68%, and 64% of study area populations, respectively.



## **B.9 PSEUDO-DESIGN VALUE DISTRIBUTION BY AVERAGE COUNTY PSEUDO-DESIGN VALUES PER 1 $\mu\text{G}/\text{M}^3$**

Figure 3-9 and Figure B-9 exhibit distributions of pseudo-DVs corresponding to study areas within each study and based on averaging pseudo-DVs. That is, for each study location, maximum 3-year pseudo-design values are averaged over study periods. Depending on the years of air quality evaluated by the study, for some locations those averages could reflect air quality that violated the current standards during part of the study period and met the current standards during part of the study period. We have examined this issue in greater detail for the studies by Di et al. (2017b) and Shi et al. (2016).

Figure B-10 and 0 present the relationship between annual pseudo-DVs averaged over the study period and the individual 3-year pseudo-DVs that contribute to those study-period averages for Di et al. (2017b). Of the 6,315 3-year pseudo-DVs available for this study, 3,915 (62%) are less than or equal to  $12.04 \mu\text{g}/\text{m}^3$  (i.e., lower than the current annual standard). Of the counties that have study-period average pseudo-DV's  $\leq 12.04 \mu\text{g}/\text{m}^3$ , 89.3% of individual 3-year pseudo-DVs are  $\leq 12.04 \mu\text{g}/\text{m}^3$  (i.e., 3,410 of 3,820 3-year pseudo-DVs).

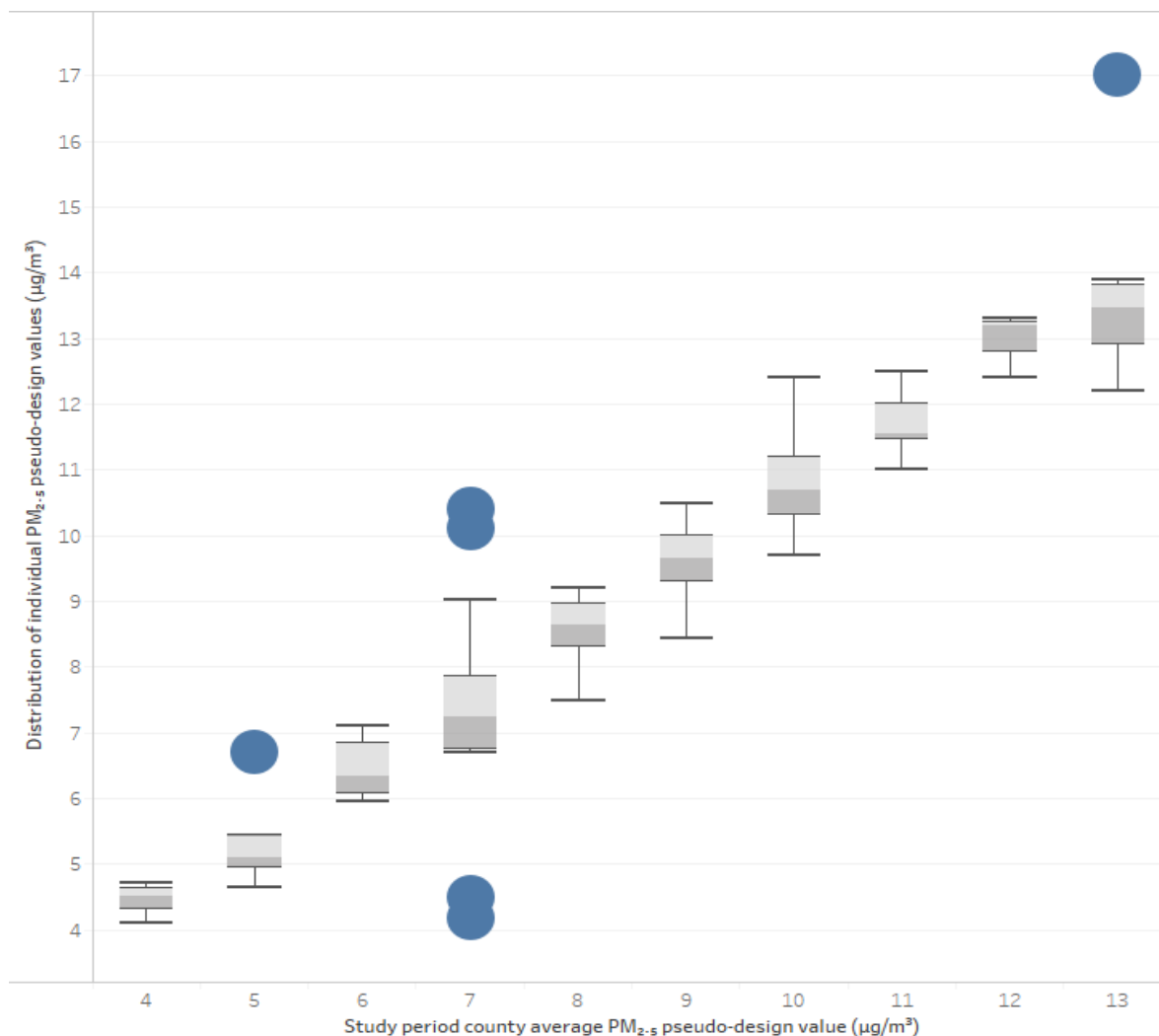


**Figure B-10. County average pseudo-DV by 1 µg/m<sup>3</sup> and distribution of individual county pseudo-DVs within each 1 µg/m<sup>3</sup> interval for study counties in Di et al., 2017b. Note: X-axis values of 11 correspond to county average pseudo-DVs from 11.0 to 12.0 µg/m<sup>3</sup>. Thus, x-axis values of 11 or below correspond to pseudo-DVs at or below the level of the current annual standard.**

**Table B-10. County average pseudo-DV by 1  $\mu\text{g}/\text{m}^3$  and distribution of county pseudo-DVs within each 1  $\mu\text{g}/\text{m}^3$  interval for study counties in Di et al., 2017b**

County average pseudo-DV $\text{PM}_{2.5}$ concentration ( $\mu\text{g}/\text{m}^3$ ) interval	Count (percent) of pseudo-DV's $\leq 12.04 \mu\text{g}/\text{m}^3$	Count (percent) of pseudo-DV's $> 12.04 \mu\text{g}/\text{m}^3$
$2.04 < \text{PM}_{2.5} \leq 3.04$	93 (100.00)	0 (0.00)
$3.04 < \text{PM}_{2.5} \leq 4.04$	117 (100.00)	0 (0.00)
$4.04 < \text{PM}_{2.5} \leq 5.04$	198 (100.00)	0 (0.00)
$5.04 < \text{PM}_{2.5} \leq 6.04$	235 (100.00)	0 (0.00)
$6.04 < \text{PM}_{2.5} \leq 7.04$	293 (99.35)	2 (0.68)
$7.04 < \text{PM}_{2.5} \leq 8.04$	283 (100.00)	0 (0.00)
$8.04 < \text{PM}_{2.5} \leq 9.04$	501 (100.00)	0 (0.00)
$9.04 < \text{PM}_{2.5} \leq 10.04$	533 (99.84)	1 (0.19)
$10.04 < \text{PM}_{2.5} \leq 11.04$	619 (92.23)	61 (8.97)
$11.04 < \text{PM}_{2.5} \leq 12.04$	538 (66.03)	346 (39.14)
$12.04 < \text{PM}_{2.5} \leq 13.04$	332 (30.46)	635 (65.67)
$13.04 < \text{PM}_{2.5} \leq 14.04$	128 (13.19)	525 (80.40)
$14.04 < \text{PM}_{2.5} \leq 15.04$	38 (5.14)	433 (91.93)
$15.04 < \text{PM}_{2.5} \leq 16.04$	7 (1.27)	228 (97.02)
$16.04 < \text{PM}_{2.5} \leq 17.04$	0 (0.47)	70 (100.00)
$17.04 < \text{PM}_{2.5} \leq 18.04$	0 (0.00)	21 (100.00)
$18.04 < \text{PM}_{2.5} \leq 19.04$	0 (0.00)	11 (100.00)
$19.04 < \text{PM}_{2.5} \leq 20.04$	0 (0.00)	33 (100.00)
$20.04 < \text{PM}_{2.5} \leq 21.04$	0 (0.00)	12 (100.00)
$21.04 < \text{PM}_{2.5} \leq 22.04$	0 (0.00)	11 (100.00)
$22.04 < \text{PM}_{2.5} \leq 23.04$	0 (0.00)	11 (100.00)
Total	3,915 (62.0)	2,400 (38.0)

Figure B-11 and Table B-11 present the relationship between annual pseudo-DVs averaged over the study period and the individual 3-year pseudo-DVs that contribute to those study-period averages for Shi et al. (2016). Of the 116 3-year pseudo-DVs available for this study, 102 (88%) are less than or equal to  $12.04 \mu\text{g}/\text{m}^3$ . Of the counties that have study-period average pseudo-DV's  $\leq 12.04 \mu\text{g}/\text{m}^3$  98.1% of individual 3-year pseudo-DVs are  $\leq 12.04 \mu\text{g}/\text{m}^3$  (i.e., 102 of 104 3-year pseudo-DVs).



**Figure B-11. County average pseudo-DV by 1 µg/m<sup>3</sup> and distribution of individual county pseudo-DVs within each 1 µg/m<sup>3</sup> interval for study counties in Shi et al., 2016. Note: X-axis values of 11 correspond to county average pseudo-DVs from 11.0 to 12.0 µg/m<sup>3</sup>. Thus, x-axis values of 11 or below correspond to pseudo-DVs at or below the level of the current annual standard.**

**Table B-11. County average pseudo-DVs by 1  $\mu\text{g}/\text{m}^3$  and distribution of county pseudo-DVs within each 1  $\mu\text{g}/\text{m}^3$  interval for study counties in Shi et al., 2016.**

County average pseudo-DV $\text{PM}_{2.5}$ concentration ( $\mu\text{g}/\text{m}^3$ ) interval	Count (percent) of pseudo-DV's $\leq 12.04 \mu\text{g}/\text{m}^3$	Count (percent) of pseudo-DV's $> 12.04 \mu\text{g}/\text{m}^3$
$4.04 < \text{PM}_{2.5} \leq 5.04$	8 (100.00)	0 (0.00)
$5.04 < \text{PM}_{2.5} \leq 6.04$	5 (100.00)	0 (0.00)
$6.04 < \text{PM}_{2.5} \leq 7.04$	7 (100.00)	0 (0.00)
$7.04 < \text{PM}_{2.5} \leq 8.04$	16 (100.00)	0 (0.00)
$8.04 < \text{PM}_{2.5} \leq 9.04$	12 (100.00)	0 (0.00)
$9.04 < \text{PM}_{2.5} \leq 10.04$	26 (100.00)	0 (0.00)
$10.04 < \text{PM}_{2.5} \leq 11.04$	21 (95.45)	1 (0.00)
$11.04 < \text{PM}_{2.5} \leq 12.04$	7 (87.50)	1 (0.00)
$12.04 < \text{PM}_{2.5} \leq 13.04$	0 (0.00)	4 (0.00)
$13.04 < \text{PM}_{2.5} \leq 14.04$	0 (0.00)	8 (0.00)
Total	102 (88.0)	14 (12.0)

## B.10 DETAILS OF KEY EPIDEMIOLOGIC STUDIES, INCLUDING STUDY DESIGN, EXPOSURE METRIC, AND STATISTICAL ANALYSIS

Table B-12 below summarizes additional details related to the designs of the U.S. and Canadian epidemiologic studies included in Figure 3-7, Figure 3-8, Figure 3-9, and Figure B-9 and the risk assessment (Table 3-4).

**Table B-12. Study characteristics from key studies.**

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Baxter et al., 2017	ST	All-cause mortality	77 US Cities	Time Series study (NCHS data)	Average daily monitored PM <sub>2.5</sub> concentration in each city. 2-day moving average (lag 0-1 days) of PM <sub>2.5</sub> conc. Included in the model.	Poisson regression model and meta-regression  In stage 1, ran single city Poisson time-series models; adjusted for temperature and dew point temperature, including variables for previous day temperature, temporal trends, and trends by age. In stage 2, meta-regression with cluster analysis (5 clusters) based on characteristics of residential infiltration.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Bell et al., 2008	ST	CVD HA Age 65+	202 US Counties with populations $\geq 200,000$	Time Series study (MEDICARE enrollees)	Daily monitored PM <sub>2.5</sub> concentrations. Used lag0 PM2.5 in the model.	2-stage Bayesian hierarchical model  In stage 1, adjusted for temperature and dew point temperature, including variables for previous day's conditions, day-of-the-week, temporal trends, and differential temporal trends by age. In stage 2, county-specific estimates were combined, accounting for their statistical uncertainty.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Bell et al., 2014	ST	CVD, Asthma, and COPD HA Age 65+	4 Counties in MA and CT	Time-series study (MEDICARE enrollees)	<p>PM<sub>2.5</sub> Teflon filter samples obtained from CT and MA DEP and used to measure PM<sub>2.5</sub> total mass.</p> <p>Fairfield County (2 monitors): Estimated exposures using population-weighted averaging of values and assigned exposure to the nearest monitor.</p> <p>Exposures were averaged, weighted by each tracts' 2000 census population. For other counties, values from the single monitor within the county were used. Explored various lags and presented lag0 PM<sub>2.5</sub> model.</p>	<p>Log-linear Poisson regression analysis</p> <p>Adjusted for temperature and dew point temperature, including previous day's temperature and dew point temperature, day-of-the-week temporal trends, and region.</p>



Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Bell et al., 2015	ST	HF HA 65+	213 U.S. Counties	Time-series study (MEDICARE enrollees)	For each county and day, PM <sub>2.5</sub> measurements for monitors within a county were averaged. Explored various lags and presented lag0 PM2.5 model.	2-stage Bayesian hierarchical model  The stage 1 model included county-specific model adjusted for weather (temperature, dew point, previous days' temperature and dew point), day-of-the-week, and temporal trends. In stage 2 county-specific effect estimates were pulled together to present overall association.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Bravo et al., 2017	ST	CVD HA Age 65+	418 U.S. Counties	Time-series study (MEDICARE enrollees)	Exposure estimated from monitoring data and monitors with multiple measurements for the same day and county were averaged. Explored various lags and distributed lags of PM2.5 exposure.	<p>2-stage Bayesian hierarchical model</p> <p>The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of PM2.5 conc simultaneously in the county-specific model.</p> <p>The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Bravo et al., 2017	ST	CVD HA Age 65+	708 U.S. Counties	Time-series study (MEDICARE enrollees)	Daily PM <sub>2.5</sub> concentrations estimated at census tracts using the downscaler method. 24-hr county-level PM <sub>2.5</sub> estimates for counties with population > 50,000 were calculated from a population-weighted average of PM <sub>2.5</sub> concentrations predicted by the downscaler at census tracts within each county using 2000 U.S. Census Data. Explored various lags and distributed lags of PM <sub>2.5</sub> exposure.	2-stage Bayesian hierarchical model  The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of PM <sub>2.5</sub> conc simultaneously in the county-specific model.  The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Burnett and Goldberg, 2003	ST	All-cause mortality	8 Canadian Cities	Time-series study	Monitored measurements	Generalized additive model (GAM) analysis to generate pooled estimate of air pollution effect among the eight cities.  The model adjusted for day-of-the-week, temporal trends, and weather variables (daily average temperature, daily average relative humidity, and barometric pressure lagged 0 and 1 days).
Burnett et al., 2004	ST	All-cause mortality	12 Canadian Cities	Time-series study (data from Statistics Canada)	Daily summary pollution exposure measurements based on averaging data over all monitors within each city. Various lags and moving average assessed and presented data for lag 1 for PM2.5.	Random-effects regression model.  Adjusted for temporal trends in mortality and effects of weather using humidex index at lag 0 and lag 1 (a measure of combined effect of temperature and humidity)

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Crouse et al., 2012	LT	All-cause mortality	11 Canadian Cities	National Cohort study (Subset of Canadian census mortality follow-up study; 43%)	Mean annual concentration from ground-based monitors averaged from 1987-2001. Participants were assigned exposure based on 11 census divisions. Another set of exposure estimate was derived from satellite remote sensing for period 2001-2006. Estimates at grid-level assigned to the cohort members by linking grid to the enumeration area of residence in 1991.	2 different modelling approach. Approach 1: Cox proportional hazards model, and Approach 2: nested, spatial random-effects Cox model with spatial clusters.  Models adjusted for individual-level covariates, urban/rural indicator, and ecological covariates (% unemployed, % without high school diploma, lowest income quintile, and rural/urban indicator).
Dai et al., 2014	ST	All-cause, CVD, and Respiratory mortality	75 U.S. Cities (with available daily mortality data and PM2.5 data for at least 400 days between 2000 and 2006)	Time-series study (NCHS)	Mean daily monitored PM <sub>2.5</sub> concentrations. For cities with more than one sampling site, concentration data were averaged. Average of 2-day lag (lag 0 and 1) PM2.5 used.	Two stage: Stage 1. City-specific season-stratified time-series analysis using Poisson regression in GAM  Model adjusted for 24-hr average temperature from closest weather station to the city center at lag0 and lag1, temporal trends, and day-of-the-week. Stage 2. Multivariate random effects meta-analysis to combined 300 (i.e. 75 cities * 4 seasons) effect estimates to obtain overall association.

Di et al., 2017b	LT	All-cause mortality 65+	US Nationwide	Open Cohort (MEDICARE enrollees)	Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of PM <sub>2.5</sub> . The neural network was fit with monitored PM <sub>2.5</sub> data and daily PM <sub>2.5</sub> concentrations were predicted for nationwide grids that were 1x1 km. For each calendar year during which a person was at risk of death the annual average PM <sub>2.5</sub> concentration was assigned according to the ZIP Code of the person's residence. As part of a sensitivity analysis, monitored PM <sub>2.5</sub> data was matched with each person in the study within a distance of 50 km of the nearest monitoring site.	Two-pollutant Cox proportional hazards model with generalized estimating equation to account for correlation between ZIP codes.
Di et al., 2017b (< 12 ug/m3)					Analysis restricted to persons-years with	Accounted for individual variables, (sex, race, Medicaid eligibility, and average age at study entry), zip code-level variables (% Hispanic, % Black, median household income, median value of housing, % > 65 living below poverty level, % > 65 with less than high school education, % of owner-occupied housing units, and population density), county-level variables (county-level BMI and % ever smokers), hospital service area-level variables ( % low-density lipoprotein level measured, % glycated hemoglobin level measured, and % >1 ambulatory visits), 32 km <sup>2</sup> gridded weather and 1 km <sup>2</sup> gridded pollution variables (annual average PM <sub>2.5</sub> concentration, annual average temperature, and annual average humidity), monitor level air pollution variables (PM <sub>2.5</sub> monitored data), and a regional dummy variable.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
					PM <sub>2.5</sub> exposures lower than 12 ug/m <sup>3</sup>	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Di et al., 2017a	ST	All-cause mortality 65+	US Nationwide	Case-crossover study (MEDICARE enrollees)	<p>Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of PM<sub>2.5</sub>. The neural network was fit with monitored PM<sub>2.5</sub> data and daily PM<sub>2.5</sub> concentrations were predicted for nationwide grids that were 1x1 km.</p> <p>For each case day (date of death) and its control days, the 24-hour PM<sub>2.5</sub> concentrations were assigned based on zip code of residence of the individual. As part of a sensitivity analysis, monitored PM<sub>2.5</sub> data was matched with each person in the study within a distance of 50 km of the nearest monitoring site.</p>	<p>Conditional logistic regression.</p> <p>“Case Day” defined as death. For the same person, compared daily air pollution exposure on the case day vs. daily air pollution exposure on “control days.” Control days were chosen (1) on the same day of the week as the case day to control for potential confounding effect by day of week; (2) before and after the case day to control for time trend; and (3) only in the same month as the case day to control for seasonal and subseasonal patterns.</p> <p>Individual-level covariates and zip code-level covariates that did not vary day to day (e.g., age, sex, race/ethnicity, SES, smoking, and other behavioral risk factors) were not considered to be confounders as they remain constant when comparing case days vs control days.</p> <p>The regression model adjusted for air and dew point temperature.</p>



Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Dominici et al., 2006	ST	HF and COPD HA 65+	204 Urban U.S. counties	Time-series study (MEDICARE enrollees)	Monitored PM <sub>2.5</sub> concentrations. Of the 204 counties, 90% had daily PM <sub>2.5</sub> data across the study period and the remaining counties had PM <sub>2.5</sub> data collected once every 3 days for at least 1 full year. Various lags and distributed lags assessed and presented.	<p>2-stage Bayesian hierarchical models to estimate county-specific, region-specific, and national-average associations.</p> <p>Stage 1 model included single lag and distributed lag over-dispersed Poisson regression models to estimate county-specific risk. Models adjusted for temperature and dew point on the same day and the 3 previous days, calendar time to control for seasonality and other time-varying influences, daily numbers of individuals at risk, and day-of-the-week. In Stage 2, to produce a national average estimate, Bayesian hierarchical models were used to combine RRs across counties and accounting for within-county statistical error and for between-county variability or heterogeneity. To produce regional estimates. The Stage 2 hierarchical models described above was used for 7 regions separately.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Franklin et al., 2007	ST	All-cause, CVD, and Respiratory mortality	27 U.S. communities (with PM <sub>2.5</sub> monitoring and daily mortality data for at least 2 years of 6-year study period 1997-2000)	Case-crossover study (NCHS)	Monitored PM <sub>2.5</sub> concentrations with data for at least 2 years of a 6-year period. Within a community, any monitor that was not well correlated with others was excluded, and values were averaged to account for true variability in concentrations across the days measured in the county. Calculated and presented various lags and averages for PM <sub>2.5</sub> .	2-stage time-stratified analysis: 1) Conditional logistic regression analysis to generate community specific estimates; 2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate.  Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as “deaths” and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1, while effect modification of community-specific characteristics including geographic location, annual PM <sub>2.5</sub> concentration > 15 ug/m <sup>3</sup> and central AC prevalence was used in stage 2.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Franklin et al., 2008	ST	All-cause, CVD, and Respiratory mortality	25 U.S communities (with PM <sub>2.5</sub> monitoring and daily mortality data for at least 4 years between 2000-2005)	Case-crossover study (NCHS)	Monitored PM <sub>2.5</sub> concentrations with data for at least 4 years of a 6-year period. Within a community, any monitor that was not well correlated with others was excluded, and values were averaged to account for true variability in concentrations across the days measured in the county. Calculated and presented various lags and averages for PM <sub>2.5</sub> .	2-stage time-stratified analysis: 1) Conditional logistic regression analysis to generate community specific estimates; 2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate.  Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as “deaths” and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Gharibvand et al., 2016	LT	Lung cancer incidence	US Nationwide	Cohort study (AHSMOG-2 study)	Using monitored PM <sub>2.5</sub> data from 2000-2001, inverse distance weighted interpolations methods, monthly pollution surfaces for PM <sub>2.5</sub> were created. Monthly exposure averages were based on daily PM <sub>2.5</sub> measurements. Participants were assigned monthly exposure based on their baseline residential address.	Cox proportional hazards model  Covariates included sex, race, smoking status, years since participant quit smoking, average number of cigarettes per day during all smoking years, and education level. Additional covariates included calendar time, alcohol consumption, family income, BMI, physical activity, and marital status. 3 variables identified a priori as either as confounders or effect modifiers: hours/day spent outdoors, years of pre-study residence length at enrollment address, and moving distance from enrollment address during follow-up.
Hart et al., 2015 (monitored)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	Calculated monthly average PM <sub>2.5</sub> from the nearest monitoring location for all addresses. Nearest monitor exposures were validated against personal exposures to PM <sub>2.5</sub> of ambient origin.	Cox proportional hazards model.  Information on potential confounders was available every two years (4 years for diet information) and each woman was assigned updated covariate values for each questionnaire cycle. Confounders examined include age, race, region,

Hart et al., 2015 (modeled)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	<p>Spatio-temporal models of PM<sub>2.5</sub> were developed to estimate monthly PM<sub>2.5</sub> exposures at each geocoded questionnaire mailing address. The model was developed using monitored data and included meteorological and GIS-derived covariates, such as urban land use within 1 km, elevation, tract- and county-level population density, distance to the nearest road for road classes A1-A3 and point-source emission density within 7.5 km.</p> <p>Modeled exposures were validated against personal exposures to PM<sub>2.5</sub> of ambient origin.</p> <p>Previous 12-month moving average of exposure either from nearest monitor or spatio-temporal models were assigned to study participants.</p>	<p>season, physical activity, BMI, hypercholesterolemia, family history of MI, smoking history, Current smoking status, diet, SES (education level, occupation of both of the nurses' parents when she was 16, marital status, and husband's education if applicable). Also adjusted for area-level SES (census tract level median income and house value), and long-term temporal trends.</p> <p>Risk set regression calibration for time-varying exposures was used to correct for bias due to exposure measurement error in the hazard ratios of all-cause mortality using the personal exposure validation data.</p>
Ito et al., 2013 <sup>14</sup>	ST	All-cause mortality	150 U.S. cities	Time-series study	24-hr average PM <sub>2.5</sub> mass data in a given city, and when data	Poisson regression analysis

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
					<p>from multiple monitors were available in a given city, computed the average of the daily values after standardizing each site's data using the mean and standard deviation of the sites data.</p> <p>Pollutant concentration is expressed in the model as a deviation from the monthly mean to reduce the influence of the seasonal cycles of the pollutants on the overall associations and help focus on the short-term associations.</p>	<p>First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach</p> <p>Adjusted for temporal trends (annual cycles and influenza epidemics), immediate and delayed temperature, and day-of-week pattern, for entire years (2001-2006) and for warm (April-September) and cold (October-March) seasons.</p> <p>In second stage, assessed effect modification using land-use variables and average air pollution levels.</p>

<sup>14</sup> This study is not referenced individually in the ISA, but is study 3 of the National Particle Component Toxicity (NPACT) Initiative published in HEI (Lippmann et al., 2013).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Jerrett et al., 2016	LT	IHD mortality 30+	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Multiple exposure estimation approaches evaluated within the study – risk assessment uses results based on an ensemble approach that incorporates chemical transport modeling, land use data, satellite data, and data from ground-based monitors	<p>Cox proportional hazards regression</p> <p>Covariates included current and former smoking status as well as smoking duration, amount, age started, second hand cigarette smoke (hours/day exposed), exposure to PM<sub>2.5</sub> in the workplace for each of the subject's major lifetime occupation, self-reported exposure to dust/fumes at work, marital status, level of education, BMI, alcohol consumption, dietary vegetable/fruit/fiber index, dietary fat index, missing nutrition information. Ecologic characteristics included median household income, percentage of people with &lt; 125% of poverty-level income, percentage of persons &gt; 16 who are unemployed, percentage of adults with &lt; 12<sup>th</sup> grade education, and percentage of population who were Black or Hispanic.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Kioumourtoglou et al., 2016	LT	All-cause mortality 65+	207 U.S. communities	Open Cohort study (MEDICARE enrollees)	City-specific annual and 2-year PM <sub>2.5</sub> averages using data from all available monitors in each city using US EPA monitors. Calculated average annual, summer and winter temperatures for each city using National Climatic Data.	<p>2-stage approach for modelling.</p> <p>In Stage 1, Cox proportional hazards model was fit for each city stratified by age, gender, race and follow-up time in study. Control for slowly varying potential confounders (e.g., SES) and confounders that vary across subjects, city, and time. City-characteristics for: proportion of city population &gt; 65, median household income, proportion in poverty, proportion of city families in poverty, proportion of white, black, and Asian residents, proportion of residents with/without high-school degrees and a college degree, and city-specific smoking and obesity rates. Population-weighted city averages were developed based on census data at the county level. Also included average annual temperature in the model.</p> <p>In stage 2, combined the city-specific estimates using a random effects meta-analysis to generate region-specific effects. Assessed effect modification by annual temperature levels, and population and city characteristics (greenness, poverty, racial composition, etc.).</p>



Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Klemm and Mason, 2003	ST	All-cause mortality	Harvard Six-City study reanalysis	Time-series study	24-hour monitored PM <sub>2.5</sub> samples in 6 communities	Generalized additive and Generalized linear models  Model adjusted for temporal trends, day-of-the-week, weather (average daily temperature and average daily dew point temperature).

Kloog et al., 2012	ST	CVD HA Age 65+	New England Area with 6 U.S. States	Mixed study design (with time series and cohort components)	<p>Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground PM<sub>2.5</sub> measurements. Incorporated land use regressions and meteorological variables (temperature, wind speed, visibility, elevation, distance to major road, percent of open space, point emissions and area emissions). Model used to predict daily PM<sub>2.5</sub> concentrations at a 10 x 10 km spatial resolution.</p> <p>Short-term exposure: used the mean of PM<sub>2.5</sub> on the day of admission and day before admission. Long-term exposure: calculated as the mean exposure in each zip-code across the study period. Short term exposure was defined as the difference between the two-day average and the long-term average.</p>	<p>Equivalence between Poisson regression and the piecewise constant proportional hazard model to model the time to a hospital admission as a function of both long-term and short-term exposure simultaneously and enabling simultaneously examination of short term and long-term associations with hospital admissions (Hierarchical mixed Poisson regression model).</p> <p>The model adjusts for temperature, age, percent minorities, median income and percent of people with no high school education.</p>
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Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Kloog et al., 2014	ST	CVD and COPD HA Age 65+	7 U.S. Mid-Atlantic States and D.C.	Case-crossover design (MEDICARE enrollees)	Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground PM <sub>2.5</sub> measurements. Incorporated land use regression (elevation, distance to major roads, percent of open space, point emissions and area emissions) and meteorological variables (temperature, wind speed, relative humidity and visibility). Model used to predict daily PM <sub>2.5</sub> concentrations at a 10 x 10 km spatial resolution. Daily predicted PM <sub>2.5</sub> exposure estimates were matched to zip codes.	Conditional logistic regression analysis  Temperature with the same moving average as PM <sub>2.5</sub> was included in the model as a potential confounder. Study design samples only cases and compares each subject's exposure experience in a time period just before a case-defining event with the subject's exposure at other times, eliminating confounding (unmeasured or measured) that do not vary over time. Cases were matched on day of the week and defined the relevant exposure time window as the mean exposure of the day of and day before the patient's hospital admission. Effect modification: 1) assessed whether subject residence within 30 km of a monitor or farther modified the PM <sub>2.5</sub> association; 2) examined interaction between exposure and income level and gender.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Krall et al., 2013	ST	All-cause mortality	72 Urban U.S. Communities	Time-series study (NCHS)	Monitored daily community-level pollutant exposure as the arithmetic mean of daily monitor observations within the community. Used lag 1 PM <sub>2.5</sub> in model.	Log-linear Poisson Regression Model  Model adjusted for temperature and previous day's temperature, long-term and seasonal trends, age, and day-of-the-week. Also included interaction term for pollutant concentration and seasons.
Lee et al., 2015a	ST	All-cause, Cardiovascular, respiratory mortality	3 U.S. Southeast States	Case-crossover design (Dept. of Pub Health data)	AOD data and predicted data at 1 km <sup>2</sup> resolution aggregated into the zip code level and assigned to resident zip code. Mean exposure was calculated using lag0 and lag1 value.  Monitored PM <sub>2.5</sub> concentrations from the nearest EPA and IMPROVE monitors from resident zip code identified. 24-hr PM measurement for lag0 and lag1 were used.	Conditional logistic regression  Model adjusted for temperature and day of the week  Also ran stratified analysis by age, sex, race, education and primary cause of death.  Analysis also restricted for zip codes where annual average of PM <sub>2.5</sub> <12 or daily average <35 separately.  Sensitivity analysis: potential non-linear relationship between temp and mortality modelled using natural spline to the temperature term.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Lepeule et al., 2012	LT	All-cause, Cardiovascular, lung cancer mortality	HARVARD 6 cities	Prospective Cohort/Longitudinal follow-up study (HARVARD 6 cities data)	PM <sub>2.5</sub> data from monitors in the participant's city. PM <sub>2.5</sub> data 1979-1986/1988 from monitors, end of monitoring to 1998 estimated from PM10 using US EPA monitors, 1999-2009 direct PM <sub>2.5</sub> measurement from US EPA monitors. 1-yr or 1-3yr or 1-5 yr. moving PM <sub>2.5</sub> averages were assigned to participants based on city of residence.	Cox proportional hazard models, Poisson survival analysis  Stratified analysis by sex, age and time in the study (1-yr interval). Confounders included: Baseline information on smoking status, smoking pack-years, education, linear and quadratic term for BMI. Also explored effect modification of PM2.5 on mortality by smoking status at enrollment, as well as time period in study.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Malig et al., 2013	ST	Respiratory morbidity (Asthma and COPD ED and HA)	35 CA counties (9 counties included for PM <sub>2.5</sub> analysis)	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	PM <sub>2.5</sub> data obtained from California Air Resources Board. Same day lag and various days lags average were calculated for PM <sub>2.5</sub> . Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid.	<p>County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis</p> <p>Time-invariant confounders and seasonal trends were controlled for given the study design.</p> <p>Other confounders included in the models were: other gaseous pollutants including ozone, linear and squared term for daily average temperature.</p> <p>Stratified analysis also by distance to monitor: within 10 km vs. 10-20 km</p>
McConnell et al., 2010	LT	Asthma Incidence	13 CA communities	Cohort Study (CHS)	PM <sub>2.5</sub> measured in central site monitors in each community and assigned to study participants.	<p>Multi-level Cox proportional hazard model accounting for residual variation in time to asthma onset and clustering of children around schools and communities</p> <p>Models adjusted for: secondhand smoke, pets in home, race/ethnicity, age at study entry, sex, and random effects for community and school.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Ostro et al., 2016	ST	Asthma and COPD ED	8 metropolitan areas/counties in CA	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	PM2.5 chemical speciation data from U.S. EPA provided by California Air Resources Board. Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid. Only participants living in zip codes within 20 km of PM2.5 constituents monitors were included.	<p>County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis</p> <p>Time-invariant confounders and seasonal trends were controlled for given the study design.</p> <p>Other confounders included in the models were: linear and squared term for lag0 temperature, day of the week.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Peng et al., 2009	ST	CVD HA Age 65+	119 U.S. Urban counties>150,000 populations	Time-series analysis (MEDICARE enrollees)	PM <sub>2.5</sub> data obtained from US EPA's AQS and STN.	<p>Log-linear Poisson Regression analysis</p> <p>Adjusted for potential confounders like: weather, day of the week, unobserved seasonal factors. In county-specific regression model, following indicators were included: indicator for the day of the weeks, a smooth function of time per calendar year to control for seasonality and long-term trends, a smooth function of current-day temperature, a smooth function of the 3-day running mean temperature, a smooth function of current-day dew-point temperature, and a smooth function of the 3-day running mean dew-point temperature. To model smooth functions we used a natural spline basis.</p>



Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Pinault et al., 2016	LT	All-cause, CVD and lung cancer mortality	Multicity Canada	Prospective Cohort Study (subset of participants of the Canadian Community Health Survey)	<p>PM<sub>2.5</sub> concentration derived from MODIS. Geographically weighted regression including monitoring and land use data was applied to the estimates from MODIS to produce average PM<sub>2.5</sub> concentration at 1 km<sup>2</sup> resolution. These model estimates extended to 1998-2003 using inter-annual variation of Boys et al.</p> <p>Participants were assigned exposure based on their postal code of residence.</p>	<p>Cox proportional hazards models</p> <p>Models were stratified by age (5-yr interval) and sex. Models adjusted for individual socioeconomic covariates and behavioral (BMI, smoking and alcohol consumption, fruit and vegetable consumption) covariates, ecological variables including neighborhood socioeconomic status (both social and material deprivation).</p>

Pope et al., 2015a	LT	All-cause, IHD mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Exposure to PM <sub>2.5</sub> was estimated by linking geocoded home addresses of the study participants to ambient PM <sub>2.5</sub> concentrations derived using a national-level hybrid land use regression (LUR) and Bayesian Maximum Entropy (BME) interpolation model (LUR-BME) that incorporated data from ground-based monitors	<p>Cox proportional hazards models</p> <p>The individual-level covariates incorporated in the models included 13 variables that characterized current and former smoking habits (including smoking status of never, former, or current smoker, linear and squared terms for years smoked and cigarettes smoked per day, indicator for starting smoking at aged &lt;18 years, and pipe/cigar smoker); 1 continuous variable that assessed exposure to second-hand cigarette smoke (hours/d exposed); 7 variables that reflected workplace PM<sub>2.5</sub> exposure in each subject's main lifetime occupation; a variable that indicated self-reported exposure to dust and fumes in the workplace; variables that represented marital status (separated/divorced/widowed or single versus married); variables that characterized the level of education (high school, more than high school versus less than high school); 2 body mass index variables (linear and squared terms for body mass index); variables that characterized the consumption of alcohol (beer, missing beer, wine, missing wine, liquor, and missing</p>
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Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
						<p>liquor); and variables that indicated quartile ranges of dietary fat index and quartile ranges of a dietary vegetable/fruit/fiber index. Ecological covariates included median household income; percentage of people with &lt;125% of poverty-level income; percentage of unemployed individual aged ≥16 years; percentage of adults with &lt;12th grade education; and percentage of the population who were black or Hispanic. These ecological covariates were included in the models using both zip code level data and zip code deviations from the county means.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Shi et al., 2016	ST and LT	Total mortality (65+)	New England Area with 6 U.S. States	Open Cohort study (MEDICARE enrollees)	Daily PM <sub>2.5</sub> was predicted at 1-km <sup>2</sup> spatial resolution from novel 3-stage statistical models. Similar 3-stage approach was used to estimate daily temperature. Participants were assigned 365-day moving average (for long-term exposure) and average lag0-1 (for short-term exposure) based on the ZIP codes of residence.	Chronic effects of air pollution assessed using Cox proportional hazard models. Acute effects of air pollution assessed using Poisson log-linear models. Both acute and chronic effects were assessed using Poisson survival analysis. Analysis performed in full-cohort as well as low exposure cohorts.  Poisson survival models were adjusted for smooth function of time, temporal covariates such as temperatures and day of the week, spatial covariates such as zip code-level socio-economic variables.
Stieb et al., 2009	ST	Cardiac and Respiratory ED visits	Seven Canadian Cities	Time series study (Hospital cases)	PM Data from National Air Pollution Surveillance (NAPS) system. City averages of the exposure were calculated by averaging stations within the city. Calculated average concentration for lag0-2.	Generalized Linear Models with natural spline functions of time to adjust for seasonal cycles in air pollution and health  Confounders included: Mean daily temperature and relative humidity at lag 0, 1, and 2 days, day of the week and holidays.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Szyszkowicz, 2009	ST	Angina ED	Seven Canadian Cities	Time series study (Hospital cases)	PM Data from National Air Pollution Surveillance (NAPS) system. City averages of the exposure were calculated by averaging stations within the city. Calculated average concentration for lag0-2.	Generalized Linear Mixed models  Models adjusted for meteorological variables such as relative humidity, temperature and atmospheric pressure (a daily 24-hr average measurements were calculated). Temperature and relative humidity in models were represented by natural splines. Stratified analysis by season as well as combined for the whole period.
Thurston et al., 2016a	LT	All-cause, CVD and respiratory mortality	6 U.S. States and 2 MSAs	Cohort study (NIH_AARP cohort)	PM Data from US EPA AQS. Census-tract estimates generated using hybrid LUR and BME models that were combined to generate monthly estimates of PM2.5.  Participants exposure was estimated at census-tract of residence and included annual mean concentration in the year of mortality, and 1-year lag average.	Cox proportional hazard models  Stratified analysis by age, sex, regions (6 states and 2 MSAs). Confounders adjusted included: race, education, marital status, BMI, alcohol consumption, smoking history, contextual variables such as median household income and % pop with less than high school education. Several interactions between PM2.5 and socio-demographics were also tested.

Turner et al., 2016	LT	Lung cancer mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Estimated PM <sub>2.5</sub> concentrations were obtained using a national-level hybrid land use regression (LUR) and Bayesian maximum entropy (BME) interpolation model. Monthly PM <sub>2.5</sub> monitoring data were collected from 1,464 sites from 1999 through 2008, with 10% reserved for cross- validation. The base LUR model that predicted PM <sub>2.5</sub> concentrations included traffic within 1 km and green space within 100 m <sup>3</sup> . Residual spatiotemporal variation in PM <sub>2.5</sub> concentrations was interpolated with a BME interpolation model. The two estimates were then combined. The cross validation R <sup>2</sup> was approximately 0.79. Mean PM <sub>2.5</sub> (1999–2004) concentrations were used here.	Cox proportional hazards model  Models were adjusted for education; marital status; BMI and BMI squared; cigarette smoking status; cigarettes per day and cigarettes per day squared; years smoked and years smoked squared; started smoking at younger than 18 years of age; passive smoking (hours); vegetable, fruit, fiber, and fat intake; beer, wine, and liquor consumption; occupational exposures; an occupational dirtiness index; and six sociodemographic ecological covariates at both the postal code and postal code minus county-level mean derived from the 1990 U.S. Census (median household income and percentage of African American residents, Hispanic residents, adults with postsecondary education, unemployment, and poverty).  Potential confounding examined by elevation, MSA size, annual average daily maximum air temperature, mean county-level residential radon concentrations, and 1980 percentage of air conditioning.
Urman et al., 2014	LT	Lung-function decline	8 Southern CA communities/counties	Cohort study (CHS)	Central monitors in each community	Linear Regression model

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
					provided data on air pollutants. Each child was assigned exposure based on the child's resident community.	Models were adjusted for demographic, socio-economic and anthropometric variables (BMI, height), study community.
Wang et al., 2017	LT	Total mortality (65+)	7 U.S. Southeast States	Open Cohort study (MEDICARE enrollees data)	<p>Three stage Hybrid model to predict daily PM<sub>2.5</sub> concentration at 1 km<sup>2</sup> resolution. Air temperature also estimated at similar scale using satellite remote sensing and land use variables.</p> <p>Participants were assigned annual averages of PM<sub>2.5</sub> by averaging estimated for all grid cells within the zip code tabulation area (ZCTA) of residence.</p>	<p>Cox Proportional hazard models</p> <p>Models were stratified by age groups, sex, race. Adjusted for variables: year of enrollment, previous admission due to CHF, COPD, MI and diabetes, numbers of days spent in ICU and CCU, state, ZCTA level socio-demographic variables such as % pop below poverty, urbanicity, lower education, median income and median home value, and behavioral variables such as % smokers and obesity at county level. Further model also included yearly mean summer temperature at ZCTA level.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Weichenthal et al., 2016c	ST	Asthma and COPD ED	15 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	Daily average concentration of PM <sub>2.5</sub> collected from fixed-monitoring stations in Ontario, part of Canada's National Air Pollution Data. Participants were assigned data based on the city of residence. Various lags assessed including lag0, lag1, lag2 and lag0-2.	Conditional logistic regression models  Models adjusted for 3-day mean temperature and relative humidity using cubic splines.
Weichenthal et al., 2016b	ST	MI ED	16 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	PM data obtained from 20 provincial monitoring sites located in 16 cities. Exposure at various lags: lag0 lag1, lag 2 and mean lag0-2 were assigned to participants based on the city of residence.	Conditional logistic regression models  Models adjusted for 3-day mean temperature and relative humidity using cubic splines.



Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Yap et al., 2013	ST	Asthma HA	12 CA counties	Time Series study (Hospital admissions)	PM <sub>2.5</sub> data was obtained from California Air Resources Board that maintains information from the National Air Monitoring Stations. 24-hr average mass concentration calculated for each county by averaging monitors within the county. Participants were assigned exposure based on their county of residence. PM at various lags lag0-lag6 were assessed.	Generalized Additive Poisson Regression analysis were run at county-level  Models adjusted for: long-term time trends and seasonality, day of the week and smoothing splines within different lags for temperature. Effect modification by single or composite area-based SES assessed.
Zanobetti et al., 2009	ST	Heart Failure and MI HA 65+	26 US communities	Time Series study (MEDICARE enrollees data)	PM <sub>2.5</sub> data obtained from US EPA AQS. Daily PM <sub>2.5</sub> data available for various monitors were averaged over the county. Generated 2-day moving average PM <sub>2.5</sub> conc..	Poisson regression analysis  Models stratified by season. Controlled for long-term trend with natural cubic spline for each season and year, day of the week, three-day average temperature and dew point temperature.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Zanobetti and Schwartz, 2009	ST	All-cause, CVD and respiratory mortality	112 US cities	Time Series study (NCHS data)	PM <sub>2.5</sub> data obtained from US EPA AQS. Daily PM <sub>2.5</sub> data available for various monitors were averaged over the county. Generated 2-day moving average (lag 0 and 1) PM <sub>2.5</sub> conc.	<p>Poisson regression analysis</p> <p>First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach in total by season and region.</p> <p>Controlled for long-term trend with natural cubic spline for each season and year, day of the week, same day and previous day temperature.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Zanobetti et al., 2014	ST	All-cause mortality 65+	121 US communities/cities	Case-Crossover Design (MEDICARE enrollees)	PM <sub>2.5</sub> data obtained from US EPA AQS. Daily PM <sub>2.5</sub> data available for various monitors were averaged over the communities. Participants were assigned 2-day moving average (lag 0 and 1) based on community of residence.	<p>Conditional logistic regression models at community level. In a second stage of analysis, the community specific results were combined using the multivariate meta-analysis techniques</p> <p>Conditional logistic regression controlled for confounders such as average temp for the same and previous day. Temperature was modelled using spline to account for nonlinear relationship. Effect modification tested for cause of prior admission due to neurological disorders or diabetes, primary or secondary hospitalization for other disease conditions. Stratified analysis by sex, age or race.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed
Zeger et al., 2008	LT	All-cause mortality 65+	668 U.S Urban counties	Retrospective Cohort Study of MEDICARE enrollees (MCAPS)	PM <sub>2.5</sub> data available from US EPA monitors. Spatially smoothed levels of 6-year average PM <sub>2.5</sub> . Participants living within 6 miles of the zip code centroid to EPA monitors were assigned exposure based on the ZIP code of residence.	Log-linear Regression model ran for specific US regions separately  Models adjusted for individual socio-demographic variables and ZIP code level SES variables (education, income, poverty etc.). Also included standardized mortality ratio for COPD as a surrogate indicator of long-term smoking pattern of its residents.

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**APPENDIX C. SUPPLEMENTAL INFORMATION  
RELATED TO THE HUMAN HEALTH RISK  
ASSESSMENT**

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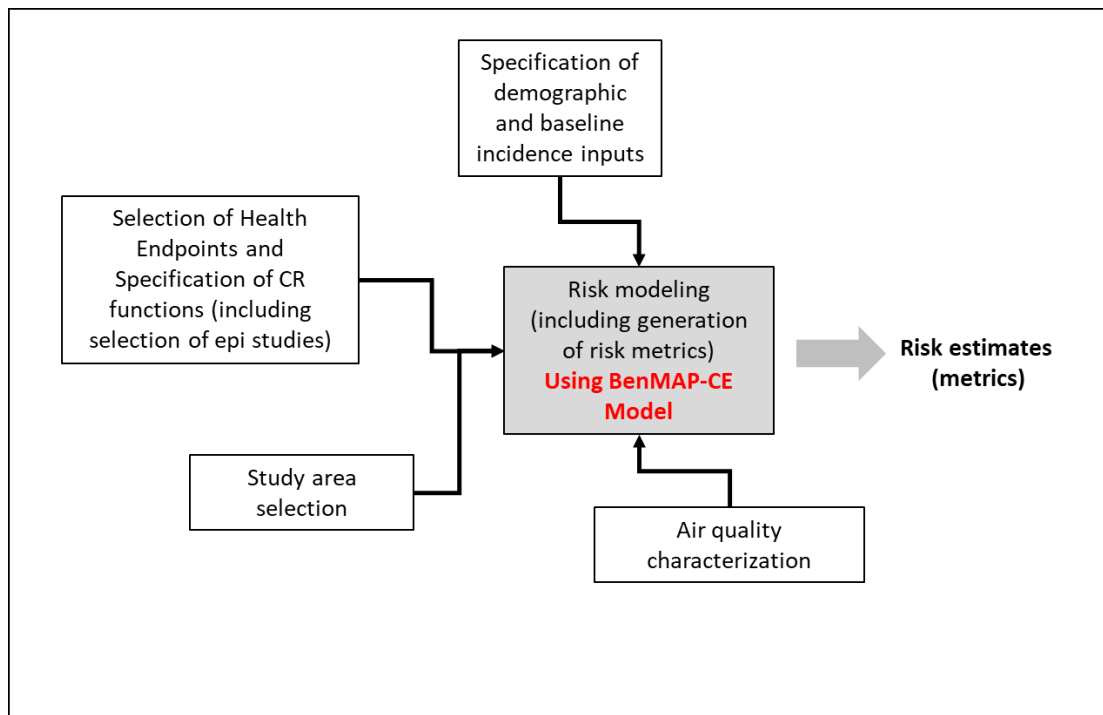
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This appendix provides supplemental information related to the risk assessment described in section 3.3 of final particulate matter (PM) policy assessment (PA), including:

- Additional technical detail on the risk assessment approach, including sources and derivation of key inputs to the risk modeling process (section C.1).
- Supplemental risk results (section C.2) intended to provide additional context for the summary risk estimates presented in the PA section 3.3.2, including:
  - The modeled risk estimates that underly summary tables presented in PA section 3.3.2 aggregated to the CBSA-level (i.e., the urban study area) (section C.2.1).
  - Additional graphics including line plots, maps and scatter plots illustrating the distribution of the grid-level risk estimates (section C.2.2).
- Characterization of variability and uncertainty related to the risk assessment (section C.3).

## **C.1 ADDITIONAL TECHNICAL DETAIL ON THE RISK ASSESSMENT APPROACH**

As discussed in section 3.3 of the PM PA, our general approach to estimating PM<sub>2.5</sub>-associated human health risks in this review utilizes concentration-response (CR) functions obtained from epidemiology studies to link ambient PM<sub>2.5</sub> exposure to risk in the form of incidence (counts) of specific health effects. The derivation and use of this type of CR function in modeling PM<sub>2.5</sub>-attributable risk is well documented both in previous PM NAAQS-related risk assessments (section 3.1.2 of U.S. EPA, 2010) and in Section C.1.1 of this appendix. Inputs required to model risk using these CR functions are identified below (Figure C-1) and include (a) the concentration-response (CR) functions themselves, which are obtained from epidemiologic studies (section C.1.1), (b) baseline health incidence data and information on population demographics (section C.1.2), and (c) modeled ambient PM<sub>2.5</sub> concentrations corresponding to air quality scenarios of interest (section C.1.5).



**Figure C-1. Key inputs to the risk assessment**

### **C.1.1 Selection of Key Health Endpoints and Specification of Concentration-Response Functions from Epidemiologic Studies**

In selecting specific CR functions for the risk assessment, we focus on health outcomes for which the PM ISA determines the evidence supports either a “causal” or a “likely to be causal” relationship with short- or long-term PM<sub>2.5</sub> exposures (U.S. EPA, 2019). As discussed in Chapter 3 of this final PA (Table 3-1), these outcomes include the following:

- mortality (resulting from long- and short-term exposure),
- cardiovascular effects (resulting from long- and short-term exposure),
- respiratory effects (resulting from long- and short-term exposure),
- cancer (resulting from long-term exposure), and
- nervous system effects (resulting from long-term exposure).

We have focused the analysis on short- and long-term PM exposure-related mortality, reflecting its clear public health importance, the large number of epidemiologic studies available for consideration, and the broad availability of baseline incidence data. The specific set of health effect endpoints included in the risk assessment are:

- *Long-term PM exposure-related mortality*: all-cause, ischemic heart disease (IHD) related, lung-cancer related



- *Short-term PM exposure-related mortality:* all-cause/non-accidental

To identify specific epidemiologic studies for potential inclusion in the risk assessment, we focus on U.S. multicity studies assessed in the ISA. These studies are identified in section 3.2.3.2.1 of this PA (Figures 3-3 to 3-6). Of these, we used the following criteria to identify the specific set of studies for inclusion in the risk assessment:

- *National-scale coverage:* We focus on epidemiology studies reporting national-level effect estimates. Epidemiology studies that focus on individual cities or regions were excluded. Focusing on national-level epidemiological studies has the benefit of characterizing PM<sub>2.5</sub>-associated risks broadly across the U.S. and in relatively large populations (compared with single-city or regional studies), which tends to improve precision in the effect estimated generated.
- *Evaluation of relatively lower ambient PM concentrations:* In selecting epidemiology studies, to the extent possible, we favored those studies which characterized the ambient PM<sub>2.5</sub>-mortality relationship at levels at or near the current NAAQS, given that the risk assessment would be focusing on evaluating risk associated with the current NAAQS.
- *Populations with available baseline incidence data:* For some populations (e.g., diesel truck drivers), it can be challenging to model risk at the national-level given uncertainties associated with specifying key inputs for risk modeling (i.e., baseline incidence rates for mortality endpoints and detailed national-level demographics). For that reason, we favored those epidemiology studies providing effect estimates for populations readily generalizable to the broader U.S. population (e.g., specific age groups not differentiated by additional socio-economic, or employment attributes).
- *Estimates of long-term PM<sub>2.5</sub> exposures based on hybrid modeling approaches:* For long-term PM<sub>2.5</sub> exposures, we focus on epidemiologic studies that estimate exposures with hybrid modeling approaches. The primary rationale for this decision is the agreement between the design of these epidemiology studies (i.e., their use of hybrid-based modeling approaches in characterizing ambient PM) and the hybrid air quality surfaces we are using in this risk assessment. This general agreement between the air modeling surfaces used in long-term mortality epidemiology studies and our air quality modeling reduces uncertainty in the risk assessment.
- *Estimates of short-term PM<sub>2.5</sub> exposures based on composite monitor data:* Short-term mortality epidemiology studies utilizing hybrid modeling approaches, which are fewer in number compared with long-term mortality studies, tend to be regional in scope and consequently, did not meet the criterion of providing national-scale effect estimates. For that reason, in modeling short-term mortality, epidemiology studies utilizing composite-monitor based exposure surrogates were used as the basis for deriving CR functions. We recognize the uncertainty introduced into the modeling of short-term mortality due to the use of effect estimated obtained from studies utilizing composite monitors. However, we

felt these use of national-scale epidemiology studies was a more important criterion for selection.<sup>1</sup>

- *Evaluation of potential confounders and effect modifiers:* Preference was given, to the extent possible, to those studies which more fully address potential confounders and effect modifiers and to those studies which utilize individual- rather than ecological measures in representing those confounders/effect modifiers. Recognizing that both single- and multi-pollutant models have advantages and disadvantages in characterizing the ambient PM-mortality relationship, to the extent possible, we include epidemiology studies (and associated effect estimates) based on both single- and multi-pollutant models.
- *Exploration of multiple approaches for estimating exposures:* For studies that estimate PM<sub>2.5</sub> exposures using hybrid modeling approaches, preference was given to studies that also explore additional methods for estimating exposures (i.e., multiple hybrid methods or hybrid methods plus monitor-based methods) and compare health effect associations across approaches.

Application of the criteria listed above resulted in the selection of the epidemiology studies presented in Table C-1 for inclusion in the risk assessment as sources of effect estimates. Table C-1 includes summary information on study design, details on the selection of effect estimates, the derivation of beta values, and specification of CR functional form based on those effect estimates for use in the risk assessment. The procedure used to derive CR functions (including specification of the beta values and mathematical forms for those functions) is described below.

The remainder of this section describes the method used in specifying the concentration-response (CR) functions used in the PM NAAQS REA (information presented in this section is drawn from BenMAP Manual, Appendix C with additional detail specific to the epidemiology studies selected for use in this risk assessment).<sup>2</sup> These CR functions translate changes in ambient PM<sub>2.5</sub> into changes in baseline incidence rates for specific disease endpoints utilizing beta ( $\beta$ ) values obtained from epidemiology studies studying the association between ambient PM<sub>2.5</sub> exposure and specific health endpoints. These beta values (and associated standard errors) are based on effect estimates obtained from the underlying epidemiology studies (equation below). In addition, the mathematical forms for the health impact functions specified for use in

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<sup>1</sup>After identifying studies for inclusion in the draft risk assessment and initiating analyses, we became aware that Di et al., 2017a uses a hybrid model-based approach to estimate PM<sub>2.5</sub> exposures. The primary effect estimate reported for this study (which reflects copollutant modeling including ozone) is larger than effect estimates selected for this risk assessment. Specifically, the copollutant model for Di et al., 2017a reports an increased daily mortality risk of 1.05% (95<sup>th</sup> CI: 0.95-1.15%) with this effect estimate being two to three times larger than similar effect estimates used in this risk assessment and has a substantially tighter confidence interval (Table C-1). Given the approximate linearity of the CR functions used, we anticipate that this difference in effect estimate would translate into a similar magnitude of difference in modeled mortality incidence (i.e., 2-3 times higher had the Di et al., 2017a effect estimate been used in the risk assessment).

<sup>2</sup> <https://www.epa.gov/benmap/benmap-ce-manual-and-appendices>

this risk assessment reflect the models used in the epidemiology studies providing those effect estimates. Consequently, derivation of the beta values based on effect estimates from underlying epidemiology studies (and specification of the form of the health impact functions) represents a key step in the design of the REA.

The majority of the epidemiology studies providing effect estimates for this PM REA utilized either Poisson or Cox proportional hazard models which result in exponential (or log-linear) forms for the CR functions, where the natural logarithm of mortality incidence is a linear function of PM<sub>2.5</sub>.<sup>3</sup> If we let  $x_0$  denote the baseline (starting) PM<sub>2.5</sub> level, and  $x_1$  denote the control (ending ) PM<sub>2.5</sub> level,  $y_0$  denote the baseline incidences rate of the health effect, and Pop the underlying population count for the applicable demographic group in the spatial unit of analysis<sup>4</sup> we can derive the following CR function specifying the relationship between the change in  $x$ ,  $\Delta x = (x_0 - x_1)$  and the corresponding change in  $y$ ,  $\Delta y$  (mortality incidence):

$$\Delta y = y_0 [1 - e^{-\beta \Delta x}] * \text{Pop}$$

Given that the epidemiology studies providing effect estimates for long-term exposure-related mortality and short-term exposure-related mortality in the context of the current PM REA (Table C-1) use different categories of models (Cox proportional hazard and Poisson/Logistic, respectively) we describe the process of deriving the betas and specifying CR functional forms separately for each of these endpoint categories. As noted earlier, the logit model utilized in Zanobetti et al., 2014, is discussed at the end of the section covering short-term PM<sub>2.5</sub>-related mortality.

#### Derivation of betas for long-term PM<sub>2.5</sub> exposure-related mortality

Cox proportional hazard models used to evaluate mortality associated with long-term PM<sub>2.5</sub> exposure are designed to model effects on population survival. This class of epidemiology model is based on a hazard function, defined as the probability that an individual die at time  $t$ , conditional on that individual having survived up to time  $t$ . As such, the hazard function represents a time-specific snapshot of the rate of mortality (events per unit time) within a study population. While the risk can vary over time, in the case of the Cox proportional hazard model, it is assumed that the hazard ratio is constant. The proportional hazard model takes the form:

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<sup>3</sup> One study. Zanobetti et al., 2014, supporting the modeling of short-term PM<sub>2.5</sub> exposure-related mortality provided a logistic-based model form, which is discussed at the end of this section.

<sup>4</sup> Spatial unit of analysis refers to the geographic scale at which the CR function is applied in generating a risk (incidence) estimate (e.g., zip code, county, 12km grid cell). Typically, the spatial unit of analysis used in a REA is based on the spatial scale reflected in the epidemiology study(s) supplying the effect estimates. For this REA, the spatial unit of analysis is the 12km grid cell.

$$h(X, t) = h_0(t)e^{X \cdot \beta} ,$$

Where X is a vector of explanatory variables,  $\beta$  is a vector of associated coefficients and  $h_0(t)$  is the baseline hazard (the risk when all covariates (X) are set to zero).

Epidemiology studies utilizing the Cox proportional hazard model in characterizing ambient PM<sub>2.5</sub>-health effects typically report hazard ratios (HRs) as the effect estimate. HRs represent the ratio of hazard functions for the baseline and control scenarios reflecting a specific difference in ambient PM<sub>2.5</sub> exposure (typically a 10 ug/m<sup>3</sup> increment). The HR simplifies as shown (with the baseline hazard ratio dropping out), allowing us to readily derive the Beta value from this effect estimate:

$$HR = \frac{h(X_0, t)}{h(X_c, t)} = \frac{h_0(t)e^{X_0 \cdot \beta}}{h_0(t)e^{X_c \cdot \beta}} = e^{\Delta PM \cdot \beta}$$

It is then possible to calculate the beta as follows:

$$\beta = \frac{\ln(HR)}{\Delta PM}$$

As noted in Sutradhar and Austin, 2018, the HR associated with a Cox-proportional hazard model may approximate the RR when the effect estimate (and consequently the beta) is relatively small. This is the case with the effect on mortality modeled for long-term exposure to ambient PM<sub>2.5</sub> (i.e., the size of the effect estimate supports an assumed equivalency between HR and RR). The near equivalency between the HR and RR, allows us to utilize the beta derived above in a CR-function based on a log-linear functional form of the type presented earlier, to model changes in mortality related to changes in ambient PM.

#### Derivation of betas for short-term PM<sub>2.5</sub> exposure-related mortality

The epidemiology studies selected for use in modeling short-term PM<sub>2.5</sub> exposure-related mortality utilize both the Poisson (log-linear) model form (Baxter et al., 2017) and the logit model form (Zanobetti et al., 2014).<sup>5</sup> In both cases, the epidemiology studies provide effects in terms of *percent increase* in mortality.

The log-linear (Poisson) model is used to evaluate effects associated with continuous (count) events. With the log-linear (Poisson) model, the relative risk is simply the ratio of the two risks:

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<sup>5</sup> Note that the Ito et al., 2013 study also utilizes a Poisson model. However, that study provides beta values (including standard errors) and for that reason the results of this study are directly applicable in modeling changes in mortality without any of the derivations presented here for the other studies.

$$RR = \frac{y_0}{y_c} = e^{\beta \cdot \Delta PM}$$

The derivation of the beta with a Poisson model specified RR is as follows. Taking the natural log of both sides, the beta coefficient in the CR function underlying the relative risk can be derived as:

$$\beta = \frac{\ln(RR)}{\Delta PM}$$

The beta derived in this fashion can then be used with a log-linear functional form (as presented earlier) to model changes in mortality related to changes in ambient PM.

The logistic model form is used to model dichotomous events. With the logistic model form, when we are provided with a RR value, as is the case here, we can make a similar assumption to that used above with the Cox proportional hazard function (i.e., that the OR and RR approach equivalency under conditions of relatively small effect levels). That observation in turn allows us to assume that

$$RR = \frac{y_0}{y_c} = (1 - y_0) \times e^{-\Delta PM \cdot \beta} + y_0$$

Then, assuming (based on the relatively small size of the baseline incidence) that:

$$e^{-\Delta PM \cdot \beta} \cong (1 - y_0) \times e^{-\Delta PM \cdot \beta} + y_0$$

$$\Rightarrow RR \cong e^{-\Delta PM \cdot \beta}$$

It is then possible to calculate the underlying beta coefficient as follows:

$$\frac{\ln(RR)}{-\Delta PM} \cong \beta$$

Since the derivation of the beta is based on the assumption of a log linear functional form, we can apply the beta in a log-liner CR function of the form described earlier:

$$\Delta y = y_0[1 - e^{-\beta \Delta x}] * \text{Pop}$$

**Table C-1. Details regarding selection of epidemiology studies and specification of concentration-response functions for the risk assessment.**

Reference and study title	Study description	Exposure Estimation Approach	CR function model	Location of study effect estimate(s) in journal article	Additional notes regarding effect estimate selection	Epi-demio-logic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
<b>Long-term exposure-related mortality studies</b>										
Di et al., 2017b  Air Pollution and Mortality in the Medicare Population	Exploring relationship between air pollution (ozone, PM <sub>2.5</sub> ) and mortality Key details: - Medicare population (65+) - ecological control for confounders - all-cause mortality only - provides CR function slopes for areas above and below the current PM NAAQS level (but model for areas below current standard only done for low ozone cells)	Exposures estimated at zip code of residence based on a neural network model that incorporates satellite data, chemical transport modeling, land-use terms, meteorology data, monitoring data, and other data	Cox proportional-hazards model with a generalized estimating equation to account for the correlation between ZIP codes	Table 2 Risk of death associated with an increase of 10 µg/m <sup>3</sup> PM <sub>2.5</sub> or an increase of 10 ppb in ozone concentration. Uses single pollutant model for full analysis.	Using single pollutant, full PM range model (model for <12 µg/m <sup>3</sup> applicable to only low-ozone days) <sup>6</sup>	Hazard ratio (95 percent CI)	All-cause	1.084 (1.081-1.086)	8.07E-03	1.18E-04
Jerrett et al., 2016  Comparing the Health Effects of Ambient Particulate Matter Estimated Using Ground-Based Versus Remote Sensing Exposure Estimates	Compares mortality effect estimates for PM <sub>2.5</sub> modeled from remote sensing to those for PM <sub>2.5</sub> modeled using ground-level information. - ACS cohort (Ages 30+) - IHD and diseases of circulatory system - individual-level confounder control	Multiple exposure estimation approaches evaluated – risk assessment uses results based on an ensemble approach that incorporates chemical transport modeling, land use data, satellite data, and data from ground-based monitors	Cox proportional hazard model	Table 4 IHD, fully adjusted (1990 ecological confounders) ensemble estimate	Used the ensemble estimate (pools effect estimates generated using different exposure estimates)	Hazard ratio (95 percent CI)	IHD	1.15 (1.11-1.19)	1.40E-02	1.78E-03

<sup>6</sup> We note that Di et al., 2017b does include a copollutant model-based effect estimate (HR 1.073, 95<sup>th</sup>%CI 1.071-1.075). Had this effect estimate been used in risk modeling (which would translate into a beta value of 7.05E-3), we would anticipate the risk estimates for all-cause mortality to be slightly less (~13% lower based on comparison of calculated betas) than those estimated based on the single-pollutant model used in this risk assessment.

Reference and study title	Study description	Exposure Estimation Approach	CR function model	Location of study effect estimate(s) in journal article	Additional notes regarding effect estimate selection	Epi-demiologic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Pope et al., 2015  Relationships Between Fine Particulate Air Pollution, Cardiometabolic Disorders, and Cardiovascular Mortality	Evaluates the relationship between long-term exposure to ambient PM <sub>2.5</sub> and CVD and cardiometabolic disease, including effect modification of the relationships by pre-existing cardiometabolic risk factors - ACS (30+) (oversampled affluent individuals) - individual-level covariates	Exposures estimated at home addresses based on a land use regression and Bayesian maximum entropy (LUR-BME) interpolation model that incorporated data from ground-based monitors	Cox proportional hazard model	Table 1. Cox model with individual-level plus ecological covariates; exposure based on LUR-BME	NA	Hazard ratio (95 percent CI)	All-cause	1.07 (1.06-1.09)	6.77E-03	7.12E-04
					NA	Hazard ratio (95 percent CI)	IHD	1.14 (1.1-1.18)	1.31E-02	1.79E-03
Thurston et al., 2016  Ambient Particulate Matter Air Pollution Exposure and Mortality in the NIH-AARP Diet and Health Cohort	Reevaluates the relationship between long-term exposure to ambient PM <sub>2.5</sub> and mortality given recent decline in U.S. ambient PM concentrations. Differentiation of risk for fossil fuel PM <sub>2.5</sub> versus total PM <sub>2.5</sub> - NIH-AARP Cohort (only select states - CA, FL, LA, NJ, NC, PA, GA MI) (55-85yrs) - CVD, all-cause - residential locations matched to census tract-level PM <sub>2.5</sub> estimates	Exposures estimated at census tract centroids based on land use data and ground-based monitors	Cox proportional hazard model	Table 2. NIH-AARP cohort time independent Cox model PM <sub>2.5</sub> mortality hazard ratios (and 95 <sup>th</sup> percentile CI) per 10 µg/m <sup>3</sup> , by cause and cohort subgroup. Cohort: ALL	NA	Hazard ratio (95 percent CI)	All-cause	1.03 (1-1.05)	2.96E-03	1.24E-03

Reference and study title	Study description	Exposure Estimation Approach	CR function model	Location of study effect estimate(s) in journal article	Additional notes regarding effect estimate selection	Epi-demiologic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Turner et al., 2016  Long-Term Ozone Exposure and Mortality in a Large Prospective Study	Evaluates the relationship between long-term exposure to ambient PM <sub>2.5</sub> and all-cause and cause-specific mortality. Also, estimated the association between PM <sub>2.5</sub> , regional PM <sub>2.5</sub> , and near-source PM <sub>2.5</sub> and mortality in single-pollutant, copollutant and multipollutant models. - ACS (30+) - Includes lung cancer (otherwise similar results to Pope et al., 2015) - county-level assessment	Exposures estimated at residential locations based on land use data and ground-based monitors	Cox proportional hazard model	Table E4. Adjusted HRs (95 <sup>th</sup> percentile CI) for all-cause and cause-specific mortality in relation to each 10 unit increase in PM <sub>2.5</sub> LUR-BME concentrations, follow-up 1982-2004, CPS-II cohort, United States (n = 669,046).	Note that the non-cancer mortality endpoints provided in table E4 appear to mirror those provided in Table 1 of Pope et al., 2015 -so will use long-cancer effect estimate from this study only.	Hazard ratio (95 percent CI)	Lung cancer	1.09 (1.03-1.16)	8.62E-03	3.03E-03
<b>Short-term exposure-related mortality studies</b>										
Baxter et al., 2017  Influence of exposure differences in city-to-city heterogeneity in PM <sub>2.5</sub> -mortality associations in U.S. cities	Uses cluster-based approach to evaluate the impact of residential infiltration factors on inter-city heterogeneity in short-term PM-mortality associations. - Mortality data from NCHS - 77 U.S. CBSAs (all ages) - non-accidental mortality - CBSA-level assessment	Exposure estimates based on data from ground-based monitors	Poisson (log-linear) at city-level then aggregated	Obtained from results section in the text. After pooling the city-specific effect estimates into an overall effect estimate, short-term PM <sub>2.5</sub> exposure was found to increase 24-hr non-accidental mortality by 0.33% (95% CI: 0.13, 0.53). Based on lag 2 (day 0-1)	NA	Percent increase in 24-hr mortality (95 percent CI)	24-hr non-accidental mortality	0.33 (0.13-0.53)	3.29E-04	1.02E-04



Reference and study title	Study description	Exposure Estimation Approach	CR function model	Location of study effect estimate(s) in journal article	Additional notes regarding effect estimate selection	Epi-demio-logic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Ito et al., 2013  NPACT study 3. Time-series analysis of mortality, hospitalizations, and ambient PM <sub>2.5</sub> and its components	Use factor analysis to characterize pollution sources, assess the association between PM <sub>2.5</sub> and PM <sub>2.5</sub> components with morbidity and mortality outcomes. Also evaluates pollution levels, land-use, and other variables as modifiers that may explain inter-city variation in PM-mortality effect estimates. - Mortality data from NCHS - 150 and 64 U.S. cities (two analyses) (all ages) - MSA-level assessment	Exposure estimates based on data from ground-based monitors	Poisson GLM	Appendix G, Table G.6 for Figure 4 - use all-year lag 1 Beta: Regression coefficients (beta) and their SE for air pollutants at lag 0 through 3 days used to compute percent excess risks in figures shown in the main text and in Appendices B and G (corresponding figures are noted).	Utilized lag-1 (all year) beta because that had the strongest effect for CVD mortality and wanted our all-cause to reflect that stronger lag-association for the CVD effect (even though focusing on all-cause)	Betas with SE (no conversion required)	24-hr all-cause mortality	Study provided beta and SE	1.45E-04	7.47E-05
Zanobetti et al., 2014  A national case-crossover analysis of the short-term effect of PM <sub>2.5</sub> on hospitalizations and mortality in subjects with diabetes and neurological disorders	Estimates the effect of short-term exposure to PM <sub>2.5</sub> on all-cause mortality. Additionally, assesses the potential for pre-existing diseases to modify the association between PM <sub>2.5</sub> and mortality (neurological disorders and diabetes) - Medicare cohort - 121 U.S. communities (65+) - Community-level assessment (community defined as the county or contiguous counties encompassing a city's population)	Exposure estimates based on data from ground-based monitors	Logistic regression	Table 2. Percent increase for 10 µg/m <sup>3</sup> increase in the two days average PM <sub>2.5</sub> : Combined across the 121 communities	NA	Percent increase (95 percent CI)	All deaths	0.64 (0.42-0.85)	6.38E-04	1.09E-04

### C.1.2 Specification of Demographic and Baseline Incidence Data Inputs

This risk analysis requires both demographic and baseline-incidence data for the mortality endpoint categories evaluated. For our analyses, these data are projected to the year 2015 since the hybrid surfaces included in the analyses are based on a 2015 model year<sup>7</sup>. The BenMAP-CE model<sup>8</sup> is used in this risk assessment and the relevant demographic and baseline incidence data for the contiguous U.S., from the sources described below, is readily available within the current version of BenMAP-CE:

- *Demographic data:* BenMAP-CE includes 2010 U.S. Census block-level age, race, ethnicity and gender-differentiated data which the program can aggregate to various grid-level definitions selected by the user, including the 12 km grid coverage used for risk modeling in this analysis. In addition, BenMAP-CE has the ability to project future demographics using county-level projections provided by Woods & Poole (2015). See BenMAP-CE manual Appendix J for additional detail.<sup>9</sup>
- *Baseline incidence data for mortality endpoints:* County-level mortality and population data from 2012-2014 for seven causes of death in the contiguous U.S. was obtained from the Centers for Disease Control (CDC) WONDER database. To estimate values for 2015, we applied annual adjustment factors, based on a series of Census Bureau projected national mortality rates for all-cause mortality. See BenMAP-CE manual Appendix D for additional detail.<sup>9</sup>

### C.1.3 Study Area Selection

In selecting U.S. study areas for inclusion in the risk assessment, we focus on the following characteristics:

- *Available ambient monitors:* We focus on areas with relatively dense ambient monitoring networks, where we have greater confidence in adjustments to modeled air quality concentrations in order to simulate “just meeting” the current and alternative primary PM<sub>2.5</sub> standards (air quality adjustments are described below in section C.1.4).
- *Geographical Diversity:* We focus on areas that represent a variety of regions across the U.S. and that include a substantial portion of the U.S. population.
- *PM<sub>2.5</sub> air quality concentrations:* We balance the value of including a broad array of study areas from across the U.S. against the larger uncertainty associated with air quality adjustments in certain areas. For example, many areas have recent air quality that meets the current primary PM<sub>2.5</sub> standards. Inclusion of such areas in the risk assessment necessitates an upward adjustment to PM<sub>2.5</sub> air quality concentrations in order to simulate

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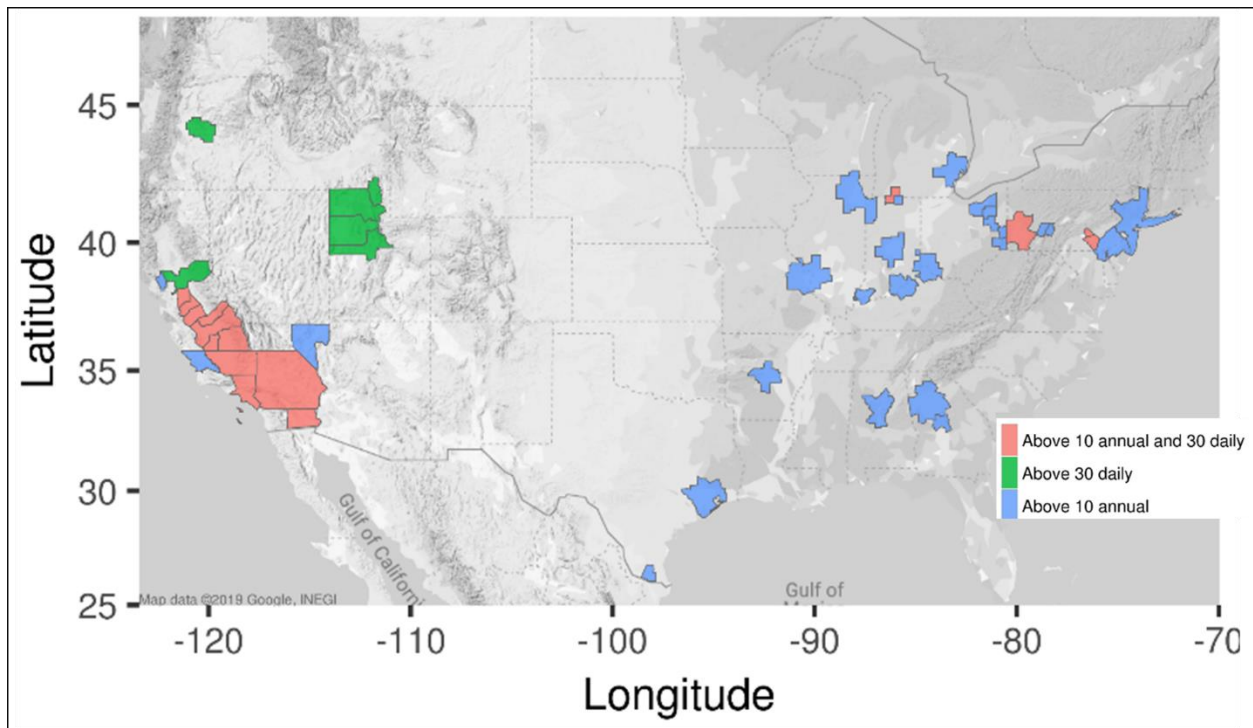
<sup>7</sup> The 2015 model year was the most recent CMAQ modeling platform available at the time of the design of the risk assessment and represents the central year of the 2014-2016 design value (DV) period. A single modeling year was used in the risk assessment, rather than modeling risk for the full three-year design value period, because model inputs for the 2016 period were not available at the time of the study (section C.1.4.3).

<sup>8</sup> <https://www.epa.gov/benmap>

<sup>9</sup> <https://www.epa.gov/benmap/benmap-ce-manual-and-appendices>

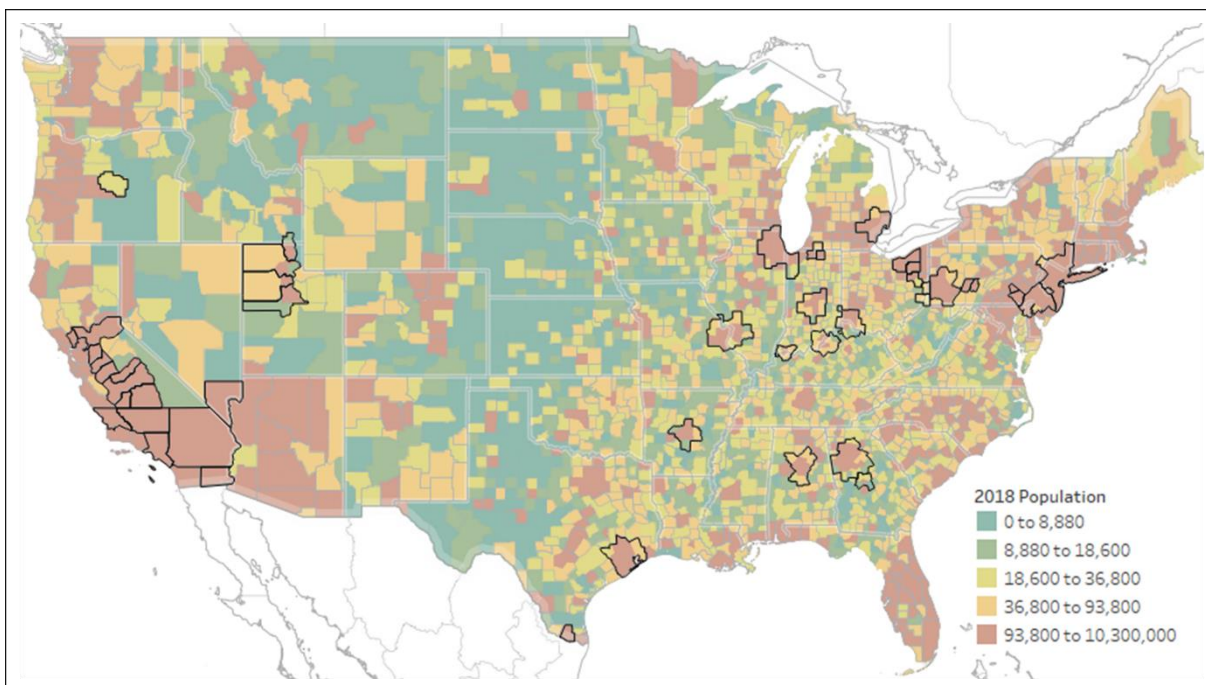
just meeting the current standards. Given uncertainty in how such increases could potentially occur, we select areas requiring either a downward adjustment to air quality or a relatively modest upward adjustment (i.e., no more than  $2.0 \mu\text{g}/\text{m}^3$  for the annual standard and  $5 \mu\text{g}/\text{m}^3$  for the 24-hour standard). In addition, as discussed further in section C.1.4.2, we excluded several areas that appeared to be strongly influenced by exceptional events.

Applying these criteria resulted in the inclusion of 47 core-based statistical areas (CBSAs) as study areas. These 47 study areas are identified in Figure C-2, with colors indicating whether they meet either or both the design value cutoffs. Green indicates areas that only exceed a 24-hr design value of  $30 \mu\text{g}/\text{m}^3$ , blue indicates areas that only exceed an annual design value of  $10 \mu\text{g}/\text{m}^3$ , and red indicates areas that exceed both cutoffs.

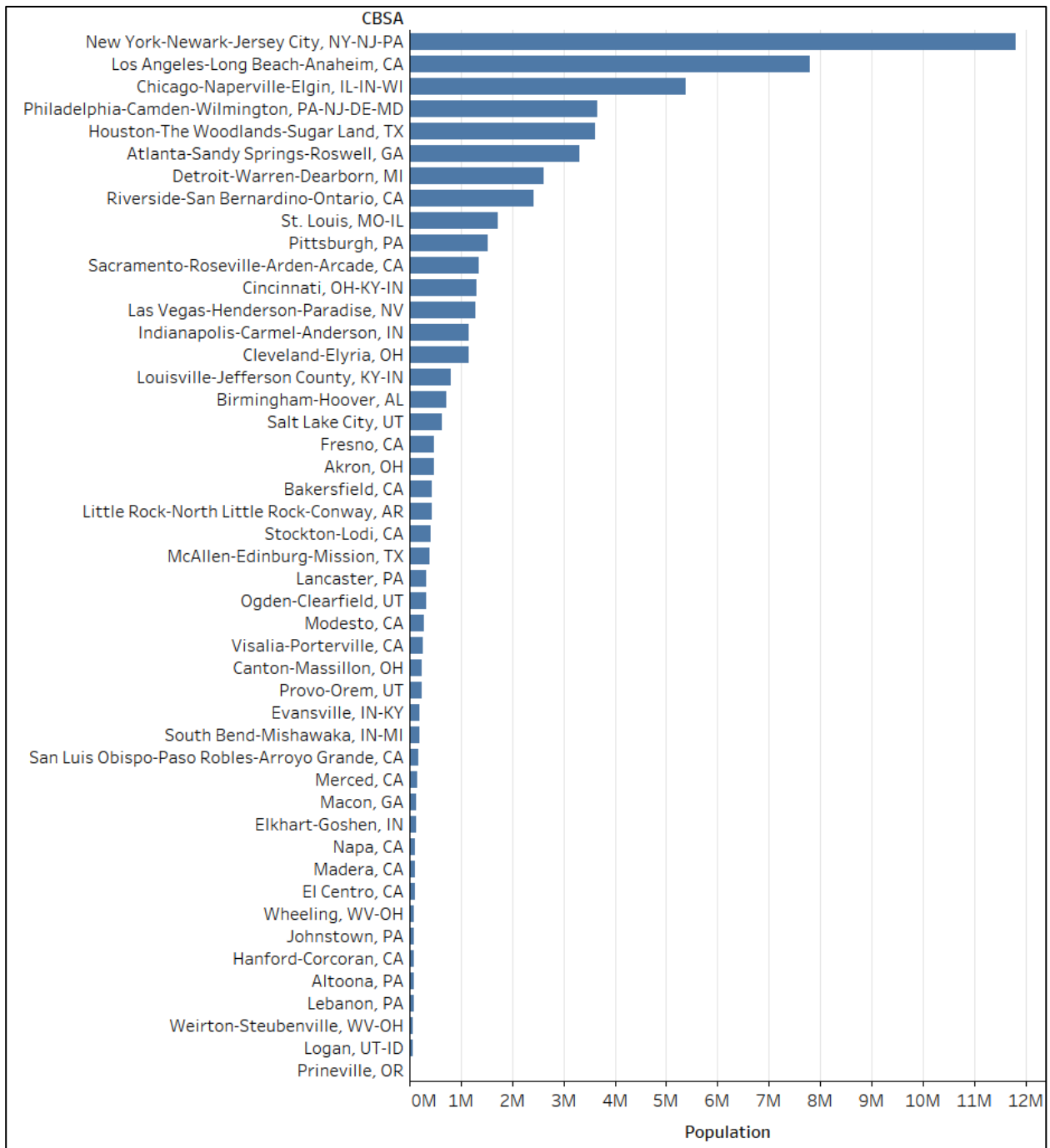


**Figure C-2. Map of the areas modeled in the risk assessment, colored by 2014-2016 PM<sub>2.5</sub> design values (DV).**

These 47 urban study areas include many highly populated CBSAs (Figure C-3 and Figure C-4). The population at or above the age of 30 in these areas includes roughly 58.4 million people, or approximately one-quarter of the total U.S. population above that age. Additional age-specific population information can be found in Table C-2.



**Figure C-3. Map of the 2018 U.S. population by CBSA, with the selected urban study areas outlined.**

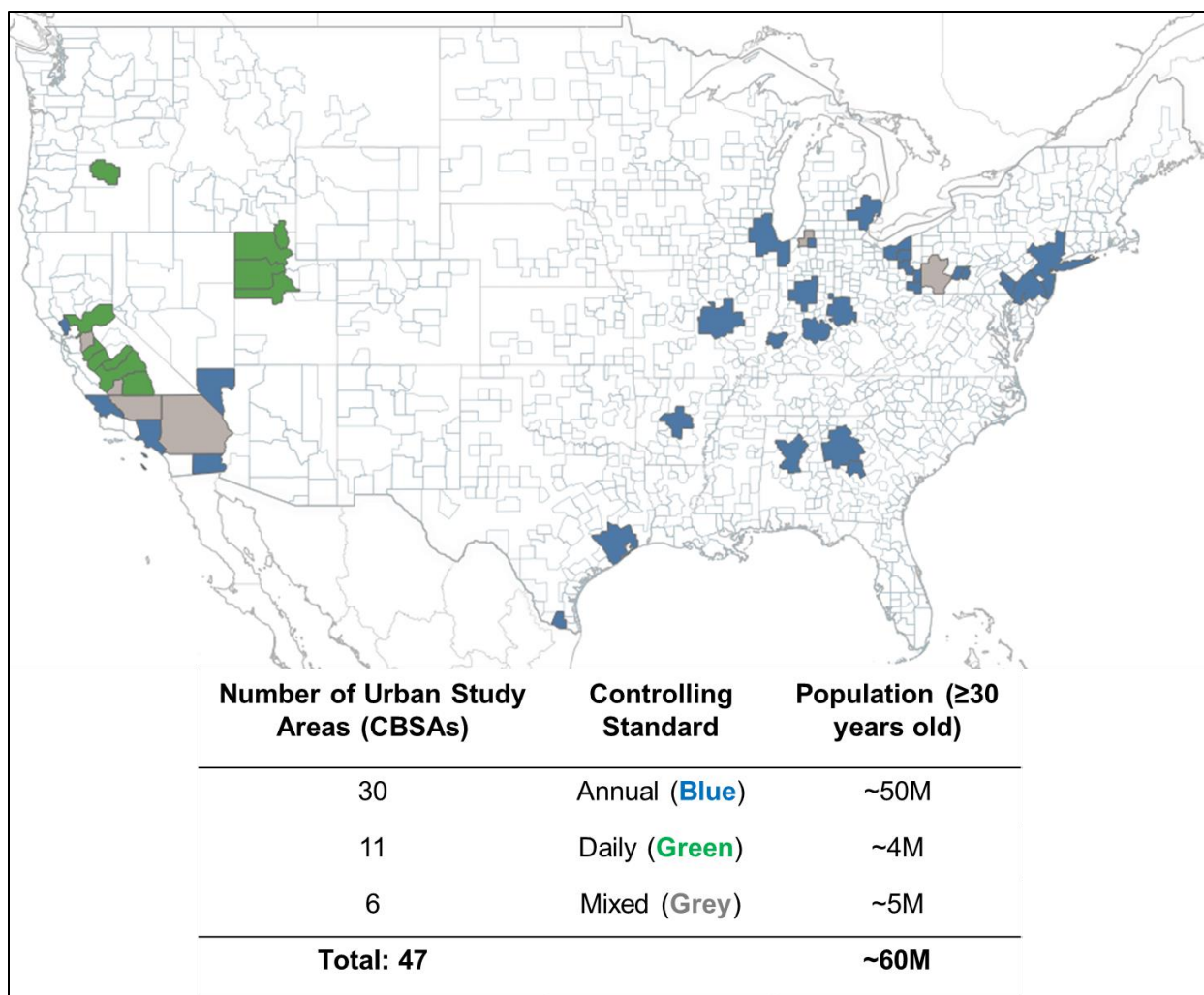


**Figure C-4. Population counts for ages 30 and above from each of the 47 CBSAs included in the risk assessment.**

**Table C-2. Population of the 47 urban study areas stratified by age.**

Population Age Range (Years)	Study Area Groupings (Millions)		
	47	30 (Annual-Controlled)	11 (24-hr-Controlled)
0-99	98.5	82.5	7.2
30-99	58.4	49.5	3.9
65-99	13.2	11.1	0.8
55-85	23.5	19.9	1.5

As noted in section 3.3 of this final PA and illustrated in Figure C-5, the 47 urban study areas include 30 study areas where just meeting the simulated standards is controlled by the current annual standard ( $12 \mu\text{g}/\text{m}^3$ ), 11 study areas where just meeting the simulated standards is controlled by the current 24-hr standard ( $35 \mu\text{g}/\text{m}^3$ ), and 6 study areas where just meeting the simulated standards is controlled by either the annual or 24-hr standard, depending on the air quality scenario and adjustment strategy (discussed more fully in section C.1.4).



**Figure C-5. Map of 47 Urban Study Areas Reflected in Risk Modeling Identifying Subsets Reflected in Risk Modeling (population estimates in millions of people).**

#### **C.1.4 Generation of Air Quality Inputs to the Risk Assessment**

As described in detail below, air quality modeling was used to develop gridded  $PM_{2.5}$  concentration fields for the risk assessment. A  $PM_{2.5}$  concentration field for 2015 was developed using a Bayesian statistical model that calibrates chemical transport model (CTM) predictions of  $PM_{2.5}$  to surface measurements (Chapter 2, section 2.3.3). The 2015  $PM_{2.5}$  concentration field was then adjusted to correspond to just meeting the existing and potential alternative standards using response factors developed from CTM modeling with emission changes relative to 2015. The modeling approach applies realistic spatial response patterns from CTM modeling to a concentration field, similar to those used in a number of recent epidemiologic studies, to characterize  $PM_{2.5}$  fields at 12 km resolution for study areas.

The adjustments to simulate just meeting the current standards and alternative standards are approximations of these air quality scenarios. In reality, changes in PM<sub>2.5</sub> in an area will depend on what emissions changes occur and the concentration gradients of PM<sub>2.5</sub> will vary across an area accordingly. For our analyses, two different adjustment approaches were applied to provide two outcomes that could represent potential bounding scenarios of PM<sub>2.5</sub> concentrations changes across the study area. The two adjustment approaches used to guide the generation of these modeled surfaces were:

- *Primary PM-based modeling approach (Pri-PM)*: This modeling approach simulates air quality scenarios of interest by preferentially adjusting direct (i.e., primary, directly-emitted) PM emissions. As such, the changes in PM<sub>2.5</sub> tend to be more localized near the direct emissions sources of PM. In locations for which air quality scenarios cannot be simulated by adjusting modeled primary emissions alone, SO<sub>2</sub> and NO<sub>x</sub> precursor emissions are additionally adjusted to simulate changes in secondarily formed PM<sub>2.5</sub>.
- *Secondary PM-based modeling approach (Sec-PM)*: This modeling approach simulates air quality scenarios of interest by preferentially adjusting SO<sub>2</sub> and NO<sub>x</sub> precursor emissions to simulate changes in secondarily formed PM<sub>2.5</sub>. In this case, the reductions in PM<sub>2.5</sub> tend to be more evenly spread across a study area. In locations for which air quality scenarios cannot be simulated by adjusting precursor emissions alone, a proportional adjustment of air quality is subsequently applied.

The air quality surfaces generated using these two approaches are not additive. Rather, they should be viewed as reflecting two different broad strategies for adjusting ambient PM<sub>2.5</sub> levels.

In addition, we also employed linear interpolation and extrapolation to simulate air quality under two additional alternative annual standard levels, 11.0 and 9.0 µg/m<sup>3</sup>, respectively (section 3.3.1 of the PA, Figure 3-11). Interpolation and extrapolation were only performed for grid cells in the subset of 30 urban study areas where the annual standard was controlling in both Pri-PM and Sec-PM simulated air quality scenarios of both 12/35 and 10/30 standard combinations. The interpolation and extrapolation were completed at the grid-cell level based on values simulated using hybrid air quality modeling to just meet the current annual standard of 12.0 µg/m<sup>3</sup> and alternative annual standard of 10.0 µg/m<sup>3</sup> (section 3.3.1 of the PA, Figure 3-11). A similar linear extrapolation/interpolation was not conducted for additional 24-hr standards due to the weaker relationship between the 98<sup>th</sup> percentile of 24-hr PM<sub>2.5</sub> concentrations, which are most relevant for simulating air quality that just meets the 24-hour standard, and the concentrations comprising the middle portion of the PM<sub>2.5</sub> air quality distribution, which are most relevant for estimating risks based on information from epidemiologic studies (i.e., discussed further in sections 3.1.2 and 3.2.3.2 in the PA).

The sections below provide more detailed information on the air quality modeling approach used to adjust air quality to simulate just meeting the current or alternative primary



PM<sub>2.5</sub> standards. Tables containing PM<sub>2.5</sub> DVs for the air quality projections can be found in section C.4.

#### **C.1.4.1 Overview of the Air Quality Modeling Approach**

To inform risk calculations, recent PM<sub>2.5</sub> measurements were analyzed to characterize the magnitude and spatial distribution of PM<sub>2.5</sub> concentrations. These data were then coupled with air quality modeling data to project ambient air quality levels corresponding to just meeting the existing and alternative PM<sub>2.5</sub> NAAQS<sup>10</sup> in specific areas. An overview of the approach is provided in Figure C-6. The process starts by acquiring PM<sub>2.5</sub> monitoring data from EPA's Air Quality System (AQS)<sup>11</sup> and simulating PM<sub>2.5</sub> concentrations with the Community Multiscale Air Quality (CMAQ)<sup>12</sup> model for base case and emission-sensitivity scenarios (Figure C-6, Box 1). The monitored and modeled data are then fused using the Downscaler model and the Software for Model Attainment Test-Community Edition (SMAT-CE)<sup>13</sup> to develop a baseline spatial field of PM<sub>2.5</sub> concentrations and relative response factors (RRFs) for projecting PM<sub>2.5</sub> concentrations, respectively (Figure C-6, Box 2). PM<sub>2.5</sub> concentrations are projected in two main steps using output from Downscaler and SMAT-CE (Figure C-6, Box 3). First, the PM<sub>2.5</sub> concentrations measured at monitoring sites in an area are iteratively projected using the RRFs to identify the percent change in anthropogenic emissions required for the highest monitored DV in the area to just meet the controlling standard. Second, gridded spatial fields of PM<sub>2.5</sub> concentrations are projected using the area-specific percent emission change<sup>14</sup> that corresponds to just meeting the standard at the controlling ambient data site. Additional details on the method are provided in (Kelly et al., 2019a; application of the method to the PM NAAQS risk assessment is described in the remainder of this appendix.

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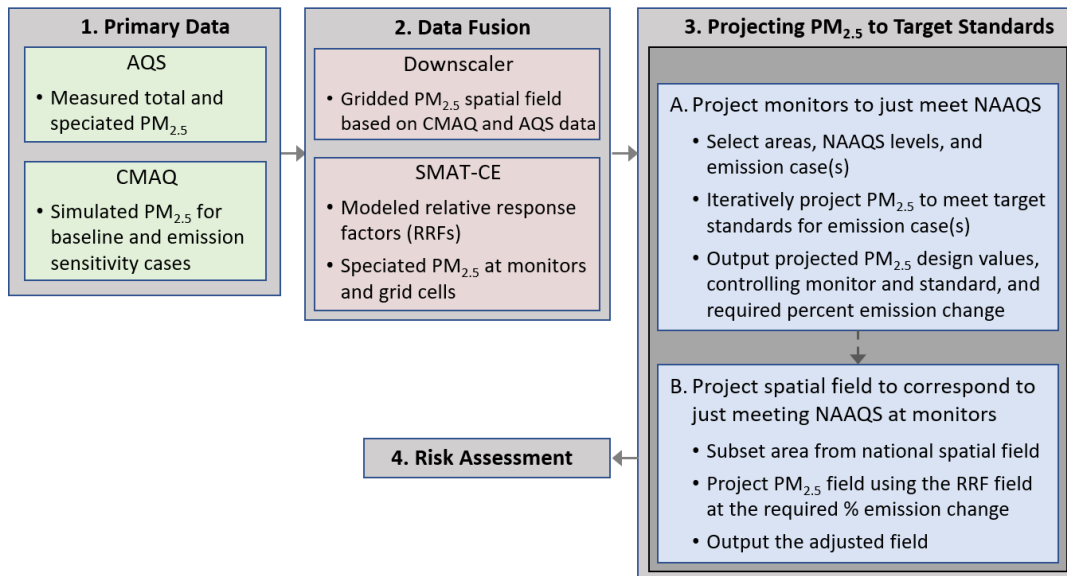
<sup>10</sup> The phrase, "just meeting the PM<sub>2.5</sub> NAAQS" is defined as the conditions where the highest design value (DV) for the controlling standard in the area equals the existing or alternative NAAQS level under consideration. DVs are statistics used in judging attainment of the NAAQS ([www.epa.gov/air-trends/air-quality-design-values](http://www.epa.gov/air-trends/air-quality-design-values)).

<sup>11</sup> [www.epa.gov/aqs](http://www.epa.gov/aqs)

<sup>12</sup> [www.epa.gov/cmaq](http://www.epa.gov/cmaq)

<sup>13</sup> [www.epa.gov/scram/photochemical-modeling-tools](http://www.epa.gov/scram/photochemical-modeling-tools)

<sup>14</sup> Scenarios based on a statistical projection approach were also developed for certain cases as discussed below.



**Figure C-6. Overview of the system for projecting PM<sub>2.5</sub> concentrations to correspond to just meeting NAAQS.** See section C.1.4.6 and Kelly et al., 2019a for more details.

#### C.1.4.2 PM<sub>2.5</sub> Monitoring Data and Area Selection

The 2014-2016 DV period was the most recent period having a complete set of total and speciated PM<sub>2.5</sub> observations available at the time of the study. PM<sub>2.5</sub> concentrations from the 2014-2016 DV period were used in selecting study areas and as the starting point for air quality projections (Figure C-6, Box 1, “AQS”). Total and speciated PM<sub>2.5</sub> concentrations for the 2014-2016 DV period were acquired from AQS. For sites in Los Angeles and Chicago, DVs were invalid during the 2014-2016 period. Los Angeles and Chicago have large populations, recent valid DVs for sites in Los Angeles are above existing standards, and Chicago is part of a CBSA that includes sites with valid 2014-2016 DVs in Indiana. For these reasons, invalid data for sites in these areas were replaced with valid data from other recent periods to enable DVs to be approximated for inclusion in the assessment. Specifically, for sites in Los Angeles and Orange Counties in California, observations from April – October 2014 were replaced with observations from the same months in 2013. For sites in Cook, DuPage, Kane, McHenry, and Will Counties in Illinois, observations from January to mid-July 2014 were replaced with observations from the same months in 2015.

Of the 56 areas initially identified as above the 10/30 selection threshold<sup>15</sup>, DVs for seven areas<sup>16</sup> appeared to meet the threshold due to the influence of wildfires. The influence of

<sup>15</sup> “10/30” indicates an annual standard level of 10 µg/ m<sup>3</sup> and a 24-hr standard level of 3 µg m<sup>-3</sup>

<sup>16</sup> Butte-Silver Bow, MT; Helena, MT; Kalispell, MT; Knoxville, TN; Medford, OR; Missoula, MT; and Yakima, WA

wildfires on DVs for these areas was estimated in part by recalculating 2014-2016 DVs with days removed that were clearly associated with summertime wildfires in the northwest. Since wildfire influence is often excluded when judging NAAQS attainment, these seven areas were excluded from further consideration. Additionally, the Eugene, OR CBSA was excluded. One monitor in the Eugene CBSA has a 24-hr 2014-2016 DV slightly above the 10/30 selection threshold<sup>17</sup>, but the monitor is in a small valley in Oakridge with very local high concentrations of PM<sub>2.5</sub> in winter that are distinct from conditions in the broader CBSA. Finally, the Phoenix-Mesa-Scottsdale, AZ CBSA was excluded. This CBSA had one monitor slightly above the 10/30 DV threshold<sup>18</sup>, but projecting concentrations for the CBSA was judged to be relatively uncertain because the annual DV is invalid at the only site that exceeded the threshold and the 24-hr DV is just above the threshold.

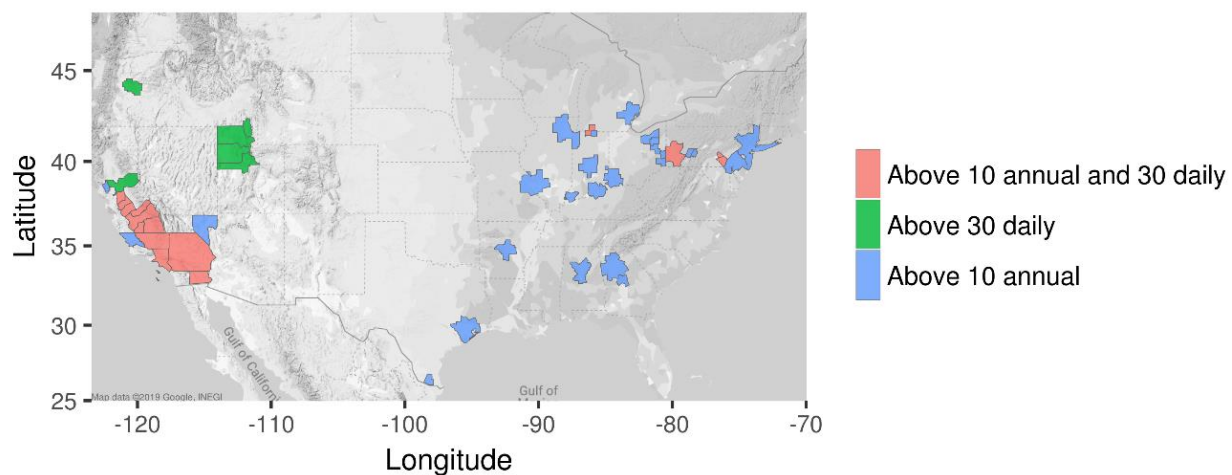
The remaining 47 CBSAs were selected for the risk assessment. These areas are shown in Figure C-7. The maximum 2014-2016 DVs and associated sites for each CBSA are provided in Table C-3, and the counties associated with the CBSAs are listed in Table C-4. DVs were calculated to an extra digit of precision for the air quality projections compared with official DVs. This approach is consistent with DV calculations in previous air quality projections (e.g., USEPA, 2012<sup>19</sup>) and provides a precise target for the iterative projection calculations.

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<sup>17</sup> The 410392013 monitor in Oakridge has a 24-hr 2014-2016 DV of 31  $\mu\text{g m}^{-3}$

<sup>18</sup> The 040213015 monitor in the Phoenix-Mesa-Scottsdale, AZ CBSA has 24-hr 2014-2016 DV of 31  $\mu\text{g m}^{-3}$

<sup>19</sup> USEPA (2012) Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division, Research Triangle Park, NC 27711. EPA-452/R-12-005 Available: <https://www3.epa.gov/ttn/ecas/regdata/RIAs/finalria.pdf>



**Figure C-7. CBSAs selected for the risk assessment.** Colors indicate whether the maximum 2014-2016 DVs in the CBSA are above the annual ( $10 \mu\text{g}/\text{m}^3$ ) and/or 24-hr ( $30 \mu\text{g}/\text{m}^3$ ) selection criteria.

**Table C-3. Maximum annual and 24-hr PM<sub>2.5</sub> DVs for 2014-2016 and associated sites for selected CBSAs.**

CBSA Name	# of Sites	Annual Max Site	Annual Max 14-16 DV	24-hr Max Site	24-hr Max 14-16 DV
Akron, OH	2	391530017	10.99	391530017	23.7
Altoona, PA	1	420130801	10.11	420130801	23.8
Atlanta-Sandy Springs-Roswell, GA	6	131210039	10.38	131210039	19.7
Bakersfield, CA	5	060290016	18.45	060290010	70.0
Birmingham-Hoover, AL	4	010732059	11.25	010730023	22.8
Canton-Massillon, OH	2	391510017	10.81	391510017	23.7
Chicago-Naperville-Elgin, IL-IN-WI <sup>a</sup>	22	170313103	11.10	170310057	26.8
Cincinnati, OH-KY-IN	9	390610014	10.70	390170020	24.2
Cleveland-Elyria, OH	8	390350065	12.17	390350038	25.0
Detroit-Warren-Dearborn, MI	11	261630033	11.30	261630033	26.8
El Centro, CA	3	060250005	12.63	060250005	33.5
Elkhart-Goshen, IN	1	180390008	10.24	180390008	28.6
Evansville, IN-KY	4	181630023	10.11	181630016	22.0
Fresno, CA	4	060195001	14.08	060190011	53.8
Hanford-Corcoran, CA	2	060310004	21.98	060310004	72.0
Houston-The Woodlands-Sugar Land, TX	4	482011035	11.19	482011035	22.4
Indianapolis-Carmel-Anderson, IN	7	180970087	11.44	180970043	26.0
Johnstown, PA	1	420210011	10.68	420210011	25.8
Lancaster, PA	2	420710012	12.83	420710012	32.7
Las Vegas-Henderson-Paradise, NV	4	320030561	10.28	320030561	24.5
Lebanon, PA	1	420750100	11.20	420750100	31.4
Little Rock-North Little Rock-Conway, AR	2	051191008	10.27	051191008	21.7
Logan, UT-ID	1	490050007	6.95	490050007	34.0
Los Angeles-Long Beach-Anaheim, CA <sup>a</sup>	9	060371103	12.38	060371103	32.8
Louisville/Jefferson County, KY-IN	7	180190006	10.64	180190006	23.9
Macon, GA	2	130210007	10.13	130210007	21.2
Madera, CA	1	060392010	13.30	060392010	45.1
McAllen-Edinburg-Mission, TX	1	482150043	10.09	482150043	25.0
Merced, CA	2	060470003	11.81	060472510	39.8
Modesto, CA	2	060990006	13.02	060990006	45.7
Napa, CA	1	060550003	10.36	060550003	25.1
New York-Newark-Jersey City, NY-NJ-PA	17	360610128	10.20	340030003	24.5
Ogden-Clearfield, UT	3	490570002	8.99	490110004	32.6
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	10	420450002	11.46	421010055	27.5
Pittsburgh, PA	10	420030064	12.82	420030064	35.8
Prineville, OR	1	410130100	8.60	410130100	37.6
Provo-Orem, UT	3	490494001	7.74	490494001	30.9
Riverside-San Bernardino-Ontario, CA	2	060658005	14.48	060658005	43.2
Sacramento--Roseville--Arden-Arcade, CA	6	060670006	9.31	060670006	31.4
Salt Lake City, UT	3	490353006	7.62	490353010	41.5
San Luis Obispo-Paso Robles-Arroyo Grande, CA	3	060792007	10.70	060792007	25.9

<b>CBSA Name</b>	<b># of Sites</b>	<b>Annual Max Site</b>	<b>Annual Max 14-16 DV</b>	<b>24-hr Max Site</b>	<b>24-hr Max 14-16 DV</b>
South Bend-Mishawaka, IN-MI	1	181410015	10.45	181410015	32.5
St. Louis, MO-IL	6	290990019	10.12	295100007	23.7
Stockton-Lodi, CA	2	060771002	12.23	060771002	38.7
Visalia-Porterville, CA	1	061072002	16.23	061072002	54.0
Weirton-Steubenville, WV-OH	4	390810017	11.75	390810017	27.2
Wheeling, WV-OH	2	540511002	10.24	540511002	22.5
<sup>a</sup> DVs for Chicago-Naperville-Elgin, IL-IN-WI and Los Angeles-Long Beach-Anaheim, CA were approximated as described in section C.1.4.2.					

**Table C-4. Counties associated with selected CBSAs**

<b>CBSA Name</b>	<b>Associated Counties</b>
Akron, OH	Portage, Summit
Altoona, PA	Blair
Atlanta-Sandy Springs-Roswell, GA	Barrow, Bartow, Butts, Carroll, Cherokee, Clayton, Cobb, Coweta, Dawson, DeKalb, Douglas, Fayette, Forsyth, Fulton, Gwinnett, Haralson, Heard, Henry, Jasper, Lamar, Meriwether, Morgan, Newton, Paulding, Pickens, Pike, Rockdale, Spalding, and Walton
Bakersfield, CA	Kern
Birmingham-Hoover, AL	Bibb, Blount, Chilton, Jefferson, St. Clair, Shelby, and Walker
Canton-Massillon, OH	Carroll, Stark
Chicago-Naperville-Elgin, IL-IN-WI	Cook, DeKalb, DuPage, Grundy, Kane, Kendall, Lake, McHenry, Will, Jasper, Lake, Newton, Porter, and Kenosha
Cincinnati, OH-KY-IN	Dearborn, Ohio, Union, Boone, Bracken, Campbell, Gallatin, Grant, Kenton, Pendleton, Brown, Butler, Clermont, Hamilton, and Warren
Cleveland-Elyria, OH	Cuyahoga, Geauga, Lake, Lorain, and Medina
Detroit-Warren-Dearborn, MI	Lapeer, Livingston, Macomb, Oakland, St. Clair, and Wayne
El Centro, CA	Imperial
Elkhart-Goshen, IN	Elkhart
Evansville, IN-KY	Posey, Vanderburgh, Warrick, and Henderson
Fresno, CA	Fresno
Hanford-Corcoran, CA	Kings
Houston-The Woodlands-Sugar Land, TX	Austin, Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller
Indianapolis-Carmel-Anderson, IN	Boone, Brown, Hamilton, Hancock, Hendricks, Johnson, Madison, Marion, Morgan, Putnam, and Shelby
Johnstown, PA	Cambria
Lancaster, PA	Lancaster
Las Vegas-Henderson-Paradise, NV	Clark
Lebanon, PA	Lebanon
Little Rock-North Little Rock-Conway, AR	Faulkner, Grant, Lonoke, Perry, Pulaski, and Saline

<b>CBSA Name</b>	<b>Associated Counties</b>
Logan, UT-ID	Franklin, Cache
Los Angeles-Long Beach-Anaheim, CA	Los Angeles and Orange
Louisville/Jefferson County, KY-IN	Clark, Floyd, Harrison, Scott, Washington, Bullitt, Henry, Jefferson, Oldham, Shelby, Spencer, and Trimble
Macon, GA	Bibb, Crawford, Jones, Monroe, and Twiggs
Madera, CA	Madera
McAllen-Edinburg-Mission, TX	Hidalgo
Merced, CA	Merced
Modesto, CA	Stanislaus
Napa, CA	Napa
New York-Newark-Jersey City, NY-NJ-PA	Bergen, Essex, Hudson, Hunterdon, Middlesex, Monmouth, Morris, Ocean, Passaic, Somerset, Sussex, Union, Bronx, Dutchess, Kings, Nassau, New York, Orange, Putnam, Queens, Richmond, Rockland, Suffolk, Westchester, and Pike
Ogden-Clearfield, UT	Box Elder, Davis, Morgan, and Weber
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	New Castle, Cecil, Burlington, Camden, Gloucester, Salem, Bucks, Chester, Delaware, Montgomery, and Philadelphia
Pittsburgh, PA	Allegheny, Armstrong, Beaver, Butler, Fayette, Washington, and Westmoreland
Prineville, OR	Crook
Provo-Orem, UT	Juab and Utah
Riverside-San Bernardino-Ontario, CA	Riverside and San Bernardino
Sacramento--Roseville--Arden-Arcade, CA	El Dorado, Placer, Sacramento, and Yolo
Salt Lake City, UT	Salt Lake, and Tooele
San Luis Obispo-Paso Robles-Arroyo Grande, CA	San Luis Obispo
South Bend-Mishawaka, IN-MI	St. Joseph and Cass
St. Louis, MO-IL	Bond, Calhoun, Clinton, Jersey, Macoupin, Madison, Monroe, St. Clair, Franklin, Jefferson, Lincoln, St. Charles, St. Louis, Warren, and St. Louis city
Stockton-Lodi, CA	San Joaquin
Visalia-Porterville, CA	Tulare
Weirton-Steubenville, WV-OH	Jefferson, Brooke, and Hancock
Wheeling, WV-OH	Belmont, Marshall, and Ohio

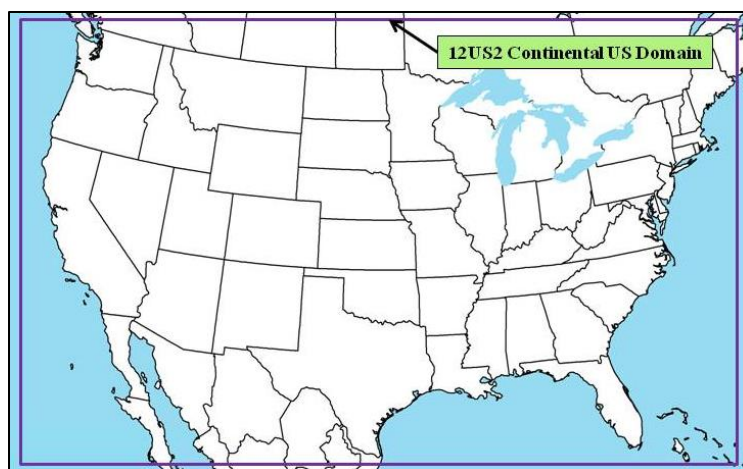
#### **C.1.4.3 Air Quality Modeling**

Air quality modeling was conducted using version 5.2.1 of the CMAQ modeling system (Appel, 2018, Pye et al., 2018) to develop a continuous national field of PM<sub>2.5</sub> concentrations and estimates of how concentrations would respond to changes in PM<sub>2.5</sub> and PM<sub>2.5</sub> precursor emissions (Figure C-6, “CMAQ”). The CMAQ modeling domain (Figure C-9) covered the contiguous U.S. with 12 km horizontal resolution and 35 vertical layers. Since 2015 was the

most recent modeling platform available at the time of the study and represents the central year of the 2014-2016 DV period, 2015 was selected as the baseline modeling year for the PM<sub>2.5</sub> projections. A single modeling year was used due to the time and resources needed to conduct photochemical grid modeling, and because model inputs for the 2016 period were not available at the time of the study.

Information on the CMAQ model configuration for the 2015 modeling is provided in Table C-5. The 2015 model simulation and its evaluation against network measurements of speciated and total PM<sub>2.5</sub> has been described in detail previously (Kelly et al., 2019b). Model performance statistics for PM<sub>2.5</sub> organic carbon, sulfate, and nitrate were generally similar to or improved compared to the performance for other recent national 12 km model simulations. One exception to the generally good model performance was identified for the Northwest region (OR, WA, and ID). Model performance statistics for this region were generally not as good as in our recent modeling due to issues related to unusually high fire influences in 2015, atmospheric mixing over sites near the Puget Sound, and other factors. However, model performance issues in the Northwest have minimal influence on the risk assessment, because only two of the 47 CBSAs are in the Northwest region (i.e., Prineville, OR and part of the Logan, UT-ID, CBSA). Also, the analysis uses ratios of model predictions rather than absolute modeled concentrations, and systematic biases associated with mixing height and fire impact estimates may largely cancel in the ratios. Moreover, fusion of monitor data with model predictions in developing PM<sub>2.5</sub> RRFs and the baseline concentration field helps mitigate the influence of biases in model predictions (as discussed below). Overall, the model performance evaluation (Kelly et al., 2019b) indicates that the 2015 CMAQ simulation provides concentration estimates that are generally as good or better than in other recent applications and are reliable for use in projecting PM<sub>2.5</sub> in the risk assessment. Model performance statistics for PM<sub>2.5</sub> by U.S. climate region and season are provided in Table C-6 and statistic definitions can be found in Table C-7.





**Figure C-9. CMAQ modeling domain.**

**Table C-5. CMAQ model configuration.**

Category	Description
Grid resolution	12 km horizontal; 35 vertical layers
Gas-phase chemistry	Carbon Bond 2006 (CB6r3)
Organic aerosol	Non-volatile treatment for primary organic aerosol; secondary organic aerosol from anthropogenic and biogenic sources
Inorganic aerosol	ISORROPIA II
NH <sub>3</sub> surface exchange	Bi-directional NH <sub>3</sub> surface exchange
Windblown dust emissions	Simulated online
Sea-spray emissions	Simulated online
Meteorology	Version 3.8 of Weather Research & Forecasting (WRF) Skamarock et al., 2005 model

**Table C-6. Model performance statistics<sup>20,21</sup> for PM<sub>2.5</sub> at AQS sites for the 2015 base case.**

Region <sup>21</sup>	Season	N	Avg. Obs. (µg m <sup>-3</sup> )	Avg. Mod. (µg m <sup>-3</sup> )	MB <sup>20</sup> (µg m <sup>-3</sup> )	NMB <sup>20</sup> (%)	RMSE <sup>20</sup> (µg m <sup>-3</sup> )	NME <sup>20</sup> (%)	r <sup>20</sup>
Northeast	Winter	13001	10.04	12.74	2.71	27.0	7.33	48.0	0.68
	Spring	13538	7.97	8.83	0.86	10.8	5.19	44.0	0.59
	Summer	13660	8.38	8.02	-0.36	-4.3	4.06	35.2	0.67
	Fall	13270	7.18	9.08	1.90	26.5	5.40	50.0	0.73
	Annual	53469	8.38	9.64	1.26	15.0	5.60	44.2	0.67
Southeast	Winter	11190	8.07	10.28	2.21	27.4	5.65	47.4	0.58
	Spring	11961	8.06	8.25	0.18	2.3	4.08	33.6	0.55
	Summer	11641	9.78	8.45	-1.33	-13.6	4.86	35.3	0.47
	Fall	11365	6.93	8.13	1.20	17.3	4.32	41.7	0.70

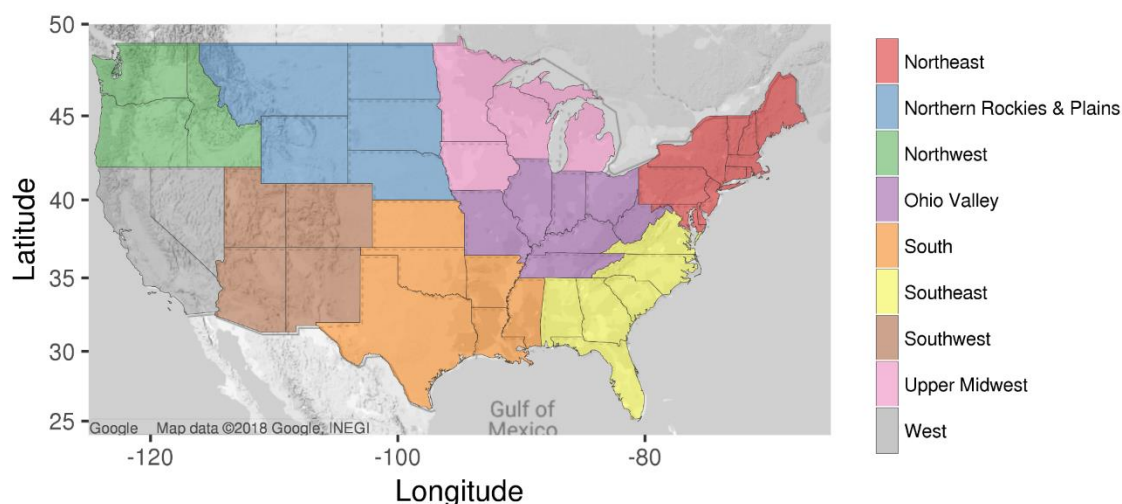
<sup>20</sup> See Table C-7 for definition of statistics.

<sup>21</sup> See Figure C-10 for definition of regions.

Region <sup>21</sup>	Season	N	Avg. Obs. ( $\mu\text{g m}^{-3}$ )	Avg. Mod. ( $\mu\text{g m}^{-3}$ )	MB <sup>20</sup> ( $\mu\text{g m}^{-3}$ )	NMB <sup>20</sup> (%)	RMSE <sup>20</sup> ( $\mu\text{g m}^{-3}$ )	NME <sup>20</sup> (%)	$r^{20}$
Ohio Valley	Annual	46157	8.22	8.76	0.54	6.6	4.75	39.1	0.55
	Winter	10323	9.49	11.60	2.10	22.1	5.75	43.2	0.63
	Spring	10867	8.90	9.85	0.95	10.6	4.60	36.3	0.65
	Summer	10714	10.95	10.56	-0.39	-3.6	5.55	34.3	0.55
	Fall	10568	8.41	10.96	2.54	30.2	6.23	47.1	0.65
	Annual	42472	9.44	10.73	1.29	13.6	5.56	39.8	0.59
Upper Midwest	Winter	6478	8.79	9.72	0.92	10.5	4.75	38.2	0.70
	Spring	6643	7.32	8.27	0.96	13.1	4.30	41.9	0.67
	Summer	6718	7.88	7.85	-0.03	-0.4	5.26	40.8	0.56
	Fall	6664	6.81	9.14	2.33	34.2	4.92	49.3	0.75
	Annual	26503	7.69	8.74	1.04	13.6	4.82	42.2	0.64
South	Winter	8041	7.53	10.13	2.60	34.5	11.81	56.6	0.36
	Spring	8369	8.08	7.12	-0.96	-11.9	4.24	36.3	0.51
	Summer	8440	10.80	8.31	-2.49	-23.0	6.04	40.3	0.34
	Fall	8340	7.55	7.99	0.44	5.9	3.76	35.5	0.63
	Annual	33190	8.50	8.37	-0.13	-1.6	7.15	41.8	0.34
Southwest	Winter	4911	7.46	7.90	0.45	6.0	6.50	55.9	0.52
	Spring	4998	4.88	5.88	1.00	20.6	3.60	48.4	0.44
	Summer	5069	6.12	4.85	-1.27	-20.8	4.15	43.1	0.59
	Fall	5091	5.31	5.90	0.59	11.1	4.35	52.2	0.49
	Annual	20069	5.93	6.12	0.19	3.2	4.77	50.2	0.52
N. Rockies & Plains	Winter	4987	5.57	3.60	-1.98	-35.5	6.80	63.4	0.23
	Spring	5380	4.57	5.00	0.44	9.6	29.58	61.6	0.20
	Summer	5260	9.98	7.68	-2.30	-23.1	17.61	57.4	0.57
	Fall	5010	5.57	5.42	-0.15	-2.7	5.65	56.4	0.44
	Annual	20637	6.43	5.45	-0.99	-15.3	18.06	59.2	0.34
Northwest	Winter	8994	7.90	7.82	-0.08	-1.0	10.20	80.9	0.25
	Spring	9306	5.02	6.84	1.82	36.2	6.65	71.5	0.48
	Summer	9993	9.17	11.12	1.95	21.2	32.40	67.7	0.46
	Fall	9868	7.03	9.39	2.37	33.7	15.33	78.3	0.31
	Annual	38161	7.31	8.85	1.55	21.2	19.26	74.3	0.43
West	Winter	10462	11.67	9.58	-2.08	-17.8	8.09	43.3	0.68
	Spring	10989	7.52	6.95	-0.57	-7.6	4.17	38.3	0.55
	Summer	11065	8.95	8.53	-0.43	-4.8	6.36	43.5	0.51
	Fall	10587	8.61	9.11	0.50	5.8	16.85	46.9	0.37
	Annual	43103	9.16	8.52	-0.64	-7.0	10.02	43.1	0.44

**Table C-7. Definition of statistics used in the CMAQ model performance evaluation.**

Statistic	Description
$MB (\mu g\ m^{-3}) = \frac{1}{n} \sum_{i=1}^n (P_i - O_i)$	Mean bias (MB) is defined as the average difference between predicted (P) and observed (O) concentrations for the total number of samples (n)
$RMSE (\mu g\ m^{-3}) = \sqrt{\sum_{i=1}^n (P_i - O_i)^2 / n}$	Root mean-squared error (RMSE)
$NMB (\%) = \frac{\sum_{i=1}^n (P_i - O_i)}{\sum_{i=1}^n O_i} \times 100$	The normalized mean bias (NMB) is defined as the sum of the difference between predictions and observations divided by the sum of observed values
$NME (\%) = \frac{\sum_{i=1}^n  P_i - O_i }{\sum_{i=1}^n O_i} \times 100$	Normalized mean error (NME) is defined as the sum of the absolute value of the difference between predictions and observations divided by the sum of observed values
$r = \frac{\sum_{i=1}^n (P_i - \bar{P})(O_i - \bar{O})}{\sqrt{\sum_{i=1}^n (P_i - \bar{P})^2} \sqrt{\sum_{i=1}^n (O_i - \bar{O})^2}}$	Pearson correlation coefficient



**Figure C-10. U.S. climate regions<sup>22</sup> used in the CMAQ model performance evaluation.**

In addition to the national model performance evaluation just described, CMAQ predictions of PM<sub>2.5</sub> concentrations were evaluated specifically for the CBSAs considered in the risk assessment. In Table C-8, model performance statistics are provided for predictions at monitors in the 47 CBSAs in 2015. Predictions generally agree well with observations over the full set of areas, with NMBs less than 10% in all seasons except Fall (NMB: 23.6%) and correlation coefficients greater than 0.60 in all seasons except Summer (r: 0.56). Model predictions are compared with observations by CBSA in Figure C-11, and NMBs at individual sites in the CBSAs are shown in Figure C-12. Predictions generally agree well with observations in the individual CBSAs, although underpredictions occurred in the Chicago-Naperville-Elgin

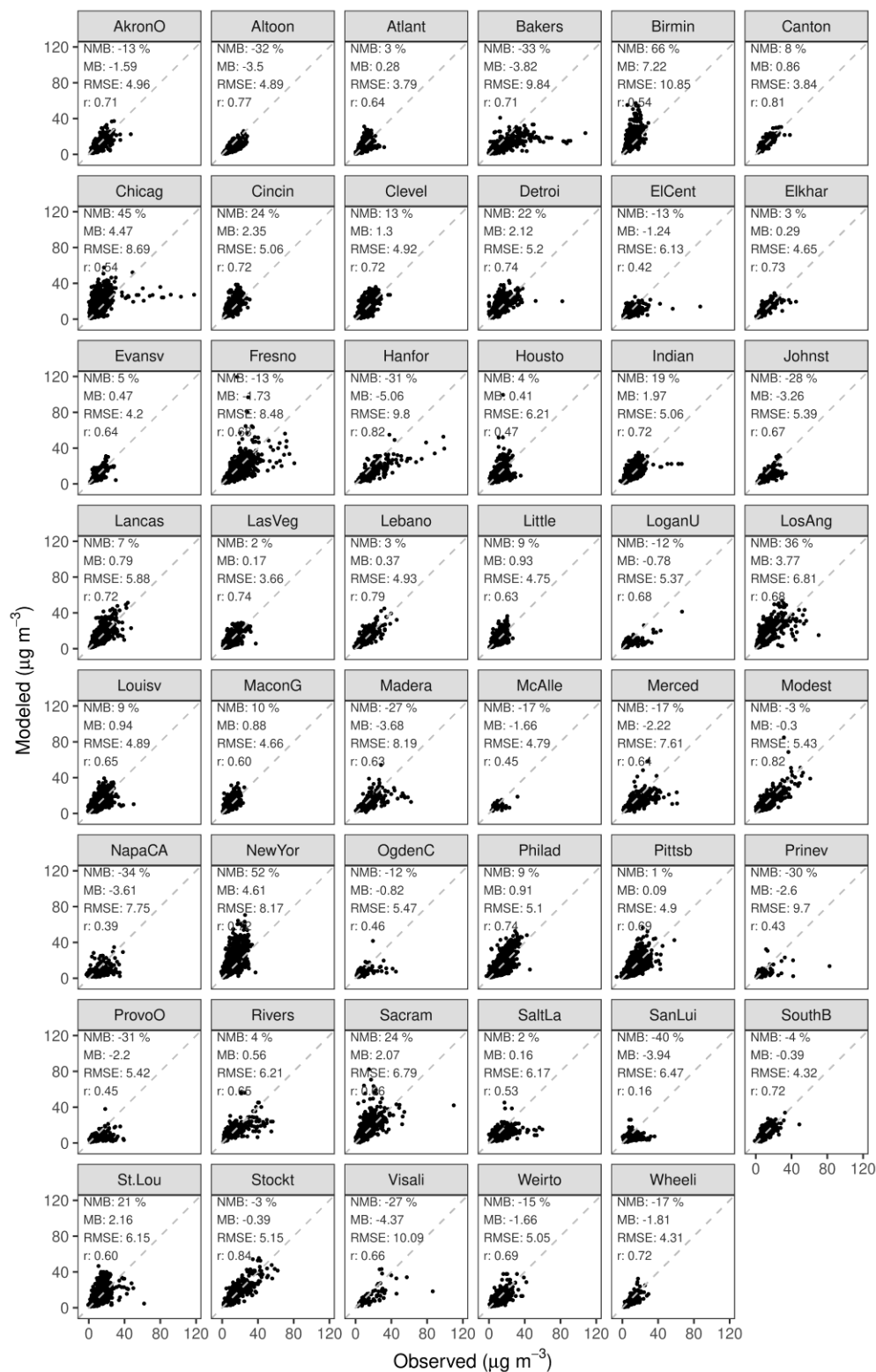
<sup>22</sup> <https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php>

CBSA when observed  $\text{PM}_{2.5}$  concentrations were  $> 40 \mu\text{g m}^{-3}$ . The high observed values in Chicago were associated with the 4<sup>th</sup> of July holiday, and the underpredictions on July 4<sup>th</sup> and 5<sup>th</sup> have small influence on the annual  $\text{PM}_{2.5}$  projections in the risk assessment. The NMB is highest for model predictions in the Birmingham-Hoover CBSA (NMB: 66%). As mentioned above, the effects of model bias are mitigated in part by use of relative response factors (i.e., the ratio model predictions from a base and emission control simulation is used in projecting  $\text{PM}_{2.5}$  concentrations, and some model bias likely cancels in the ratio). For the risk assessment projections, the key aspect of the CMAQ modeling is the spatial pattern of  $\text{PM}_{2.5}$  response to changes in emissions. The spatial response pattern was examined in the 47 CBSAs and found to be reasonable even in areas with relatively high bias, such as Birmingham. In Figure C-13, the spatial response pattern associated with the 10/30 projection case for the Birmingham-Hoover CBSA is compared for the proportional projection method and the primary PM projection case based on CMAQ modeling. Relatively high  $\text{PM}_{2.5}$  responsiveness occurred in the urban part of Birmingham and along arterial roads in the CMAQ-based approach. This spatial pattern is consistent with the location of  $\text{PM}_{2.5}$  emission sources in Birmingham and provides a realistic spatial response pattern despite the relatively high bias in the concentration predictions. Overall, both the national model performance evaluation and the evaluation for the 47 CBSAs of the risk assessment support use of the CMAQ modeling in this application.

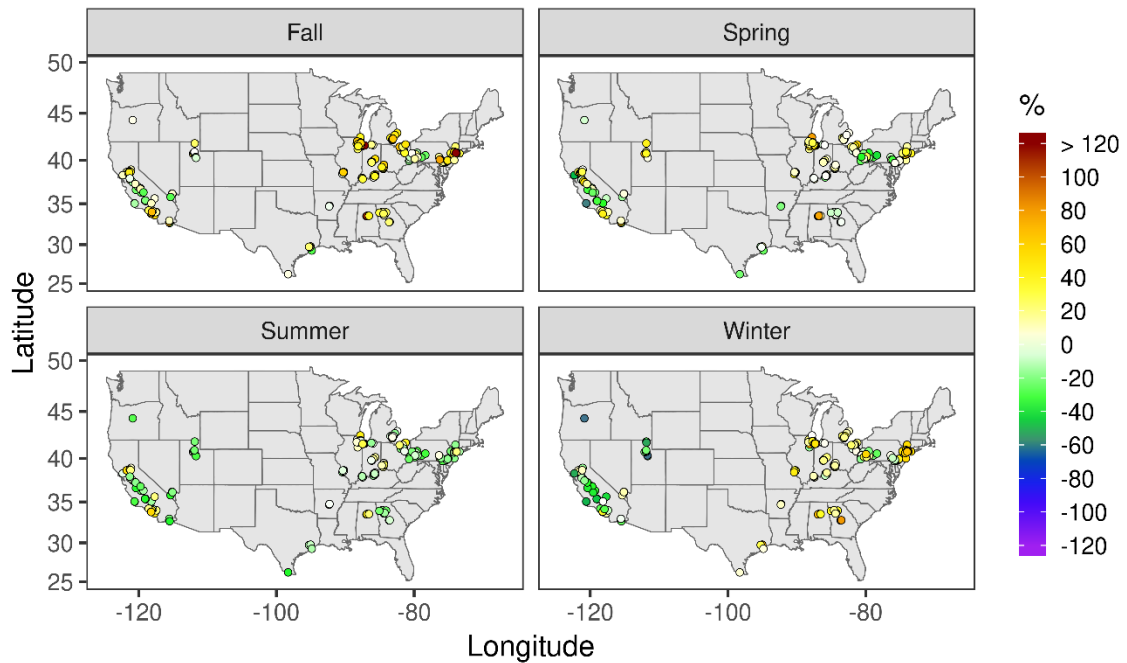
To inform  $\text{PM}_{2.5}$  projections, annual CMAQ modeling was conducted using the same configuration and inputs as the 2015 base case simulation but with anthropogenic emissions of primary  $\text{PM}_{2.5}$  or  $\text{NOx}$  and  $\text{SO}_2$  scaled by fixed percentages. Specifically, seven simulations were conducted with changes in anthropogenic  $\text{NOx}$  and  $\text{SO}_2$  emissions (i.e., combined  $\text{NOx}$  and  $\text{SO}_2$ , not separate  $\text{NOx}$  and  $\text{SO}_2$  simulations) of -100%, -75%, -50%, -25%, +25%, +50%, and +75%. Two simulations were conducted with changes in anthropogenic  $\text{PM}_{2.5}$  emissions of -50% and +50%. The sensitivity simulations were based on emission changes applied to all anthropogenic sources throughout the year. These “across-the-board” emission changes facilitate projecting the baseline concentrations to just meet a relatively wide range of standards in areas throughout the U.S. using a feasible number of national sensitivity simulations.

**Table C-8. Performance statistics for CMAQ predictions at monitoring sites in the 47 CBSAs considered in the risk assessment.**

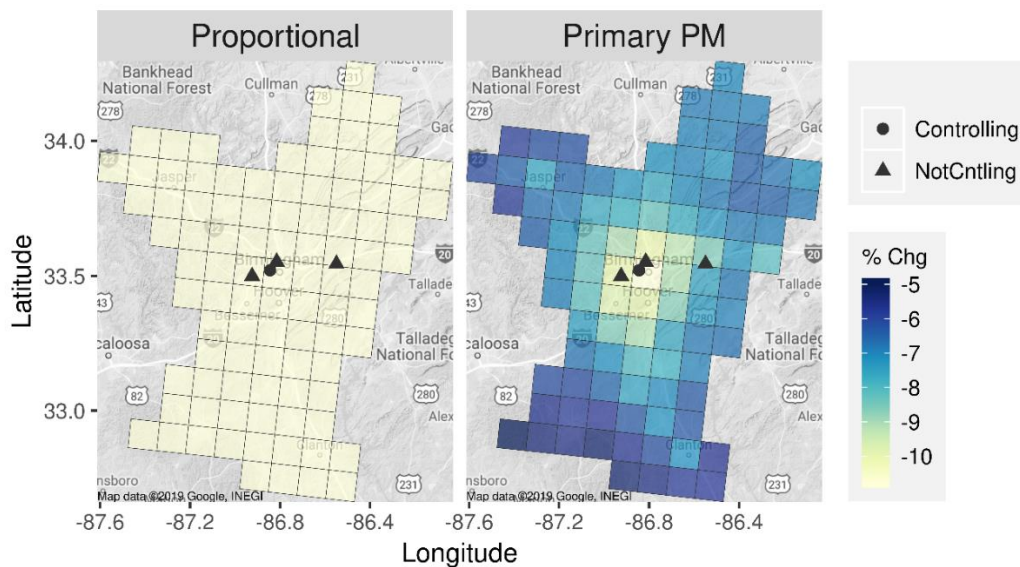
Season	Average Observed ( $\mu\text{g m}^{-3}$ )	Average Modeled ( $\mu\text{g m}^{-3}$ )	MB ( $\mu\text{g m}^{-3}$ )	NMB (%)	RMSE ( $\mu\text{g m}^{-3}$ )	NME (%)	<i>r</i>
Winter	12.40	13.45	1.05	8.5	8.03	42.4	0.61
Spring	9.17	9.94	0.77	8.4	5.15	38.6	0.62
Summer	10.35	10.08	-0.27	-2.6	5.51	34.6	0.56
Fall	9.00	11.11	2.12	23.6	6.26	45.6	0.67



**Figure C-11. Comparison of CMAQ predictions and observations at monitoring sites in the 47 CBSAs considered in the risk assessment.**



**Figure C-12. NMB for CMAQ PM<sub>2.5</sub> predictions at monitoring sites in the 47 CBSAs by season in 2015.**



**Figure C-13. Percent change in 2015 annual average PM<sub>2.5</sub> over the Birmingham CBSA associated with projecting 2014–2016 DVs at monitors to just meet an alternative NAAQS of 10/30 using the proportional projection method and the primary PM<sub>2.5</sub>, CMAQ-based projection method.**

The two emission sensitivity scenarios (primary PM<sub>2.5</sub> and NO<sub>x</sub> and SO<sub>2</sub>) were selected to span a wide range of possible PM<sub>2.5</sub> spatial response patterns. NO<sub>x</sub> and SO<sub>2</sub> emission changes influence concentrations of ammonium nitrate and ammonium sulfate, which are secondary pollutants that often have broad spatial distributions. Primary PM<sub>2.5</sub> emission changes have the greatest influence on PM<sub>2.5</sub> concentrations close to emission sources. The two distinctly different PM<sub>2.5</sub> response patterns for primary PM<sub>2.5</sub> and NO<sub>x</sub> and SO<sub>2</sub> emission changes enable PM<sub>2.5</sub> to be projected for a wide range of conditions. Projecting PM<sub>2.5</sub> for a wide range of conditions is desirable in this study because many PM<sub>2.5</sub> spatial response patterns can cause PM<sub>2.5</sub> concentrations to just meet NAAQS.

#### C.1.4.4 Relative Response Factors for PM<sub>2.5</sub> Projection

The 2015 base case and sensitivity modeling results were used to develop RRFs for projecting PM<sub>2.5</sub> concentrations to correspond to just meeting NAAQS (Figure C-6, Box 2, “SMAT-CE”). Baseline PM<sub>2.5</sub> concentrations are projected by multiplication with RRFs. The RRF for a PM<sub>2.5</sub> species is calculated as the ratio of the concentration in the sensitivity simulation to that in the base case:

$$RRF_{species} = \frac{C_{sensitivity,species}}{C_{base,species}} \quad (1)$$

where  $C_{sensitivity,species}$  is the concentration of the PM<sub>2.5</sub> species in the sensitivity simulation, and  $C_{base,species}$  is the concentration of the PM<sub>2.5</sub> species in the base case simulation. RRFs were calculated for each monitor, grid cell, calendar quarter, standard (annual or 24-hr), species, and sensitivity simulation using SMAT-CE version 1.2.1. RRFs are used in projecting air quality to help mitigate the influence of systematic biases in model predictions (National Resources Council, U.S. EPA, 2018a). More details on the RRF projection method are provided in EPA’s modeling guidance document (U.S. EPA, 2018a) and the user’s guide for the predecessor to the SMAT-CE software (Abt Associates, 2014).

To apply the RRF approach for the risk assessment projections, RRFs for total PM<sub>2.5</sub> were calculated from RRFs for the individual PM<sub>2.5</sub> species using observation-based estimates of PM<sub>2.5</sub> species concentrations in SMAT-CE output. Specifically, total PM<sub>2.5</sub> RRFs ( $RRF_{Tot,PM2.5}$ ) were calculated as the weighted average of the speciated RRFs using the observation-based species concentrations ( $C_{species}$ ) as weights:

$$RRF_{Tot,PM2.5} = \frac{\sum RRF_{species} C_{species}}{\sum C_{species}} \quad (2)$$

Total PM<sub>2.5</sub> RRFs were used to project base-case PM<sub>2.5</sub> concentrations as follows:

$$PM_{2.5,projected} = RRF_{Tot,PM2.5} PM_{2.5,base} \quad (3)$$

The species concentrations used in calculating the total PM<sub>2.5</sub> RRFs were generally based on application of the Sulfate, Adjusted Nitrate, Derived Water, Inferred Carbonaceous material balance approach (SANDWICH) (Frank, 2006) to measurements of PM<sub>2.5</sub> species



concentrations from the Chemical Speciation Network (CSN)<sup>23</sup> and the Interagency Monitoring of Protected Visual Environments (IMPROVE)<sup>24</sup> network. The SANDWICH method corrects for different artifacts in the measurements for PM<sub>2.5</sub> species and total PM<sub>2.5</sub>. An alternative approach to calculating total PM<sub>2.5</sub> RRFs was applied for monitors and grid cells in California due to factors including missing data at the Bakersfield speciation monitor<sup>25</sup> throughout 2014 and part of 2015. For projections in California, RRFs were calculated directly from the ratio of CMAQ PM<sub>2.5</sub> concentration predictions in the sensitivity simulation to the base simulation.

By default, PM<sub>2.5</sub> RRFs for the annual standard are calculated using average concentrations over all modeled days in the quarter, and RRFs for the 24-hr standard are calculated using average concentrations over days with the top 10% of modeled PM<sub>2.5</sub> concentration in the quarter. The default approach was generally followed here, with exceptions for counties in the San Joaquin Valley (SJV) of California and Utah. In these counties<sup>26</sup>, the average concentration over all days in the quarter was used to calculate RRFs for both the 24-hr and annual standards for sites with valid 24-hr and annual DVs. This approach was used to provide stability in projections of annual fields due the variability in the 24-hr and annual RRFs<sup>27</sup>. Also, RRFs were set to one<sup>28</sup> in the third quarter (July-September) for select counties in the San Joaquin Valley and Utah<sup>29</sup> to better reflect the seasonal nature of PM<sub>2.5</sub> in these areas (i.e., PM<sub>2.5</sub> concentrations are relatively high in winter).

RRFs were calculated for each combination of emission sensitivity simulation and the 2015 base case. RRFs corresponding to the percent change in emissions for each sensitivity simulation were then interpolated across the range of emission changes from -100 to +100% to facilitate iterative projections of PM<sub>2.5</sub> concentrations to the nearest percent emission change. PM<sub>2.5</sub> RRFs are shown in Figure C-14 and Figure C-15 as a function of changes in anthropogenic primary PM<sub>2.5</sub> and NO<sub>x</sub> and SO<sub>2</sub> emissions for monitors in the U.S. during the first and third

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<sup>23</sup> [www.epa.gov/amtic/chemical-speciation-network-csn](http://www.epa.gov/amtic/chemical-speciation-network-csn)

<sup>24</sup> <http://vista.cira.colostate.edu/Improve/>

<sup>25</sup> Site identification number: 060290014

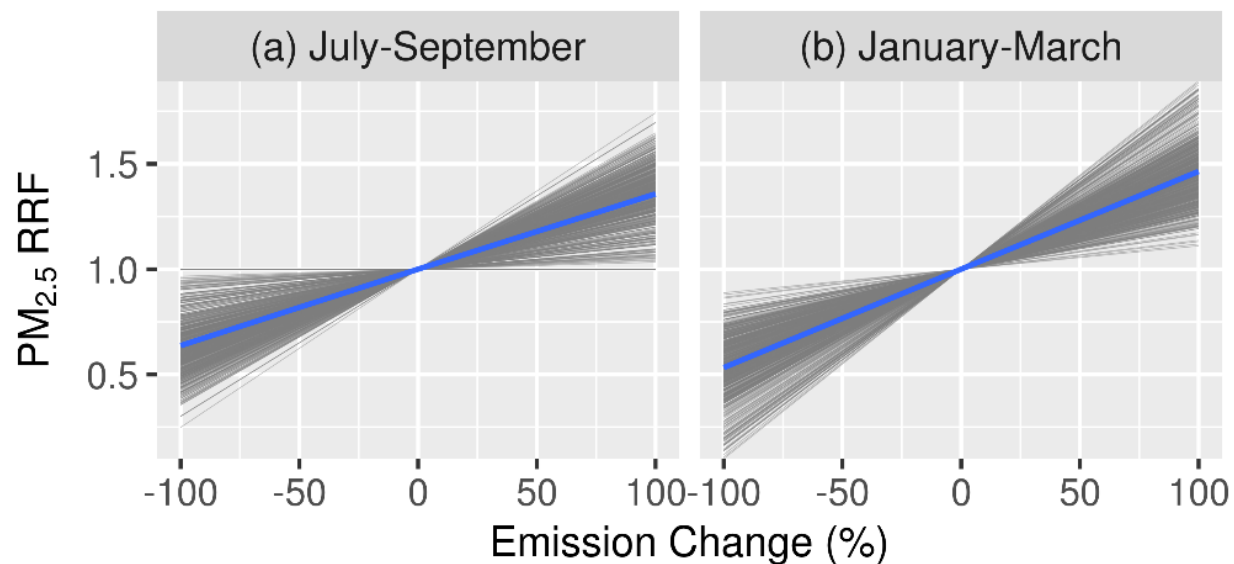
<sup>26</sup> SJV counties: Fresno, Stanislaus, Kern, Merced, Madera, Tulare, San Joaquin, and Kings; Utah counties: Cache, Box Elder, Davis, Morgan, Weber, Juab, Utah, Salt Lake, and Tooele.

<sup>27</sup> This variability is less of an issue in regional modeling applications where emission changes can be targeted to time periods of elevated PM<sub>2.5</sub> concentrations in the area.

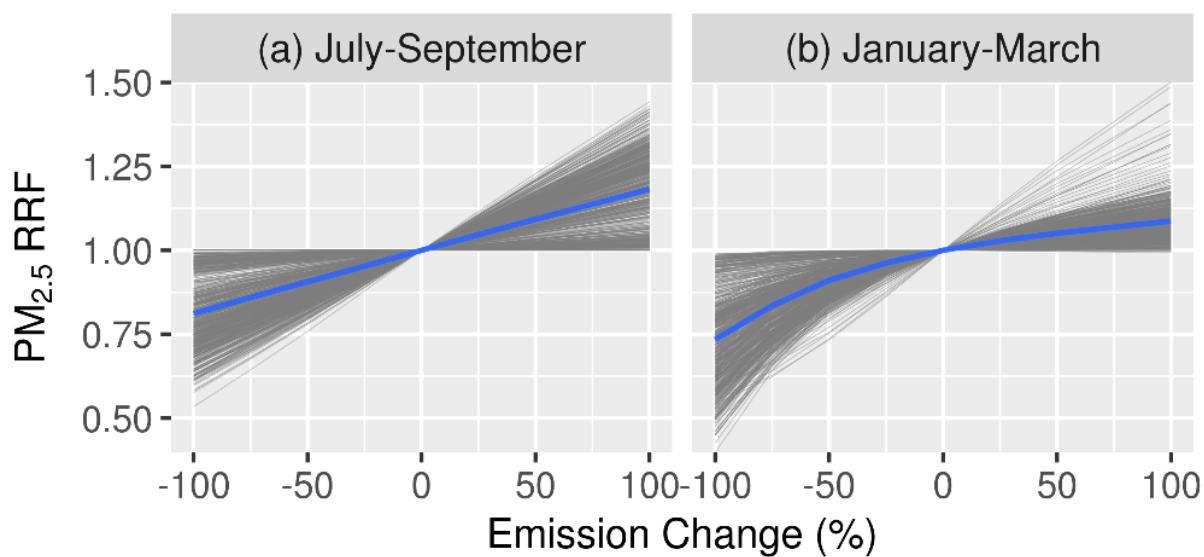
<sup>28</sup> When the RRF is 1, the projected concentration equals the base concentration (Equation 3).

<sup>29</sup> SJV counties: Fresno, Stanislaus, Kern, Merced, and Madera; Utah counties: Cache, Box Elder, Davis, Morgan, Weber, Juab, Utah, Salt Lake, and Tooele. This approach was not applied for Kings, Tulare, and San Joaquin counties in SJV because the percent exceedance of the annual standard was within 10% of the exceedance of the 24-hr standard suggesting that relatively uniform PM<sub>2.5</sub> concentrations occur throughout the year compared with the other SJV counties.

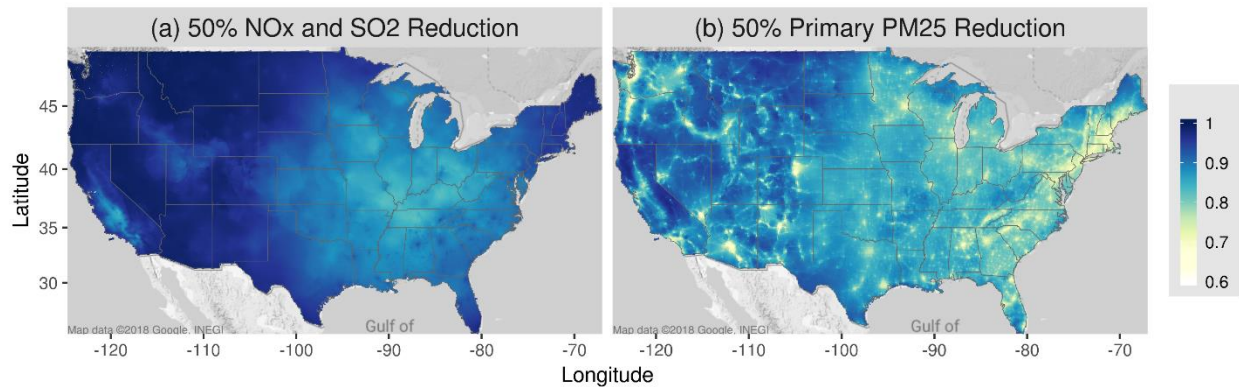
calendar quarters. Spatial fields of  $\text{PM}_{2.5}$  RRFs for 50% reductions in anthropogenic primary  $\text{PM}_{2.5}$  and  $\text{NO}_x$  and  $\text{SO}_2$  emissions are shown in Figure C-16.



**Figure C-14. Annual standard  $\text{PM}_{2.5}$  RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic primary  $\text{PM}_{2.5}$  emissions for monitoring sites in the contiguous U.S.**



**Figure C-15. Annual standard  $\text{PM}_{2.5}$  RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic  $\text{NO}_x$  and  $\text{SO}_2$  emissions for monitoring sites in the contiguous U.S.**



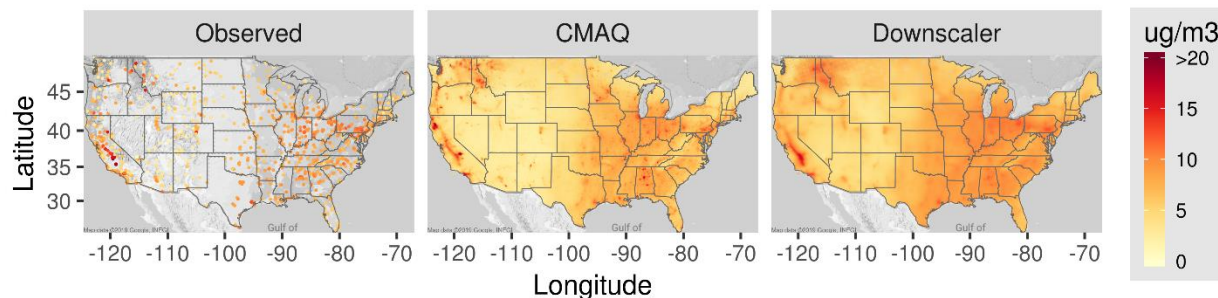
**Figure C-16. Annual average PM<sub>2.5</sub> RRFs at CMAQ grid-cell centers for 50% reductions in anthropogenic (a) NO<sub>x</sub> and SO<sub>2</sub> and (b) primary PM<sub>2.5</sub> emissions.**

#### **C.1.4.5 2015 PM<sub>2.5</sub> Concentration Fields**

To develop a baseline gridded PM<sub>2.5</sub> concentration field for projection with PM<sub>2.5</sub> RRFs, a Bayesian statistical model (i.e., Downscaler) was applied (Figure C-6, Box 2, “Downscaler”) (Berrocal et al., 2012). Downscaler makes predictions of PM<sub>2.5</sub> concentrations to a spatial field of receptor points using PM<sub>2.5</sub> monitoring data and CMAQ model predictions as inputs. Downscaler takes advantage of the accuracy of the monitoring data and the spatial coverage of the CMAQ predictions to develop new predictions of PM<sub>2.5</sub> concentration over the U.S.

The Downscaler model is routinely applied by U.S. EPA to predict 24-hr average PM<sub>2.5</sub> concentrations at the centroids of census tracts in the contiguous U.S. (U.S. EPA, 2018b). The model configuration used here is generally consistent with the previous applications, but here predictions were made to the centers of the CMAQ model grid cells rather than to census-tract centroids. Also, PM<sub>2.5</sub> measurements from the IMPROVE monitoring network were used in addition to measurements included in the AQS database. 24-hr average PM<sub>2.5</sub> concentrations were predicted for the 2015 period, and the 24-hr PM<sub>2.5</sub> fields were averaged to the quarterly periods of the PM<sub>2.5</sub> RRFs for use in projection.

Annual average PM<sub>2.5</sub> concentrations from the monitoring network and CMAQ simulation that were used in model fitting are shown in Figure C-17 along with the resulting Downscaler predictions. Cross-validation statistics are provided in Table C-9 based on comparisons of Downscaler predictions against the 10% of the observations that were randomly withheld from model fitting.



**Figure C-17. Annual average of the 2015 PM<sub>2.5</sub> observations and CMAQ predictions used in the Downscaler model, and the annual average of the Downscaler PM<sub>2.5</sub> predictions.**

**Table C-9. Cross-validation statistics associated with the 2015 Downscaler predictions.**

Number of Monitors	Mean Bias <sup>a</sup> ( $\mu\text{g m}^{-3}$ )	Root Mean Squared Error <sup>b</sup> ( $\mu\text{g m}^{-3}$ )	Mean Coverage <sup>c</sup>
1101	0.37	3.17	0.95
<sup>a</sup> The mean of all biases across the CV cases, where the bias of each prediction is the downscaler prediction minus the observed value. <sup>b</sup> The bias is squared for each CV prediction, then the square root of the mean of all squared biases across all CV predictions is obtained. <sup>c</sup> A value of 1 is assigned if the measured value lies in the 95 <sup>th</sup> percentile CI of the Downscaler prediction (the Downscaler prediction $\pm$ the Downscaler standard error), and 0 otherwise. This column is the mean of all those 0's and 1's.			

#### C.1.4.6 Projecting PM<sub>2.5</sub> to Just Meet the Standards

PM<sub>2.5</sub> was projected from baseline concentrations to levels corresponding to just meeting NAAQS using the monitoring data (section C.1.4.2), RRFs (section C.1.4.4), and baseline concentration fields (section C.1.4.5) described above. The projection was done in two steps as shown in Box 3 of Figure C-6. Projections were performed for the existing (12/35)<sup>30</sup> and alternative (10/30)<sup>31</sup> standards.

First, monitors in the CBSA of interest were identified, and concentrations from these monitors were subset from the national monitoring dataset. The measured concentrations were then projected using the corresponding PM<sub>2.5</sub> RRF. PM<sub>2.5</sub> DVs were calculated using the projected concentrations, and the difference between the maximum projected DV and target standard was determined. DV projections over the complete range of percent emission changes (-100 to 100%) were performed using bisection iteration until the difference between the

<sup>30</sup> Annual standard level of 12  $\mu\text{g m}^{-3}$  and 24-hr standard level of 35  $\mu\text{g m}^{-3}$

<sup>31</sup> Annual standard level of 10  $\mu\text{g m}^{-3}$  and 24-hr standard level of 30  $\mu\text{g m}^{-3}$

maximum projected DV in the CBSA and the standard level was zero or within the difference associated with a 1% emission change. Iterative projections of annual and 24-hr DVs were performed separately, and the controlling standard was determined as the standard requiring the greater percent emission change<sup>32</sup>. In cases where the emission change needed to just meet the target annual or 24-hr standard was outside of the  $\pm 100\%$  range, the standard could not be met using the modeled air quality scenarios. If neither the annual nor 24-hr standard could be just met with emission changes within  $\pm 100\%$ , then an alternative projection approach was used (discussed below).

Second, 2015 PM<sub>2.5</sub> concentration fields developed with Downscaler were projected according to the percent emission change required for the maximum projected DV to just meet the controlling standard. The projection was done by multiplying the gridded spatial fields of quarterly average PM<sub>2.5</sub> concentrations based on Downscaler modeling with the gridded spatial fields of quarterly PM<sub>2.5</sub> RRFs corresponding to the percent emission change required to just meet the controlling standard. The projected fields of quarterly average PM<sub>2.5</sub> concentrations were then averaged to produce the annual average projected field.

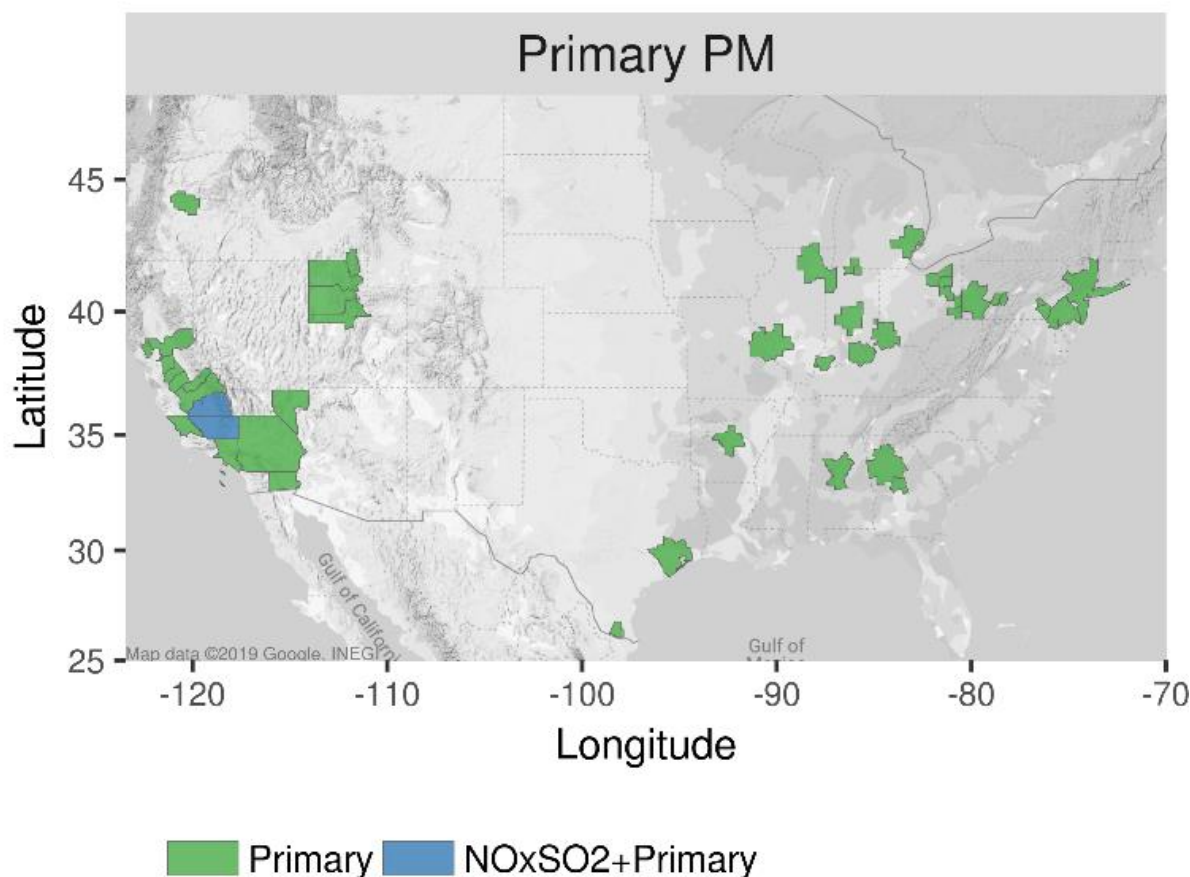
Since PM<sub>2.5</sub> concentrations can be projected in multiple ways to just meet a standard, projections were done for two scenarios that provide results for a range of PM<sub>2.5</sub> conditions. The first scenario is referred to as “Primary PM” or Pri-PM because projections were largely based on RRFs developed using CMAQ sensitivity simulations with primary PM<sub>2.5</sub> emission changes. For three CBSAs<sup>33</sup>, standards could not be met using primary PM<sub>2.5</sub> emission reductions alone. PM<sub>2.5</sub> concentrations were projected for these areas using a combination of primary PM<sub>2.5</sub> and NO<sub>x</sub> and SO<sub>2</sub> emission reductions in the Primary PM scenario<sup>34</sup> (Figure C-18).

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<sup>32</sup> Note that calculations are performed in terms of percent emission reduction. Therefore, in cases where DVs are projected to just meet standards greater than the baseline DVs, the required percent emission reduction is negative (i.e., an emission increase is required), and the smaller absolute percent emission change is selected as the controlling case. For example, the annual standard would be selected as controlling in a case where a 10% emission increase is needed to meet the annual standard and a 50% emission increase is needed to meet the 24-hr standard (because -10 is greater than -50).

<sup>33</sup> Bakersfield, Hanford-Corcoran, and Visalia-Porterville (all in California)

<sup>34</sup> This approach was applied by using RRFs from the NO<sub>x</sub> and SO<sub>2</sub> emission sensitivity simulations to eliminate a fraction of the difference between the maximum base DV and the standard level and then using RRFs from the primary PM<sub>2.5</sub> emission sensitivity simulations to eliminate the remainder of the difference. The fraction of the difference eliminated with NO<sub>x</sub> and SO<sub>2</sub> emission reductions was as follows: 0.4 for Bakersfield, 0.5 for Visalia-Porterville, and 0.6 for Hanford-Corcoran



**Figure C-18. Projection method used for each CBSA in the “Primary PM” projection case.**  
See text for details.

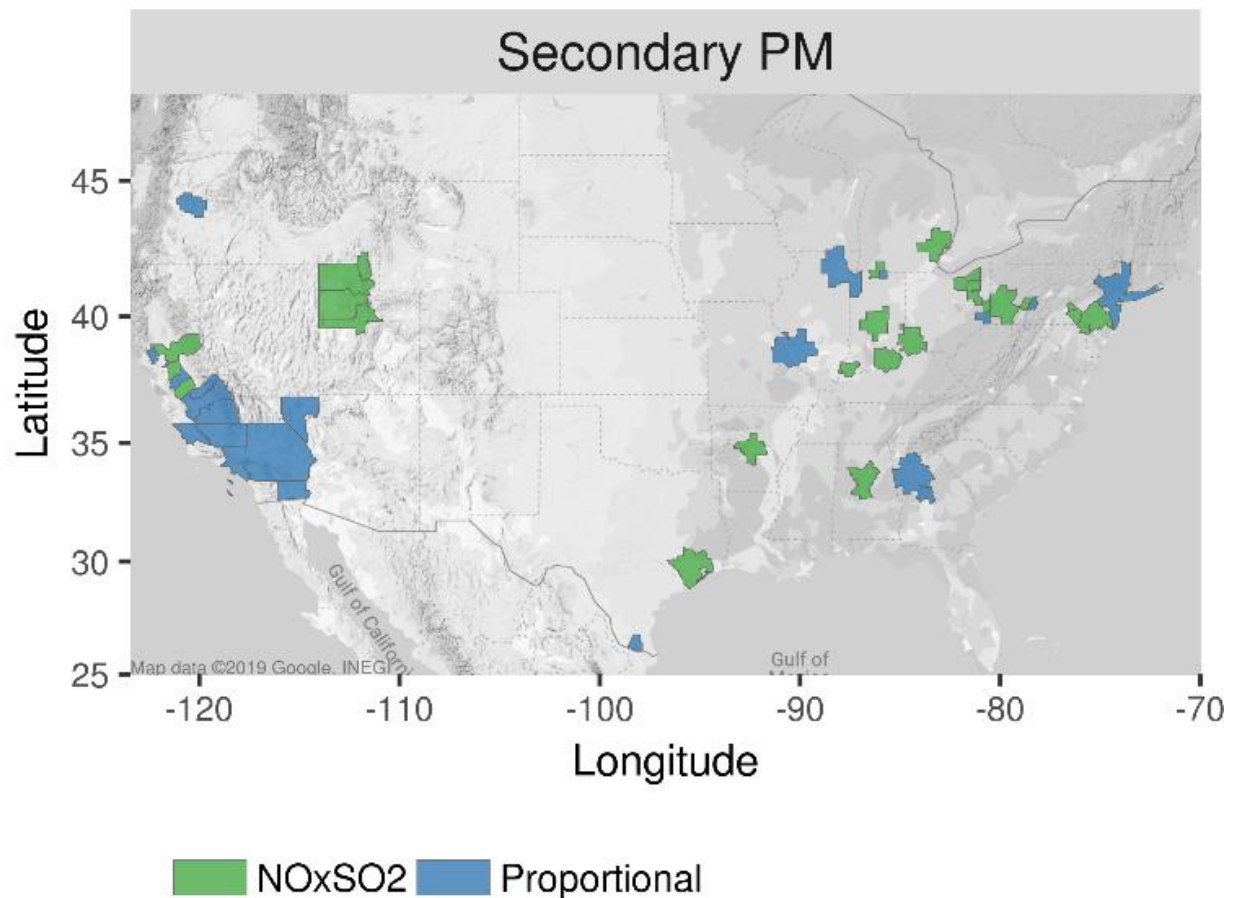
The second scenario is referred to as “Secondary PM” or Sec-PM because projections were largely based on RRFs developed using CMAQ modeling with NO<sub>x</sub> and SO<sub>2</sub> emission changes, which affect concentrations of secondary PM components such as ammonium nitrate and ammonium sulfate. For 22 CBSAs<sup>35</sup>, standards could not be just met using NO<sub>x</sub> and SO<sub>2</sub> emission changes alone. These areas were projected using the proportional scaling method<sup>36</sup> (Figure C-19). The proportional method was selected to gap-fill the Secondary PM case because

<sup>35</sup> Altoona, PA; Atlanta-Sandy Springs-Roswell, GA; Bakersfield, CA; Chicago-Naperville-Elgin, IL-IN-WI; El Centro, CA; Elkhart-Goshen, IN; Fresno, CA; Hanford-Corcoran, CA; Las Vegas-Henderson-Paradise, NV; Los Angeles-Long Beach-Anaheim, CA; Macon, GA; Madera, CA; McAllen-Edinburg-Mission, TX; Modesto, CA; Napa, CA; New York-Newark-Jersey City, NY-NJ-PA; Prineville, OR; Riverside-San Bernardino-Ontario, CA; St. Louis, MO-IL; San Luis Obispo-Paso Robles-Arroyo Grande, CA; Visalia-Porterville, CA; Wheeling, WV-OH

<sup>36</sup> In the proportional method, the spatial field is uniformly scaled by a fixed percentage that corresponds to the percent difference between the controlling standard level and maximum PM<sub>2.5</sub> DV for the controlling standard. The controlling standard (annual or 24-hr) is identified as the one with the greater percent difference between the maximum DV and the standard level.



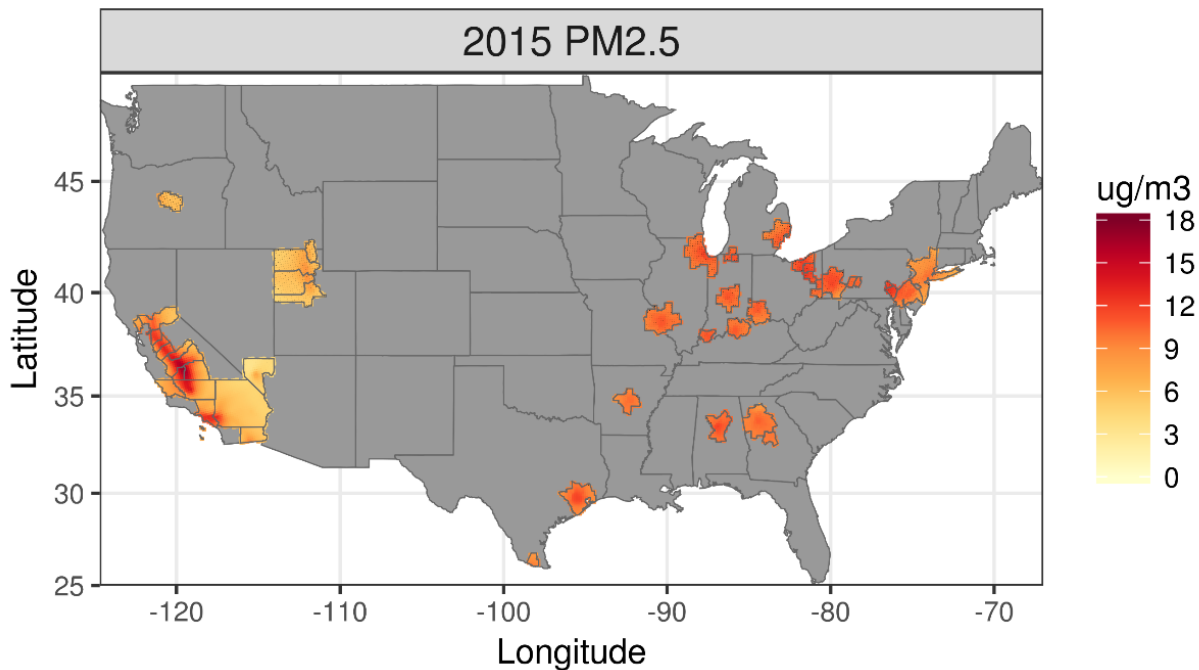
it is based on a spatially uniform percent change in  $PM_{2.5}$  over the area that is like the conceptually broad spatial response pattern of  $PM_{2.5}$  to changes in secondary  $PM_{2.5}$  components. The proportional method has been used previously in the Risk and Exposure Assessment for the 2012 PM NAAQS review (U.S. EPA, 2010).



**Figure C-19. Projection method used for each CBSA in the “Secondary PM” projection case.**

The baseline 2015 concentration in the 47 CBSAs is shown in Figure C-20. These concentrations are the same as those in Figure C-17 but are shown only for the CBSAs included in the projections. In Figure C-21, the difference in annual concentration projected for the 12/35 case and the 2015 baseline concentration is shown. The positive and negative differences reflect areas where concentrations were projected to higher and lower levels to just meet the standard, respectively. In Figure C-22, the difference between the annual concentration projected for the 10/30 case and the 2015 baseline concentration. Negative values indicate that concentrations

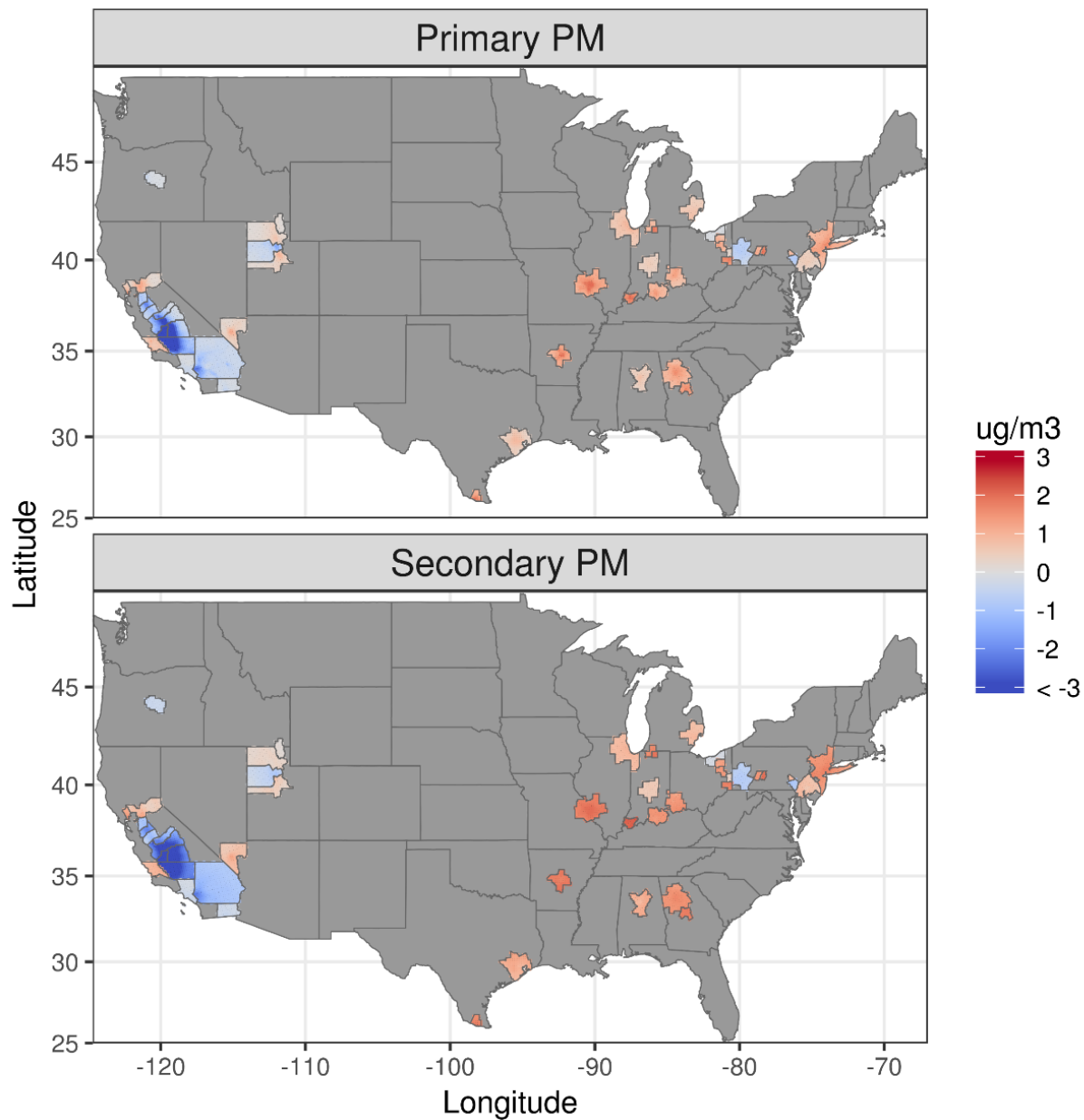
were projected to lower levels in all cases for the areas. The difference in projected concentrations for the 10/30 and 12/35 fields is shown in Figure C-23. Baseline and projected PM<sub>2.5</sub> DVs for monitors in the 47 CBSAs are provided in Table C-33, Table C-34, Table C-35, and Table C-36 in section C.4.<sup>37</sup>



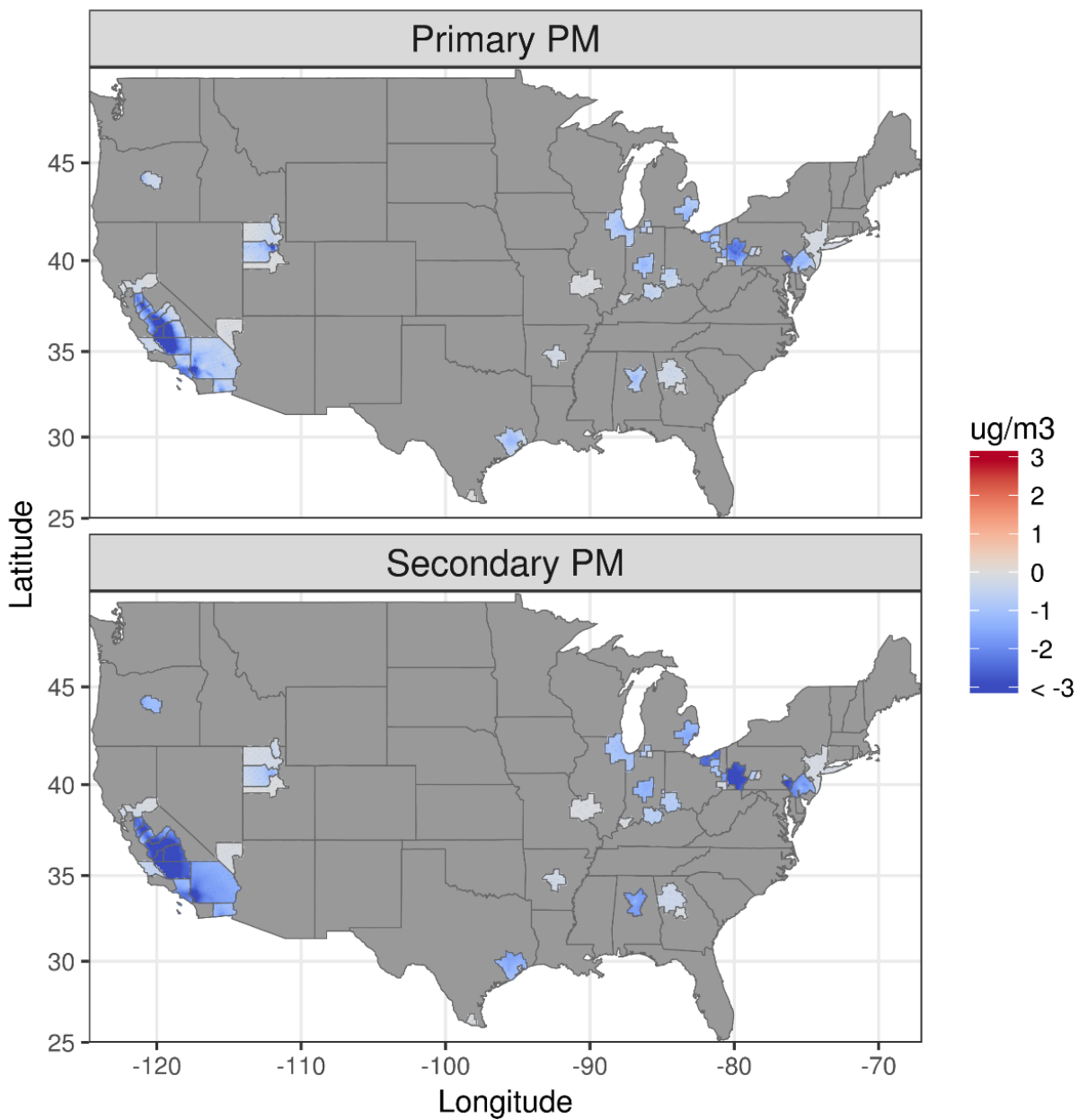
**Figure C-20. Annual average 2015 PM<sub>2.5</sub> concentrations in the 47 CBSAs based on Downscaler modeling.**

<sup>37</sup> The tables report the percent emission reduction associated with just meeting standards in the current modeling. These values should not be interpreted as the percent emission reductions that would be required to meet the standards in other application (e.g., attainment demonstrations for state implementation plans). The modeling done here was designed to quickly project PM<sub>2.5</sub> fields throughout the U.S. with a broad range of model response patterns, rather than to apply model configurations and emission scenarios specific to just meeting standards most efficiently in particular regions.

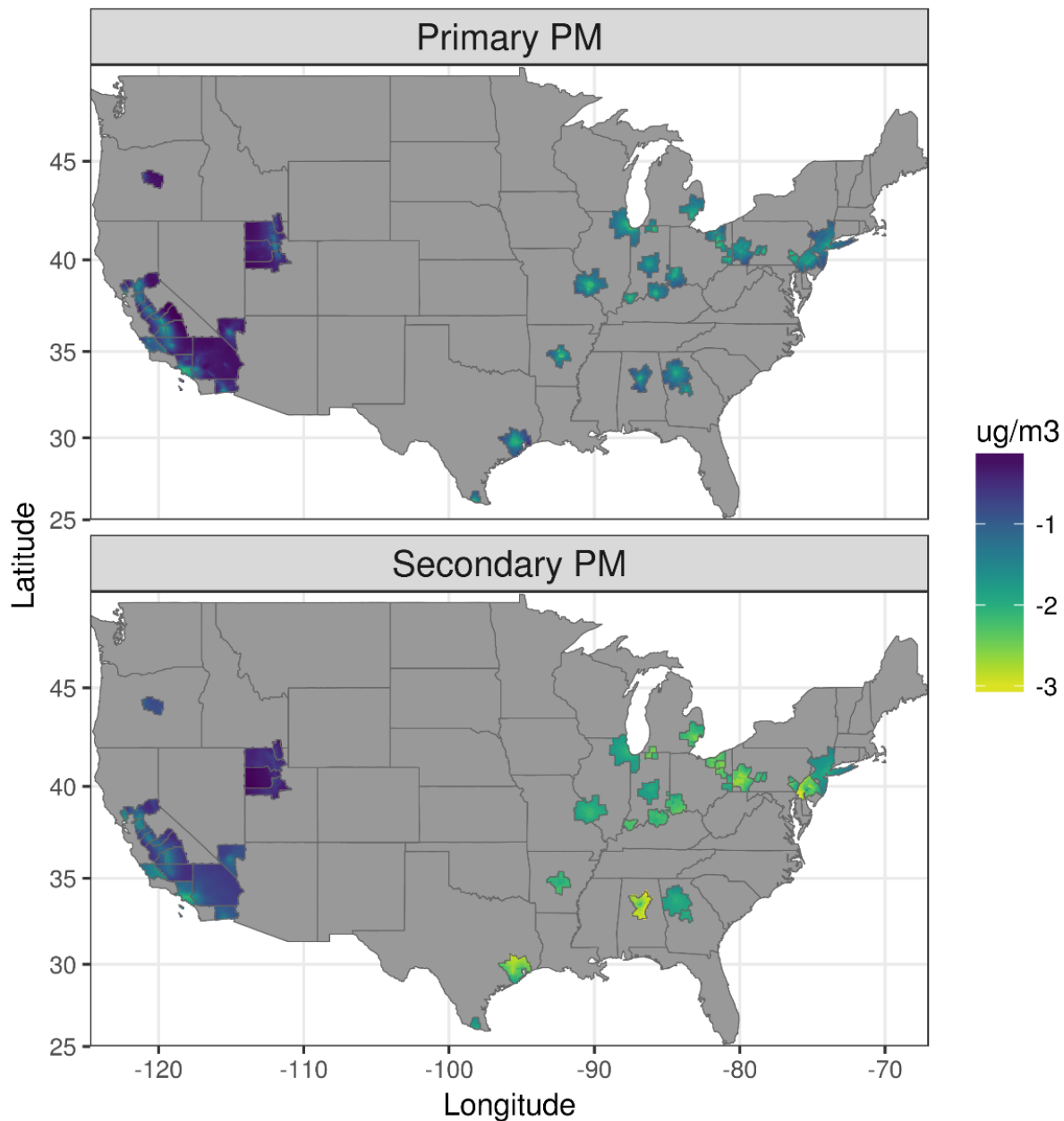




**Figure C-21. Difference between the annual average projected PM<sub>2.5</sub> concentrations and the 2015 baseline concentrations for the 12/35 projection cases (i.e., 12/35 – baseline).**



**Figure C-22. Difference between the annual average projected PM<sub>2.5</sub> concentrations and the 2015 baseline concentrations for the 10/30 projection cases (i.e., 10/30 – baseline).**



**Figure C-23. Difference between the annual average projected PM<sub>2.5</sub> concentrations in the 10/30 and 12/35 cases (i.e., 10/30 – 12/35) for the Primary PM and Secondary PM projection cases.**

#### **C.1.4.7 Limitations**

There are several limitations associated with the air quality projections. First, the baseline and projected concentrations rely on model predictions. Although state-of-the-science modeling methods were applied, and model performance was generally good, there is uncertainty associated with the model predictions. Second, due to the national scale of the assessment, the

modeling scenarios are based on “across-the-board” emission changes in which emissions of primary PM<sub>2.5</sub> or NO<sub>x</sub> and SO<sub>2</sub> from all anthropogenic sources throughout the U.S. are scaled by fixed percentages. Although this approach tends to target the key sources in each area, it does not tailor emission changes to specific periods or sources. More refined emission scenarios could be beneficial for projections in areas with relatively large seasonal and/or spatial variability in PM<sub>2.5</sub>. Similarly, fine scale simulations (e.g., 4 km or less), which are not possible due to the national scale of the assessment, would be beneficial in areas with complex terrain and relatively large spatial gradients in PM<sub>2.5</sub>. A third limitation arises because many emission cases could be applied to project PM<sub>2.5</sub> concentrations to just meet standards. We applied two projection cases that span a wide range of possible conditions, but these cases are necessarily a subset of the full set of possible projection cases.

### **C.1.5 Risk Modeling Approach**

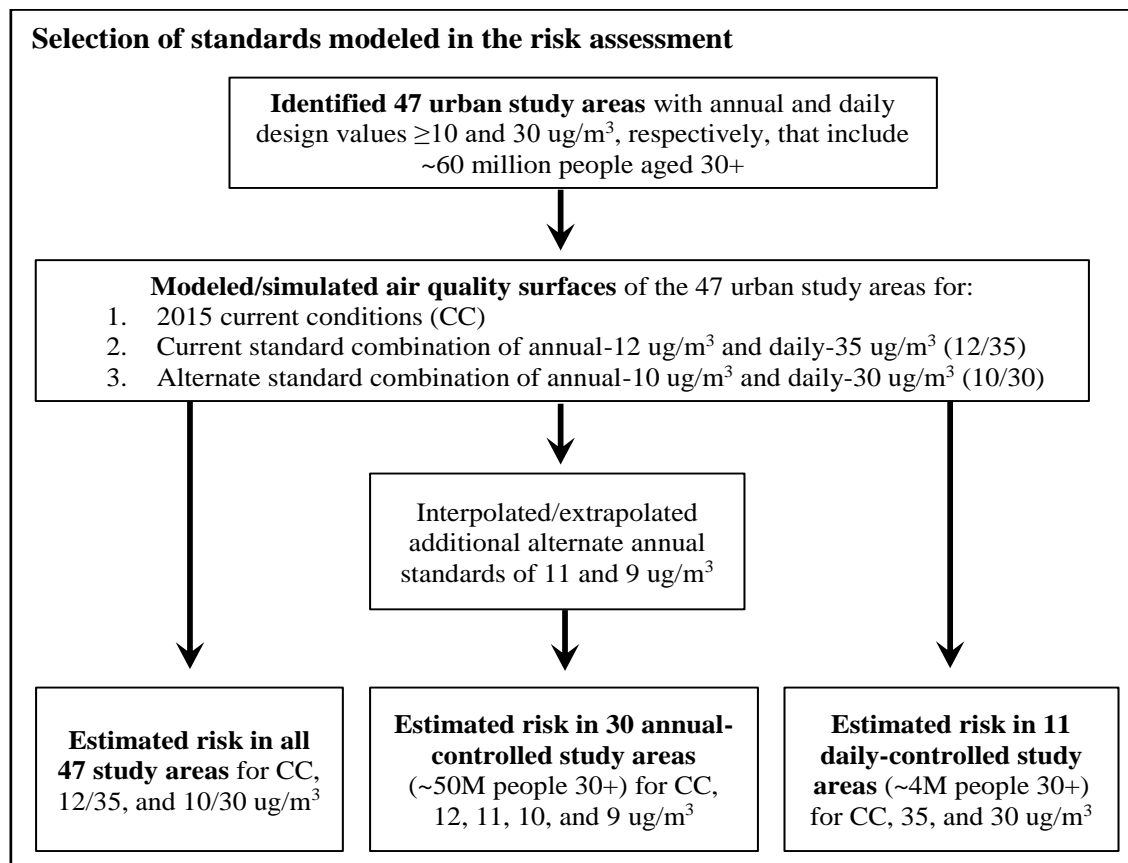
Risk modeling for this assessment was completed using the EPA’s Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE) version 1.4.14.1.<sup>38</sup> BenMAP-CE was used to estimate risk at the 12 km grid cell level for grid cells intersected by the 47 urban study area CBSAs included in risk modeling. BenMAP-CE is an open-source computer program that calculates the number and economic value of air pollution-related deaths and illnesses. The software incorporates a database that includes many of the concentration-response relationships, population files, and health and economic data needed to quantify these impacts. BenMAP-CE also allows the user to import customized datasets for any of the inputs used in modeling risk. For this analysis, CR functions developed specifically for this assessment were imported into BenMAP-CE (section C.1.1). The BenMAP-CE tool estimates the number of health impacts resulting from changes in air quality—specifically, ground-level ozone and fine particles. BenMAP-CE can also translate these incidence estimates into monetized benefits, although that functionality was not employed for this risk assessment. Inputs to BenMAP-CE used for this risk assessment are identified above in Figure C-1 and described in detail in sections C.1.1, C.1.2, C.1.3, and C.1.4.

An overall flow diagram of the risk assessment approach is provided in Figure C-24. Application of this approach resulted in separate sets of risk estimates being generated for three groupings of urban study areas including: (a) the full set of 47, (b) the 30 areas controlled by the annual standard, and (c) the 11 areas controlled by the 24-hr standard. Risk estimates are presented and discussed for each of these groupings in PA section 3.3.2, with greater emphasis being placed on results generated for the full set of 47 urban study areas and 30 annual-

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<sup>38</sup> BenMAP-CE is a free program which can be downloaded from: <https://www.epa.gov/benmap>.

controlled study areas given interest in national representation and on those study areas where we could also consider the alternative annual standards of 9 and 11  $\mu\text{g}/\text{m}^3$ .



**Figure C-24. Flow diagram of risk assessment technical approach.**

## C.2 SUPPLEMENTAL RISK RESULTS

As noted earlier, this appendix presents more granular risk information that supplements the aggregated risk estimates presented and discussed in section 3.3.2 of the PA. This supplemental information is intended to provide additional context for the interpretation of summary risk estimates presented in section 3.3.2 above, and includes:

- Modeled risk estimates that underly summary tables presented in PA section 3.3.2 aggregated to the CBSA-level (i.e., the urban study area) (section C.2.1).* Here we begin by presenting the summary table for the full set of 47 study areas followed by the CBSA-level data underlying each summary table. We then present the summary table for the 30-annual-controlled study areas, followed by the CBSA-level data underlying those summary tables.

- *Additional graphics including line plots, maps and scatter plots illustrating the distribution of the grid-level risk estimates (section C.2.2).* These graphics allow the reader to consider different aspects of the grid-level data underlying the summary tables presented in the PA (e.g., spatial distribution of risk across the cities included in the risk assessment, how the distribution of grid-cell level risk estimates shifts as lower alternative standards are considered).

Note that at the end of section C.2 we present key observations from consideration both of the CBSA-level risk estimates presented in section C.2.1 and the graphics illustrating the distribution of grid-level risk estimates in section C.2.2.

### **C.2.1 Risk Summary Tables and Underlying CBSA-Level Risk Estimates**

This section presents the full results of the risk assessment conducted in support of this review of the PM NAAQS. This includes aggregate results for all 47 urban study areas across each of the endpoints modeled, as well as the underlying results for individual cities for each endpoint. The aggregate results are consistent with those reported above in the summary tables in Chapter 3 (section 3.3.2). The more refined results for each urban study area presented below reflect the detailed 12 km grid-level risk estimates aggregated to the CBSA-level (i.e., the urban study area).

The results are organized as follows: the summary tables for the full set of 47 urban study areas, followed by tables of the associated CBSA-level risk estimates, are presented in section C.2.1.1. Then, in section C.2.1.2, we break out the 30 annual-controlled study areas (both in summary form and by the associated CBSA-level risk estimates) to show the results of simulating alternative annual standard levels of  $11.0 \mu\text{g}/\text{m}^3$  and  $9.0 \mu\text{g}/\text{m}^3$ . We do not report the results for the 11 daily-controlled areas separately, as readers can find the CBSA-level results for these areas within the tables presented for the full set of 47 study areas.<sup>39</sup> In reviewing the CBSA-level risk estimates, it is important to consider several details related to these tables including:

- In addition to the information on current and alternative standards presented in PA section 3.3.2, the tables below include information on 2015 current conditions.
- The CBSA tables are organized by health endpoint (i.e., each table presenting risk estimates for a specific endpoint). Then within a given CBSA table, the columns

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<sup>39</sup> The set of 11 daily-controlled study areas is shown in Figure C-5 and includes the following study areas: Fresno, CA, Logan, UT-ID, Madera, CA, Merced, CA, Modesto, CA, Ogden-Clearfield, UT, Prineville, OR, Provo-Orem, UT, Sacramento-Roseville-Arden-Arcade, CA, Salt Lake City, UT, Visalia-Porterville, CA.

present risk estimates for specific air quality scenarios (e.g., current conditions, current standard and so on) with the rows presenting risks for individual CBSAs. To aid cross-walk comparison between the summary tables and the CBSAs, the order of the standards presented in the CBSA tables matches the order of standards presented in the summary tables.

- Each CBSA table includes a “total” as the last row in the table, which provides the sum for that air quality scenario/health endpoint combination across all study areas. This total value can be used as a cross-check with the matching value presented in the summary table for a particular air quality scenario/health endpoint combination.
- Given the national-scale of the effect estimates used in modeling mortality risks, greater confidence is associated with aggregated (cross-city) risk estimates (as presented in PA section 3.3) than with individual CBSA-level results.

#### **C.2.1.1 CBSA-Level Results for the 47 Urban Study Areas**

Here we begin by presenting the summary tables of absolute risk and risk reduction for the full set of 47 study areas (Table C-10 and Table C-11). Then we provide tables of individual endpoint- and study- specific CBSA-level risk estimates (Table C-12, Table C-13, Table C-14, Table C-15, Table C-16, Table C-17, Table C-18, Table C-19, and Table C-20).

**Table C-10. Absolute risk summary table of the 47 urban study areas, including current conditions (2015).**

Endpoint	Study	Absolute Risk				
		Current Conditions	Simulation Method*	Current Annual Standard (12 µg/m3)	Alternative Annual Standard (10 µg/m <sup>3</sup> )	Alternative 24-hr Standard (30 µg/m <sup>3</sup> )
Long-term exposure related mortality						
IHD	Jerrett 2016	15,800 (12,100-19,400)	Pri-PM	16,500 (12,600-20,300)	14,400 (11,000-17,700)	16,400 (12,500-20,000)
			Sec-PM	16,800 (12,800-20,500)	14,200 (10,900-17,500)	16,500 (12,600-20,200)
	Pope 2015	14,900 (11,100-18,500)	Pri-PM	15,600 (11,600-19,400)	13,600 (10,100-17,000)	15,400 (11,500-19,200)
			Sec-PM	15,800 (11,800-19,600)	13,400 (9,970-16,700)	15,600 (11,600-19,400)
All-cause	Di 2017	44,100 (42,900-45,300)	Pri-PM	46,200 (45,000-47,500)	40,300 (39,200-41,400)	45,700 (44,500-47,000)
			Sec-PM	46,900 (45,600-48,200)	39,700 (38,600-40,800)	46,200 (44,900-47,500)
	Pope 2015	49,000 (39,200-58,700)	Pri-PM	51,300 (41,000-61,400)	44,700 (35,700-53,500)	50,700 (40,500-60,700)
			Sec-PM	52,100 (41,600-62,300)	44,000 (35,100-52,700)	51,300 (41,000-61,400)
	Thurston 2015	12,900 (2,250-23,100)	Pri-PM	13,500 (2,360-24,200)	11,700 (2,050-21,100)	13,300 (2,330-24,000)
			Sec-PM	13,700 (2,400-24,600)	11,500 (2,010-20,700)	13,500 (2,360-24,200)
Lung cancer	Turner 2016	3,700 (1,180-6,060)	Pri-PM	3,890 (1,240-6,360)	3,390 (1,080-5,560)	3,850 (1,230-6,300)
			Sec-PM	3,950 (1,260-6,460)	3,330 (1,060-5,470)	3,890 (1,240-6,370)
Short-term exposure related mortality						
All cause	Baxter 2017	2,380 (936-3,810)	Pri-PM	2,490 (983-4,000)	2,160 (850-3,460)	2,460 (970-3,950)
			Sec-PM	2,530 (998-4,060)	2,120 (837-3,400)	2,490 (982-3,990)
	Ito 2013	1,120 (-15-2,260)	Pri-PM	1,180 (-16-2,370)	1,020 (-14-2,050)	1,160 (-16-2,340)
			Sec-PM	1,200 (-16-2,400)	1,000 (-14-2,020)	1,180 (-16-2,370)
	Zanobetti 2014	3,630 (2,410-4,840)	Pri-PM	3,810 (2,530-5,080)	3,300 (2,190-4,400)	3,760 (2,500-5,020)
			Sec-PM	3,870 (2,570-5,160)	3,250 (2,160-4,330)	3,810 (2,530-5,070)

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)



**Table C-11. Summary of risk reduction in the 47 urban study areas when simulating a change in air quality from the current standards to an alternative suite of standards.**

Endpoint	Study	Simulation Method*	Risk Reduction (Relative to Current Standard)		% Risk Reduction (Relative to Current Standard)	
			Alternative Annual Standard (12-10 µg/m³)	Alternative 24-hr Standard (35-30 µg/m³)	Alternative Annual Standard (12-10 µg/m³)	Alternative 24-hr Standard (35-30 µg/m³)
Long-term exposure related mortality						
IHD	Jerrett 2016	Pri-PM	2,390 (1,800-2,970)	200 (150-249)	12.6	1.1
		Sec-PM	2,870 (2,160-3,570)	266 (200-331)	15.0	1.4
	Pope 2015	Pri-PM	2,240 (1,640-2,830)	187 (137-237)	12.7	1.1
		Sec-PM	2,690 (1,970-3,400)	250 (183-315)	15.1	1.4
All-cause	Di 2017	Pri-PM	6,440 (6,260-6,630)	573 (557-589)	12.9	1.2
		Sec-PM	7,800 (7,580-8,020)	772 (750-793)	15.4	1.5
	Pope 2015	Pri-PM	7,100 (5,640-8,550)	644 (511-776)	13.0	1.2
		Sec-PM	8,630 (6,860-10,400)	828 (658-997)	15.6	1.5
	Thurston 2015	Pri-PM	1,830 (316-3,320)	168 (29-305)	13.2	1.2
		Sec-PM	2,230 (387-4,060)	209 (36-381)	15.9	1.5
Lung cancer	Turner 2016	Pri-PM	548 (170-921)	42 (13-70)	13.0	1.0
		Sec-PM	670 (208-1,120)	61 (19-102)	15.6	1.4
Short-term exposure related mortality						
All cause	Baxter 2017	Pri-PM	335 (132-537)	30 (12-48)	13.5	1.3
		Sec-PM	408 (160-654)	39 (15-62)	16.1	1.6
	Ito 2013	Pri-PM	158 (-2-317)	14 (0-29)	13.4	1.2
		Sec-PM	192 (-3-386)	18 (0-37)	16.1	1.5
	Zanobetti 2014	Pri-PM	513 (341-684)	46 (30-61)	13.4	1.2
		Sec-PM	622 (413-830)	62 (41-82)	16.0	1.6

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-12. CBSA level results for the 47 urban study areas using the Jerrett et al., 2016 long-term IHD mortality CR function.**

CBSA	Absolute Risk							Risk Reduction (Relative to Current Standard)			
	Current Conditions (2015)	Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	161	173	177	150	147	173	177	27	35	0	0
Altoona, PA	31	36	36	31	31	36	36	6	6	0	0
Atlanta-Sandy Springs-Roswell, GA	414	462	475	403	401	462	475	68	84	0	0
Bakersfield, CA	137	83	89	69	89	83	78	15	0	0	13
Birmingham-Hoover, AL	163	171	177	150	142	171	177	24	41	0	0
Canton-Massillon, OH	90	99	101	85	84	99	101	17	19	0	0
Chicago-Naperville-Elgin, IL-IN-WI	1,330	1,420	1,430	1,220	1,210	1,420	1,430	226	255	0	0
Cincinnati, OH-KY-IN	332	365	373	315	312	365	373	57	71	0	0
Cleveland-Elyria, OH	436	433	431	379	347	433	431	62	95	0	0
Detroit-Warren-Dearborn, MI	1,030	1,090	1,110	926	892	1,090	1,110	183	242	0	0
El Centro, CA	21	20	20	17	17	20	20	4	4	0	0
Elkhart-Goshen, IN	42	49	49	41	41	49	49	9	9	0	0
Evansville, IN-KY	61	70	72	60	60	70	72	12	13	0	0
Fresno, CA	182	141	139	141	139	123	127	0	0	21	14
Hanford-Corcoran, CA	22	12	11	10	11	12	10	3	0	0	2
Houston-The Woodlands-Sugar Land, TX	682	723	746	624	600	723	746	114	167	0	0
Indianapolis-Carmel-Anderson, IN	282	293	296	254	248	293	296	45	54	0	0
Johnstown, PA	39	43	44	37	37	43	44	7	9	0	0
Lancaster, PA	109	103	101	87	83	103	101	18	22	0	0
Las Vegas-Henderson-Paradise, NV	163	186	189	159	159	186	189	30	33	0	0
Lebanon, PA	25	27	27	23	23	27	27	5	5	0	0
Little Rock-North Little Rock-Conway, AR	100	116	117	98	98	116	117	21	22	0	0
Logan, UT-ID	6	6	6	6	6	6	6	0	0	1	1
Los Angeles-Long Beach-Anaheim, CA	2,250	2,190	2,190	1,870	1,850	2,190	2,190	365	388	0	0
Louisville/Jefferson County, KY-IN	184	204	208	176	174	204	208	32	40	0	0
Macon, GA	41	48	48	41	41	48	48	8	9	0	0
Madera, CA	36	31	31	31	31	28	28	0	0	3	3
McAllen-Edinburg-Mission, TX	94	110	110	93	93	110	110	19	20	0	0
Merced, CA	44	41	41	41	41	37	37	0	0	5	4
Modesto, CA	117	99	99	99	99	90	90	0	0	11	10
Napa, CA	23	27	27	23	23	27	27	4	5	0	0
New York-Newark-Jersey City, NY-NJ-PA	3,540	4,020	4,130	3,480	3,480	4,020	4,130	616	730	0	0
Ogden-Clearfield, UT	44	47	46	47	46	42	43	0	0	6	4
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	1,000	1,040	1,070	898	846	1,040	1,070	167	251	0	0
Pittsburgh, PA	622	587	584	502	584	587	449	96	0	0	151
Prineville, OR	3	3	3	3	3	3	2	0	0	0	0
Provo-Orem, UT	20	22	21	22	21	20	20	0	0	3	2
Riverside-San Bernardino-Ontario, CA	586	498	486	498	415	443	486	0	78	61	0
Sacramento-Roseville-Arden-Arcade, CA	327	359	352	359	352	319	321	0	0	46	35
Salt Lake City, UT	65	55	59	55	59	45	55	0	0	10	4
San Luis Obispo-Paso Robles-Arroyo Grande, CA	29	33	33	28	28	33	33	6	6	0	0
South Bend-Mishawaka, IN-MI	59	64	68	64	68	56	55	0	0	10	14
St. Louis, MO-IL	569	656	668	564	565	656	668	106	119	0	0
Stockton-Lodi, CA	118	111	110	111	96	99	110	0	16	14	0
Visalia-Porterville, CA	96	66	65	66	65	57	57	0	0	10	10
Weirton-Steubenville, WV-OH	44	44	45	38	37	44	45	7	9	0	0
Wheeling, WV-OH	48	56	56	47	47	56	56	10	10	0	0
<b>Totals</b>	<b>15,800</b>	<b>16,500</b>	<b>16,800</b>	<b>14,400</b>	<b>14,200</b>	<b>16,400</b>	<b>16,500</b>	<b>2,390</b>	<b>2,870</b>	<b>200</b>	<b>266</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

Table C-13. CBSA level results for the 47 urban study areas using the Pope et al., 2015 long-term IHD mortality CR function.

CBSA	Absolute Risk							Risk Reduction (Relative to Current Standard)			
	Current Conditions (2015)	Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	152	163	167	141	138	163	167	25	33	0	0
Altoona, PA	29	34	34	29	29	34	34	6	6	0	0
Atlanta-Sandy Springs-Roswell, GA	390	435	447	379	378	435	447	64	79	0	0
Bakersfield, CA	129	78	84	65	84	78	73	14	0	0	12
Birmingham-Hoover, AL	154	162	167	142	133	162	167	23	38	0	0
Canton-Massillon, OH	85	93	95	80	79	93	95	16	18	0	0
Chicago-Naperville-Elgin, IL-IN-WI	1,250	1,340	1,350	1,150	1,140	1,340	1,350	213	239	0	0
Cincinnati, OH-KY-IN	313	344	352	297	293	344	352	54	67	0	0
Cleveland-Elyria, OH	411	408	406	357	327	408	406	58	89	0	0
Detroit-Warren-Dearborn, MI	967	1,020	1,040	871	839	1,020	1,040	172	227	0	0
El Centro, CA	20	19	19	16	16	19	19	3	3	0	0
Elkhart-Goshen, IN	40	46	46	39	39	46	46	8	8	0	0
Evansville, IN-KY	57	66	67	57	57	66	67	11	13	0	0
Fresno, CA	171	133	131	133	131	116	119	0	0	19	13
Hanford-Corcoran, CA	21	12	11	9	11	12	9	2	0	0	2
Houston-The Woodlands-Sugar Land, TX	642	682	703	588	564	682	703	107	157	0	0
Indianapolis-Carmel-Anderson, IN	266	276	279	239	234	276	279	42	51	0	0
Johnstown, PA	37	40	42	35	34	40	42	6	8	0	0
Lancaster, PA	103	97	96	82	78	97	96	16	20	0	0
Las Vegas-Henderson-Paradise, NV	153	175	178	149	150	175	178	28	31	0	0
Lebanon, PA	24	26	26	22	22	26	26	4	5	0	0
Little Rock-North Little Rock-Conway, AR	94	109	110	92	92	109	110	19	20	0	0
Logan, UT-ID	6	6	6	6	6	5	5	0	0	1	0
Los Angeles-Long Beach-Anaheim, CA	2,120	2,070	2,060	1,760	1,740	2,070	2,060	342	364	0	0
Louisville/Jefferson County, KY-IN	174	192	196	165	163	192	196	30	37	0	0
Macon, GA	39	45	46	39	39	45	46	7	8	0	0
Madera, CA	34	29	29	29	29	27	26	0	0	3	3
McAllen-Edinburg-Mission, TX	88	103	104	88	88	103	104	18	18	0	0
Merced, CA	42	39	39	39	39	35	35	0	0	5	4
Modesto, CA	110	93	93	93	93	84	84	0	0	10	10
Napa, CA	22	25	25	21	21	25	25	4	4	0	0
New York-Newark-Jersey City, NY-NJ-PA	3,330	3,790	3,890	3,280	3,280	3,790	3,890	578	685	0	0
Ogden-Clearfield, UT	42	45	43	45	43	39	40	0	0	6	3
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	944	984	1,010	845	796	984	1,010	156	236	0	0
Pittsburgh, PA	586	553	550	473	550	553	423	90	0	0	141
Prineville, OR	3	3	3	3	3	2	2	0	0	0	0
Provo-Orem, UT	19	21	20	21	20	19	19	0	0	2	1
Riverside-San Bernardino-Ontario, CA	551	468	457	468	390	416	457	0	74	57	0
Sacramento-Roseville-Arden-Arcade, CA	308	338	331	338	331	301	302	0	0	43	33
Salt Lake City, UT	61	51	55	51	55	42	52	0	0	10	3
San Luis Obispo-Paso Robles-Arroyo Grande, CA	28	31	31	26	26	31	31	5	5	0	0
South Bend-Mishawaka, IN-MI	56	60	64	60	64	52	52	0	0	9	14
St. Louis, MO-IL	536	618	629	531	532	618	629	99	112	0	0
Stockton-Lodi, CA	111	104	104	104	91	93	104	0	15	13	0
Visalia-Porterville, CA	91	62	62	62	62	54	53	0	0	9	9
Weirton-Stebenville, WV-OH	41	42	42	36	35	42	42	7	8	0	0
Wheeling, WV-OH	45	52	53	44	44	52	53	9	9	0	0
Totals	14,900	15,600	15,800	13,600	13,400	15,400	15,600	2,240	2,690	187	250

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-14. CBSA level results for the 47 urban study areas using the Di et al., 2017b long-term all-cause mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	547	589	602	507	496	589	602	90	117	0	0
Altoona, PA	104	123	123	103	104	123	123	21	21	0	0
Atlanta-Sandy Springs-Roswell, GA	1,940	2,180	2,240	1,890	1,880	2,180	2,240	314	387	0	0
Bakersfield, CA	333	199	214	166	214	199	186	35	0	0	30
Birmingham-Hoover, AL	709	745	770	649	613	745	770	104	170	0	0
Canton-Massillon, OH	300	329	335	281	278	329	335	53	63	0	0
Chicago-Naperville-Elgin, IL-IN-WI	4,220	4,520	4,570	3,870	3,840	4,520	4,570	698	789	0	0
Cincinnati, OH-KY-IN	1,160	1,280	1,300	1,100	1,080	1,280	1,300	196	240	0	0
Cleveland-Elyria, OH	1,290	1,280	1,280	1,120	1,020	1,280	1,280	178	274	0	0
Detroit-Warren-Dearborn, MI	2,430	2,570	2,620	2,180	2,100	2,570	2,620	421	562	0	0
El Centro, CA	51	48	48	40	41	48	48	8	8	0	0
Elkhart-Goshen, IN	114	133	133	112	112	133	133	23	23	0	0
Evansville, IN-KY	207	242	247	206	206	242	247	39	45	0	0
Fresno, CA	506	389	383	389	383	338	348	0	0	56	37
Hanford-Corcoran, CA	64	35	33	28	33	35	28	7	0	0	5
Houston-The Woodlands-Sugar Land, TX	2,130	2,260	2,340	1,940	1,870	2,260	2,340	347	510	0	0
Indianapolis-Carmel-Anderson, IN	950	989	997	852	832	989	997	148	178	0	0
Johnstown, PA	120	133	136	114	112	133	136	21	26	0	0
Lancaster, PA	397	374	370	317	299	374	370	62	76	0	0
Las Vegas-Henderson-Paradise, NV	543	622	633	529	531	622	633	98	108	0	0
Lebanon, PA	95	102	102	86	86	102	102	17	18	0	0
Little Rock-North Little Rock-Conway, AR	354	411	415	345	346	411	415	71	75	0	0
Logan, UT-ID	26	27	27	27	27	25	25	0	0	3	2
Los Angeles-Long Beach-Anaheim, CA	5,280	5,150	5,140	4,380	4,320	5,150	5,140	832	887	0	0
Louisville/Jefferson County, KY-IN	731	813	829	695	688	813	829	127	152	0	0
Macon, GA	129	149	152	128	128	149	152	23	26	0	0
Madera, CA	88	76	75	76	75	69	68	0	0	7	8
McAllen-Edinburg-Mission, TX	213	251	252	212	212	251	252	42	44	0	0
Merced, CA	115	106	107	106	107	95	97	0	0	13	11
Modesto, CA	268	226	225	226	225	204	204	0	0	24	23
Napa, CA	87	99	100	84	84	99	100	16	17	0	0
New York-Newark-Jersey City, NY-NJ-PA	7,690	8,770	9,020	7,570	7,580	8,770	9,020	1,290	1,560	0	0
Ogden-Clearfield, UT	178	191	186	191	186	168	173	0	0	24	14
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3,260	3,400	3,480	2,910	2,740	3,400	3,480	530	798	0	0
Pittsburgh, PA	1,870	1,760	1,750	1,500	1,750	1,760	1,340	281	0	0	441
Prineville, OR	12	11	11	11	11	10	10	0	0	1	2
Provo-Orem, UT	97	107	103	107	103	96	96	0	0	12	7
Riverside-San Bernardino-Ontario, CA	1,510	1,280	1,250	1,280	1,060	1,140	1,250	0	198	153	0
Sacramento-Roseville-Arden-Arcade, CA	990	1,090	1,070	1,090	1,070	965	972	0	0	136	103
Salt Lake City, UT	304	256	276	256	276	210	260	0	0	48	17
San Luis Obispo-Paso Robles-Arroyo Grande, CA	108	120	121	101	101	120	121	20	21	0	0
South Bend-Mishawaka, IN-MI	197	213	226	213	226	184	183	0	0	31	47
St. Louis, MO-IL	1,590	1,840	1,870	1,570	1,580	1,840	1,870	287	325	0	0
Stockton-Lodi, CA	357	333	331	333	289	296	331	0	46	40	0
Visalia-Porterville, CA	247	166	166	166	166	144	143	0	0	24	24
Weirton-Steubenville, WV-OH	102	104	104	89	86	104	104	16	20	0	0
Wheeling, WV-OH	124	144	145	122	122	144	145	24	25	0	0
<b>Totals</b>	<b>44,100</b>	<b>46,200</b>	<b>46,900</b>	<b>40,300</b>	<b>39,700</b>	<b>45,700</b>	<b>46,200</b>	<b>6,440</b>	<b>7,800</b>	<b>573</b>	<b>772</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-15. CBSA level results for the 47 urban study areas using the Pope et al., 2015 long-term all-cause mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	595	641	656	551	539	641	656	97	126	0	0
Altoona, PA	107	126	126	106	106	126	126	22	22	0	0
Atlanta-Sandy Springs-Roswell, GA	2,310	2,590	2,660	2,240	2,230	2,590	2,660	371	457	0	0
Bakersfield, CA	404	240	258	200	258	240	224	42	0	0	36
Birmingham-Hoover, AL	831	874	903	761	717	874	903	121	198	0	0
Canton-Massillon, OH	318	349	355	297	294	349	355	56	66	0	0
Chicago-Naperville-Elgin, IL-IN-WI	4,660	4,990	5,040	4,270	4,230	4,990	5,040	767	866	0	0
Cincinnati, OH-KY-IN	1,310	1,440	1,480	1,240	1,220	1,440	1,480	220	270	0	0
Cleveland-Elyria, OH	1,390	1,380	1,370	1,200	1,100	1,380	1,370	191	293	0	0
Detroit-Warren-Dearborn, MI	2,720	2,880	2,940	2,440	2,350	2,880	2,940	469	625	0	0
El Centro, CA	59	56	56	47	47	56	56	10	10	0	0
Elkhart-Goshen, IN	125	146	146	123	123	146	146	25	25	0	0
Evansville, IN-KY	229	268	273	228	228	268	273	43	49	0	0
Fresno, CA	573	441	432	441	432	382	393	0	0	62	42
Hanford-Corcoran, CA	78	43	39	35	39	43	34	9	0	0	6
Houston-The Woodlands-Sugar Land, TX	2,590	2,760	2,850	2,360	2,270	2,760	2,850	421	617	0	0
Indianapolis-Carmel-Anderson, IN	1,080	1,130	1,130	968	946	1,130	1,130	168	201	0	0
Johnstown, PA	126	139	143	119	118	139	143	21	27	0	0
Lancaster, PA	402	378	373	320	301	378	373	62	77	0	0
Las Vegas-Henderson-Paradise, NV	631	723	737	615	617	723	737	113	125	0	0
Lebanon, PA	97	104	105	88	87	104	105	17	19	0	0
Little Rock-North Little Rock-Conway, AR	414	481	486	404	405	481	486	83	87	0	0
Logan, UT-ID	27	28	28	28	28	25	26	0	0	3	2
Los Angeles-Long Beach-Anaheim, CA	5,800	5,660	5,650	4,810	4,740	5,660	5,650	909	969	0	0
Louisville/Jefferson County, KY-IN	841	935	954	799	791	935	954	145	174	0	0
Macon, GA	153	177	180	151	151	177	180	27	31	0	0
Madera, CA	104	88	88	88	88	81	79	0	0	8	9
McAllen-Edinburg-Mission, TX	243	286	288	241	241	286	288	47	49	0	0
Merced, CA	135	124	125	124	125	110	113	0	0	15	13
Modesto, CA	307	258	257	258	257	233	233	0	0	27	26
Napa, CA	89	102	103	87	86	102	103	17	18	0	0
New York-Newark-Jersey City, NY-NJ-PA	8,230	9,400	9,670	8,100	8,110	9,400	9,670	1,380	1,660	0	0
Ogden-Clearfield, UT	195	209	203	209	203	184	189	0	0	27	16
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3,570	3,730	3,820	3,190	3,000	3,730	3,820	578	872	0	0
Pittsburgh, PA	1,950	1,830	1,820	1,560	1,820	1,830	1,390	291	0	0	457
Prineville, OR	12	12	11	12	11	11	10	0	0	1	2
Provo-Orem, UT	105	116	112	116	112	104	104	0	0	13	8
Riverside-San Bernardino-Ontario, CA	1,740	1,470	1,430	1,470	1,220	1,300	1,430	0	226	177	0
Sacramento-Roseville-Arden-Arcade, CA	1,090	1,210	1,180	1,210	1,180	1,070	1,070	0	0	149	114
Salt Lake City, UT	350	294	317	294	317	241	298	0	0	55	19
San Luis Obispo-Paso Robles-Arroyo Grande, CA	112	125	125	105	105	125	125	21	21	0	0
South Bend-Mishawaka, IN-MI	214	231	246	231	246	200	198	0	0	34	50
St. Louis, MO-IL	1,750	2,030	2,070	1,740	1,740	2,030	2,070	314	356	0	0
Stockton-Lodi, CA	413	385	382	385	333	342	382	0	52	46	0
Visalia-Porterville, CA	289	193	193	193	193	167	166	0	0	28	28
Weirton-Steubenville, WV-OH	112	114	115	98	94	114	115	17	22	0	0
Wheeling, WV-OH	129	150	151	127	127	150	151	25	26	0	0
<b>Totals</b>	<b>49,000</b>	<b>51,300</b>	<b>52,100</b>	<b>44,700</b>	<b>44,000</b>	<b>50,700</b>	<b>51,300</b>	<b>7,100</b>	<b>8,630</b>	<b>644</b>	<b>828</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-16. CBSA level results for the 47 urban study areas using the Thurston et al., 2016 long-term all-cause mortality CR function.**

CBSA	Absolute Risk							Risk Reduction (Relative to Current Standard)			
	Current Conditions (2015)	Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	157	169	173	145	142	169	173	25	33	0	0
Altoona, PA	27	32	33	27	27	32	33	5	5	0	0
Atlanta-Sandy Springs-Roswell, GA	644	725	746	626	624	725	746	102	126	0	0
Bakersfield, CA	114	67	72	56	72	67	63	11	0	0	10
Birmingham-Hoover, AL	231	243	252	211	199	243	252	33	55	0	0
Canton-Massillon, OH	84	92	94	78	77	92	94	14	17	0	0
Chicago-Naperville-Elgin, IL-IN-WI	1,220	1,310	1,320	1,120	1,110	1,310	1,320	197	223	0	0
Cincinnati, OH-KY-IN	353	390	400	334	330	390	400	58	72	0	0
Cleveland-Elyria, OH	359	357	355	310	282	357	355	48	75	0	0
Detroit-Warren-Dearborn, MI	717	761	776	643	618	761	776	121	162	0	0
El Centro, CA	16	16	16	13	13	16	16	3	3	0	0
Elkhart-Goshen, IN	33	39	39	33	33	39	39	6	7	0	0
Evansville, IN-KY	62	72	74	61	61	72	74	11	13	0	0
Fresno, CA	150	114	112	114	112	99	102	0	0	16	11
Hanford-Corcoran, CA	22	12	11	9	11	12	9	2	0	0	2
Houston-The Woodlands-Sugar Land, TX	729	776	803	664	636	776	803	116	171	0	0
Indianapolis-Carmel-Anderson, IN	293	305	308	262	256	305	308	45	54	0	0
Johnstown, PA	31	34	35	29	29	34	35	5	7	0	0
Lancaster, PA	97	91	90	77	72	91	90	15	18	0	0
Las Vegas-Henderson-Paradise, NV	186	214	218	181	182	214	218	33	37	0	0
Lebanon, PA	25	26	26	22	22	26	26	4	5	0	0
Little Rock-North Little Rock-Conway, AR	116	135	137	113	113	135	137	23	24	0	0
Logan, UT-ID	7	7	7	7	7	6	6	0	0	1	1
Los Angeles-Long Beach-Anaheim, CA	1,470	1,430	1,430	1,210	1,190	1,430	1,430	225	240	0	0
Louisville/Jefferson County, KY-IN	231	258	263	220	217	258	263	39	47	0	0
Macon, GA	43	51	52	43	43	51	52	8	9	0	0
Madera, CA	28	24	24	24	24	22	22	0	0	2	2
McAllen-Edinburg-Mission, TX	66	78	79	66	66	78	79	13	13	0	0
Merced, CA	36	33	33	33	33	29	30	0	0	4	3
Modesto, CA	84	70	70	70	70	63	63	0	0	7	7
Napa, CA	22	25	26	21	21	25	26	4	4	0	0
New York-Newark-Jersey City, NY-NJ-PA	2,070	2,370	2,440	2,030	2,040	2,370	2,440	343	410	0	0
Ogden-Clearfield, UT	50	54	52	54	52	47	48	0	0	7	4
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	912	953	977	812	763	953	977	145	219	0	0
Pittsburgh, PA	490	461	458	391	458	461	348	72	0	0	113
Prineville, OR	4	3	3	3	3	3	3	0	0	0	0
Provo-Orem, UT	26	29	28	29	28	26	26	0	0	3	2
Riverside-San Bernardino-Ontario, CA	480	404	395	404	335	357	395	0	61	48	0
Sacramento-Roseville-Arden-Arcade, CA	288	318	311	318	311	281	282	0	0	38	30
Salt Lake City, UT	89	75	80	75	80	61	76	0	0	14	5
San Luis Obispo-Paso Robles-Arroyo Grande, CA	27	30	30	25	25	30	30	5	5	0	0
South Bend-Mishawaka, IN-MI	55	60	64	60	64	52	51	0	0	9	13
St. Louis, MO-IL	463	539	550	460	460	539	550	82	93	0	0
Stockton-Lodi, CA	111	103	102	103	89	91	102	0	14	12	0
Visalia-Porterville, CA	77	51	51	51	51	44	44	0	0	7	7
Weirton-Steubenville, WV-OH	31	32	32	27	26	32	32	5	6	0	0
Wheeling, WV-OH	34	40	40	34	34	40	40	7	7	0	0
<b>Totals</b>	<b>12,900</b>	<b>13,500</b>	<b>13,700</b>	<b>11,700</b>	<b>11,500</b>	<b>13,300</b>	<b>13,500</b>	<b>1,830</b>	<b>2,230</b>	<b>168</b>	<b>209</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-17. CBSA level results for the 47 urban study areas using the Turner et al., 2016 long-term lung cancer mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	48	51	52	44	43	51	52	8	10	0	0
Altoona, PA	6	7	7	6	6	7	7	1	1	0	0
Atlanta-Sandy Springs-Roswell, GA	183	204	210	178	177	204	210	29	36	0	0
Bakersfield, CA	27	16	17	13	17	16	15	3	0	0	2
Birmingham-Hoover, AL	63	66	69	58	55	66	69	9	15	0	0
Canton-Massillon, OH	25	28	28	24	24	28	28	5	5	0	0
Chicago-Naperville-Elgin, IL-IN-WI	379	406	410	348	345	406	410	63	71	0	0
Cincinnati, OH-KY-IN	122	134	137	115	114	134	137	20	26	0	0
Cleveland-Elyria, OH	111	111	110	96	88	111	110	15	24	0	0
Detroit-Warren-Dearborn, MI	220	233	237	198	190	233	237	38	51	0	0
El Centro, CA	4	4	4	3	3	4	4	1	1	0	0
Elkhart-Goshen, IN	10	11	11	9	9	11	11	2	2	0	0
Evansville, IN-KY	19	22	23	19	19	22	23	4	4	0	0
Fresno, CA	35	27	26	27	26	23	24	0	0	4	3
Hanford-Corcoran, CA	5	3	2	2	2	3	2	1	0	0	0
Houston-The Woodlands-Sugar Land, TX	194	206	213	177	170	206	213	31	47	0	0
Indianapolis-Carmel-Anderson, IN	102	106	107	91	89	106	107	16	19	0	0
Johnstown, PA	8	9	9	8	8	9	9	1	2	0	0
Lancaster, PA	28	26	26	22	21	26	26	4	5	0	0
Las Vegas-Henderson-Paradise, NV	55	63	64	53	53	63	64	10	11	0	0
Lebanon, PA	9	9	9	8	8	9	9	2	2	0	0
Little Rock-North Little Rock-Conway, AR	37	43	43	36	36	43	43	7	8	0	0
Logan, UT-ID	1	1	1	1	1	1	1	0	0	0	0
Los Angeles-Long Beach-Anaheim, CA	360	351	351	299	295	351	351	57	61	0	0
Louisville/Jefferson County, KY-IN	82	91	93	78	78	91	93	14	17	0	0
Macon, GA	13	15	15	13	13	15	15	2	3	0	0
Madera, CA	7	6	6	6	6	5	5	0	0	1	1
McAllen-Edinburg-Mission, TX	11	13	13	11	11	13	13	2	2	0	0
Merced, CA	9	9	9	9	9	8	8	0	0	1	1
Modesto, CA	21	18	17	18	17	16	16	0	0	2	2
Napa, CA	7	8	8	6	6	8	8	1	1	0	0
New York-Newark-Jersey City, NY-NJ-PA	590	672	691	580	581	672	691	99	119	0	0
Ogden-Clearfield, UT	8	8	8	8	8	7	7	0	0	1	1
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	284	296	303	253	238	296	303	46	70	0	0
Pittsburgh, PA	153	145	144	123	144	145	110	23	0	0	36
Prineville, OR	1	1	1	1	1	1	1	0	0	0	0
Provo-Orem, UT	3	3	3	3	3	3	3	0	0	0	0
Riverside-San Bernardino-Ontario, CA	120	102	99	102	85	90	99	0	16	12	0
Sacramento-Roseville-Arden-Arcade, CA	79	87	86	87	86	77	78	0	0	11	8
Salt Lake City, UT	14	12	13	12	13	10	12	0	0	2	1
San Luis Obispo-Paso Robles-Arroyo Grande, CA	8	9	9	7	7	9	9	1	2	0	0
South Bend-Mishawaka, IN-MI	17	18	20	18	20	16	16	0	0	3	4
St. Louis, MO-IL	158	182	186	156	157	182	186	28	32	0	0
Stockton-Lodi, CA	29	27	27	27	23	24	27	0	4	3	0
Visalia-Porterville, CA	18	12	12	12	12	11	10	0	0	2	2
Weirton-Steubenville, WV-OH	9	10	10	8	8	10	10	1	2	0	0
Wheeling, WV-OH	11	12	12	10	10	12	12	2	2	0	0
<b>Totals</b>	<b>3,700</b>	<b>3,890</b>	<b>3,950</b>	<b>3,390</b>	<b>3,330</b>	<b>3,850</b>	<b>3,890</b>	<b>548</b>	<b>670</b>	<b>42</b>	<b>61</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-18. CBSA level results for the 47 urban study areas using the Baxter et al., 2017 all-cause short-term mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	29	31	32	27	26	31	32	5	6	0	0
Altoona, PA	5	6	6	5	5	6	6	1	1	0	0
Atlanta-Sandy Springs-Roswell, GA	111	126	129	108	108	126	129	18	22	0	0
Bakersfield, CA	20	11	12	9	12	11	11	2	0	0	2
Birmingham-Hoover, AL	40	42	44	37	35	42	44	6	9	0	0
Canton-Massillon, OH	15	17	17	14	14	17	17	3	3	0	0
Chicago-Naperville-Elgin, IL-IN-WI	228	245	248	208	206	245	248	37	41	0	0
Cincinnati, OH-KY-IN	63	70	71	59	59	70	71	10	13	0	0
Cleveland-Elyria, OH	68	67	67	58	53	67	67	9	14	0	0
Detroit-Warren-Dearborn, MI	132	140	143	118	113	140	143	22	30	0	0
El Centro, CA	3	3	3	2	2	3	3	0	0	0	0
Elkhart-Goshen, IN	6	7	7	6	6	7	7	1	1	0	0
Evansville, IN-KY	11	13	13	11	11	13	13	2	2	0	0
Fresno, CA	28	22	21	22	21	19	19	0	0	3	2
Hanford-Corcoran, CA	4	2	2	2	2	2	2	0	0	0	0
Houston-The Woodlands-Sugar Land, TX	126	134	139	114	109	134	139	20	29	0	0
Indianapolis-Carmel-Anderson, IN	52	54	55	47	46	54	55	8	9	0	0
Johnstown, PA	6	7	7	6	6	7	7	1	1	0	0
Lancaster, PA	20	18	18	16	15	18	18	3	4	0	0
Las Vegas-Henderson-Paradise, NV	30	34	35	29	29	34	35	5	6	0	0
Lebanon, PA	5	5	5	4	4	5	5	1	1	0	0
Little Rock-North Little Rock-Conway, AR	20	23	24	20	20	23	24	4	4	0	0
Logan, UT-ID	1	1	1	1	1	1	1	0	0	0	0
Los Angeles-Long Beach-Anaheim, CA	284	277	277	234	231	277	277	43	46	0	0
Louisville/Jefferson County, KY-IN	41	45	46	38	38	45	46	7	8	0	0
Macon, GA	7	9	9	7	7	9	9	1	1	0	0
Madera, CA	5	4	4	4	4	4	4	0	0	0	0
McAllen-Edinburg-Mission, TX	12	14	14	12	12	14	14	2	2	0	0
Merced, CA	6	6	6	6	6	5	5	0	0	1	1
Modesto, CA	15	13	13	13	13	11	11	0	0	1	1
Napa, CA	4	5	5	4	4	5	5	1	1	0	0
New York-Newark-Jersey City, NY-NJ-PA	401	459	473	394	394	459	473	66	79	0	0
Ogden-Clearfield, UT	9	10	10	10	10	9	9	0	0	1	1
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	172	180	184	153	144	180	184	27	41	0	0
Pittsburgh, PA	94	88	88	74	88	88	66	14	0	0	21
Prineville, OR	1	1	1	1	1	0	0	0	0	0	0
Provo-Orem, UT	5	6	5	6	5	5	5	0	0	1	0
Riverside-San Bernardino-Ontario, CA	85	71	69	71	59	63	69	0	11	8	0
Sacramento-Roseville-Arden-Arcade, CA	52	58	57	58	57	51	51	0	0	7	5
Salt Lake City, UT	16	14	15	14	15	11	14	0	0	3	1
San Luis Obispo-Paso Robles-Arroyo Grande, CA	5	6	6	5	5	6	6	1	1	0	0
South Bend-Mishawaka, IN-MI	10	11	12	11	12	10	10	0	0	2	2
St. Louis, MO-IL	84	98	100	83	83	98	100	15	17	0	0
Stockton-Lodi, CA	20	19	19	19	16	17	19	0	2	2	0
Visalia-Porterville, CA	14	9	9	9	9	8	8	0	0	1	1
Weirton-Steubenville, WV-OH	5	5	6	5	4	5	6	1	1	0	0
Wheeling, WV-OH	6	7	7	6	6	7	7	1	1	0	0
<b>Totals</b>	<b>2,380</b>	<b>2,490</b>	<b>2,530</b>	<b>2,160</b>	<b>2,120</b>	<b>2,460</b>	<b>2,490</b>	<b>335</b>	<b>408</b>	<b>30</b>	<b>39</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)



**Table C-19. CBSA level results for the 47 urban study areas using the Ito et al., 2013 all-cause short-term mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	14	15	15	13	12	15	15	2	3	0	0
Altoona, PA	2	3	3	2	2	3	3	0	0	0	0
Atlanta-Sandy Springs-Roswell, GA	53	60	62	52	52	60	62	8	10	0	0
Bakersfield, CA	10	6	6	5	6	6	5	1	0	0	1
Birmingham-Hoover, AL	19	20	21	18	17	20	21	3	4	0	0
Canton-Massillon, OH	7	8	8	7	7	8	8	1	1	0	0
Chicago-Naperville-Elgin, IL-IN-WI	107	115	116	98	97	115	116	17	19	0	0
Cincinnati, OH-KY-IN	30	33	34	28	28	33	34	5	6	0	0
Cleveland-Elyria, OH	32	31	31	27	25	31	31	4	7	0	0
Detroit-Warren-Dearborn, MI	62	66	68	56	54	66	68	10	14	0	0
El Centro, CA	1	1	1	1	1	1	1	0	0	0	0
Elkhart-Goshen, IN	3	3	3	3	3	3	3	1	1	0	0
Evansville, IN-KY	5	6	6	5	5	6	6	1	1	0	0
Fresno, CA	14	10	10	10	10	9	9	0	0	1	1
Hanford-Corcoran, CA	2	1	1	1	1	1	1	0	0	0	0
Houston-The Woodlands-Sugar Land, TX	61	65	67	55	53	65	67	10	14	0	0
Indianapolis-Carmel-Anderson, IN	25	26	26	22	22	26	26	4	5	0	0
Johnstown, PA	3	3	3	3	3	3	3	0	1	0	0
Lancaster, PA	9	9	9	7	7	9	9	1	2	0	0
Las Vegas-Henderson-Paradise, NV	14	16	17	14	14	16	17	3	3	0	0
Lebanon, PA	2	2	2	2	2	2	2	0	0	0	0
Little Rock-North Little Rock-Conway, AR	10	11	11	9	9	11	11	2	2	0	0
Logan, UT-ID	1	1	1	1	1	1	1	0	0	0	0
Los Angeles-Long Beach-Anaheim, CA	133	130	129	109	108	130	129	20	22	0	0
Louisville/Jefferson County, KY-IN	19	22	22	18	18	22	22	3	4	0	0
Macon, GA	4	4	4	3	3	4	4	1	1	0	0
Madera, CA	2	2	2	2	2	2	2	0	0	0	0
McAllen-Edinburg-Mission, TX	6	7	7	6	6	7	7	1	1	0	0
Merced, CA	3	3	3	3	3	3	3	0	0	0	0
Modesto, CA	7	6	6	6	6	5	5	0	0	1	1
Napa, CA	2	2	2	2	2	2	2	0	0	0	0
New York-Newark-Jersey City, NY-NJ-PA	187	214	220	184	184	214	220	31	37	0	0
Ogden-Clearfield, UT	5	5	5	5	5	4	4	0	0	1	0
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	82	86	88	73	68	86	88	13	19	0	0
Pittsburgh, PA	44	42	41	35	41	42	31	6	0	0	10
Prineville, OR	0	0	0	0	0	0	0	0	0	0	0
Provo-Orem, UT	2	3	3	3	3	2	2	0	0	0	0
Riverside-San Bernardino-Ontario, CA	40	34	33	34	28	30	33	0	5	4	0
Sacramento-Roseville-Arden-Arcade, CA	25	28	27	28	27	24	25	0	0	3	3
Salt Lake City, UT	8	7	7	7	7	6	7	0	0	1	0
San Luis Obispo-Paso Robles-Arroyo Grande, CA	3	3	3	2	2	3	3	0	0	0	0
South Bend-Mishawaka, IN-MI	5	5	6	5	6	5	5	0	0	1	1
St. Louis, MO-IL	40	47	48	40	40	47	48	7	8	0	0
Stockton-Lodi, CA	10	9	9	9	8	8	9	0	1	1	0
Visalia-Porterville, CA	7	4	4	4	4	4	4	0	0	1	1
Weirton-Steubenville, WV-OH	3	3	3	2	2	3	3	0	0	0	0
Wheeling, WV-OH	3	3	3	3	3	3	3	1	1	0	0
<b>Totals</b>	<b>1,120</b>	<b>1,180</b>	<b>1,200</b>	<b>1,020</b>	<b>1,000</b>	<b>1,160</b>	<b>1,180</b>	<b>158</b>	<b>192</b>	<b>14</b>	<b>18</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-20. CBSA level results for the 47 urban study areas using the Zanobetti et al., 2014 all-cause short-term mortality CR function.**

CBSA	Current Conditions (2015)	Absolute Risk						Risk Reduction (Relative to Current Standard)			
		Current Annual Standard (12 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative 24-hr (30 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative 24-hr Standard (35-30 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	45	49	50	42	41	49	50	7	9	0	0
Altoona, PA	9	10	10	9	9	10	10	2	2	0	0
Atlanta-Sandy Springs-Roswell, GA	159	180	185	155	154	180	185	25	31	0	0
Bakersfield, CA	28	16	17	13	17	16	15	3	0	0	2
Birmingham-Hoover, AL	58	62	64	53	50	62	64	8	14	0	0
Canton-Massillon, OH	25	27	28	23	23	27	28	4	5	0	0
Chicago-Naperville-Elgin, IL-IN-WI	348	373	377	318	315	373	377	56	63	0	0
Cincinnati, OH-KY-IN	95	105	108	90	89	105	108	16	19	0	0
Cleveland-Elyria, OH	106	106	105	92	83	106	105	14	22	0	0
Detroit-Warren-Dearborn, MI	200	212	216	179	172	212	216	34	45	0	0
El Centro, CA	4	4	4	3	3	4	4	1	1	0	0
Elkhart-Goshen, IN	9	11	11	9	9	11	11	2	2	0	0
Evansville, IN-KY	17	20	21	17	17	20	21	3	4	0	0
Fresno, CA	42	32	32	32	32	28	29	0	0	4	3
Hanford-Corcoran, CA	5	3	3	2	3	3	2	1	0	0	0
Houston-The Woodlands-Sugar Land, TX	175	187	193	160	153	187	193	28	41	0	0
Indianapolis-Carmel-Anderson, IN	78	82	82	70	68	82	82	12	14	0	0
Johnstown, PA	10	11	11	9	9	11	11	2	2	0	0
Lancaster, PA	33	31	31	26	24	31	31	5	6	0	0
Las Vegas-Henderson-Paradise, NV	44	51	52	43	43	51	52	8	9	0	0
Lebanon, PA	8	8	8	7	7	8	8	1	1	0	0
Little Rock-North Little Rock-Conway, AR	29	34	34	28	28	34	34	6	6	0	0
Logan, UT-ID	2	2	2	2	2	2	2	0	0	0	0
Los Angeles-Long Beach-Anaheim, CA	435	425	424	359	354	425	424	66	71	0	0
Louisville/Jefferson County, KY-IN	60	67	69	57	57	67	69	10	12	0	0
Macon, GA	11	12	13	11	11	12	13	2	2	0	0
Madera, CA	7	6	6	6	6	6	6	0	0	1	1
McAllen-Edinburg-Mission, TX	17	21	21	17	17	21	21	3	3	0	0
Merced, CA	10	9	9	9	9	8	8	0	0	1	1
Modesto, CA	22	19	19	19	19	17	17	0	0	2	2
Napa, CA	7	8	8	7	7	8	8	1	1	0	0
New York-Newark-Jersey City, NY-NJ-PA	630	722	743	619	620	722	743	103	124	0	0
Ogden-Clearfield, UT	15	16	15	16	15	14	14	0	0	2	1
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	268	280	287	238	224	280	287	42	64	0	0
Pittsburgh, PA	154	145	144	123	144	145	109	22	0	0	35
Prineville, OR	1	1	1	1	1	1	1	0	0	0	0
Provo-Orem, UT	8	9	8	9	8	8	8	0	0	1	1
Riverside-San Bernardino-Ontario, CA	124	104	102	104	86	92	102	0	16	12	0
Sacramento-Roseville-Arden-Arcade, CA	81	90	88	90	88	79	80	0	0	11	8
Salt Lake City, UT	25	21	22	21	22	17	21	0	0	4	1
San Luis Obispo-Paso Robles-Arroyo Grande, CA	9	10	10	8	8	10	10	2	2	0	0
South Bend-Mishawaka, IN-MI	16	18	19	18	19	15	15	0	0	2	4
St. Louis, MO-IL	131	152	155	129	130	152	155	23	26	0	0
Stockton-Lodi, CA	30	28	27	28	24	24	27	0	4	3	0
Visalia-Porterville, CA	21	14	14	14	14	12	12	0	0	2	2
Weirton-Steubenville, WV-OH	8	9	9	7	7	9	9	1	2	0	0
Wheeling, WV-OH	10	12	12	10	10	12	12	2	2	0	0
<b>Totals</b>	<b>3,630</b>	<b>3,810</b>	<b>3,870</b>	<b>3,300</b>	<b>3,250</b>	<b>3,760</b>	<b>3,810</b>	<b>513</b>	<b>622</b>	<b>46</b>	<b>62</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

### C.2.1.2 CBSA-Level Results for the 30 Annual-Controlled Urban Study Areas

Here we begin by presenting the summary tables of absolute risk and risk reduction for the 30 annual-controlled study areas (Table C-21 and Table C-22) where the annual standard was controlling. Then we provide tables of individual endpoint- and study- specific CBSA-level risk estimates (Table C-23, Table C-24, Table C-25, Table C-26, Table C-27, Table C-28, Table C-29, Table C-30, and Table C-31).

**Table C-21. Absolute risk summary table of the 30 urban study areas, including current conditions (2015).**

Endpoint	Study	Absolute Risk						
		Current Conditions (2015)	Simulation Method*	Current Annual Standard (12 µg/m³)	Alternative Annual Standard (11 µg/m³)	Alternative Annual Standard (10 µg/m³)	Alternative Annual Standard (9 µg/m³)	
Long-term exposure related mortality								
IHD	Jerrett 2016	13,300	Pri-PM	14,300 (10,900-17,500)	13,300 (10,200-16,300)	12,300 (9,400-15,100)	11,300 (8,610-13,900)	
		(10,200-16,300)	Sec-PM	14,600 (11,100-17,800)	13,300 (10,200-16,400)	12,100 (9,240-14,900)	10,900 (8,280-13,400)	
	Pope 2015	12,500	Pri-PM	13,500 (10,100-16,800)	12,500 (9,340-15,600)	11,600 (8,620-14,500)	10,600 (7,900-13,300)	
		(9,340-15,600)	Sec-PM	13,700 (10,200-17,000)	12,600 (9,360-15,600)	11,400 (8,480-14,200)	10,200 (7,590-12,800)	
All-cause	Di 2017	37,000	Pri-PM	39,800 (38,700-40,900)	36,900 (35,900-38,000)	34,100 (33,200-35,000)	31,200 (30,400-32,100)	
		(36,000-38,000)	Sec-PM	40,500 (39,400-41,600)	37,000 (36,000-38,000)	33,500 (32,600-34,400)	29,900 (29,100-30,800)	
	Pope 2015	41,000	Pri-PM	44,200 (35,300-52,800)	41,000 (32,800-49,100)	37,800 (30,200-45,300)	34,600 (27,600-41,500)	
		(32,800-49,100)	Sec-PM	45,000 (35,900-53,800)	41,000 (32,800-49,100)	37,100 (29,600-44,500)	33,200 (26,500-39,700)	
	Thurston 2015	10,700	Pri-PM	11,600 (2,030-20,800)	10,700 (1,880-19,300)	9,900 (1,730-17,800)	9,050 (1,580-16,300)	
		(1,880-19,300)	Sec-PM	11,800 (2,070-21,200)	10,800 (1,880-19,400)	9,710 (1,700-17,500)	8,650 (1,510-15,600)	
	Lung cancer	Turner 2016	3,150	Pri-PM	3,400 (1,080-5,550)	3,160 (1,010-5,170)	2,920 (927-4,790)	2,670 (847-4,400)
			(1,000-5,160)	Sec-PM	3,460 (1,110-5,650)	3,160 (1,010-5,180)	2,860 (908-4,700)	2,560 (809-4,210)
Short-term exposure related mortality								
All-cause	Baxter 2017	1,990	Pri-PM	2,150 (846-3,440)	1,990 (784-3,190)	1,830 (721-2,930)	1,670 (658-2,680)	
		(784-3,190)	Sec-PM	2,190 (862-3,510)	1,990 (785-3,190)	1,790 (707-2,880)	1,600 (630-2,560)	
	Ito 2013	940	Pri-PM	1,010 (-14-2,040)	939 (-13-1,880)	864 (-12-1,730)	789 (-11-1,580)	
		(-13-1,890)	Sec-PM	1,030 (-14-2,070)	940 (-13-1,890)	847 (-11-1,700)	754 (-10-1,510)	
	Zanobetti 2014	3,040	Pri-PM	3,280 (2,180-4,370)	3,040 (2,020-4,050)	2,790 (1,860-3,730)	2,550 (1,700-3,400)	
		(2,020-4,050)	Sec-PM	3,340 (2,220-4,450)	3,040 (2,020-4,050)	2,740 (1,820-3,650)	2,440 (1,620-3,260)	

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-22. Summary of risk reduction in the 30 urban study areas when simulating a change in air quality from the current standards to alternative annual standards.**

Endpoint	Study	Simulation Method*	Risk Reduction (Relative to Current Standard)			Percent Risk Reduction (Relative to Current Standard)		
			Alternative Annual Standard (12-11 µg/m³)	Alternative Annual Standard (12-10 µg/m³)	Alternative Annual Standard (12-9 µg/m³)	Alternative Annual Standard (12-11 µg/m³)	Alternative Annual Standard (12-10 µg/m³)	Alternative Annual Standard (12-9 µg/m³)
Long-term exposure related mortality								
IHD	Jerrett 2016	Pri-PM	1,140 (859-1,420)	2,270 (1,710-2,830)	3,390 (2,550-4,210)	7%	14%	21%
		Sec-PM	1,400 (1,050-1,740)	2,770 (2,090-3,450)	4,130 (3,110-5,130)	8%	17%	25%
	Pope 2014	Pri-PM	1,070 (785-1,360)	2,130 (1,560-2,690)	3,180 (2,340-4,010)	7%	14%	21%
		Sec-PM	1,310 (960-1,660)	2,600 (1,910-3,280)	3,880 (2,850-4,890)	8%	17%	25%
All-cause	Di 2017	Pri-PM	3,070 (2,980-3,160)	6,120 (5,950-6,300)	9,150 (8,890-9,410)	7%	14%	21%
		Sec-PM	3,800 (3,690-3,900)	7,560 (7,340-7,770)	11,300 (11,000-11,600)	9%	17%	26%
	Pope 2014	Pri-PM	3,390 (2,690-4,080)	6,760 (5,370-8,140)	10,100 (8,030-12,200)	7%	14%	22%
		Sec-PM	4,190 (3,330-5,050)	8,350 (6,640-10,100)	12,500 (9,930-15,000)	9%	17%	26%
	Thurston 2015	Pri-PM	871 (151-1,590)	1,740 (301-3,170)	2,610 (452-4,740)	7%	15%	22%
		Sec-PM	1,080 (187-1,970)	2,160 (374-3,930)	3,230 (561-5,870)	9%	18%	27%
Lung cancer	Turner 2016	Pri-PM	262 (81-441)	522 (162-877)	780 (243-1,310)	7%	14%	21%
		Sec-PM	327 (101-550)	651 (202-1,090)	972 (303-1,630)	9%	17%	26%
Short-term exposure related mortality								
All-cause	Baxter 2017	Pri-PM	160 (63-256)	319 (126-512)	478 (188-767)	7%	15%	22%
		Sec-PM	197 (78-316)	394 (155-632)	592 (233-948)	9%	18%	27%
	Ito 2013	Pri-PM	75 (-1-151)	150 (-2-302)	226 (-3-453)	7%	15%	22%
		Sec-PM	93 (-1-187)	186 (-2-374)	279 (-4-561)	9%	18%	27%
	Zanobetti 2014	Pri-PM	244 (162-325)	487 (324-650)	731 (486-975)	7%	15%	22%
		Sec-PM	301 (200-402)	603 (400-804)	904 (600-1,210)	9%	18%	27%

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-23. CBSA level results for the 30 annual-controlled urban study areas using the Jerrett et al., 2016 long-term IHD mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	161	173	177	162	162	150	147	138	131	14	18	27	35	40	53
Altoona, PA	31	36	36	33	34	31	31	28	28	3	3	6	6	10	10
Atlanta-Sandy Springs-Roswell, GA	414	462	475	433	438	403	401	372	364	34	42	68	84	102	126
Birmingham-Hoover, AL	163	171	177	161	160	150	142	140	123	12	21	24	41	36	60
Canton-Massillon, OH	90	99	101	92	92	85	84	78	76	8	10	17	19	25	29
Chicago-Naperville-Elgin, IL-IN-WI	1,330	1,420	1,430	1,320	1,320	1,220	1,210	1,120	1,100	114	128	226	255	338	380
Cincinnati, OH-KY-IN	332	365	373	341	343	315	312	290	280	29	36	57	71	86	106
Cleveland-Elyria, OH	436	433	431	406	389	379	347	351	304	31	48	62	95	92	142
Detroit-Warren-Dearborn, MI	1,030	1,090	1,110	1,010	1,000	926	892	844	783	92	122	183	242	273	360
El Centro, CA	21	20	20	19	19	17	17	15	15	2	2	4	4	5	5
Elkhart-Goshen, IN	42	49	49	45	45	41	41	38	38	4	4	9	9	13	13
Evansville, IN-KY	61	70	72	65	66	60	60	55	54	6	7	12	13	18	20
Houston-The Woodlands-Sugar Land, TX	682	723	746	674	673	624	600	574	525	58	84	114	167	170	249
Indianapolis-Carmel-Anderson, IN	282	293	296	274	272	254	248	234	224	23	27	45	54	67	81
Johnstown, PA	39	43	44	40	40	37	37	34	33	3	4	7	9	10	13
Lancaster, PA	109	103	101	95	92	87	83	80	73	9	11	18	22	26	32
Las Vegas-Henderson-Paradise, NV	163	186	189	172	174	159	159	145	144	15	17	30	33	44	49
Lebanon, PA	25	27	27	25	25	23	23	21	21	2	3	5	5	7	7
Little Rock-North Little Rock-Conway, AR	100	116	117	107	107	98	98	89	88	10	11	21	22	31	32
Los Angeles-Long Beach-Anaheim, CA	2,250	2,190	2,190	2,030	2,020	1,870	1,850	1,710	1,680	184	195	365	388	544	578
Louisville/Jefferson County, KY-IN	184	204	208	190	191	176	174	161	156	16	20	32	40	48	59
Macon, GA	41	48	48	44	45	41	41	38	37	4	4	8	9	11	13
McAllen-Edinburg-Mission, TX	94	110	110	101	102	93	93	85	85	9	10	19	20	28	29
Napa, CA	23	27	27	25	25	23	23	21	20	2	2	4	5	7	7
New York-Newark-Jersey City, NY-NJ-PA	3,540	4,020	4,130	3,750	3,810	3,480	3,480	3,200	3,160	310	368	616	730	918	1,090
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	1,000	1,040	1,070	971	958	898	846	823	732	84	127	167	251	249	374
San Luis Obispo-Paso Robles-Arroyo Grande, CA	29	33	33	30	30	28	28	25	25	3	3	6	6	8	9
St. Louis, MO-IL	569	656	668	610	617	564	565	518	512	53	60	106	119	158	178
Weirton-Steubenville, WV-OH	44	44	45	41	41	38	37	35	33	4	4	7	9	10	13
Wheeling, WV-OH	48	56	56	51	52	47	47	43	43	5	5	10	10	14	15
<b>Totals</b>	<b>13,300</b>	<b>14,300</b>	<b>14,600</b>	<b>13,300</b>	<b>13,300</b>	<b>12,300</b>	<b>12,100</b>	<b>11,300</b>	<b>10,900</b>	<b>1,140</b>	<b>1,400</b>	<b>2,270</b>	<b>2,770</b>	<b>3,390</b>	<b>4,130</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-24. CBSA level results for the 30 annual-controlled urban study areas using the Pope et al., 2015 long-term IHD mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	152	163	167	152	153	141	138	130	123	13	17	25	33	38	50
Altoona, PA	29	34	34	31	32	29	29	26	26	3	3	6	6	9	9
Atlanta-Sandy Springs-Roswell, GA	390	435	447	407	413	379	378	350	342	32	40	64	79	96	118
Birmingham-Hoover, AL	154	162	167	152	150	142	133	131	116	12	19	23	38	34	57
Canton-Massillon, OH	85	93	95	87	87	80	79	73	71	8	9	16	18	23	27
Chicago-Naperville-Elgin, IL-IN-WI	1,250	1,340	1,350	1,240	1,250	1,150	1,140	1,050	1,030	107	120	213	239	317	356
Cincinnati, OH-KY-IN	313	344	352	321	323	297	293	273	263	27	34	54	67	80	99
Cleveland-Elyria, OH	411	408	406	382	367	357	327	331	286	29	45	58	89	87	133
Detroit-Warren-Dearborn, MI	967	1,020	1,040	947	941	871	839	794	736	86	115	172	227	256	338
El Centro, CA	20	19	19	18	18	16	16	14	15	2	2	3	3	5	5
Elkhart-Goshen, IN	40	46	46	42	43	39	39	35	35	4	4	8	8	12	12
Evansville, IN-KY	57	66	67	61	62	57	57	52	51	6	6	11	13	16	19
Houston-The Woodlands-Sugar Land, TX	642	682	703	635	634	588	564	540	494	54	79	107	157	160	234
Indianapolis-Carmel-Anderson, IN	266	276	279	258	256	239	234	220	211	21	26	42	51	63	76
Johnstown, PA	37	40	42	38	38	35	34	32	31	3	4	6	8	10	12
Lancaster, PA	103	97	96	90	87	82	78	75	69	8	10	16	20	25	30
Las Vegas-Henderson-Paradise, NV	153	175	178	162	164	149	150	136	135	14	16	28	31	42	46
Lebanon, PA	24	26	26	24	24	22	22	20	20	2	2	4	5	6	7
Little Rock-North Little Rock-Conway, AR	94	109	110	101	101	92	92	83	83	10	10	19	20	29	30
Los Angeles-Long Beach-Anaheim, CA	2,120	2,070	2,060	1,920	1,900	1,760	1,740	1,610	1,580	172	183	342	364	510	543
Louisville/Jefferson County, KY-IN	174	192	196	179	180	165	163	152	147	15	19	30	37	45	56
Macon, GA	39	45	46	42	42	39	39	35	35	4	4	7	8	11	12
McAllen-Edinburg-Mission, TX	88	103	104	96	96	88	88	80	80	9	9	18	18	26	27
Napa, CA	22	25	25	23	23	21	21	19	19	2	2	4	4	6	7
New York-Newark-Jersey City, NY-NJ-PA	3,330	3,790	3,890	3,530	3,590	3,280	3,280	3,020	2,970	290	345	578	685	862	1,020
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	944	984	1,010	915	902	845	796	775	688	79	119	156	236	233	351
San Luis Obispo-Paso Robles-Arroyo Grande, CA	28	31	31	28	28	26	26	24	23	3	3	5	5	8	8
St. Louis, MO-IL	536	618	629	575	581	531	532	487	482	50	56	99	112	148	167
Weirton-Steubenville, WV-OH	41	42	42	39	38	36	35	33	31	3	4	7	8	10	12
Wheeling, WV-OH	45	52	53	48	49	44	44	40	40	5	5	9	9	13	14
<b>Totals</b>	<b>12,500</b>	<b>13,500</b>	<b>13,700</b>	<b>12,500</b>	<b>12,600</b>	<b>11,600</b>	<b>11,400</b>	<b>10,600</b>	<b>10,200</b>	<b>1,070</b>	<b>1,310</b>	<b>2,130</b>	<b>2,600</b>	<b>3,180</b>	<b>3,880</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-25. CBSA level results for the 30 annual-controlled urban study areas using the Di et al., 2017b long-term all-cause mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	547	589	602	548	549	507	496	465	441	45	59	90	117	134	174
Altoona, PA	104	123	123	113	113	103	104	94	94	11	11	21	21	32	32
Atlanta-Sandy Springs-Roswell, GA	1,940	2,180	2,240	2,030	2,060	1,890	1,880	1,740	1,700	158	194	314	387	470	578
Birmingham-Hoover, AL	709	745	770	697	692	649	613	601	533	52	85	104	170	156	253
Canton-Massillon, OH	300	329	335	305	307	281	278	256	249	27	31	53	63	80	93
Chicago-Naperville-Elgin, IL-IN-WI	4,220	4,520	4,570	4,200	4,200	3,870	3,840	3,550	3,470	350	396	698	789	1,040	1,180
Cincinnati, OH-KY-IN	1,160	1,280	1,300	1,190	1,190	1,100	1,080	1,000	970	98	120	196	240	293	358
Cleveland-Elyria, OH	1,290	1,280	1,280	1,200	1,150	1,120	1,020	1,030	891	89	138	178	274	266	410
Detroit-Warren-Dearborn, MI	2,430	2,570	2,620	2,380	2,360	2,180	2,100	1,990	1,840	211	283	421	562	630	840
El Centro, CA	51	48	48	44	45	40	41	36	37	4	4	8	8	12	12
Elkhart-Goshen, IN	114	133	133	122	123	112	112	101	101	11	12	23	23	34	35
Evansville, IN-KY	207	242	247	224	226	206	206	188	185	20	22	39	45	59	66
Houston-The Woodlands-Sugar Land, TX	2,130	2,260	2,340	2,100	2,100	1,940	1,870	1,780	1,630	174	256	347	510	519	761
Indianapolis-Carmel-Anderson, IN	950	989	997	921	915	852	832	783	749	74	89	148	178	221	266
Johnstown, PA	120	133	136	123	124	114	112	104	100	10	13	21	26	31	39
Lancaster, PA	397	374	370	346	334	317	299	288	263	31	38	62	76	93	114
Las Vegas-Henderson-Paradise, NV	543	622	633	575	582	529	531	482	479	49	54	98	108	146	161
Lebanon, PA	95	102	102	94	94	86	86	78	77	8	9	17	18	25	27
Little Rock-North Little Rock-Conway, AR	354	411	415	378	381	345	346	312	311	36	37	71	75	107	111
Los Angeles-Long Beach-Anaheim, CA	5,280	5,150	5,140	4,770	4,730	4,380	4,320	3,990	3,900	418	445	832	887	1,240	1,330
Louisville/Jefferson County, KY-IN	731	813	829	754	759	695	688	636	617	64	77	127	152	190	228
Macon, GA	129	149	152	138	140	128	128	117	115	12	13	23	26	35	39
McAllen-Edinburg-Mission, TX	213	251	252	231	232	212	212	192	192	21	22	42	44	62	65
Napa, CA	87	99	100	92	92	84	84	77	76	8	9	16	17	24	26
New York-Newark-Jersey City, NY-NJ-PA	7,690	8,770	9,020	8,170	8,310	7,570	7,580	6,960	6,850	649	781	1,290	1,560	1,940	2,320
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3,260	3,400	3,480	3,160	3,110	2,910	2,740	2,660	2,360	266	401	530	798	792	1,190
San Luis Obispo-Paso Robles-Arroyo Grande, CA	108	120	121	111	111	101	101	92	91	10	10	20	21	30	31
St. Louis, MO-IL	1,590	1,840	1,870	1,710	1,730	1,570	1,580	1,440	1,420	144	163	287	325	429	485
Weirton-Steubenville, WV-OH	102	104	104	96	95	89	86	82	76	8	10	16	20	24	30
Wheeling, WV-OH	124	144	145	133	133	122	122	110	110	12	13	24	25	36	37
<b>Totals</b>	<b>37,000</b>	<b>39,800</b>	<b>40,500</b>	<b>36,900</b>	<b>37,000</b>	<b>34,100</b>	<b>33,500</b>	<b>31,200</b>	<b>29,900</b>	<b>3,070</b>	<b>3,800</b>	<b>6,120</b>	<b>7,560</b>	<b>9,150</b>	<b>11,300</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-26. CBSA level results for the 30 annual-controlled urban study areas using the Pope et al., 2015 long-term all-cause mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	595	641	656	596	598	551	539	506	479	49	63	97	126	145	188
Altoona, PA	107	126	126	116	116	106	106	96	96	11	11	22	22	32	33
Atlanta-Sandy Springs-Roswell, GA	2,310	2,590	2,660	2,420	2,450	2,240	2,230	2,070	2,020	186	229	371	457	555	683
Birmingham-Hoover, AL	831	874	903	817	811	761	717	704	623	61	100	121	198	181	296
Canton-Massillon, OH	318	349	355	323	325	297	294	271	263	28	33	56	66	84	98
Chicago-Naperville-Elgin, IL-IN-WI	4,660	4,990	5,040	4,630	4,640	4,270	4,230	3,910	3,820	384	434	767	866	1,150	1,290
Cincinnati, OH-KY-IN	1,310	1,440	1,480	1,340	1,350	1,240	1,220	1,130	1,100	110	136	220	270	329	404
Cleveland-Elyria, OH	1,390	1,380	1,370	1,290	1,230	1,200	1,100	1,110	956	96	147	191	293	285	438
Detroit-Warren-Dearborn, MI	2,720	2,880	2,940	2,660	2,640	2,440	2,350	2,220	2,050	235	314	469	625	702	933
El Centro, CA	59	56	56	51	52	47	47	42	42	5	5	10	10	14	14
Elkhart-Goshen, IN	125	146	146	134	135	123	123	111	111	12	13	25	25	37	38
Evansville, IN-KY	229	268	273	248	250	228	228	207	205	22	25	43	49	65	73
Houston-The Woodlands-Sugar Land, TX	2,590	2,760	2,850	2,560	2,560	2,360	2,270	2,170	1,980	211	310	421	617	629	922
Indianapolis-Carmel-Anderson, IN	1,080	1,130	1,130	1,050	1,040	968	946	889	851	84	101	168	201	251	300
Johnstown, PA	126	139	143	129	130	119	118	109	105	11	14	21	27	32	40
Lancaster, PA	402	378	373	349	337	320	301	290	265	31	38	62	77	93	114
Las Vegas-Henderson-Paradise, NV	631	723	737	669	677	615	617	560	557	57	63	113	125	170	187
Lebanon, PA	97	104	105	96	96	88	87	80	79	9	9	17	19	26	28
Little Rock-North Little Rock-Conway, AR	414	481	486	443	446	404	405	365	364	42	44	83	87	124	130
Los Angeles-Long Beach-Anaheim, CA	5,800	5,660	5,650	5,230	5,200	4,810	4,740	4,380	4,280	456	486	909	969	1,360	1,450
Louisville/Jefferson County, KY-IN	841	935	954	867	872	799	791	730	708	73	88	145	174	217	261
Macon, GA	153	177	180	164	166	151	151	139	137	14	16	27	31	41	46
McAllen-Edinburg-Mission, TX	243	286	288	264	265	241	241	219	218	24	25	47	49	71	74
Napa, CA	89	102	103	94	95	87	86	79	78	8	9	17	18	25	26
New York-Newark-Jersey City, NY-NJ-PA	8,230	9,400	9,670	8,750	8,890	8,100	8,110	7,450	7,330	694	831	1,380	1,660	2,070	2,480
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	3,570	3,730	3,820	3,460	3,410	3,190	3,000	2,910	2,580	290	438	578	872	864	1,300
San Luis Obispo-Paso Robles-Arroyo Grande, CA	112	125	125	115	115	105	105	95	95	10	11	21	21	31	32
St. Louis, MO-IL	1,750	2,030	2,070	1,880	1,900	1,740	1,740	1,590	1,570	158	179	314	356	470	532
Weirton-Steubenville, WV-OH	112	114	115	106	105	98	94	90	84	9	11	17	22	26	33
Wheeling, WV-OH	129	150	151	138	139	127	127	115	114	13	13	25	26	38	39
<b>Totals</b>	<b>41,000</b>	<b>44,200</b>	<b>45,000</b>	<b>41,000</b>	<b>41,000</b>	<b>37,800</b>	<b>37,100</b>	<b>34,600</b>	<b>33,200</b>	<b>3,390</b>	<b>4,190</b>	<b>6,760</b>	<b>8,350</b>	<b>10,100</b>	<b>12,500</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)



**Table C-27. CBSA level results for the 30 annual-controlled urban study areas using the Thurston et al., 2016 long-term all-cause mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	157	169	173	157	157	145	142	133	126	13	16	25	33	37	49
Altoona, PA	27	32	33	30	30	27	27	25	25	3	3	5	5	8	8
Atlanta-Sandy Springs-Roswell, GA	644	725	746	676	685	626	624	577	562	51	63	102	126	152	188
Birmingham-Hoover, AL	231	243	252	227	225	211	199	195	172	16	27	33	55	49	82
Canton-Massillon, OH	84	92	94	85	86	78	77	71	69	7	9	14	17	22	26
Chicago-Naperville-Elgin, IL-IN-WI	1,220	1,310	1,320	1,210	1,210	1,120	1,110	1,020	996	99	112	197	223	295	334
Cincinnati, OH-KY-IN	353	390	400	362	365	334	330	306	294	29	36	58	72	87	108
Cleveland-Elyria, OH	359	357	355	333	319	310	282	286	246	24	37	48	75	73	112
Detroit-Warren-Dearborn, MI	717	761	776	702	697	643	618	583	538	61	81	121	162	182	243
El Centro, CA	16	16	16	14	14	13	13	12	12	1	1	3	3	4	4
Elkhart-Goshen, IN	33	39	39	36	36	33	33	29	29	3	3	6	7	10	10
Evansville, IN-KY	62	72	74	67	68	61	61	56	55	6	7	11	13	17	19
Houston-The Woodlands-Sugar Land, TX	729	776	803	720	720	664	636	607	552	58	86	116	171	174	256
Indianapolis-Carmel-Anderson, IN	293	305	308	284	282	262	256	240	230	22	27	45	54	67	80
Johnstown, PA	31	34	35	32	32	29	29	27	26	3	3	5	7	8	10
Lancaster, PA	97	91	90	84	81	77	72	69	63	7	9	15	18	22	27
Las Vegas-Henderson-Paradise, NV	186	214	218	197	200	181	182	165	164	17	18	33	37	50	55
Lebanon, PA	25	26	26	24	24	22	22	20	20	2	2	4	5	6	7
Little Rock-North Little Rock-Conway, AR	116	135	137	124	125	113	113	102	102	11	12	23	24	34	36
Los Angeles-Long Beach-Anaheim, CA	1,470	1,430	1,430	1,320	1,310	1,210	1,190	1,100	1,080	113	120	225	240	338	360
Louisville/Jefferson County, KY-IN	231	258	263	239	240	220	217	201	194	20	24	39	47	59	71
Macon, GA	43	51	52	47	47	43	43	39	39	4	4	8	9	11	13
McAllen-Edinburg-Mission, TX	66	78	79	72	72	66	66	59	59	6	7	13	13	19	20
Napa, CA	22	25	26	23	24	21	21	19	19	2	2	4	4	6	6
New York-Newark-Jersey City, NY-NJ-PA	2,070	2,370	2,440	2,200	2,240	2,030	2,040	1,870	1,840	172	205	343	410	514	615
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	912	953	977	883	870	812	763	741	655	73	110	145	219	217	328
San Luis Obispo-Paso Robles-Arroyo Grande, CA	27	30	30	27	27	25	25	23	23	2	3	5	5	7	8
St. Louis, MO-IL	463	539	550	499	505	460	460	420	415	41	46	82	93	122	139
Weirton-Steubenville, WV-OH	31	32	32	30	29	27	26	25	23	2	3	5	6	7	9
Wheeling, WV-OH	34	40	40	37	37	34	34	30	30	3	3	7	7	10	10
<b>Totals</b>	<b>10,700</b>	<b>11,600</b>	<b>11,800</b>	<b>10,700</b>	<b>10,800</b>	<b>9,900</b>	<b>9,710</b>	<b>9,050</b>	<b>8,650</b>	<b>871</b>	<b>1,080</b>	<b>1,740</b>	<b>2,160</b>	<b>2,610</b>	<b>3,230</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-28. CBSA level results for the 30 annual-controlled urban study areas using the Turner et al., 2016 long-term lung cancer mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	48	51	52	48	48	44	43	41	38	4	5	8	10	12	15
Altoona, PA	6	7	7	7	7	6	6	6	6	1	1	1	1	2	2
Atlanta-Sandy Springs-Roswell, GA	183	204	210	191	194	178	177	164	160	15	18	29	36	44	54
Birmingham-Hoover, AL	63	66	69	62	62	58	55	54	47	5	8	9	15	14	23
Canton-Massillon, OH	25	28	28	26	26	24	24	22	21	2	3	5	5	7	8
Chicago-Naperville-Elgin, IL-IN-WI	379	406	410	377	378	348	345	319	312	32	36	63	71	94	106
Cincinnati, OH-KY-IN	122	134	137	125	126	115	114	106	102	10	13	20	26	31	38
Cleveland-Elyria, OH	111	111	110	103	99	96	88	89	77	8	12	15	24	23	35
Detroit-Warren-Dearborn, MI	220	233	237	215	214	198	190	180	166	19	26	38	51	57	76
El Centro, CA	4	4	4	3	3	3	3	3	3	0	0	1	1	1	1
Elkhart-Goshen, IN	10	11	11	10	10	9	9	9	9	1	1	2	2	3	3
Evansville, IN-KY	19	22	23	21	21	19	19	17	17	2	2	4	4	5	6
Houston-The Woodlands-Sugar Land, TX	194	206	213	191	191	177	170	162	148	16	24	31	47	47	70
Indianapolis-Carmel-Anderson, IN	102	106	107	99	98	91	89	84	80	8	10	16	19	24	29
Johnstown, PA	8	9	9	9	9	8	8	7	7	1	1	1	2	2	3
Lancaster, PA	28	26	26	24	23	22	21	20	18	2	3	4	5	6	8
Las Vegas-Henderson-Paradise, NV	55	63	64	58	59	53	53	49	48	5	5	10	11	15	16
Lebanon, PA	9	9	9	8	8	8	8	7	7	1	1	2	2	2	2
Little Rock-North Little Rock-Conway, AR	37	43	43	39	40	36	36	33	33	4	4	7	8	11	12
Los Angeles-Long Beach-Anaheim, CA	360	351	351	325	323	299	295	272	266	29	30	57	61	85	91
Louisville/Jefferson County, KY-IN	82	91	93	85	85	78	78	72	69	7	9	14	17	21	26
Macon, GA	13	15	15	14	14	13	13	11	11	1	1	2	3	3	4
McAllen-Edinburg-Mission, TX	11	13	13	12	12	11	11	10	10	1	1	2	2	3	3
Napa, CA	7	8	8	7	7	6	6	6	6	1	1	1	1	2	2
New York-Newark-Jersey City, NY-NJ-PA	590	672	691	626	637	580	581	534	525	50	60	99	119	148	178
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	284	296	303	275	271	253	238	232	205	23	35	46	70	69	104
San Luis Obispo-Paso Robles-Arroyo Grande, CA	8	9	9	8	8	7	7	7	7	1	1	1	2	2	2
St. Louis, MO-IL	158	182	186	169	171	156	157	143	142	14	16	28	32	42	48
Weirton-Steubenville, WV-OH	9	10	10	9	9	8	8	8	7	1	1	1	2	2	3
Wheeling, WV-OH	11	12	12	11	11	10	10	9	9	1	1	2	2	3	3
<b>Totals</b>	<b>3,150</b>	<b>3,400</b>	<b>3,460</b>	<b>3,160</b>	<b>3,160</b>	<b>2,920</b>	<b>2,860</b>	<b>2,670</b>	<b>2,560</b>	<b>262</b>	<b>327</b>	<b>522</b>	<b>651</b>	<b>780</b>	<b>972</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-29. CBSA level results for the 30 annual-controlled urban study areas using the Baxter et al., 2017 all-cause short-term mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	29	31	32	29	29	27	26	25	23	2	3	5	6	7	9
Altoona, PA	5	6	6	6	6	5	5	5	5	1	1	1	1	2	2
Atlanta-Sandy Springs-Roswell, GA	111	126	129	117	119	108	108	100	97	9	11	18	22	26	32
Birmingham-Hoover, AL	40	42	44	40	39	37	35	34	30	3	5	6	9	9	14
Canton-Massillon, OH	15	17	17	16	16	14	14	13	13	1	2	3	3	4	5
Chicago-Naperville-Elgin, IL-IN-WI	228	245	248	227	227	208	206	190	186	18	21	37	41	55	62
Cincinnati, OH-KY-IN	63	70	71	64	65	59	59	54	52	5	6	10	13	15	19
Cleveland-Elyria, OH	68	67	67	63	60	58	53	54	46	5	7	9	14	14	21
Detroit-Warren-Dearborn, MI	132	140	143	129	128	118	113	107	99	11	15	22	30	33	44
El Centro, CA	3	3	3	2	2	2	2	2	2	0	0	0	0	1	1
Elkhart-Goshen, IN	6	7	7	7	7	6	6	5	5	1	1	1	1	2	2
Evansville, IN-KY	11	13	13	12	12	11	11	10	10	1	1	2	2	3	3
Houston-The Woodlands-Sugar Land, TX	126	134	139	124	124	114	109	104	95	10	15	20	29	30	44
Indianapolis-Carmel-Anderson, IN	52	54	55	51	50	47	46	43	41	4	5	8	9	12	14
Johnstown, PA	6	7	7	6	6	6	6	5	5	0	1	1	1	1	2
Lancaster, PA	20	18	18	17	16	16	15	14	13	1	2	3	4	4	5
Las Vegas-Henderson-Paradise, NV	30	34	35	32	32	29	29	26	26	3	3	5	6	8	9
Lebanon, PA	5	5	5	5	5	4	4	4	4	0	0	1	1	1	1
Little Rock-North Little Rock-Conway, AR	20	23	24	21	22	20	20	18	18	2	2	4	4	6	6
Los Angeles-Long Beach-Anaheim, CA	284	277	277	255	254	234	231	212	208	22	23	43	46	65	69
Louisville/Jefferson County, KY-IN	41	45	46	42	42	38	38	35	34	3	4	7	8	10	12
Macon, GA	7	9	9	8	8	7	7	7	7	1	1	1	1	2	2
McAllen-Edinburg-Mission, TX	12	14	14	13	13	12	12	11	11	1	1	2	2	3	4
Napa, CA	4	5	5	5	5	4	4	4	4	0	0	1	1	1	1
New York-Newark-Jersey City, NY-NJ-PA	401	459	473	427	434	394	394	361	355	33	39	66	79	99	118
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	172	180	184	166	164	153	144	139	123	14	21	27	41	41	61
San Luis Obispo-Paso Robles-Arroyo Grande, CA	5	6	6	5	5	5	5	5	4	0	0	1	1	1	1
St. Louis, MO-IL	84	98	100	90	91	83	83	76	75	7	8	15	17	22	25
Weirton-Steubenville, WV-OH	5	5	6	5	5	5	4	4	4	0	1	1	1	1	2
Wheeling, WV-OH	6	7	7	7	7	6	6	6	6	1	1	1	1	2	2
<b>Totals</b>	<b>1,990</b>	<b>2,150</b>	<b>2,190</b>	<b>1,990</b>	<b>1,990</b>	<b>1,830</b>	<b>1,790</b>	<b>1,670</b>	<b>1,600</b>	<b>160</b>	<b>197</b>	<b>319</b>	<b>394</b>	<b>478</b>	<b>592</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-30. CBSA level results for the 30 annual-controlled urban study areas using the Ito et al., 2013 all-cause short-term mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	14	15	15	14	14	13	12	12	11	1	1	2	3	3	4
Altoona, PA	2	3	3	3	3	2	2	2	2	0	0	0	0	1	1
Atlanta-Sandy Springs-Roswell, GA	53	60	62	56	57	52	52	48	46	4	5	8	10	13	15
Birmingham-Hoover, AL	19	20	21	19	19	18	17	16	14	1	2	3	4	4	7
Canton-Massillon, OH	7	8	8	7	7	7	7	6	6	1	1	1	1	2	2
Chicago-Naperville-Elgin, IL-IN-WI	107	115	116	106	106	98	97	89	87	9	10	17	19	26	29
Cincinnati, OH-KY-IN	30	33	34	31	31	28	28	26	25	2	3	5	6	7	9
Cleveland-Elyria, OH	32	31	31	29	28	27	25	25	22	2	3	4	7	6	10
Detroit-Warren-Dearborn, MI	62	66	68	61	61	56	54	51	47	5	7	10	14	16	21
El Centro, CA	1	1	1	1	1	1	1	1	1	0	0	0	0	0	0
Elkhart-Goshen, IN	3	3	3	3	3	3	3	3	3	0	0	1	1	1	1
Evansville, IN-KY	5	6	6	6	6	5	5	5	5	0	1	1	1	1	2
Houston-The Woodlands-Sugar Land, TX	61	65	67	60	60	55	53	50	46	5	7	10	14	14	21
Indianapolis-Carmel-Anderson, IN	25	26	26	24	24	22	22	20	20	2	2	4	5	6	7
Johnstown, PA	3	3	3	3	3	3	3	2	2	0	0	0	1	1	1
Lancaster, PA	9	9	9	8	8	7	7	7	6	1	1	1	2	2	3
Las Vegas-Henderson-Paradise, NV	14	16	17	15	15	14	14	13	13	1	1	3	3	4	4
Lebanon, PA	2	2	2	2	2	2	2	2	2	0	0	0	0	1	1
Little Rock-North Little Rock-Conway, AR	10	11	11	10	10	9	9	8	8	1	1	2	2	3	3
Los Angeles-Long Beach-Anaheim, CA	133	130	129	120	119	109	108	99	97	10	11	20	22	30	32
Louisville/Jefferson County, KY-IN	19	22	22	20	20	18	18	17	16	2	2	3	4	5	6
Macon, GA	4	4	4	4	4	3	3	3	3	0	0	1	1	1	1
McAllen-Edinburg-Mission, TX	6	7	7	6	6	6	6	5	5	1	1	1	1	2	2
Napa, CA	2	2	2	2	2	2	2	2	2	0	0	0	0	1	1
New York-Newark-Jersey City, NY-NJ-PA	187	214	220	199	202	184	184	168	165	15	18	31	37	46	55
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	82	86	88	79	78	73	68	66	59	6	10	13	19	19	29
San Luis Obispo-Paso Robles-Arroyo Grande, CA	3	3	3	3	3	2	2	2	2	0	0	0	0	1	1
St. Louis, MO-IL	40	47	48	43	44	40	40	36	36	4	4	7	8	11	12
Weirton-Steubenville, WV-OH	3	3	3	2	2	2	2	2	2	0	0	0	0	1	1
Wheeling, WV-OH	3	3	3	3	3	3	3	3	3	0	0	1	1	1	1
<b>Totals</b>	<b>940</b>	<b>1,010</b>	<b>1,030</b>	<b>939</b>	<b>940</b>	<b>864</b>	<b>847</b>	<b>789</b>	<b>754</b>	<b>75</b>	<b>93</b>	<b>150</b>	<b>186</b>	<b>226</b>	<b>279</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

**Table C-31. CBSA level results for the 30 annual-controlled urban study areas using the Zanobetti et al., 2014 all-cause short-term mortality CR function.**

CBSA	Absolute Risk									Risk Reduction (Relative to Current Standard)					
	Current Conditions (2015)	Current Standard (12 µg/m³)		Alternative Annual Standard (11 µg/m³)		Alternative Annual Standard (10 µg/m³)		Alternative Annual Standard (9 µg/m³)		Alternative Annual Standard (12-11 µg/m³)		Alternative Annual Standard (12-10 µg/m³)		Alternative Annual Standard (12-9 µg/m³)	
		Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM	Pri-PM	Sec-PM
Akron, OH	45	49	50	45	45	42	41	38	36	4	5	7	9	11	14
Altoona, PA	9	10	10	9	9	9	9	8	8	1	1	2	2	3	3
Atlanta-Sandy Springs-Roswell, GA	159	180	185	167	170	155	154	142	139	13	15	25	31	38	46
Birmingham-Hoover, AL	58	62	64	57	57	53	50	49	44	4	7	8	14	12	20
Canton-Massillon, OH	25	27	28	25	25	23	23	21	20	2	3	4	5	6	7
Chicago-Naperville-Elgin, IL-IN-WI	348	373	377	345	346	318	315	290	284	28	32	56	63	83	94
Cincinnati, OH-KY-IN	95	105	108	98	98	90	89	82	79	8	10	16	19	23	29
Cleveland-Elyria, OH	106	106	105	99	94	92	83	85	73	7	11	14	22	21	33
Detroit-Warren-Dearborn, MI	200	212	216	196	194	179	172	162	149	17	22	34	45	50	67
El Centro, CA	4	4	4	4	4	3	3	3	3	0	0	1	1	1	1
Elkhart-Goshen, IN	9	11	11	10	10	9	9	8	8	1	1	2	2	3	3
Evansville, IN-KY	17	20	21	19	19	17	17	15	15	2	2	3	4	5	5
Houston-The Woodlands-Sugar Land, TX	175	187	193	173	173	160	153	146	133	14	20	28	41	41	61
Indianapolis-Carmel-Anderson, IN	78	82	82	76	75	70	68	64	61	6	7	12	14	18	21
Johnstown, PA	10	11	11	10	10	9	9	9	8	1	1	2	2	2	3
Lancaster, PA	33	31	31	28	28	26	24	24	21	2	3	5	6	7	9
Las Vegas-Henderson-Paradise, NV	44	51	52	47	47	43	43	39	39	4	4	8	9	12	13
Lebanon, PA	8	8	8	8	8	7	7	6	6	1	1	1	1	2	2
Little Rock-North Little Rock-Conway, AR	29	34	34	31	31	28	28	26	25	3	3	6	6	9	9
Los Angeles-Long Beach-Anaheim, CA	435	425	424	392	389	359	354	326	319	33	35	66	71	99	106
Louisville/Jefferson County, KY-IN	60	67	69	62	63	57	57	52	50	5	6	10	12	15	18
Macon, GA	11	12	13	11	12	11	11	10	9	1	1	2	2	3	3
McAllen-Edinburg-Mission, TX	17	21	21	19	19	17	17	16	16	2	2	3	3	5	5
Napa, CA	7	8	8	8	8	7	7	6	6	1	1	1	1	2	2
New York-Newark-Jersey City, NY-NJ-PA	630	722	743	671	682	619	620	568	559	52	62	103	124	154	186
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	268	280	287	259	255	238	224	217	192	21	32	42	64	63	96
San Luis Obispo-Paso Robles-Arroyo Grande, CA	9	10	10	9	9	8	8	7	7	1	1	2	2	2	2
St. Louis, MO-IL	131	152	155	141	142	129	130	118	117	11	13	23	26	34	39
Weirton-Steubenville, WV-OH	8	9	9	8	8	7	7	7	6	1	1	1	2	2	2
Wheeling, WV-OH	10	12	12	11	11	10	10	9	9	1	1	2	2	3	3
<b>Totals</b>	<b>3,040</b>	<b>3,280</b>	<b>3,340</b>	<b>3,040</b>	<b>3,040</b>	<b>2,790</b>	<b>2,740</b>	<b>2,550</b>	<b>2,440</b>	<b>244</b>	<b>301</b>	<b>487</b>	<b>603</b>	<b>731</b>	<b>904</b>

\* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

### **C.2.2 Impact of Alternative Standards on the Distribution of Risk Across Ambient PM<sub>2.5</sub> Levels**

The tables of risk results presented in section C.2.1 illustrate the estimated risk of premature death under current and alternative PM<sub>2.5</sub> standards. As the city-specific results indicate, both total risk and risk reductions estimated to occur under alternative standards can vary substantially by urban area. This is due to differences in underlying demographics (e.g., size and age of population), health status (e.g., underlying death rates) and exposure (air quality conditions). Furthermore, each of these CBSA estimates represents an aggregation of underlying 12 km grid cell results, masking the underlying variability in the distribution of risk under different scenarios. Thus, it can be challenging to understand how patterns of risk are changing under air quality simulated to just meet the current or alternative standards.

To better illustrate the distribution of risk under the current standards, and how that distribution changes under potential alternative standards, this section presents graphics depicting these changes both in aggregate and at the grid-cell level. It would be possible to illustrate these changes separately for each endpoint and CR function, as was done numerically in the tables in section C.2.1. However, because the pattern of risk and risk reduction is similar across endpoints, we have chosen to focus on a single endpoint to illustrate the changes graphically. Consequently, as with the graphics presented in the PA section 3.3.2, the graphics presented in this section are also based on long-term exposure-related IHD mortality modeled using effect estimates obtained from Jerrett et al. (2016). The first set of graphics presented in this section (Figure C-25, Figure C-26, Figure C-27, Figure C-28, and Figure C-29) include results for the full set of 47 urban study areas and the second set (Figure C-30 and Figure C-31) include results for the 30 annual-controlled study areas. These graphical plots include:

- Line graphs showing the distribution of gridded risk estimates across annual-averaged PM<sub>2.5</sub> concentrations (Figure C-25 and Figure C-30). These figures allow the reader to consider how the distribution of risk shifts when simulating air quality that just meets the current standard (12/35 µg/m<sup>3</sup>) relative to 2015 current conditions and subsequently how that distribution of risk shifts downward when simulating air quality that just meets alternative standards of 10/30 µg/m<sup>3</sup>.
- Maps showing the 12 km grid-level risk estimates associated with each of the 47 urban study areas. In these representative maps each grid cell is shown as a square, with the color of the square going from green (lower risk estimates) to red (higher risk estimate) colors. The center of the color scales (the beginning of yellow) has been set to a risk estimate of two premature deaths. This means that green squares represent grid cells where 0-1 premature deaths are estimated,

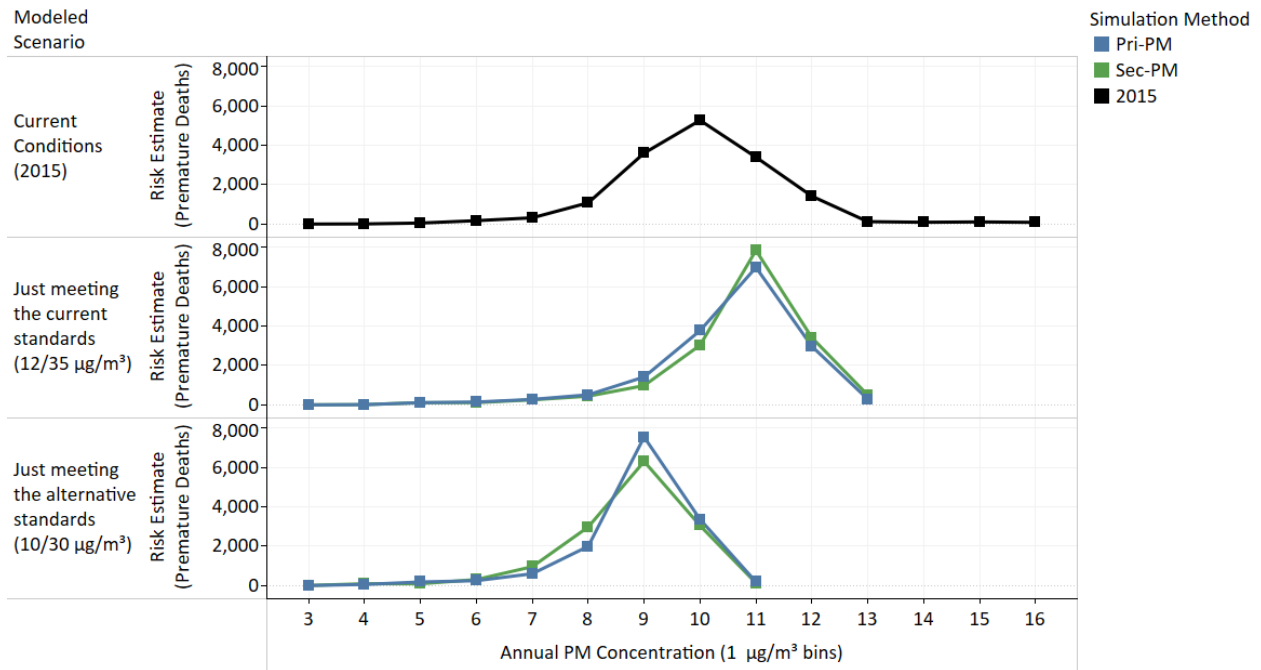
yellow squares represent grid cells in which at least two premature deaths are estimated, and as the color graduation approaches red the number of estimated premature deaths increases. Separate maps are presented for (a) the unadjusted 2015 current conditions simulation (Figure C-26), (b) simulation of the current standard ( $12/35 \mu\text{g}/\text{m}^3$ ) (Figure C-27), and (c) simulation of the change (delta) in risk between the current and alternative standards ( $10/30 \mu\text{g}/\text{m}^3$ ) (Figure C-28). These maps are not repeated for just the 30 area set, as those areas are included in the 47 area maps.

- Scatter plots depicting the distribution of modeled risk by annual-average  $\text{PM}_{2.5}$  concentration (Figure C-29 and Figure C-31). While these scatter plots present similar distributional information as the line graphs, the scatter plots allow for a more detailed consideration of the nature of the risk distribution in relation to ambient  $\text{PM}_{2.5}$  levels. In these figures, each grid cell is shown as a dot, with the frequency of dots shown on a color scale from cool (green – lower frequency) to hot (red – higher frequency) colors.<sup>40</sup> Consequently, it is possible to consider whether, for example, a shift in risk involves a change in the magnitude of risk across higher-risk cells, or in a change in the density of lower risk cells.

Key observations resulting from review of these graphics as well as the CBSA tables presented in section C.2.1 are presented below, following the graphics.

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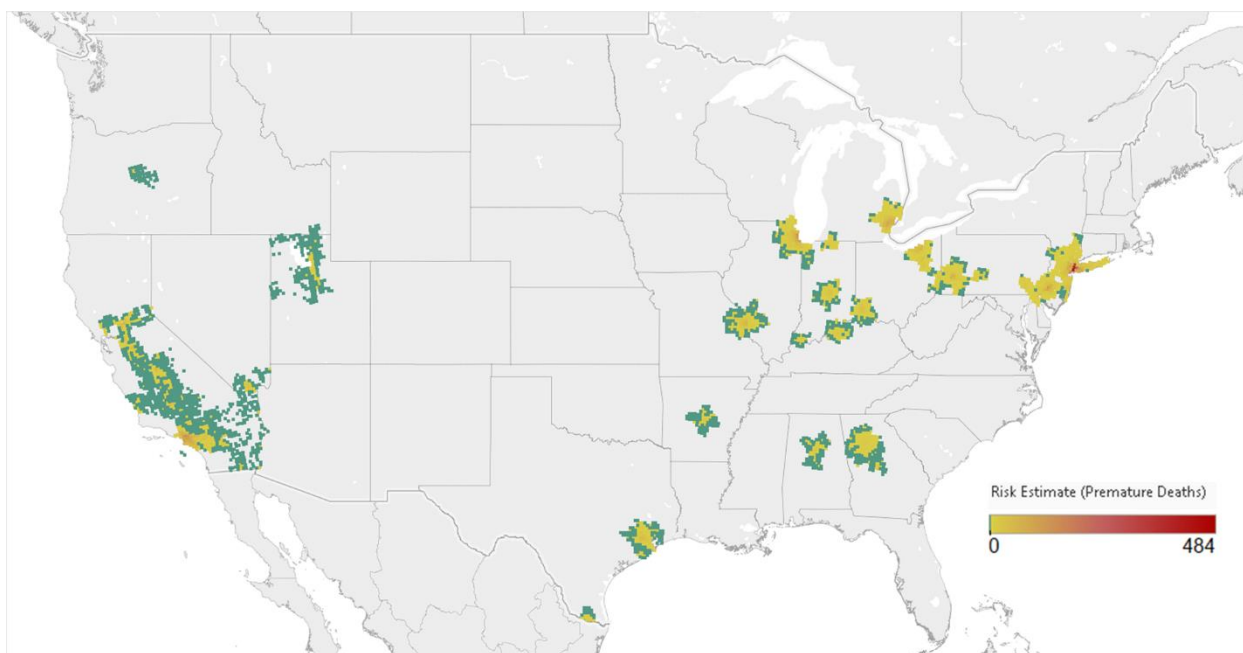
<sup>40</sup> For adjusted air quality, a small amount of risk is estimated at concentrations higher than the level of the annual standard (e.g., some risk is estimated at an average concentration of  $13 \mu\text{g}/\text{m}^3$  when air quality is adjusted to just meet the current standard). This can result because risk estimates are for a single year (i.e., 2015) within the 3-year design value period (i.e., 2014 to 2016). While the three-year average design value is  $12.0 \mu\text{g}/\text{m}^3$ , a single year can have grid cells with annual average concentrations above or below  $12.0 \mu\text{g}/\text{m}^3$ .



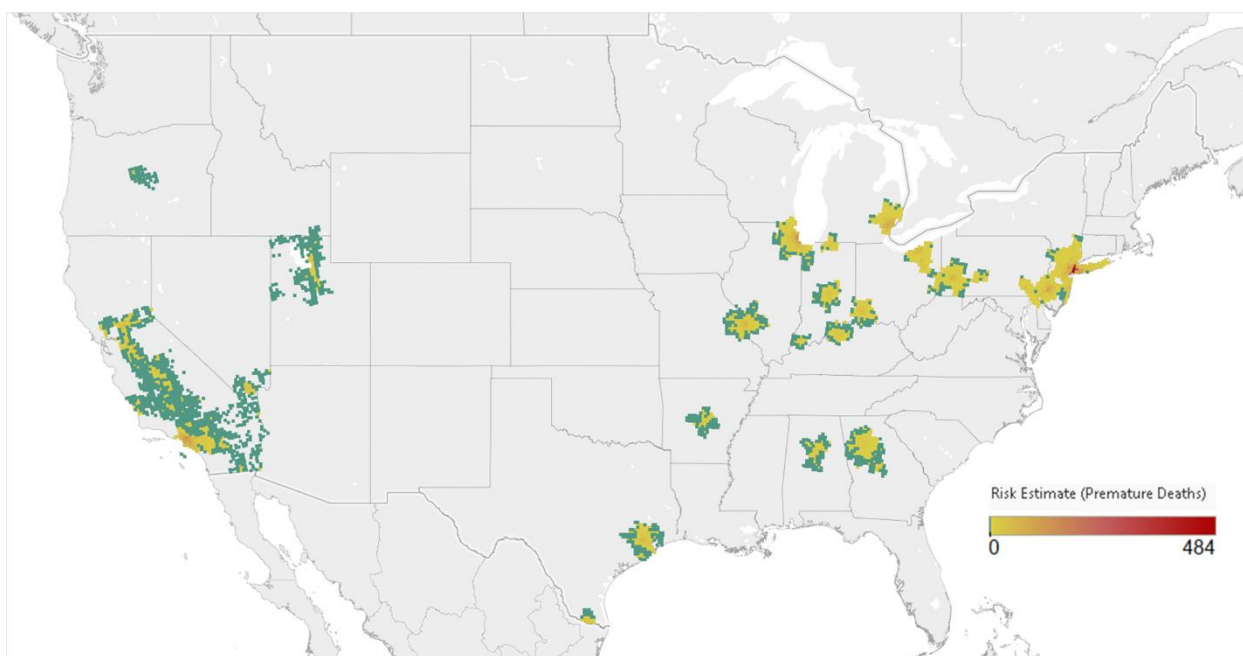
**Figure C-25. Distribution of estimated PM<sub>2.5</sub>-associated mortality for current conditions (2015), current standards (12/35 µg/m<sup>3</sup>), and alternative standards (10/30 µg/m<sup>3</sup>) simulated for all 47 urban study areas.<sup>41</sup>**

<sup>41</sup> Risk is rounded toward zero into whole PM<sub>2.5</sub> concentration values (e.g., risk estimate at 10 µg/m<sup>3</sup> includes risk occurring at 10.0-10.9 µg/m<sup>3</sup>). Blue lines represent the Pri-PM risk estimates, green lines represent the Sec-PM risk estimates, and black lines represent the 2015 current conditions risk estimates.

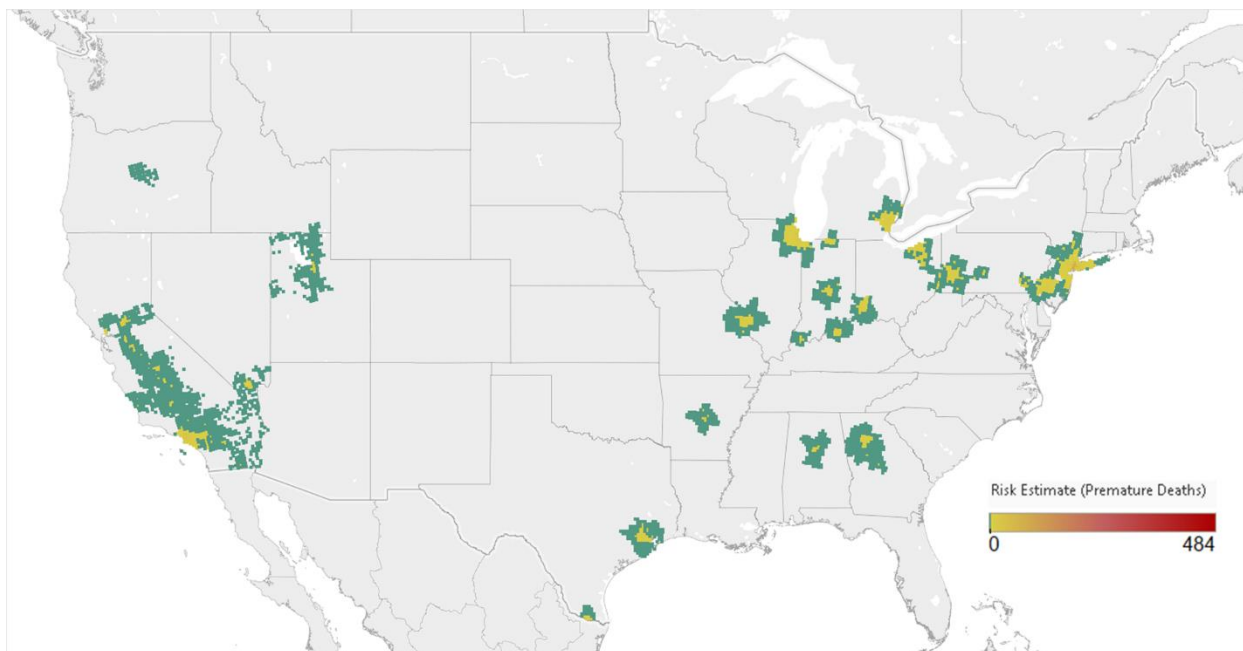




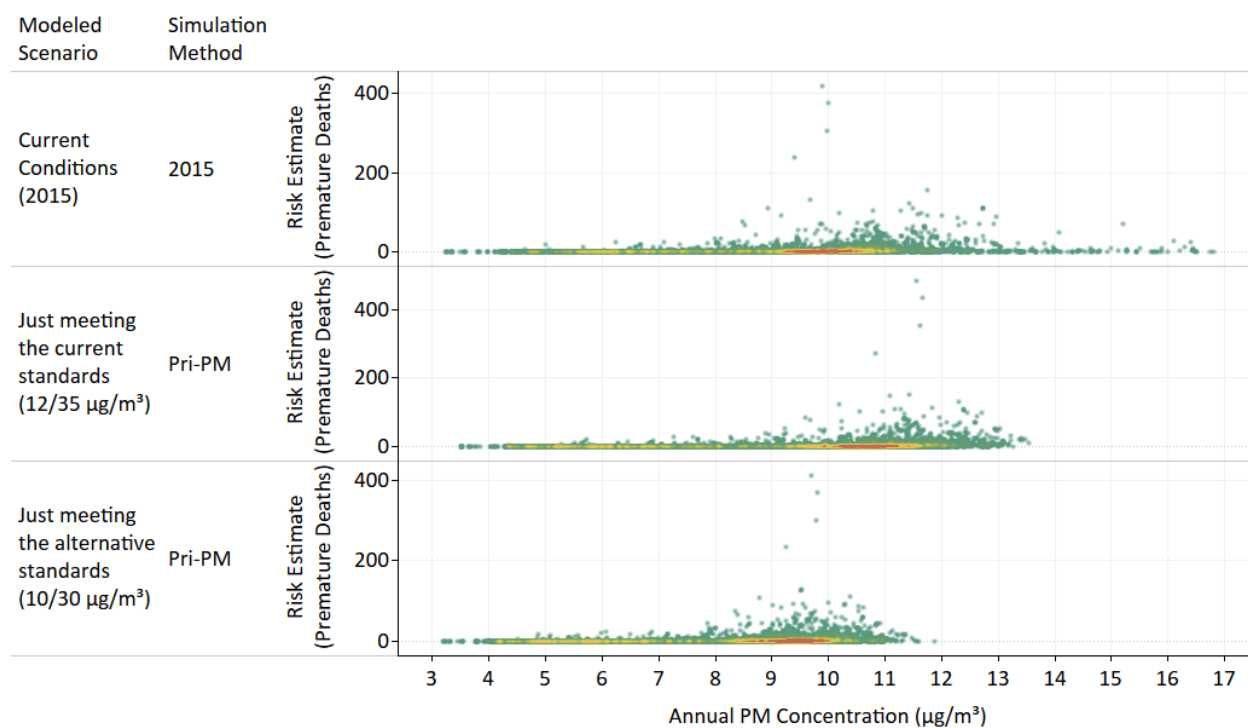
**Figure C-26. Estimated number of premature deaths (by 12 km grid cell) under 2015 current conditions in all 47 study areas.**



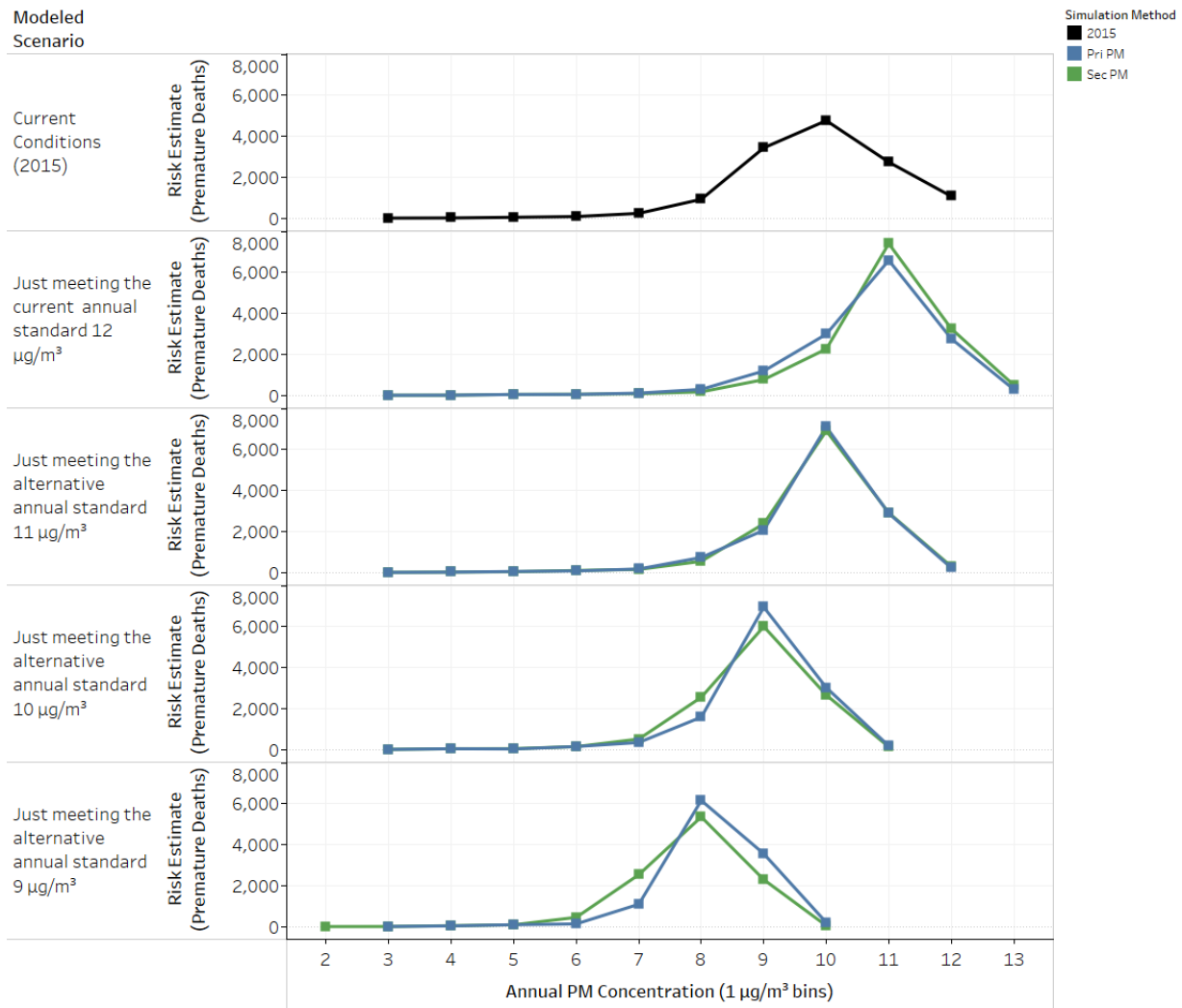
**Figure C-27. Estimated number of premature deaths (by 12 km grid cell) when just meeting the current PM standards (12/35) in all 47 study areas (Pri-PM simulation).**



**Figure C-28. Estimated reduction in the number of premature deaths (by 12 km grid cell) when going from just meeting the current standards (12/35) to just meeting the alternative standards (10/30) in all 47 study areas (Pri-PM simulation).**

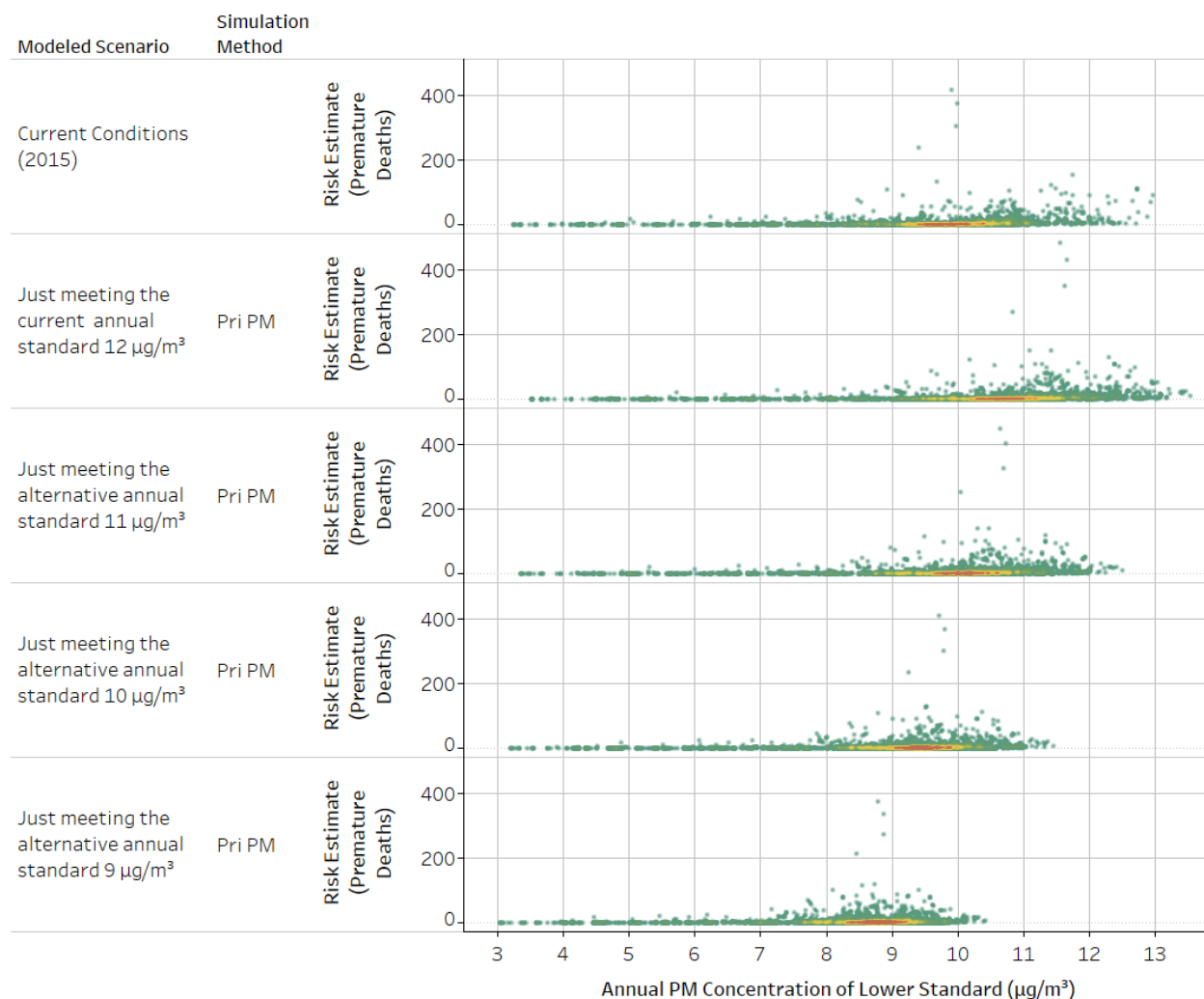


**Figure C-29. Distribution of estimated premature death (by 12 km grid cell) for the current standards (12/35  $\mu\text{g}/\text{m}^3$ ), alternative standards (10/30  $\mu\text{g}/\text{m}^3$ ), and current conditions (2015) for all 47 urban study areas (Pri-PM simulation).**



**Figure C-30. Distribution of estimated PM<sub>2.5</sub>-associated mortality for current conditions (2015), the current annual standard (12/35 µg/m³), and alternative standards (9.0, 10.0, and 11.0 µg/m³) simulated for the 30 annual-controlled urban study areas.<sup>42</sup>**

<sup>42</sup> Risk is rounded toward zero into whole PM<sub>2.5</sub> concentration values (e.g., risk estimate at 10 µg/m³ includes risk occurring at 10.0-10.9 µg/m³). Blue lines represent the Pri-PM risk estimates, green lines represent the Sec-PM risk estimates, and black lines represent the 2015 current conditions risk estimates.



**Figure C-31. Distribution of estimated premature death (by 12 km grid cell) for current conditions (2015), the current annual standard (12.0  $\mu\text{g}/\text{m}^3$ ), alternative annual standards (9.0, 10.0, 11.0  $\mu\text{g}/\text{m}^3$ ), and for all 47 urban study areas (Pri-PM simulation).**

Review of the CBSA-level risk estimates presented in Section C.2.1 along with the distributional risk estimates presented in Section C.2.2 further support the key observations presented in PA section 3.2. Briefly, these observations include:

- Under simulation of the current  $\text{PM}_{2.5}$  standards, long-term annual mortality ranges up to 52,100 premature deaths (all-cause, based on Pope et al., 2015), including 16,800 IHD-related deaths (based on Jerrett et al., 2016) and 3,950 lung cancer deaths (based on Turner et al., 2016) for the full set of 47 urban study areas. Estimates of short-term all-cause annual mortality range up to 3,870 deaths (based on Zanobetti et al., 2014) for the full set of 47 urban study areas (Table C-10).

- In considering the alternative suite of standards (10/30  $\mu\text{g}/\text{m}^3$ ) modeled for the full set of 47 urban study areas, we note that larger risk reductions are estimated for those urban study areas controlled by annual standards, relative to those controlled by the 24-hour standard (Table C-10 and Table C-11).
- Across the full set of alternative annual standards modeled including 11, 10 and 9  $\mu\text{g}/\text{m}^3$  (each evaluated for the 30 annually-controlled study areas), we see a consistent reduction in mortality (Table C-21 and Table C-22). In addition, we note that these risk reductions are associated with iteratively lower ambient  $\text{PM}_{2.5}$  concentrations, such that with the lowest annual standard considered (9  $\mu\text{g}/\text{m}^3$ ) the majority of remaining risk occurs in grid cells with ambient  $\text{PM}_{2.5}$  concentrations between 7 and 9  $\mu\text{g}/\text{m}^3$ . In contrast, most of the risk occurring under the current standard occurs in grid cells with ambient concentrations in the range of 10-12  $\mu\text{g}/\text{m}^3$  (Figure C-29).
- Patterns of risk reduction seen in the summary (aggregated) risk results tables presented both in PA section 3.3 and in section C.2.1 are driven by considerable underlying variability across both CBSAs and across the 12km grid-level risk estimates. Specifically, if we consider the detailed CBSA-level risk estimates presented in section C.2.1, we observe significant variation in the magnitude of modeled risk across the 47 urban study areas. Similarly, if we consider both the maps and scatter plots presented in section C.2.2, we see considerable spread (i.e., variability) in the grid-level risk estimates. We note that this underlying variability in risk (both across CBSAs and across underlying 12km grid cells) reflects local patterns of population density, baseline incidence and modeled ambient  $\text{PM}_{2.5}$  levels. However, it is important to also note that the underlying variability does not result from differences in CR functions, since for all mortality endpoints modeled in this analysis, national-level effect estimates were utilized.
- When considering the shift in the distribution of risks for the alternative standards (Figure C-29 and Figure C-31), we note that risk reductions are estimated in grid cells encompassing a wide range of  $\text{PM}_{2.5}$  concentrations. This includes grid cells with typical (i.e. frequently occurring) concentrations (as seen in red) as well as cells with concentrations that occur relatively infrequently (as seen in green). Furthermore, these shifts reflect reductions both in areas with relatively few estimated premature deaths (as represented by points near the bottom of each of the scatter plots) and in areas with much larger numbers of estimated deaths (points higher on the y-axis in these scatter plots).

### **C.3 CHARACTERIZING VARIABILITY AND UNCERTAINTY IN RISK ESTIMATES**

An important component of the risk assessment is the characterization of variability and uncertainty. Variability refers to the heterogeneity of a variable of interest within a population or across different populations. Variability is inherent and cannot be reduced through further research. Hence, the design of a population-level risk assessment is often focused on effectively characterizing variability in estimated risks across populations. Uncertainty refers to the lack of knowledge regarding the actual values of inputs to an analysis. In contrast to variability, uncertainty can be reduced through improved measurement of key variables and ongoing model refinement. This section discusses our approaches to addressing key sources of variability and uncertainty in the PM<sub>2.5</sub> risk assessment.

Variability in the risk of PM<sub>2.5</sub>-associated mortality could result from a number of factors. These can include variation in PM<sub>2.5</sub> exposures within and across populations (e.g., due to differences in behavior patterns, building characteristics, air quality patterns etc.) and in the health responses to those exposures (e.g., because some groups are at increased risk of PM-related health effects). There is also variation over space and time in both PM<sub>2.5</sub> itself (e.g., concentrations, air quality patterns) and in the ambient pollutants that co-occur with PM<sub>2.5</sub>. In the PM<sub>2.5</sub> risk assessment discussed in this PA, we account for these and other sources of variability, in part, by estimating risks based on CR functions from a number of epidemiologic studies. These studies evaluate PM<sub>2.5</sub> health effect associations for either annual or daily PM<sub>2.5</sub> exposures across various time periods; in numerous geographic locations, encompassing much or all of the U.S.; in various populations, including some with the potential to be at higher risk than the general population (e.g., older adults); and using a variety of methods to estimate PM<sub>2.5</sub> exposures (e.g., hybrid modeling approaches, monitors) and to control for potential confounders. In selecting areas in which to estimate PM<sub>2.5</sub>-associated risks, we include areas that cover multiple regions of the U.S., with varying population demographics. Additionally, we use two different strategies for adjusting PM<sub>2.5</sub> air quality, reflecting the potential for changes in ambient PM<sub>2.5</sub> concentrations to be influenced by changes in primary PM<sub>2.5</sub> emissions and by changes in precursor emissions that contribute to secondary particle formation.

Beyond the reliance on information from multiple epidemiologic studies to account for the variability in key risk assessment inputs, we use a combination of quantitative and qualitative approaches to more explicitly characterize the remaining uncertainty in risk estimates. The characterization of uncertainty associated with risk assessments is often addressed in the regulatory context using a tiered approach in which progressively more sophisticated methods are used to evaluate and characterize sources of uncertainty depending on the overall complexity of the risk assessment (WHO, 2008). Guidance documents developed by EPA for assessing air

toxics-related risk and Superfund Site risks (U.S. EPA, 2004 U.S. EPA, 2001) as well as recent guidance from the World Health Organization (WHO, 2008) specify multitiered approaches for addressing uncertainty. The WHO guidance presents a four-tiered approach, where the decision to proceed to the next tier is based on the outcome of the previous tier's assessment. The four tiers described in the WHO guidance include:

- Tier 0 – recommended for routine screening assessments, uses default uncertainty factors (rather than developing site-specific uncertainty characterizations);
- Tier 1 – the lowest level of site-specific uncertainty characterization, involves qualitative characterization of sources of uncertainty (e.g., a qualitative assessment of the general magnitude and direction of the effect on risk results);
- Tier 2 – site-specific deterministic quantitative analysis involving sensitivity analysis, interval-based assessment, and possibly probability bound (high- and low-end) assessment; and
- Tier 3 – uses probabilistic methods to characterize the effects on risk estimates of sources of uncertainty, individually and combined.

With this four-tiered approach, the WHO framework provides a means for systematically linking the characterization of uncertainty to the sophistication of the underlying risk assessment. Ultimately, the decision as to which tier of uncertainty characterization to include in a risk assessment will depend both on the overall sophistication of the risk assessment and the availability of information for characterizing the various sources of uncertainty. EPA staff used the WHO guidance as a framework for developing the approach used for characterizing uncertainty in this risk assessment. The overall analysis in the PM NAAQS risk assessment is relatively complex, thereby warranting consideration of a full probabilistic (WHO Tier 3) uncertainty analysis. However, limitations in available information prevent this level of analysis from being completed at this time. In particular, the incorporation of uncertainty related to key elements of CR functions (e.g., alternative functional forms, etc.) into a full probabilistic WHO Tier 3 analysis would require that probabilities be assigned to each competing specification of a given model element (with each probability reflecting a subjective assessment of the probability that the given specification is the “correct” description of reality). However, for many model elements there is insufficient information on which to base these probabilities. One approach that has been taken in such cases is expert elicitation; however, this approach is resource- and time-intensive and consequently, it was not feasible to use this technique in the current PM NAAQS review to support a WHO Tier 3 analysis.

For most elements of this risk assessment, rather than conducting a full probabilistic uncertainty analysis, we have included qualitative discussions of the potential impact of uncertainty on risk results (WHO Tier1) and/or completed sensitivity analyses assessing the potential impact of sources of uncertainty on risk results. The remainder of this section is



organized as follows. Those sources of uncertainty addressed quantitatively in the risk assessment are discussed in section C.3.1. Those sources of uncertainty addressed qualitatively in the risk assessment are discussed in section C.3.2. Below we summarize key findings from both the qualitative and quantitative assessments of variability and uncertainty in the context of assessing overall confidence in the risk assessment and its estimates.

### **C.3.1 Quantitative Assessment of Uncertainty**

The risk assessment includes three components which allow us to quantitatively evaluate the impact of potentially important sources of uncertainty on the risk estimates generated. Each of these is discussed below including conclusions drawn from each assessment regarding the potential importance of each source of uncertainty:

- *95 percent CIs around point estimates of mortality risk:* Each of the point estimates presented in the results section includes 95 percent CIs generated by BenMAP-CE, reflecting the standard error (SE) associated with the underlying effect estimate (i.e., a measure of the statistical precision of the effect estimate). There is considerable variation in the range of 95 percent CIs associated with the point estimates generated for this analysis, with some health endpoint/study combinations displaying substantially greater variability than others (e.g., short-term PM<sub>2.5</sub> exposure and all-cause mortality based on effect estimates from Ito et al., 2013 versus long-term PM<sub>2.5</sub> exposure IHD mortality estimates based on Jerrett et al., 2016, respectively—see tables presenting risk estimates in section 3.3.2 of this PA). There are a number of factors potentially responsible for the varying degrees of statistical precision in effect estimates, including sample size, exposure measurement error, degree of control for confounders/effect modifiers, and variability in PM<sub>2.5</sub> concentrations.
- *Inclusion of range of mortality estimates reflecting variation in effect estimates across studies:* For some mortality endpoints, we include a range of risk estimates reflecting different epidemiology studies and associated study designs (e.g., age ranges, methods for controlling potential confounders). In some instances, we find that the effect estimate used has only a small impact on risk estimates (i.e., modeling of IHD mortality using effect estimates from Jerrett et al., 2016 and Pope et al., 2015, Table 3-5 in PA section 3.3.2). By contrast, for other mortality endpoints, such as all-cause mortality associated with long-term exposures (e.g., Di et al., 2017b versus Thurston et al., 2016), the use of different effect estimates can have a larger impact (section 3.3.2, Table 3-5). The degree to which different CR functions result in different risk estimates could reflect differences in study design and/or study populations evaluated, as well as other factors. For example, the examination of different cohorts in Di et al., 2017b) and Thurston et al., 2016) could contribute to greater divergence in risk estimates. Details regarding the design of epidemiology studies providing effect estimates for this risk assessment are presented in Table C-1).
- *Evaluation of two different strategies for simulating air quality scenarios:* As noted above, we use two methods to adjust air quality in order to simulate just meeting the current and alternative standards (i.e., the Pri-PM-based method and the Sec-PM based method). Our evaluation of these methods reflects the fact that there is variability, and

uncertainty, in how emissions in a particular area could change such that the area “just meets” either the current or alternative standards. By modeling risks based on adjusted primary PM<sub>2.5</sub> emissions and based on adjusted precursor emissions that contribute to secondary PM<sub>2.5</sub> formation, the risk assessment provides insight into the potential significance of this source of uncertainty. As discussed in section 3.3.2 of this PA, the approach to adjusting air quality had relatively modest impacts on overall risk estimates. Specifically, the difference between the absolute risk estimates from two air quality modeling approach methods was generally less than 5% (Table 3-5 in PA section 3.3.2).

### **C.3.2 Qualitative Uncertainty Analysis**

While the methods described above address some of the potentially important sources of uncertainty and variability in the risk assessment, there are a range of additional sources that cannot be analyzed quantitatively due to limitations in data, methods and/or resources. We have addressed these additional sources of uncertainty qualitatively (Table C-32).

In describing each source of uncertainty, we attempt to characterize both the magnitude and direction of impact on mortality risk estimates, including our rationale for these characterizations. The categories used in describing the potential magnitude of impact (i.e., low, medium, or high) reflect EPA staff judgments on the degree to which a particular source of uncertainty could produce a sufficient impact on risk estimates to influence the interpretation of those estimates in the context of the PM NAAQS review. Sources classified as having a *low* impact would not be expected to influence conclusions from the risk assessment. Sources classified as having a *medium* impact have the potential to affect such conclusions and sources classified as *high* are likely to influence conclusions. Because this classification of the potential magnitude of impact of sources of uncertainty is qualitative, it is not possible to place a quantitative level of impact on each of the categories.

**Table C-32. Qualitative analysis of sources of uncertainty and assessment of potential impact on risk assessment.**

Source of Uncertainty	Description	Direction	Magnitude	Comments
a) Simulating just meeting current and alternative standards using model-based (Downscaler) methods	<p>a) The baseline and adjusted concentration fields were developed using modeling to fill spatial and temporal gaps in monitoring and to explore air quality scenarios of policy interest. State-of-the-science modeling methods were used, but model-related biases and errors can introduce uncertainty into the PM<sub>2.5</sub> concentration estimates.</p> <p>b) Due to the national scale of the assessment, the modeling scenarios are based on “across-the-board” emission changes in which emissions of primary PM<sub>2.5</sub> or NO<sub>x</sub> and SO<sub>2</sub> from all anthropogenic sources throughout the U.S. are scaled by fixed percentages. Although this approach tends to target the key sources in each area, it does not tailor emission changes to specific periods or sources.</p> <p>c) Two adjustment cases were applied that span a wide range of emission conditions, but these cases are necessarily a subset of the full set of possible emission cases that could be used to adjust PM<sub>2.5</sub> concentrations to just meet standards.</p>	This source of uncertainty could bias results in either direction.	Medium	Use of state-of-the-science modeling systems with the relative response factor adjustment approach provides confidence in the broad features of the simulated national PM <sub>2.5</sub> distributions and how the distributions shift with changing standards levels. Due to challenges in modeling local features in the national annual simulations, quantitative results for individual areas or small subsets of grid cells are relatively uncertain compared with broad features of the national PM <sub>2.5</sub> distributions.

Source of Uncertainty	Description	Direction	Magnitude	Comments
b) Simulating just meeting alternative annual standards with levels of 9.0 and 11.0 ug/m <sup>3</sup> using linear extrapolation/interpolation	The use of extrapolation/interpolation in simulating just meeting annual standards introduces uncertainty into the risk assessment since this approach does not fully capture potential non-linearities associated with the formation of secondary PM <sub>2.5</sub> .	Both	Medium	Extrapolation to generate the surface for 9.0 µg/m <sup>3</sup> is subject to greater uncertainty than interpolation to 11.0 µg/m <sup>3</sup> (i.e., since the former estimates concentrations below those in modeled surfaces, while the latter estimates a surface between two sets of modeled results). In addition, linear extrapolation/interpolation based on the primary-PM modeled surfaces (for current standard and 10.0 µg/m <sup>3</sup> ) is likely subject to less uncertainty than extrapolation/interpolation based on the secondary-PM modeled surfaces since the latter focus on secondary formation which could involve a higher degree of non-linearity.
c) Exposure measurement error in epidemiologic studies assessing the relationship between mortality and exposure to ambient PM <sub>2.5</sub>	Epidemiologic studies have employed a variety of approaches to estimate population-level PM <sub>2.5</sub> exposures (e.g., stationary monitors, hybrid modeling approaches). These approaches are based on using measured or predicted ambient PM <sub>2.5</sub> concentrations as surrogates for population exposures. As such, exposure estimates in epidemiologic studies are subject to exposure error. This error in the underlying epidemiologic studies contributes to uncertainty in the risk estimates that are based on concentration-response relationships in those studies.	Both	Low	Available studies indicate that PM <sub>2.5</sub> health effect associations are robust across various approaches to estimating PM <sub>2.5</sub> exposures. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates the potential for exposure error in epidemiologic studies, such error does not call into question the findings of key PM <sub>2.5</sub> epidemiologic studies. The ISA notes that, while bias in either direction can occur, exposure error tends to result in underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study Hart et al. (2015) reports that correction for PM <sub>2.5</sub> exposure error using personal exposure information results in a moderately larger effect estimate for long-term PM <sub>2.5</sub> exposure and mortality (though with wider confidence intervals). While most PM <sub>2.5</sub> epidemiologic studies have not employed similar corrections for exposure error, several studies report that restricting analyses to populations in close proximity to a monitor (i.e., in order to reduce exposure error) result in larger PM <sub>2.5</sub>

Source of Uncertainty	Description	Direction	Magnitude	Comments
				effect estimates (e.g., Willis et al., 2003; Kloog et al., 2013). Thus, to the extent key PM <sub>2.5</sub> epidemiologic studies are subject to exposure error, correction for that error would likely result in larger effect estimates, and thus larger estimates of PM <sub>2.5</sub> -associated mortality incidence in the risk assessment.
d) Representing population-level exposure with 12 km grid cell spatial framework (in context of modeling long-term exposure-related mortality)	The risk assessment utilizes a 12 km grid structure in modeling risk. A source of uncertainty associated with this approach is the mismatch between the 12 km grid cell framework and the exposure estimation approaches used in the epidemiology studies providing effect estimates for the risk assessment. This mismatch can introduce additional exposure error to risk estimates, beyond the error in the underlying epidemiologic study itself.	Both	Medium	There are a variety of spatial templates used across the five epidemiology studies providing effect estimates used in the risk assessment and that none of them are an exact match with the 12km grid cell template used in the risk assessment. For example, the Jerrett et al. (2013) effect estimate is an ensemble model which integrates results from a range of spatial templates (e.g., 1 km, 9.8, 30 km and 36 km grids) while Pope et al. (2015) utilized a county-level design. Differences between the exposure metric used in the risk assessment and those used in the underlying epidemiologic studies introduce uncertainty into risk estimates.
e) Representing population-level exposure with 12 km grid cell spatial framework (in context of modeling short-term exposure-related mortality)	As with long-term exposure-related mortality, short-term exposure-related mortality endpoints were also modeled using the same 12 km grid cell template. The disconnect between the spatial template used in the underlying short-term epidemiology studies and the 12 km grid template used in the risk assessment introduces uncertainty into risk estimates.	Both	Medium-High	The three studies providing effect estimates for short-term exposure-related mortality in the risk assessment all utilized some form of urban-level spatial unit in characterizing exposure (e.g., Baxter et al. (2017) utilizes the CBSA, Ito et al. (2013), utilizes the MSA), which are larger (less spatially differentiated) in general than the 12 km grid cells used in modeling risk. This means that we are generally modeling short-term exposure-related mortality at a finer level of spatial resolution in the risk assessment than reflected in the epidemiology studies supplying the effect estimates, which does introduce uncertainty into the analysis.
f) Temporal mismatch between	Several of the epidemiology studies for long-term exposure-	Both	Low	This approach can be reasonable in the context of an epidemiologic study evaluating health effect associations

Source of Uncertainty	Description	Direction	Magnitude	Comments
ambient air quality data characterizing exposure and mortality in long-term exposure-related epidemiology studies	related mortality have a mismatch between the time period associated with ambient PM <sub>2.5</sub> concentrations used to characterize population-level exposure and mortality data (i.e., the ambient PM <sub>2.5</sub> data reflects a period near the end of the mortality period for Jerrett et al. (2016) and Pope et al. (2015)).			with long-term PM <sub>2.5</sub> exposures, under the assumption that spatial patterns in PM <sub>2.5</sub> concentrations are not appreciably different during time periods for which air quality information is not available (e.g., Chen et al. (2016)), Thus, as long as the overall spatial pattern of ambient PM <sub>2.5</sub> levels in relation to population-level exposure and mortality rates has held relatively stable over time, then a temporal disconnect between the time-period associated with mortality and the ambient PM <sub>2.5</sub> level used in characterizing exposure would not be expected to introduce significant uncertainty into the epidemiology studies and associated effect estimates.
g) Shape and corresponding statistical uncertainty around the CR function for long-term and short-term exposure-related mortality (especially at lower ambient PM levels)	Interpreting the shapes of concentration-response relationships, particularly at PM <sub>2.5</sub> concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and “linearize” population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015, section 6.c).	Both	Medium-High	With regard to long-term exposure-related (nonaccidental) mortality, the ISA concludes that the majority of evidence supports a linear, no-threshold concentration-response relationship, though there is initial evidence indicating that the slope of the concentration-response curve may be steeper at lower concentrations for cardiovascular mortality (U.S. EPA, 2019, section 1.5.3.2). For long-term exposure-related mortality, the ISA notes that there is less certainty in the shape of the concentration-response curve at mean annual PM <sub>2.5</sub> concentrations generally below 8 µg/m <sup>3</sup> because data density is reduced below this concentration (section 11.2.4). Given that a portion of risk modeling in the risk assessment does involve locations with ambient PM <sub>2.5</sub> concentrations below 8 ug/m <sup>3</sup> (although most of the population modeled is associated with level above this), we note the potential for significant uncertainty being introduced into the risk assessment (particularly for that portion of risk modeled at or below 8 ug/m <sup>3</sup> ). With regard to short-term exposure-related mortality, the ISA concludes that, while difficulties remain in assessing the shape of the PM <sub>2.5</sub> -mortality concentration-response relationship, as identified in the 2009 PM ISA, and studies have not conducted systematic evaluations of alternatives to linearity, recent

Source of Uncertainty	Description	Direction	Magnitude	Comments
				studies continue to provide evidence of a no-threshold linear relationship, with less confidence at concentrations lower than 5 µg/m <sup>3</sup> .
h) Potential confounding of the PM <sub>2.5</sub> -mortality effect	Factors are considered potential confounders if demonstrated in the scientific literature to be related to health effects and correlated with PM. Omitting potential confounders from analyses could either increase or decrease the magnitude of PM <sub>2.5</sub> effect estimates (e.g., Di et al., 2017b, Figure S2 in Supplementary Materials). Thus, not accounting for confounders can introduce uncertainty into effect estimates and, consequently, into the risk estimates generated using those effect estimates. Confounders vary according to study design, exposure duration, and health effect. While a range of approaches to control for potential confounders have been adopted across the studies used in the risk assessment, and across the broader body of PM <sub>2.5</sub> epidemiologic studies assessed in the ISA, no individual study adjusts for all potential confounders.	Both	Medium	<p>Long-term PM<sub>2.5</sub> exposure and mortality studies: For studies of long-term exposures, potential confounders are those that vary spatially. These may include socioeconomic status, race, age, medication use, smoking status, stress, noise, occupational exposures, and copollutant concentrations. Cohort studies used to characterize the PM<sub>2.5</sub>-mortality relationship used a variety of approaches to account for these and other potential confounders (e.g., see Appendix B, Table B-12). Across studies, a variety of study designs and statistical approaches have been used to account for potential confounding in the PM<sub>2.5</sub>-mortality relationship. The fact that across this diverse body of evidence epidemiologic studies continue to report consistently positive associations that are often similar in magnitude, adds support the conclusion that the PM<sub>2.5</sub>-mortality association is robust. Specifically regarding copollutants, the final PM ISA notes that, overall, associations remained relatively unchanged in copollutant models for total (nonaccidental) mortality, cardiovascular, and respiratory adjusted for ozone (Figure 11-20). Studies focusing on copollutant models with NO<sub>2</sub>, PM<sub>10-2.5</sub>, SO<sub>2</sub> and benzene were examined in individual studies, and across these studies the PM<sub>2.5</sub>-mortality association was relatively unchanged (Figure 11-21).</p> <p>Short-term PM<sub>2.5</sub> exposure and mortality studies: For studies of short-term exposures, potential confounders are those that vary temporally. These may include meteorology (e.g., temperature, humidity), day of week, season, medication use, allergen exposure, copollutant concentrations, and long-term temporal trends. Some recent studies have</p>

Source of Uncertainty	Description	Direction	Magnitude	Comments
				expanded the examination of potential confounders, including long-term temporal trends, weather, and copollutants. Overall, the ISA concludes that alternative approaches to controlling for long-term temporal trends and for the potential confounding effects of weather may influence the magnitude of the association between PM <sub>2.5</sub> exposures and mortality, but have not been found to influence the direction of the observed association (U.S. EPA, 2019, section 11.1.5.1). With regard to copollutants, recent studies conducted outside the U.S. provide additional evidence that associations between short-term PM <sub>2.5</sub> exposures and mortality remain positive and relatively unchanged in copollutant models with both gaseous pollutants and PM <sub>10-2.5</sub> (U.S. EPA, 2019, Section 11.1.4).
i) Compositional and source differences in PM	The composition of PM <sub>2.5</sub> can differ across study areas reflecting underlying differences in primary and secondary PM <sub>2.5</sub> sources (both natural and anthropogenic). If these compositional differences lead to differences in public health impacts (per unit concentration in ambient air) for PM <sub>2.5</sub> , then uncertainty may be introduced into risk estimates that are based on concentration-response relationships for PM <sub>2.5</sub> mass.	Both	Low	The Integrated Synthesis chapter of the final ISA (Chapter 1, U.S. EPA, 2019) states that, the assessment of PM sources and components confirms and continues to support the conclusion from the 2009 PM ISA: Many PM <sub>2.5</sub> components and sources are associated with health effects, and the evidence does not indicate that any one source or component is more strongly related with health effects than PM <sub>2.5</sub> mass.
j) Lag structure in short-term exposure-related mortality	It can be challenging to characterize the timing associated with specific PM <sub>2.5</sub> -related health effects and consequently specify the lag-structure that should be	Both	Low-Medium	Given the emphasis placed in the risk assessment on mortality (and specifically, IHD mortality), we focus here on lags associated with cardiovascular-related mortality. The ISA notes that the immediate effect of PM <sub>2.5</sub> on cardiovascular morbidity outcomes, specifically those



Source of Uncertainty	Description	Direction	Magnitude	Comments
epidemiology studies	used in modeling those health effects. This can introduce uncertainty into the modeling of risk for short-term exposure-related endpoints.			related to ischemic events, are consistent with the lag structure of associations observed in studies of cardiovascular mortality that report immediate effects (i.e., lag 0-1 day). (final PM ISA, section 1.5.2.2, U.S. EPA, 2019)
k) Use of associations reported in epidemiologic studies to estimate how mortality incidence may change with changing PM <sub>2.5</sub> air quality.	The ISA's determination that the evidence supports a causal relationship between PM <sub>2.5</sub> exposure and mortality is based on assessing a broad body of evidence from epidemiologic and experimental studies. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate how mortality incidence may change with changing PM <sub>2.5</sub> air quality is subject to uncertainty.	Both	Low	The ISA assesses a longstanding body of health evidence supporting relationships between PM <sub>2.5</sub> exposures (short- and long-term) and mortality. Much of this evidence comes from epidemiologic studies conducted in North America, Europe, or Asia that demonstrate generally positive, and often statistically significant, associations between PM <sub>2.5</sub> exposures and total or cause-specific mortality. In addition, recent experimental evidence, as well as evidence from panel studies, strengthens support for potential biological pathways through which PM <sub>2.5</sub> exposures could lead to serious health outcomes, including mortality. While this broad body of evidence from across disciplines provides the foundation for the ISA's conclusions, the risk assessment necessarily focuses on a small number of individual studies. Although the studies selected for the risk assessment are part of the evidence base supporting the ISA's causality determinations for mortality, the concentration-response relationship in any given study reflects the particular time period, locations, air quality distribution and populations evaluated in that study. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate mortality incidence across the U.S. for populations, locations and PM <sub>2.5</sub> air quality distributions different from those present during the study period is subject to uncertainty.

### **C.3.3 Conclusion**

To increase overall confidence in the risk assessment, a deliberative process has been used in specifying each of the analytical elements comprising the risk model, including selection of urban study areas as well as specification of other inputs such as CR functions. This deliberative process involved rigorous review of available literature addressing both PM<sub>2.5</sub> exposure and risk combined with the application of a formal set of criteria to guide development of each of the key analytical elements in the risk assessment. In addition, the risk assessment design reflects consideration of CASAC and public comments on the Integrated Review Plan (IRP) for the PM NAAQS (U.S. EPA, 2016). The application of this deliberative process increases overall confidence in the risk estimates by ensuring that the estimates are based on the best available science and data characterizing PM<sub>2.5</sub> exposure and risk, and that they reflect consideration of input from experts on PM exposure and risk through CASAC and public reviews.

## C.4 PM<sub>2.5</sub> DESIGN VALUES FOR THE AIR QUALITY PROJECTIONS

**Table C-33. PM<sub>2.5</sub> DVs for the Primary PM projection case and 12/35 standard level.**

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
AkronO	391530017	Annual	Yes	0	-18	10.99	11.99	23.7	25.4
AkronO	391530023	Annual	No	0	-18	9.16	9.90	20.2	21.4
Altoon	420130801	Annual	Yes	0	-41	10.11	12.02	23.8	29.5
Atlant	131210039	Annual	Yes	0	-27	10.38	11.99	19.7	22.6
Atlant	132230003	Annual	No	0	-27	7.82	8.62	16.2	17.5
Atlant	131350002	Annual	No	0	-27	8.84	10.05	17.9	20.2
Atlant	130890002	Annual	No	0	-27	9.34	10.63	19.2	21.7
Atlant	130670003	Annual	No	0	-27	9.51	10.79	18.6	21.0
Atlant	130630091	Annual	No	0	-27	9.86	11.19	19.1	21.6
Bakers	060290010	24-hr	Yes	79	77	16.52	10.23	70.0	35.4
Bakers	060290016	24-hr	No	79	77	18.45	11.45	61.3	31.7
Bakers	060290015	24-hr	No	79	77	5.15	3.97	15.8	13.6
Bakers	060290014	24-hr	No	79	77	16.53	9.81	61.4	31.7
Bakers	060290011	24-hr	No	79	77	6.06	4.84	19.6	16.6
Birmin	010732059	Annual	Yes	0	-10	11.25	12.00	22.3	23.9
Birmin	010732003	Annual	No	0	-10	10.08	10.70	19.0	20.1
Birmin	010731010	Annual	No	0	-10	9.78	10.30	19.2	20.1
Birmin	010730023	Annual	No	0	-10	10.94	11.66	22.8	24.2
Canton	391510017	Annual	Yes	0	-23	10.81	12.04	23.7	26.1
Canton	391510020	Annual	No	0	-23	9.91	10.96	22.0	23.6
Chicag	170313103	Annual	Yes	0	-15	11.10	12.00	22.6	24.2
Chicag	550590019	Annual	No	0	-15	8.04	8.56	20.4	21.5
Chicag	181270024	Annual	No	0	-15	9.51	10.30	22.4	24.1
Chicag	180892004	Annual	No	0	-15	9.84	10.71	24.7	26.7
Chicag	180890031	Annual	No	0	-15	10.12	11.01	23.6	25.6
Chicag	180890026	Annual	No	0	-15	-	-	25.2	27.1
Chicag	180890022	Annual	No	0	-15	-	-	22.7	24.8
Chicag	180890006	Annual	No	0	-15	10.03	10.93	23.1	25.2
Chicag	171971011	Annual	No	0	-15	8.36	8.85	18.4	19.3
Chicag	171971002	Annual	No	0	-15	7.69	8.23	20.0	21.2
Chicag	170890007	Annual	No	0	-15	8.94	9.55	19.2	20.5
Chicag	170890003	Annual	No	0	-15	-	-	19.2	20.0
Chicag	170434002	Annual	No	0	-15	8.87	9.48	19.9	20.7
Chicag	170316005	Annual	No	0	-15	10.79	11.66	24.1	26.1

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Chicag	170314201	Annual	No	0	-15	9.00	9.61	21.4	22.6
Chicag	170314007	Annual	No	0	-15	9.49	10.17	-	-
Chicag	170313301	Annual	No	0	-15	10.37	11.18	23.5	25.2
Chicag	170310076	Annual	No	0	-15	10.18	10.96	22.5	24.0
Chicag	170310057	Annual	No	0	-15	11.03	11.89	26.8	28.4
Chicag	170310052	Annual	No	0	-15	10.00	10.78	23.3	24.9
Chicag	170310022	Annual	No	0	-15	10.38	11.30	22.4	23.9
Chicag	170310001	Annual	No	0	-15	10.13	10.88	21.7	23.4
Cincin	390610014	Annual	Yes	0	-24	10.70	12.02	22.9	24.7
Cincin	390610042	Annual	No	0	-24	10.29	11.47	22.6	24.5
Cincin	390610040	Annual	No	0	-24	9.45	10.53	21.0	22.9
Cincin	390610010	Annual	No	0	-24	9.43	10.41	21.3	22.9
Cincin	390610006	Annual	No	0	-24	9.46	10.56	20.3	21.8
Cincin	390170020	Annual	No	0	-24	-	-	24.2	26.5
Cincin	390170019	Annual	No	0	-24	10.24	11.51	22.0	23.8
Cincin	390170016	Annual	No	0	-24	9.79	10.91	22.1	23.7
Cincin	210373002	Annual	No	0	-24	9.06	10.00	20.9	22.6
Clevel	390350065	Annual	Yes	0	2	12.17	12.03	24.9	24.6
Clevel	391030004	Annual	No	0	2	8.73	8.66	19.6	19.5
Clevel	390933002	Annual	No	0	2	8.10	8.03	20.2	20.1
Clevel	390850007	Annual	No	0	2	7.88	7.82	17.4	17.3
Clevel	390351002	Annual	No	0	2	8.86	8.78	19.5	19.4
Clevel	390350045	Annual	No	0	2	10.61	10.50	22.9	22.7
Clevel	390350038	Annual	No	0	2	11.38	11.25	25.0	24.8
Clevel	390350034	Annual	No	0	2	8.87	8.79	20.4	20.2
Detroi	261630033	Annual	Yes	0	-15	11.30	12.04	26.8	28.4
Detroi	261630039	Annual	No	0	-15	9.11	9.63	22.3	23.7
Detroi	261630036	Annual	No	0	-15	8.68	9.13	21.8	23.2
Detroi	261630025	Annual	No	0	-15	8.98	9.54	24.1	25.2
Detroi	261630019	Annual	No	0	-15	9.18	9.75	22.4	24.1
Detroi	261630016	Annual	No	0	-15	9.62	10.19	24.4	25.4
Detroi	261630015	Annual	No	0	-15	11.19	11.91	25.5	27.0
Detroi	261630001	Annual	No	0	-15	9.50	10.14	23.3	24.9
Detroi	261470005	Annual	No	0	-15	8.89	9.34	24.3	25.4
Detroi	261250001	Annual	No	0	-15	8.86	9.41	24.2	25.7
Detroi	260990009	Annual	No	0	-15	8.80	9.29	26.2	27.6
EICent	060250005	Annual	Yes	0	12	12.63	12.00	33.5	31.3
EICent	060251003	Annual	No	0	12	7.44	7.01	19.8	18.5

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ElCent	060250007	Annual	No	0	12	8.37	7.99	21.5	20.8
Elkhar	180390008	Annual	Yes	0	-47	10.24	12.01	28.6	33.2
Evansv	181630023	Annual	Yes	0	-44	10.11	12.03	21.5	24.0
Evansv	211010014	Annual	No	0	-44	9.64	11.32	20.7	22.3
Evansv	181630021	Annual	No	0	-44	9.84	11.68	21.6	23.3
Evansv	181630016	Annual	No	0	-44	10.02	11.91	22.0	24.0
Fresno	060195001	24-hr	Yes	0	70	14.08	10.87	49.3	35.4
Fresno	060195025	24-hr	No	0	70	13.63	9.98	47.9	31.7
Fresno	060192009	24-hr	No	0	70	8.47	7.26	31.3	25.1
Fresno	060190011	24-hr	No	0	70	14.07	10.01	53.8	34.4
Hanfor	060310004	24-hr	Yes	65	79	21.98	11.79	72.0	35.4
Hanfor	060311004	24-hr	No	65	79	16.49	9.68	58.9	30.7
Housto	482011035	Annual	Yes	0	-14	11.19	12.04	22.4	24.0
Housto	482011039	Annual	No	0	-14	9.22	9.82	21.7	23.1
Housto	482010058	Annual	No	0	-14	9.67	10.37	22.3	23.8
Housto	481671034	Annual	No	0	-14	7.36	7.57	20.3	20.8
Indian	180970087	Annual	Yes	0	-10	11.44	12.01	25.9	26.8
Indian	180970083	Annual	No	0	-10	11.06	11.59	23.9	24.9
Indian	180970081	Annual	No	0	-10	11.07	11.61	25.0	26.0
Indian	180970078	Annual	No	0	-10	10.14	10.60	24.4	24.9
Indian	180970043	Annual	No	0	-10	-	-	26.0	26.4
Indian	180950011	Annual	No	0	-10	9.05	9.40	21.8	22.3
Indian	180570007	Annual	No	0	-10	9.02	9.39	21.4	22.1
Johnst	420210011	Annual	Yes	0	-25	10.68	12.03	25.8	30.3
Lancas	420710012	Annual	Yes	0	12	12.83	12.00	32.7	30.4
Lancas	420710007	Annual	No	0	12	10.57	9.88	29.8	27.4
LasVeg	320030561	Annual	Yes	0	-22	10.28	11.98	24.5	29.4
LasVeg	320032002	Annual	No	0	-22	9.79	11.38	19.8	23.4
LasVeg	320031019	Annual	No	0	-22	5.18	5.70	11.5	12.2
LasVeg	320030540	Annual	No	0	-22	8.80	10.21	21.7	25.9
Lebano	420750100	Annual	Yes	0	-15	11.20	12.02	31.4	33.9
Little	051191008	Annual	Yes	0	-41	10.27	12.03	21.7	24.7
Little	051190007	Annual	No	0	-41	9.78	11.76	20.5	24.0
LoganU	490050007	24-hr	Yes	0	-7	6.95	7.15	34.0	35.4
LosAng	060371103	Annual	Yes	0	5	12.38	12.03	32.8	32.1
LosAng	060592022	Annual	No	0	5	7.48	7.33	15.3	15.0
LosAng	060590007	Annual	No	0	5	9.63	9.37	-	-
LosAng	060374004	Annual	No	0	5	10.25	9.97	27.3	26.7

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LosAng	060374002	Annual	No	0	5	11.06	10.76	29.2	28.6
LosAng	060371602	Annual	No	0	5	11.86	11.52	32.3	31.5
LosAng	060371302	Annual	No	0	5	11.99	11.64	31.5	30.8
LosAng	060371201	Annual	No	0	5	9.46	9.24	25.6	25.0
LosAng	060370002	Annual	No	0	5	10.52	10.27	29.2	28.6
Louisv	180190006	Annual	Yes	0	-27	10.64	12.04	23.9	26.2
Louisv	211110075	Annual	No	0	-27	10.42	11.84	22.3	24.3
Louisv	211110067	Annual	No	0	-27	9.55	10.78	21.4	23.6
Louisv	211110051	Annual	No	0	-27	10.29	11.48	21.8	23.7
Louisv	211110043	Annual	No	0	-27	10.37	11.72	22.0	24.1
Louisv	180431004	Annual	No	0	-27	9.96	11.20	22.0	24.2
Louisv	180190008	Annual	No	0	-27	8.72	9.69	20.1	21.5
MaconG	130210007	Annual	Yes	0	-39	10.13	12.01	21.2	24.8
MaconG	130210012	Annual	No	0	-39	7.68	8.90	16.6	18.6
Madera	060392010	24-hr	Yes	0	56	13.30	11.03	45.1	35.3
McAlle	482150043	Annual	Yes	0	-67	10.09	12.02	25.0	27.4
Merced	060470003	24-hr	Yes	0	28	11.81	10.97	39.0	35.4
Merced	060472510	24-hr	No	0	28	11.68	10.57	39.8	35.1
Modest	060990006	24-hr	Yes	0	51	13.02	10.70	45.7	35.3
Modest	060990005	24-hr	No	0	51	-	-	38.8	32.5
NapaCA	060550003	Annual	Yes	0	-47	10.36	12.03	25.1	29.1
NewYor	360610128	Annual	Yes	0	-26	10.20	12.00	23.9	27.8
NewYor	361030002	Annual	No	0	-26	7.18	8.10	18.8	21.0
NewYor	360810124	Annual	No	0	-26	7.52	8.65	19.5	22.4
NewYor	360710002	Annual	No	0	-26	6.95	7.81	17.5	19.6
NewYor	360610134	Annual	No	0	-26	9.70	11.38	21.6	25.0
NewYor	360610079	Annual	No	0	-26	8.42	9.82	22.8	25.6
NewYor	360470122	Annual	No	0	-26	8.66	10.10	20.5	23.7
NewYor	360050133	Annual	No	0	-26	9.05	10.53	24.0	28.0
NewYor	360050110	Annual	No	0	-26	7.39	8.56	19.4	22.8
NewYor	340392003	Annual	No	0	-26	8.59	9.87	23.6	26.3
NewYor	340390004	Annual	No	0	-26	9.87	11.40	24.2	27.3
NewYor	340310005	Annual	No	0	-26	8.42	9.63	22.2	24.7
NewYor	340292002	Annual	No	0	-26	7.23	8.04	18.1	19.8
NewYor	340273001	Annual	No	0	-26	6.78	7.56	17.1	18.8
NewYor	340171003	Annual	No	0	-26	8.79	10.15	23.4	26.9
NewYor	340130003	Annual	No	0	-26	8.89	10.21	23.8	27.3
NewYor	340030003	Annual	No	0	-26	8.90	10.22	24.5	27.4

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OgdenC	490110004	24-hr	Yes	0	-18	7.28	7.77	32.6	35.4
OgdenC	490570002	24-hr	No	0	-18	8.99	9.73	-	-
OgdenC	490030003	24-hr	No	0	-18	6.35	6.76	-	-
Philad	420450002	Annual	Yes	0	-8	11.46	12.04	26.0	27.2
Philad	421010057	Annual	No	0	-8	10.86	11.37	27.0	28.4
Philad	421010055	Annual	No	0	-8	11.43	12.03	27.5	29.0
Philad	421010048	Annual	No	0	-8	10.27	10.77	25.6	27.0
Philad	420290100	Annual	No	0	-8	9.64	10.03	23.9	25.1
Philad	340150004	Annual	No	0	-8	8.33	8.69	20.6	21.5
Philad	340071007	Annual	No	0	-8	8.84	9.23	21.0	22.0
Philad	340070002	Annual	No	0	-8	10.19	10.61	23.5	24.6
Philad	240150003	Annual	No	0	-8	8.70	9.02	22.6	23.4
Philad	100031012	Annual	No	0	-8	9.04	9.40	23.0	23.8
Pittsb	420030064	Annual	Yes	0	13	12.82	12.00	35.8	32.8
Pittsb	421290008	Annual	No	0	13	8.65	8.15	19.6	18.9
Pittsb	421255001	Annual	No	0	13	8.35	7.89	17.8	17.2
Pittsb	421250200	Annual	No	0	13	8.95	8.44	19.3	18.2
Pittsb	421250005	Annual	No	0	13	11.02	10.38	22.7	21.2
Pittsb	420070014	Annual	No	0	13	10.11	9.48	21.9	20.5
Pittsb	420050001	Annual	No	0	13	11.03	10.30	21.9	20.5
Pittsb	420031301	Annual	No	0	13	11.00	10.30	24.8	23.0
Pittsb	420031008	Annual	No	0	13	9.78	9.16	20.5	19.3
Pittsb	420030008	Annual	No	0	13	9.50	8.85	20.5	19.0
Prinev	410130100	24-hr	Yes	0	10	8.60	8.17	37.6	35.3
ProvoO	490494001	24-hr	Yes	0	-30	7.74	8.57	30.9	35.3
ProvoO	490495010	24-hr	No	0	-30	6.73	7.52	-	-
ProvoO	490490002	24-hr	No	0	-30	7.41	8.31	28.9	33.2
Rivers	060658005	24-hr	Yes	0	36	14.48	11.51	43.2	35.3
Rivers	060658001	24-hr	No	0	36	-	-	36.5	29.6
Sacram	060670006	24-hr	Yes	0	-23	9.31	10.40	31.4	35.4
Sacram	061131003	24-hr	No	0	-23	6.62	7.19	15.8	17.3
Sacram	060670012	24-hr	No	0	-23	7.30	8.01	19.8	21.2
Sacram	060670010	24-hr	No	0	-23	8.67	9.65	26.5	29.9
Sacram	060610006	24-hr	No	0	-23	7.58	8.47	20.3	22.3
Sacram	060610003	24-hr	No	0	-23	6.71	7.26	19.3	20.2
SaltLa	490353010	24-hr	Yes	0	44	-	-	41.5	35.3
SaltLa	490353006	24-hr	No	0	44	7.62	6.19	36.8	30.2
SaltLa	490351001	24-hr	No	0	44	7.07	5.85	32.1	25.8

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SanLui	060792007	Annual	Yes	0	-46	10.70	12.04	25.9	29.4
SanLui	060798002	Annual	No	0	-46	5.71	6.33	-	-
SanLui	060792004	Annual	No	0	-46	8.25	9.26	19.8	21.4
SouthB	181410015	24-hr	Yes	0	-23	10.45	11.37	32.5	35.4
St.Lou	290990019	Annual	Yes	0	-39	10.12	12.02	22.8	24.9
St.Lou	295100094	Annual	No	0	-39	9.57	11.38	23.3	25.9
St.Lou	295100093	Annual	No	0	-39	-	-	23.7	26.6
St.Lou	295100085	Annual	No	0	-39	10.10	12.01	23.6	26.2
St.Lou	295100007	Annual	No	0	-39	9.78	11.52	23.7	26.4
St.Lou	291893001	Annual	No	0	-39	9.85	11.72	22.4	25.2
Stockt	060771002	24-hr	Yes	0	17	12.23	11.30	38.7	35.4
Stockt	060772010	24-hr	No	0	17	10.74	9.96	37.3	34.3
Visali	061072002	24-hr	Yes	48	56	16.23	10.93	54.0	35.4
Weirto	390810017	Annual	Yes	0	-5	11.75	12.02	27.2	27.8
Weirto	540090011	Annual	No	0	-5	9.75	9.95	22.8	23.5
Weirto	540090005	Annual	No	0	-5	10.52	10.74	22.4	22.9
Weirto	390810021	Annual	No	0	-5	9.29	9.47	22.2	22.6
Wheeli	540511002	Annual	Yes	0	-44	10.24	12.02	22.5	25.4
Wheeli	540690010	Annual	No	0	-44	9.61	11.32	19.7	22.6
<sup>a</sup> CBSA names are the first six characters of the full CBSAs names in Table C-3. <sup>b</sup> Percent reduction in NOx and SO <sub>2</sub> emissions associated with just meeting the standard in this case. <sup>c</sup> Percent reduction in Primary PM <sub>2.5</sub> emissions associated with just meeting the standard in this case.									



**Table C-34. PM<sub>2.5</sub> DVs for the Secondary PM projection case and 12/35 standard level.**

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NO <sub>x</sub> & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
AkronO	391530017	Annual	Yes	-67	0	10.99	12.04	23.7	26.8
AkronO	391530023	Annual	No	-67	0	9.16	10.20	20.2	21.8
Altoon	420130801	Annual	Yes	N/A	N/A	10.11	12.04	23.8	28.3
Atlant	131210039	Annual	Yes	N/A	N/A	10.38	12.04	19.7	22.9
Atlant	132230003	Annual	No	N/A	N/A	7.82	9.07	16.2	18.8
Atlant	131350002	Annual	No	N/A	N/A	8.84	10.25	17.9	20.8
Atlant	130890002	Annual	No	N/A	N/A	9.34	10.83	19.2	22.3
Atlant	130670003	Annual	No	N/A	N/A	9.51	11.03	18.6	21.6
Atlant	130630091	Annual	No	N/A	N/A	9.86	11.44	19.1	22.2
Bakers	060290010	24-hr	Yes	N/A	N/A	16.52	10.40	70.0	35.4
Bakers	060290016	24-hr	No	N/A	N/A	18.45	11.61	61.3	31.0
Bakers	060290015	24-hr	No	N/A	N/A	5.15	3.24	15.8	8.0
Bakers	060290014	24-hr	No	N/A	N/A	16.53	10.40	61.4	31.1
Bakers	060290011	24-hr	No	N/A	N/A	6.06	3.81	19.6	9.9
Birmin	010732059	Annual	Yes	-56	0	11.25	12.03	22.3	24.2
Birmin	010732003	Annual	No	-56	0	10.08	10.86	19.0	21.5
Birmin	010731010	Annual	No	-56	0	9.78	10.68	19.2	21.4
Birmin	010730023	Annual	No	-56	0	10.94	11.73	22.8	25.3
Canton	391510017	Annual	Yes	-78	0	10.81	12.04	23.7	26.1
Canton	391510020	Annual	No	-78	0	9.91	11.14	22.0	24.8
Chicag	170313103	Annual	Yes	N/A	N/A	11.10	12.04	22.6	24.5
Chicag	550590019	Annual	No	N/A	N/A	8.04	8.72	20.4	22.1
Chicag	181270024	Annual	No	N/A	N/A	9.51	10.32	22.4	24.3
Chicag	180892004	Annual	No	N/A	N/A	9.84	10.67	24.7	26.8
Chicag	180890031	Annual	No	N/A	N/A	10.12	10.98	23.6	25.6
Chicag	180890026	Annual	No	N/A	N/A	-	-	25.2	27.3
Chicag	180890022	Annual	No	N/A	N/A	-	-	22.7	24.6
Chicag	180890006	Annual	No	N/A	N/A	10.03	10.88	23.1	25.1
Chicag	171971011	Annual	No	N/A	N/A	8.36	9.07	18.4	20.0
Chicag	171971002	Annual	No	N/A	N/A	7.69	8.34	20.0	21.7
Chicag	170890007	Annual	No	N/A	N/A	8.94	9.70	19.2	20.8
Chicag	170890003	Annual	No	N/A	N/A	-	-	19.2	20.8
Chicag	170434002	Annual	No	N/A	N/A	8.87	9.62	19.9	21.6
Chicag	170316005	Annual	No	N/A	N/A	10.79	11.70	24.1	26.1
Chicag	170314201	Annual	No	N/A	N/A	9.00	9.76	21.4	23.2
Chicag	170314007	Annual	No	N/A	N/A	9.49	10.29	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	11.25	23.5	25.5

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Chicag	170310076	Annual	No	N/A	N/A	10.18	11.04	22.5	24.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	11.96	26.8	29.1
Chicag	170310052	Annual	No	N/A	N/A	10.00	10.85	23.3	25.3
Chicag	170310022	Annual	No	N/A	N/A	10.38	11.26	22.4	24.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	10.99	21.7	23.5
Cincin	390610014	Annual	Yes	-72	0	10.70	12.04	22.9	26.1
Cincin	390610042	Annual	No	-72	0	10.29	11.66	22.6	26.2
Cincin	390610040	Annual	No	-72	0	9.45	10.79	21.0	25.4
Cincin	390610010	Annual	No	-72	0	9.43	10.75	21.3	24.4
Cincin	390610006	Annual	No	-72	0	9.46	10.75	20.3	24.3
Cincin	390170020	Annual	No	-72	0	-	-	24.2	27.8
Cincin	390170019	Annual	No	-72	0	10.24	11.40	22.0	24.5
Cincin	390170016	Annual	No	-72	0	9.79	11.06	22.1	25.1
Cincin	210373002	Annual	No	-72	0	9.06	10.42	20.9	25.1
Clevel	390350065	Annual	Yes	6	0	12.17	12.04	24.9	24.7
Clevel	391030004	Annual	No	6	0	8.73	8.61	19.6	19.2
Clevel	390933002	Annual	No	6	0	8.10	7.99	20.2	19.9
Clevel	390850007	Annual	No	6	0	7.88	7.78	17.4	17.1
Clevel	390351002	Annual	No	6	0	8.86	8.74	19.5	19.2
Clevel	390350045	Annual	No	6	0	10.61	10.49	22.9	22.6
Clevel	390350038	Annual	No	6	0	11.38	11.26	25.0	24.7
Clevel	390350034	Annual	No	6	0	8.87	8.75	20.4	20.1
Detroi	261630033	Annual	Yes	-56	0	11.30	12.04	26.8	30.2
Detroi	261630039	Annual	No	-56	0	9.11	9.88	22.3	24.8
Detroi	261630036	Annual	No	-56	0	8.68	9.39	21.8	23.4
Detroi	261630025	Annual	No	-56	0	8.98	9.75	24.1	26.5
Detroi	261630019	Annual	No	-56	0	9.18	9.97	22.4	24.1
Detroi	261630016	Annual	No	-56	0	9.62	10.38	24.4	27.4
Detroi	261630015	Annual	No	-56	0	11.19	11.97	25.5	28.2
Detroi	261630001	Annual	No	-56	0	9.50	10.20	23.3	25.0
Detroi	261470005	Annual	No	-56	0	8.89	9.50	24.3	26.1
Detroi	261250001	Annual	No	-56	0	8.86	9.65	24.2	26.7
Detroi	260990009	Annual	No	-56	0	8.80	9.48	26.2	28.4
ElCent	060250005	Annual	Yes	N/A	N/A	12.63	12.04	33.5	31.9
ElCent	060251003	Annual	No	N/A	N/A	7.44	7.09	19.8	18.9
ElCent	060250007	Annual	No	N/A	N/A	8.37	7.98	21.5	20.5
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	12.04	28.6	33.6
Evansv	181630023	Annual	Yes	-89	0	10.11	12.03	21.5	32.5

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Evansv	211010014	Annual	No	-89	0	9.64	11.58	20.7	30.2
Evansv	181630021	Annual	No	-89	0	9.84	11.79	21.6	32.4
Evansv	181630016	Annual	No	-89	0	10.02	11.95	22.0	32.8
Fresno	060190011	24-hr	Yes	N/A	N/A	14.07	10.46	53.8	35.4
Fresno	060195025	24-hr	No	N/A	N/A	13.63	10.13	47.9	31.5
Fresno	060195001	24-hr	No	N/A	N/A	14.08	10.47	49.3	32.4
Fresno	060192009	24-hr	No	N/A	N/A	8.47	6.30	31.3	20.6
Hanford	060310004	24-hr	Yes	N/A	N/A	21.98	10.81	72.0	35.4
Hanford	060311004	24-hr	No	N/A	N/A	16.49	8.11	58.9	29.0
Houston	482011035	Annual	Yes	-91	0	11.19	12.04	22.4	25.2
Houston	482011039	Annual	No	-91	0	9.22	10.16	21.7	24.9
Houston	482010058	Annual	No	-91	0	9.67	10.52	22.3	24.8
Houston	481671034	Annual	No	-91	0	7.36	8.27	20.3	23.3
Indian	180970087	Annual	Yes	-24	0	11.44	12.02	25.9	27.5
Indian	180970083	Annual	No	-24	0	11.06	11.64	23.9	25.2
Indian	180970081	Annual	No	-24	0	11.07	11.65	25.0	26.7
Indian	180970078	Annual	No	-24	0	10.14	10.72	24.4	26.2
Indian	180970043	Annual	No	-24	0	-	-	26.0	27.6
Indian	180950011	Annual	No	-24	0	9.05	9.51	21.8	23.1
Indian	180570007	Annual	No	-24	0	9.02	9.52	21.4	22.8
Johnst	420210011	Annual	Yes	-86	0	10.68	12.04	25.8	27.9
Lancas	420710012	Annual	Yes	40	0	12.83	12.03	32.7	31.6
Lancas	420710007	Annual	No	40	0	10.57	9.78	29.8	28.5
LasVeg	320030561	Annual	Yes	N/A	N/A	10.28	12.04	24.5	28.7
LasVeg	320032002	Annual	No	N/A	N/A	9.79	11.47	19.8	23.2
LasVeg	320031019	Annual	No	N/A	N/A	5.18	6.07	11.5	13.5
LasVeg	320030540	Annual	No	N/A	N/A	8.80	10.31	21.7	25.4
Lebano	420750100	Annual	Yes	-61	0	11.20	12.04	31.4	32.4
Little	051191008	Annual	Yes	-98	0	10.27	12.04	21.7	26.7
Little	051190007	Annual	No	-98	0	9.78	11.40	20.5	25.5
LoganU	490050007	24-hr	Yes	-28	0	6.95	7.12	34.0	35.4
LosAng	060371103	Annual	Yes	N/A	N/A	12.38	12.04	32.8	31.9
LosAng	060592022	Annual	No	N/A	N/A	7.48	7.27	15.3	14.9
LosAng	060590007	Annual	No	N/A	N/A	9.63	9.37	-	-
LosAng	060374004	Annual	No	N/A	N/A	10.25	9.97	27.3	26.6
LosAng	060374002	Annual	No	N/A	N/A	11.06	10.76	29.2	28.4
LosAng	060371602	Annual	No	N/A	N/A	11.86	11.53	32.3	31.4
LosAng	060371302	Annual	No	N/A	N/A	11.99	11.66	31.5	30.6

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
LosAng	060371201	Annual	No	N/A	N/A	9.46	9.20	25.6	24.9
LosAng	060370002	Annual	No	N/A	N/A	10.52	10.23	29.2	28.4
Louisv	180190006	Annual	Yes	-65	0	10.64	12.04	23.9	28.4
Louisv	211110075	Annual	No	-65	0	10.42	11.76	22.3	26.4
Louisv	211110067	Annual	No	-65	0	9.55	10.84	21.4	25.4
Louisv	211110051	Annual	No	-65	0	10.29	11.67	21.8	25.9
Louisv	211110043	Annual	No	-65	0	10.37	11.71	22.0	26.1
Louisv	180431004	Annual	No	-65	0	9.96	11.32	22.0	25.8
Louisv	180190008	Annual	No	-65	0	8.72	10.07	20.1	24.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	12.04	21.2	25.2
MaconG	130210012	Annual	No	N/A	N/A	7.68	9.13	16.6	19.7
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	11.15	45.1	35.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	12.04	25.0	29.8
Merced	060472510	24-hr	Yes	32	0	11.68	10.79	39.8	35.4
Merced	060470003	24-hr	No	32	0	11.81	10.89	39.0	34.1
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	10.82	45.7	35.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	30.1
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	12.04	25.1	29.2
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	12.04	23.9	28.2
NewYor	361030002	Annual	No	N/A	N/A	7.18	8.48	18.8	22.2
NewYor	360810124	Annual	No	N/A	N/A	7.52	8.88	19.5	23.0
NewYor	360710002	Annual	No	N/A	N/A	6.95	8.20	17.5	20.7
NewYor	360610134	Annual	No	N/A	N/A	9.70	11.45	21.6	25.5
NewYor	360610079	Annual	No	N/A	N/A	8.42	9.94	22.8	26.9
NewYor	360470122	Annual	No	N/A	N/A	8.66	10.22	20.5	24.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	10.68	24.0	28.3
NewYor	360050110	Annual	No	N/A	N/A	7.39	8.72	19.4	22.9
NewYor	340392003	Annual	No	N/A	N/A	8.59	10.14	23.6	27.9
NewYor	340390004	Annual	No	N/A	N/A	9.87	11.65	24.2	28.6
NewYor	340310005	Annual	No	N/A	N/A	8.42	9.94	22.2	26.2
NewYor	340292002	Annual	No	N/A	N/A	7.23	8.53	18.1	21.4
NewYor	340273001	Annual	No	N/A	N/A	6.78	8.00	17.1	20.2
NewYor	340171003	Annual	No	N/A	N/A	8.79	10.38	23.4	27.6
NewYor	340130003	Annual	No	N/A	N/A	8.89	10.49	23.8	28.1
NewYor	340030003	Annual	No	N/A	N/A	8.90	10.51	24.5	28.9
OgdenC	490110004	24-hr	Yes	-53	0	7.28	7.65	32.6	35.4
OgdenC	490570002	24-hr	No	-53	0	8.99	9.37	-	-
OgdenC	490030003	24-hr	No	-53	0	6.35	6.70	-	-

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Philad	420450002	Annual	Yes	-75	0	11.46	12.04	26.0	27.4
Philad	421010057	Annual	No	-75	0	10.86	11.54	27.0	28.1
Philad	421010055	Annual	No	-75	0	11.43	12.03	27.5	28.8
Philad	421010048	Annual	No	-75	0	10.27	10.91	25.6	27.4
Philad	420290100	Annual	No	-75	0	9.64	10.38	23.9	25.2
Philad	340150004	Annual	No	-75	0	8.33	8.94	20.6	23.2
Philad	340071007	Annual	No	-75	0	8.84	9.51	21.0	21.9
Philad	340070002	Annual	No	-75	0	10.19	10.95	23.5	24.6
Philad	240150003	Annual	No	-75	0	8.70	9.47	22.6	23.7
Philad	100031012	Annual	No	-75	0	9.04	9.81	23.0	23.6
Pittsb	420030064	Annual	Yes	30	0	12.82	12.02	35.8	34.8
Pittsb	421290008	Annual	No	30	0	8.65	8.06	19.6	18.0
Pittsb	421255001	Annual	No	30	0	8.35	7.78	17.8	16.4
Pittsb	421250200	Annual	No	30	0	8.95	8.32	19.3	18.2
Pittsb	421250005	Annual	No	30	0	11.02	10.30	22.7	21.7
Pittsb	420070014	Annual	No	30	0	10.11	9.52	21.9	20.6
Pittsb	420050001	Annual	No	30	0	11.03	10.45	21.9	20.4
Pittsb	420031301	Annual	No	30	0	11.00	10.28	24.8	23.6
Pittsb	420031008	Annual	No	30	0	9.78	9.20	20.5	19.0
Pittsb	420030008	Annual	No	30	0	9.50	8.89	20.5	19.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	8.10	37.6	35.4
ProvoO	490494001	24-hr	Yes	-76	0	7.74	8.29	30.9	35.4
ProvoO	490495010	24-hr	No	-76	0	6.73	7.21	-	-
ProvoO	490490002	24-hr	No	-76	0	7.41	7.95	28.9	33.2
Rivers	060658005	24-hr	Yes	N/A	N/A	14.48	11.87	43.2	35.4
Rivers	060658001	24-hr	No	N/A	N/A	-	-	36.5	29.9
Sacram	060670006	24-hr	Yes	-99	0	9.31	10.04	31.4	35.3
Sacram	061131003	24-hr	No	-99	0	6.62	7.08	15.8	19.0
Sacram	060670012	24-hr	No	-99	0	7.30	7.85	19.8	21.3
Sacram	060670010	24-hr	No	-99	0	8.67	9.30	26.5	30.2
Sacram	060610006	24-hr	No	-99	0	7.58	8.08	20.3	22.2
Sacram	060610003	24-hr	No	-99	0	6.71	7.04	19.3	20.7
SaltLa	490353010	24-hr	Yes	58	0	-	-	41.5	35.4
SaltLa	490353006	24-hr	No	58	0	7.62	6.91	36.8	31.5
SaltLa	490351001	24-hr	No	58	0	7.07	6.30	32.1	25.8
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	12.04	25.9	29.1
SanLui	060798002	Annual	No	N/A	N/A	5.71	6.43	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	9.28	19.8	22.3

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NO <sub>x</sub> & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
SouthB	181410015	Annual	Yes	-92	0	10.45	12.04	32.5	34.8
St.Lou	290990019	Annual	Yes	N/A	N/A	10.12	12.04	22.8	27.1
St.Lou	295100094	Annual	No	N/A	N/A	9.57	11.39	23.3	27.7
St.Lou	295100093	Annual	No	N/A	N/A	-	-	23.7	28.2
St.Lou	295100085	Annual	No	N/A	N/A	10.10	12.02	23.6	28.1
St.Lou	295100007	Annual	No	N/A	N/A	9.78	11.64	23.7	28.2
St.Lou	291893001	Annual	No	N/A	N/A	9.85	11.72	22.4	26.6
Stockt	060771002	24-hr	Yes	42	0	12.23	11.41	38.7	35.4
Stockt	060772010	24-hr	No	42	0	10.74	9.96	37.3	34.3
Visali	061072002	24-hr	Yes	N/A	N/A	16.23	10.64	54.0	35.4
Weirto	390810017	Annual	Yes	-14	0	11.75	12.03	27.2	27.5
Weirto	540090011	Annual	No	-14	0	9.75	10.02	22.8	23.6
Weirto	540090005	Annual	No	-14	0	10.52	10.80	22.4	23.1
Weirto	390810021	Annual	No	-14	0	9.29	9.55	22.2	22.8
Wheeli	540511002	Annual	Yes	N/A	N/A	10.24	12.04	22.5	26.5
Wheeli	540690010	Annual	No	N/A	N/A	9.61	11.30	19.7	23.2

<sup>a</sup> CBSA names are the first six characters of the full CBSAs names in Table C-3.

<sup>b</sup> Percent reduction in NO<sub>x</sub> and SO<sub>2</sub> emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

<sup>c</sup> Percent reduction in Primary PM<sub>2.5</sub> emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

**Table C-35. PM<sub>2.5</sub> DVs for the Primary PM projection case and 10/30 standard level.**

<b>CBSA <sup>a</sup></b>	<b>Site</b>	<b>Controlling Standard</b>	<b>Controlling Site?</b>	<b>NOx &amp; SO<sub>2</sub> Reduction (%) <sup>b</sup></b>	<b>Primary PM<sub>2.5</sub> Reduction (%) <sup>c</sup></b>	<b>Base Annual DV (µg m<sup>-3</sup>)</b>	<b>Projected Annual DV (µg m<sup>-3</sup>)</b>	<b>Base 24-hr DV (µg m<sup>-3</sup>)</b>	<b>Projected 24-hr DV (µg m<sup>-3</sup>)</b>
AkronO	391530017	Annual	Yes	0	17	10.99	10.03	23.7	22.6
AkronO	391530023	Annual	No	0	17	9.16	8.46	20.2	19.1
Altoon	420130801	Annual	Yes	0	2	10.11	10.02	23.8	23.5
Atlant	131210039	Annual	Yes	0	6	10.38	10.01	19.7	19.0
Atlant	132230003	Annual	No	0	6	7.82	7.64	16.2	15.9
Atlant	131350002	Annual	No	0	6	8.84	8.57	17.9	17.3
Atlant	130890002	Annual	No	0	6	9.34	9.04	19.2	18.7
Atlant	130670003	Annual	No	0	6	9.51	9.22	18.6	18.2
Atlant	130630091	Annual	No	0	6	9.86	9.56	19.1	18.5
Bakers	060290016	Annual	Yes	91	100	18.45	10.01	61.3	29.1
Bakers	060290015	Annual	No	91	100	5.15	3.66	15.8	13.6
Bakers	060290014	Annual	No	91	100	16.53	8.37	61.4	26.0
Bakers	060290011	Annual	No	91	100	6.06	4.58	19.6	15.9
Bakers	060290010	Annual	No	91	100	16.52	8.87	70.0	27.9
Birmin	010732059	Annual	Yes	0	16	11.25	10.03	22.3	19.8
Birmin	010732003	Annual	No	0	16	10.08	9.06	19.0	17.2
Birmin	010731010	Annual	No	0	16	9.78	8.94	19.2	17.7
Birmin	010730023	Annual	No	0	16	10.94	9.77	22.8	20.6
Canton	391510017	Annual	Yes	0	15	10.81	10.01	23.7	22.6
Canton	391510020	Annual	No	0	15	9.91	9.21	22.0	21.0
Chicag	170313103	Annual	Yes	0	18	11.10	10.01	22.6	21.0
Chicag	550590019	Annual	No	0	18	8.04	7.42	20.4	18.8
Chicag	181270024	Annual	No	0	18	9.51	8.55	22.4	20.4
Chicag	180892004	Annual	No	0	18	9.84	8.78	24.7	22.8
Chicag	180890031	Annual	No	0	18	10.12	9.05	23.6	21.1
Chicag	180890026	Annual	No	0	18	-	-	25.2	22.8
Chicag	180890022	Annual	No	0	18	-	-	22.7	20.4
Chicag	180890006	Annual	No	0	18	10.03	8.93	23.1	20.5
Chicag	171971011	Annual	No	0	18	8.36	7.78	18.4	17.4
Chicag	171971002	Annual	No	0	18	7.69	7.04	20.0	18.7
Chicag	170890007	Annual	No	0	18	8.94	8.21	19.2	17.8
Chicag	170890003	Annual	No	0	18	-	-	19.2	18.1
Chicag	170434002	Annual	No	0	18	8.87	8.13	19.9	18.9
Chicag	170316005	Annual	No	0	18	10.79	9.73	24.1	21.7
Chicag	170314201	Annual	No	0	18	9.00	8.25	21.4	19.9
Chicag	170314007	Annual	No	0	18	9.49	8.66	-	-
Chicag	170313301	Annual	No	0	18	10.37	9.38	23.5	21.3

<b>CBSA <sup>a</sup></b>	<b>Site</b>	<b>Controlling Standard</b>	<b>Controlling Site?</b>	<b>NOx &amp; SO<sub>2</sub> Reduction (%) <sup>b</sup></b>	<b>Primary PM<sub>2.5</sub> Reduction (%) <sup>c</sup></b>	<b>Base Annual DV (µg m<sup>-3</sup>)</b>	<b>Projected Annual DV (µg m<sup>-3</sup>)</b>	<b>Base 24-hr DV (µg m<sup>-3</sup>)</b>	<b>Projected 24-hr DV (µg m<sup>-3</sup>)</b>
Chicag	170310076	Annual	No	0	18	10.18	9.24	22.5	20.7
Chicag	170310057	Annual	No	0	18	11.03	9.99	26.8	25.1
Chicag	170310052	Annual	No	0	18	10.00	9.06	23.3	21.4
Chicag	170310022	Annual	No	0	18	10.38	9.28	22.4	20.9
Chicag	170310001	Annual	No	0	18	10.13	9.22	21.7	19.7
Cincin	390610014	Annual	Yes	0	12	10.70	10.04	22.9	21.8
Cincin	390610042	Annual	No	0	12	10.29	9.69	22.6	21.6
Cincin	390610040	Annual	No	0	12	9.45	8.91	21.0	20.0
Cincin	390610010	Annual	No	0	12	9.43	8.93	21.3	20.5
Cincin	390610006	Annual	No	0	12	9.46	8.91	20.3	19.5
Cincin	390170020	Annual	No	0	12	-	-	24.2	23.3
Cincin	390170019	Annual	No	0	12	10.24	9.60	22.0	21.1
Cincin	390170016	Annual	No	0	12	9.79	9.22	22.1	21.2
Cincin	210373002	Annual	No	0	12	9.06	8.58	20.9	20.0
Clevel	390350065	Annual	Yes	0	33	12.17	10.00	24.9	21.3
Clevel	391030004	Annual	No	0	33	8.73	7.57	19.6	17.8
Clevel	390933002	Annual	No	0	33	8.10	6.95	20.2	18.7
Clevel	390850007	Annual	No	0	33	7.88	6.84	17.4	15.4
Clevel	390351002	Annual	No	0	33	8.86	7.64	19.5	17.5
Clevel	390350045	Annual	No	0	33	10.61	8.84	22.9	20.1
Clevel	390350038	Annual	No	0	33	11.38	9.37	25.0	22.0
Clevel	390350034	Annual	No	0	33	8.87	7.58	20.4	18.2
Detroi	261630033	Annual	Yes	0	26	11.30	10.00	26.8	24.9
Detroi	261630039	Annual	No	0	26	9.11	8.21	22.3	20.3
Detroi	261630036	Annual	No	0	26	8.68	7.88	21.8	19.8
Detroi	261630025	Annual	No	0	26	8.98	7.99	24.1	21.7
Detroi	261630019	Annual	No	0	26	9.18	8.18	22.4	19.7
Detroi	261630016	Annual	No	0	26	9.62	8.63	24.4	22.6
Detroi	261630015	Annual	No	0	26	11.19	9.94	25.5	22.8
Detroi	261630001	Annual	No	0	26	9.50	8.39	23.3	20.4
Detroi	261470005	Annual	No	0	26	8.89	8.11	24.3	22.4
Detroi	261250001	Annual	No	0	26	8.86	7.90	24.2	22.2
Detroi	260990009	Annual	No	0	26	8.80	7.94	26.2	23.8
ElCent	060250005	Annual	Yes	0	50	12.63	10.01	33.5	25.0
ElCent	060251003	Annual	No	0	50	7.44	5.67	19.8	14.6
ElCent	060250007	Annual	No	0	50	8.37	6.80	21.5	18.5
Elkhar	180390008	Annual	Yes	0	6	10.24	10.01	28.6	27.8
Evansv	181630023	Annual	Yes	0	2	10.11	10.02	21.5	21.5



CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Evansv	211010014	Annual	No	0	2	9.64	9.56	20.7	20.7
Evansv	181630021	Annual	No	0	2	9.84	9.76	21.6	21.5
Evansv	181630016	Annual	No	0	2	10.02	9.94	22.0	21.9
Fresno	060195001	24-hr	Yes	0	100	14.08	9.49	49.3	30.3
Fresno	060195025	24-hr	No	0	100	13.63	8.41	47.9	26.4
Fresno	060192009	24-hr	No	0	100	8.47	6.74	31.3	22.2
Fresno	060190011	24-hr	No	0	100	14.07	8.27	53.8	27.1
Hanfor	060310004	Annual	Yes	82	98	21.98	10.00	72.0	29.5
Hanfor	060311004	Annual	No	82	98	16.49	8.36	58.9	25.2
Housto	482011035	Annual	Yes	0	19	11.19	10.01	22.4	20.2
Housto	482011039	Annual	No	0	19	9.22	8.40	21.7	19.6
Housto	482010058	Annual	No	0	19	9.67	8.70	22.3	20.3
Housto	481671034	Annual	No	0	19	7.36	7.07	20.3	19.6
Indian	180970087	Annual	Yes	0	25	11.44	10.01	25.9	24.2
Indian	180970083	Annual	No	0	25	11.06	9.72	23.9	22.5
Indian	180970081	Annual	No	0	25	11.07	9.71	25.0	23.4
Indian	180970078	Annual	No	0	25	10.14	8.97	24.4	22.8
Indian	180970043	Annual	No	0	25	-	-	26.0	24.6
Indian	180950011	Annual	No	0	25	9.05	8.17	21.8	20.7
Indian	180570007	Annual	No	0	25	9.02	8.07	21.4	20.0
Johnst	420210011	Annual	Yes	0	12	10.68	10.02	25.8	23.5
Lancas	420710012	Annual	Yes	0	41	12.83	9.98	32.7	25.5
Lancas	420710007	Annual	No	0	41	10.57	8.20	29.8	22.0
LasVeg	320030561	Annual	Yes	0	4	10.28	9.97	24.5	23.6
LasVeg	320032002	Annual	No	0	4	9.79	9.50	19.8	19.2
LasVeg	320031019	Annual	No	0	4	5.18	5.08	11.5	11.3
LasVeg	320030540	Annual	No	0	4	8.80	8.55	21.7	20.9
Lebano	420750100	Annual	Yes	0	21	11.20	10.04	31.4	28.0
Little	051191008	Annual	Yes	0	6	10.27	10.00	21.7	21.3
Little	051190007	Annual	No	0	6	9.78	9.48	20.5	20.1
LoganU	490050007	24-hr	Yes	0	19	6.95	6.40	34.0	30.3
LosAng	060371103	Annual	Yes	0	34	12.38	9.99	32.8	27.8
LosAng	060592022	Annual	No	0	34	7.48	6.43	15.3	13.3
LosAng	060590007	Annual	No	0	34	9.63	7.84	-	-
LosAng	060374004	Annual	No	0	34	10.25	8.36	27.3	23.7
LosAng	060374002	Annual	No	0	34	11.06	9.02	29.2	24.9
LosAng	060371602	Annual	No	0	34	11.86	9.55	32.3	26.5
LosAng	060371302	Annual	No	0	34	11.99	9.64	31.5	27.0

<b>CBSA <sup>a</sup></b>	<b>Site</b>	<b>Controlling Standard</b>	<b>Controlling Site?</b>	<b>NOx &amp; SO<sub>2</sub> Reduction (%) <sup>b</sup></b>	<b>Primary PM<sub>2.5</sub> Reduction (%) <sup>c</sup></b>	<b>Base Annual DV (µg m<sup>-3</sup>)</b>	<b>Projected Annual DV (µg m<sup>-3</sup>)</b>	<b>Base 24-hr DV (µg m<sup>-3</sup>)</b>	<b>Projected 24-hr DV (µg m<sup>-3</sup>)</b>
LosAng	060371201	Annual	No	0	34	9.46	7.93	25.6	21.6
LosAng	060370002	Annual	No	0	34	10.52	8.81	29.2	25.0
Louisv	180190006	Annual	Yes	0	12	10.64	10.01	23.9	22.8
Louisv	211110075	Annual	No	0	12	10.42	9.79	22.3	21.4
Louisv	211110067	Annual	No	0	12	9.55	8.99	21.4	20.5
Louisv	211110051	Annual	No	0	12	10.29	9.76	21.8	21.2
Louisv	211110043	Annual	No	0	12	10.37	9.77	22.0	21.2
Louisv	180431004	Annual	No	0	12	9.96	9.41	22.0	21.0
Louisv	180190008	Annual	No	0	12	8.72	8.29	20.1	19.5
MaconG	130210007	Annual	Yes	0	2	10.13	10.03	21.2	21.0
MaconG	130210012	Annual	No	0	2	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	0	84	13.30	9.89	45.1	30.4
McAlle	482150043	Annual	Yes	0	2	10.09	10.03	25.0	24.9
Merced	060470003	24-hr	Yes	0	65	11.81	9.87	39.0	30.4
Merced	060472510	24-hr	No	0	65	11.68	9.11	39.8	28.8
Modest	060990006	24-hr	Yes	0	77	13.02	9.52	45.7	30.3
Modest	060990005	24-hr	No	0	77	-	-	38.8	29.2
NapaCA	060550003	Annual	Yes	0	9	10.36	10.04	25.1	24.6
NewYor	360610128	Annual	Yes	0	3	10.20	9.99	23.9	23.5
NewYor	361030002	Annual	No	0	3	7.18	7.07	18.8	18.6
NewYor	360810124	Annual	No	0	3	7.52	7.39	19.5	19.1
NewYor	360710002	Annual	No	0	3	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	0	3	9.70	9.51	21.6	21.2
NewYor	360610079	Annual	No	0	3	8.42	8.26	22.8	22.5
NewYor	360470122	Annual	No	0	3	8.66	8.49	20.5	20.2
NewYor	360050133	Annual	No	0	3	9.05	8.87	24.0	23.6
NewYor	360050110	Annual	No	0	3	7.39	7.25	19.4	19.1
NewYor	340392003	Annual	No	0	3	8.59	8.44	23.6	23.2
NewYor	340390004	Annual	No	0	3	9.87	9.69	24.2	23.8
NewYor	340310005	Annual	No	0	3	8.42	8.28	22.2	21.9
NewYor	340292002	Annual	No	0	3	7.23	7.13	18.1	17.9
NewYor	340273001	Annual	No	0	3	6.78	6.69	17.1	16.9
NewYor	340171003	Annual	No	0	3	8.79	8.64	23.4	22.9
NewYor	340130003	Annual	No	0	3	8.89	8.73	23.8	23.4
NewYor	340030003	Annual	No	0	3	8.90	8.75	24.5	24.1
OgdenC	490110004	24-hr	Yes	0	15	7.28	6.89	32.6	30.3
OgdenC	490570002	24-hr	No	0	15	8.99	8.39	-	-
OgdenC	490030003	24-hr	No	0	15	6.35	6.02	-	-

<b>CBSA <sup>a</sup></b>	<b>Site</b>	<b>Controlling Standard</b>	<b>Controlling Site?</b>	<b>NOx &amp; SO<sub>2</sub> Reduction (%) <sup>b</sup></b>	<b>Primary PM<sub>2.5</sub> Reduction (%) <sup>c</sup></b>	<b>Base Annual DV (µg m<sup>-3</sup>)</b>	<b>Projected Annual DV (µg m<sup>-3</sup>)</b>	<b>Base 24-hr DV (µg m<sup>-3</sup>)</b>	<b>Projected 24-hr DV (µg m<sup>-3</sup>)</b>
Philad	420450002	Annual	Yes	0	20	11.46	9.99	26.0	22.9
Philad	421010057	Annual	No	0	20	10.86	9.56	27.0	23.4
Philad	421010055	Annual	No	0	20	11.43	9.94	27.5	24.2
Philad	421010048	Annual	No	0	20	10.27	9.00	25.6	22.7
Philad	420290100	Annual	No	0	20	9.64	8.66	23.9	21.2
Philad	340150004	Annual	No	0	20	8.33	7.43	20.6	18.2
Philad	340071007	Annual	No	0	20	8.84	7.86	21.0	18.8
Philad	340070002	Annual	No	0	20	10.19	9.11	23.5	20.6
Philad	240150003	Annual	No	0	20	8.70	7.90	22.6	20.5
Philad	100031012	Annual	No	0	20	9.04	8.15	23.0	21.1
Pittsb	420030064	Annual	Yes	0	44	12.82	10.04	35.8	26.2
Pittsb	421290008	Annual	No	0	44	8.65	6.96	19.6	16.9
Pittsb	421255001	Annual	No	0	44	8.35	6.78	17.8	15.7
Pittsb	421250200	Annual	No	0	44	8.95	7.22	19.3	15.7
Pittsb	421250005	Annual	No	0	44	11.02	8.85	22.7	18.0
Pittsb	420070014	Annual	No	0	44	10.11	7.98	21.9	17.5
Pittsb	420050001	Annual	No	0	44	11.03	8.58	21.9	17.8
Pittsb	420031301	Annual	No	0	44	11.00	8.64	24.8	18.7
Pittsb	420031008	Annual	No	0	44	9.78	7.68	20.5	16.1
Pittsb	420030008	Annual	No	0	44	9.50	7.30	20.5	16.3
Prinev	410130100	24-hr	Yes	0	33	8.60	7.19	37.6	30.4
ProvoO	490494001	24-hr	Yes	0	3	7.74	7.65	30.9	30.4
ProvoO	490495010	24-hr	No	0	3	6.73	6.65	-	-
ProvoO	490490002	24-hr	No	0	3	7.41	7.32	28.9	28.4
Rivers	060658005	24-hr	Yes	0	58	14.48	9.69	43.2	30.4
Rivers	060658001	24-hr	No	0	58	-	-	36.5	25.4
Sacram	060670006	24-hr	Yes	0	6	9.31	9.02	31.4	30.4
Sacram	061131003	24-hr	No	0	6	6.62	6.47	15.8	15.4
Sacram	060670012	24-hr	No	0	6	7.30	7.11	19.8	19.4
Sacram	060670010	24-hr	No	0	6	8.67	8.41	26.5	25.7
Sacram	060610006	24-hr	No	0	6	7.58	7.34	20.3	19.9
Sacram	060610003	24-hr	No	0	6	6.71	6.56	19.3	19.0
SaltLa	490353010	24-hr	Yes	0	85	-	-	41.5	30.4
SaltLa	490353006	24-hr	No	0	85	7.62	4.85	36.8	23.8
SaltLa	490351001	24-hr	No	0	85	7.07	4.72	32.1	21.0
SanLui	060792007	Annual	Yes	0	22	10.70	10.04	25.9	24.9
SanLui	060798002	Annual	No	0	22	5.71	5.42	-	-
SanLui	060792004	Annual	No	0	22	8.25	7.76	19.8	19.2

<b>CBSA <sup>a</sup></b>	<b>Site</b>	<b>Controlling Standard</b>	<b>Controlling Site?</b>	<b>NOx &amp; SO<sub>2</sub> Reduction (%) <sup>b</sup></b>	<b>Primary PM<sub>2.5</sub> Reduction (%) <sup>c</sup></b>	<b>Base Annual DV (µg m<sup>-3</sup>)</b>	<b>Projected Annual DV (µg m<sup>-3</sup>)</b>	<b>Base 24-hr DV (µg m<sup>-3</sup>)</b>	<b>Projected 24-hr DV (µg m<sup>-3</sup>)</b>
SouthB	181410015	24-hr	Yes	0	18	10.45	9.72	32.5	30.3
St.Lou	290990019	Annual	Yes	0	2	10.12	10.02	22.8	22.7
St.Lou	295100094	Annual	No	0	2	9.57	9.48	23.3	23.2
St.Lou	295100093	Annual	No	0	2	-	-	23.7	23.5
St.Lou	295100085	Annual	No	0	2	10.10	10.00	23.6	23.4
St.Lou	295100007	Annual	No	0	2	9.78	9.69	23.7	23.6
St.Lou	291893001	Annual	No	0	2	9.85	9.76	22.4	22.3
Stockt	060771002	24-hr	Yes	0	43	12.23	9.86	38.7	30.3
Stockt	060772010	24-hr	No	0	43	10.74	8.75	37.3	29.6
Visali	061072002	24-hr	Yes	58	74	16.23	9.67	54.0	30.4
Weirto	390810017	Annual	Yes	0	33	11.75	10.00	27.2	22.6
Weirto	540090011	Annual	No	0	33	9.75	8.42	22.8	19.8
Weirto	540090005	Annual	No	0	33	10.52	9.07	22.4	19.8
Weirto	390810021	Annual	No	0	33	9.29	8.06	22.2	19.3
Wheeli	540511002	Annual	Yes	0	5	10.24	10.03	22.5	22.1
Wheeli	540690010	Annual	No	0	5	9.61	9.42	19.7	19.4

<sup>a</sup> CBSA names are the first six characters of the full CBSAs names in Table C-3.

<sup>b</sup> Percent reduction in NOx and SO<sub>2</sub> emissions associated with just meeting the standard in this case.

<sup>c</sup> Percent reduction in Primary PM<sub>2.5</sub> emissions associated with just meeting the standard in this case.

**Table C-36. PM<sub>2.5</sub> DVs for the Secondary PM projection case and 10/30 standard level.**

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NO <sub>x</sub> & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
AkronO	391530017	Annual	Yes	45	0	10.99	10.04	23.7	20.8
AkronO	391530023	Annual	No	45	0	9.16	8.24	20.2	17.7
Altoon	420130801	Annual	Yes	N/A	N/A	10.11	10.04	23.8	23.6
Atlant	131210039	Annual	Yes	N/A	N/A	10.38	10.04	19.7	19.1
Atlant	132230003	Annual	No	N/A	N/A	7.82	7.56	16.2	15.7
Atlant	131350002	Annual	No	N/A	N/A	8.84	8.55	17.9	17.3
Atlant	130890002	Annual	No	N/A	N/A	9.34	9.03	19.2	18.6
Atlant	130670003	Annual	No	N/A	N/A	9.51	9.20	18.6	18.0
Atlant	130630091	Annual	No	N/A	N/A	9.86	9.54	19.1	18.5
Bakers	060290010	24-hr	Yes	N/A	N/A	16.52	8.99	70.0	30.4
Bakers	060290016	24-hr	No	N/A	N/A	18.45	10.04	61.3	26.6
Bakers	060290015	24-hr	No	N/A	N/A	5.15	2.80	15.8	6.9
Bakers	060290014	24-hr	No	N/A	N/A	16.53	9.00	61.4	26.7
Bakers	060290011	24-hr	No	N/A	N/A	6.06	3.30	19.6	8.5
Birmin	010732059	Annual	Yes	71	0	11.25	10.04	22.3	20.2
Birmin	010732003	Annual	No	71	0	10.08	8.86	19.0	16.1
Birmin	010731010	Annual	No	71	0	9.78	8.39	19.2	16.6
Birmin	010730023	Annual	No	71	0	10.94	9.72	22.8	20.3
Canton	391510017	Annual	Yes	36	0	10.81	10.04	23.7	21.7
Canton	391510020	Annual	No	36	0	9.91	9.13	22.0	19.4
Chicag	170313103	Annual	Yes	N/A	N/A	11.10	10.04	22.6	20.4
Chicag	550590019	Annual	No	N/A	N/A	8.04	7.27	20.4	18.5
Chicag	181270024	Annual	No	N/A	N/A	9.51	8.60	22.4	20.3
Chicag	180892004	Annual	No	N/A	N/A	9.84	8.90	24.7	22.3
Chicag	180890031	Annual	No	N/A	N/A	10.12	9.15	23.6	21.3
Chicag	180890026	Annual	No	N/A	N/A	-	-	25.2	22.8
Chicag	180890022	Annual	No	N/A	N/A	-	-	22.7	20.5
Chicag	180890006	Annual	No	N/A	N/A	10.03	9.07	23.1	20.9
Chicag	171971011	Annual	No	N/A	N/A	8.36	7.56	18.4	16.6
Chicag	171971002	Annual	No	N/A	N/A	7.69	6.96	20.0	18.1
Chicag	170890007	Annual	No	N/A	N/A	8.94	8.09	19.2	17.4
Chicag	170890003	Annual	No	N/A	N/A	-	-	19.2	17.4
Chicag	170434002	Annual	No	N/A	N/A	8.87	8.02	19.9	18.0
Chicag	170316005	Annual	No	N/A	N/A	10.79	9.76	24.1	21.8
Chicag	170314201	Annual	No	N/A	N/A	9.00	8.14	21.4	19.4
Chicag	170314007	Annual	No	N/A	N/A	9.49	8.58	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	9.38	23.5	21.3

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Chicag	170310076	Annual	No	N/A	N/A	10.18	9.21	22.5	20.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	9.98	26.8	24.2
Chicag	170310052	Annual	No	N/A	N/A	10.00	9.05	23.3	21.1
Chicag	170310022	Annual	No	N/A	N/A	10.38	9.39	22.4	20.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	9.16	21.7	19.6
Cincin	390610014	Annual	Yes	28	0	10.70	10.03	22.9	21.2
Cincin	390610042	Annual	No	28	0	10.29	9.61	22.6	20.8
Cincin	390610040	Annual	No	28	0	9.45	8.78	21.0	19.0
Cincin	390610010	Annual	No	28	0	9.43	8.78	21.3	19.6
Cincin	390610006	Annual	No	28	0	9.46	8.82	20.3	18.4
Cincin	390170020	Annual	No	28	0	-	-	24.2	22.5
Cincin	390170019	Annual	No	28	0	10.24	9.66	22.0	20.6
Cincin	390170016	Annual	No	28	0	9.79	9.16	22.1	20.1
Cincin	210373002	Annual	No	28	0	9.06	8.38	20.9	18.9
Clevel	390350065	Annual	Yes	79	0	12.17	10.04	24.9	20.5
Clevel	391030004	Annual	No	79	0	8.73	6.75	19.6	13.9
Clevel	390933002	Annual	No	79	0	8.10	6.28	20.2	13.8
Clevel	390850007	Annual	No	79	0	7.88	6.10	17.4	12.9
Clevel	390351002	Annual	No	79	0	8.86	6.81	19.5	14.4
Clevel	390350045	Annual	No	79	0	10.61	8.50	22.9	17.0
Clevel	390350038	Annual	No	79	0	11.38	9.33	25.0	19.7
Clevel	390350034	Annual	No	79	0	8.87	6.90	20.4	15.4
Detroi	261630033	Annual	Yes	60	0	11.30	10.03	26.8	24.3
Detroi	261630039	Annual	No	60	0	9.11	7.82	22.3	18.8
Detroi	261630036	Annual	No	60	0	8.68	7.43	21.8	19.1
Detroi	261630025	Annual	No	60	0	8.98	7.63	24.1	19.1
Detroi	261630019	Annual	No	60	0	9.18	7.83	22.4	20.3
Detroi	261630016	Annual	No	60	0	9.62	8.33	24.4	21.3
Detroi	261630015	Annual	No	60	0	11.19	9.88	25.5	22.0
Detroi	261630001	Annual	No	60	0	9.50	8.26	23.3	20.1
Detroi	261470005	Annual	No	60	0	8.89	7.81	24.3	20.6
Detroi	261250001	Annual	No	60	0	8.86	7.49	24.2	20.5
Detroi	260990009	Annual	No	60	0	8.80	7.57	26.2	21.8
ElCent	060250005	Annual	Yes	N/A	N/A	12.63	10.04	33.5	26.6
ElCent	060251003	Annual	No	N/A	N/A	7.44	5.91	19.8	15.7
ElCent	060250007	Annual	No	N/A	N/A	8.37	6.65	21.5	17.1
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	10.04	28.6	28.0
Evansv	181630023	Annual	Yes	3	0	10.11	10.03	21.5	21.2

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Evansv	211010014	Annual	No	3	0	9.64	9.56	20.7	20.3
Evansv	181630021	Annual	No	3	0	9.84	9.76	21.6	21.2
Evansv	181630016	Annual	No	3	0	10.02	9.95	22.0	21.7
Fresno	060190011	24-hr	Yes	N/A	N/A	14.07	9.48	53.8	30.4
Fresno	060195025	24-hr	No	N/A	N/A	13.63	9.18	47.9	27.1
Fresno	060195001	24-hr	No	N/A	N/A	14.08	9.49	49.3	27.9
Fresno	060192009	24-hr	No	N/A	N/A	8.47	5.71	31.3	17.7
Hanford	060310004	24-hr	Yes	N/A	N/A	21.98	9.28	72.0	30.4
Hanford	060311004	24-hr	No	N/A	N/A	16.49	6.96	58.9	24.9
Housto	482011035	Annual	Yes	84	0	11.19	10.04	22.4	19.6
Housto	482011039	Annual	No	84	0	9.22	8.09	21.7	18.7
Housto	482010058	Annual	No	84	0	9.67	8.57	22.3	19.1
Housto	481671034	Annual	No	84	0	7.36	6.29	20.3	17.8
Indian	180970087	Annual	Yes	48	0	11.44	10.03	25.9	21.8
Indian	180970083	Annual	No	48	0	11.06	9.64	23.9	21.4
Indian	180970081	Annual	No	48	0	11.07	9.66	25.0	20.8
Indian	180970078	Annual	No	48	0	10.14	8.73	24.4	19.9
Indian	180970043	Annual	No	48	0	-	-	26.0	20.9
Indian	180950011	Annual	No	48	0	9.05	7.86	21.8	18.3
Indian	180570007	Annual	No	48	0	9.02	7.75	21.4	17.8
Johnst	420210011	Annual	Yes	31	0	10.68	10.04	25.8	25.1
Lancas	420710012	Annual	Yes	98	0	12.83	10.01	32.7	26.2
Lancas	420710007	Annual	No	98	0	10.57	7.81	29.8	23.4
LasVeg	320030561	Annual	Yes	N/A	N/A	10.28	10.04	24.5	23.9
LasVeg	320032002	Annual	No	N/A	N/A	9.79	9.56	19.8	19.3
LasVeg	320031019	Annual	No	N/A	N/A	5.18	5.06	11.5	11.2
LasVeg	320030540	Annual	No	N/A	N/A	8.80	8.59	21.7	21.2
Lebano	420750100	Annual	Yes	53	0	11.20	10.03	31.4	28.6
Little	051191008	Annual	Yes	11	0	10.27	10.04	21.7	21.1
Little	051190007	Annual	No	11	0	9.78	9.57	20.5	19.9
LoganU	490050007	24-hr	Yes	56	0	6.95	6.51	34.0	30.4
LosAng	060371103	Annual	Yes	N/A	N/A	12.38	10.04	32.8	26.6
LosAng	060592022	Annual	No	N/A	N/A	7.48	6.07	15.3	12.4
LosAng	060590007	Annual	No	N/A	N/A	9.63	7.81	-	-
LosAng	060374004	Annual	No	N/A	N/A	10.25	8.31	27.3	22.1
LosAng	060374002	Annual	No	N/A	N/A	11.06	8.97	29.2	23.7
LosAng	060371602	Annual	No	N/A	N/A	11.86	9.62	32.3	26.2
LosAng	060371302	Annual	No	N/A	N/A	11.99	9.72	31.5	25.5

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
LosAng	060371201	Annual	No	N/A	N/A	9.46	7.67	25.6	20.8
LosAng	060370002	Annual	No	N/A	N/A	10.52	8.53	29.2	23.7
Louisv	180190006	Annual	Yes	24	0	10.64	10.02	23.9	22.0
Louisv	211110075	Annual	No	24	0	10.42	9.83	22.3	20.3
Louisv	211110067	Annual	No	24	0	9.55	8.96	21.4	19.9
Louisv	211110051	Annual	No	24	0	10.29	9.68	21.8	20.2
Louisv	211110043	Annual	No	24	0	10.37	9.77	22.0	20.2
Louisv	180431004	Annual	No	24	0	9.96	9.37	22.0	20.4
Louisv	180190008	Annual	No	24	0	8.72	8.13	20.1	18.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	10.04	21.2	21.0
MaconG	130210012	Annual	No	N/A	N/A	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	10.04	45.1	30.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	10.04	25.0	24.9
Merced	060472510	24-hr	Yes	68	0	11.68	9.74	39.8	30.4
Merced	060470003	24-hr	No	68	0	11.81	9.82	39.0	29.8
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	9.75	45.7	30.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	25.8
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	10.04	25.1	24.3
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	10.04	23.9	23.5
NewYor	361030002	Annual	No	N/A	N/A	7.18	7.07	18.8	18.5
NewYor	360810124	Annual	No	N/A	N/A	7.52	7.40	19.5	19.2
NewYor	360710002	Annual	No	N/A	N/A	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	N/A	N/A	9.70	9.55	21.6	21.3
NewYor	360610079	Annual	No	N/A	N/A	8.42	8.29	22.8	22.4
NewYor	360470122	Annual	No	N/A	N/A	8.66	8.52	20.5	20.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	8.91	24.0	23.6
NewYor	360050110	Annual	No	N/A	N/A	7.39	7.27	19.4	19.1
NewYor	340392003	Annual	No	N/A	N/A	8.59	8.46	23.6	23.2
NewYor	340390004	Annual	No	N/A	N/A	9.87	9.72	24.2	23.8
NewYor	340310005	Annual	No	N/A	N/A	8.42	8.29	22.2	21.9
NewYor	340292002	Annual	No	N/A	N/A	7.23	7.12	18.1	17.8
NewYor	340273001	Annual	No	N/A	N/A	6.78	6.67	17.1	16.8
NewYor	340171003	Annual	No	N/A	N/A	8.79	8.65	23.4	23.0
NewYor	340130003	Annual	No	N/A	N/A	8.89	8.75	23.8	23.4
NewYor	340030003	Annual	No	N/A	N/A	8.90	8.76	24.5	24.1
OgdenC	490110004	24-hr	Yes	29	0	7.28	7.01	32.6	30.4
OgdenC	490570002	24-hr	No	29	0	8.99	8.71	-	-
OgdenC	490030003	24-hr	No	29	0	6.35	6.10	-	-



CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NOx & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
Philad	420450002	Annual	Yes	86	0	11.46	10.04	26.0	22.3
Philad	421010057	Annual	No	86	0	10.86	9.12	27.0	22.5
Philad	421010055	Annual	No	86	0	11.43	9.95	27.5	23.9
Philad	421010048	Annual	No	86	0	10.27	8.70	25.6	21.1
Philad	420290100	Annual	No	86	0	9.64	7.87	23.9	19.5
Philad	340150004	Annual	No	86	0	8.33	6.99	20.6	16.9
Philad	340071007	Annual	No	86	0	8.84	7.23	21.0	17.1
Philad	340070002	Annual	No	86	0	10.19	8.40	23.5	20.2
Philad	240150003	Annual	No	86	0	8.70	6.90	22.6	17.5
Philad	100031012	Annual	No	86	0	9.04	7.21	23.0	17.7
Pittsb	420030064	24-hr	Yes	100	0	12.82	9.22	35.8	30.4
Pittsb	421290008	24-hr	No	100	0	8.65	6.04	19.6	12.9
Pittsb	421255001	24-hr	No	100	0	8.35	5.90	17.8	11.1
Pittsb	421250200	24-hr	No	100	0	8.95	6.10	19.3	13.7
Pittsb	421250005	24-hr	No	100	0	11.02	7.78	22.7	18.1
Pittsb	420070014	24-hr	No	100	0	10.11	7.38	21.9	15.2
Pittsb	420050001	24-hr	No	100	0	11.03	8.39	21.9	15.5
Pittsb	420031301	24-hr	No	100	0	11.00	7.79	24.8	19.7
Pittsb	420031008	24-hr	No	100	0	9.78	7.11	20.5	14.7
Pittsb	420030008	24-hr	No	100	0	9.50	6.81	20.5	14.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	6.95	37.6	30.4
ProvoO	490494001	24-hr	Yes	6	0	7.74	7.68	30.9	30.4
ProvoO	490495010	24-hr	No	6	0	6.73	6.68	-	-
ProvoO	490490002	24-hr	No	6	0	7.41	7.36	28.9	28.4
Rivers	060658005	Annual	Yes	N/A	N/A	14.48	10.04	43.2	30.0
Rivers	060658001	Annual	No	N/A	N/A	-	-	36.5	25.3
Sacram	060670006	24-hr	Yes	18	0	9.31	9.11	31.4	30.4
Sacram	061131003	24-hr	No	18	0	6.62	6.50	15.8	15.1
Sacram	060670012	24-hr	No	18	0	7.30	7.17	19.8	19.3
Sacram	060670010	24-hr	No	18	0	8.67	8.50	26.5	25.5
Sacram	060610006	24-hr	No	18	0	7.58	7.45	20.3	19.9
Sacram	060610003	24-hr	No	18	0	6.71	6.63	19.3	18.9
SaltLa	490353010	24-hr	Yes	79	0	-	-	41.5	30.3
SaltLa	490353006	24-hr	No	79	0	7.62	6.46	36.8	29.3
SaltLa	490351001	24-hr	No	79	0	7.07	5.88	32.1	23.2
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	10.04	25.9	24.3
SanLui	060798002	Annual	No	N/A	N/A	5.71	5.36	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	7.74	19.8	18.6

CBSA <sup>a</sup>	Site	Controlling Standard	Controlling Site?	NO <sub>x</sub> & SO <sub>2</sub> Reduction (%) <sup>b</sup>	Primary PM <sub>2.5</sub> Reduction (%) <sup>c</sup>	Base Annual DV (µg m <sup>-3</sup> )	Projected Annual DV (µg m <sup>-3</sup> )	Base 24-hr DV (µg m <sup>-3</sup> )	Projected 24-hr DV (µg m <sup>-3</sup> )
SouthB	181410015	24-hr	Yes	30	0	10.45	9.68	32.5	30.4
St.Lou	290990019	Annual	Yes	N/A	N/A	10.12	10.04	22.8	22.6
St.Lou	295100094	Annual	No	N/A	N/A	9.57	9.49	23.3	23.1
St.Lou	295100093	Annual	No	N/A	N/A	-	-	23.7	23.5
St.Lou	295100085	Annual	No	N/A	N/A	10.10	10.02	23.6	23.4
St.Lou	295100007	Annual	No	N/A	N/A	9.78	9.70	23.7	23.5
St.Lou	291893001	Annual	No	N/A	N/A	9.85	9.77	22.4	22.2
Stockt	060771002	Annual	Yes	97	0	12.23	10.04	38.7	29.7
Stockt	060772010	Annual	No	97	0	10.74	8.69	37.3	28.4
Visali	061072002	24-hr	Yes	N/A	N/A	16.23	9.14	54.0	30.4
Weirto	390810017	Annual	Yes	62	0	11.75	10.02	27.2	23.8
Weirto	540090011	Annual	No	62	0	9.75	8.14	22.8	19.9
Weirto	540090005	Annual	No	62	0	10.52	8.82	22.4	18.8
Weirto	390810021	Annual	No	62	0	9.29	7.68	22.2	18.5
Wheeli	540511002	Annual	Yes	N/A	N/A	10.24	10.04	22.5	22.1
Wheeli	540690010	Annual	No	N/A	N/A	9.61	9.42	19.7	19.3

<sup>a</sup> CBSA names are the first six characters of the full CBSAs names in Table C-3.

<sup>b</sup> Percent reduction in NO<sub>x</sub> and SO<sub>2</sub> emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

<sup>c</sup> Percent reduction in Primary PM<sub>2.5</sub> emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

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# APPENDIX D. QUANTITATIVE ANALYSES FOR VISIBILITY IMPAIRMENT

## D.1 BACKGROUND

To inform the EPA's decision in the last review on the adequacy of protection provided by the secondary PM standards the EPA conducted a technical analysis of the relationships between a 3-year average daily visibility metric and the 24-hour PM<sub>2.5</sub> mass-based standard (Kelly et al., 2012). The 3-year visibility metric was calculated as the 3-year average of the 90th percentile of daily visibility index values.<sup>1</sup> Light extinction coefficient ( $b_{ext}$ ) values for the visibility index were calculated using the original IMPROVE equation (Equation D-1 in section D.2.2 below), which at the time of the last review, the EPA considered to be better suited to urban sites that were the focus of the analysis than other versions of the IMPROVE equation, with a few modifications to the equation: excluding the coarse mass<sup>2</sup> and sea salt<sup>3</sup> terms in the equation and using a multiplier of 1.6 for converting OC to OM.<sup>4</sup>

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<sup>1</sup> The visibility index is a logarithmic transformation of the light extinction coefficient,  $b_{ext}$ , the use of which ensures that increases or decreases in light extinction coefficient always produce, respectively, increases or decreases in visibility index (Kelly et al., 2012).

<sup>2</sup> PM<sub>2.5</sub> is the size fraction of PM responsible for most of the visibility impairment in urban areas (U.S. EPA, 2009, section 9.2.2.2). Data available at the time of the last review suggested that, generally, PM<sub>10-2.5</sub> was a minor contributor to visibility impairment most of the time (U.S. EPA, 2010) although the coarse fraction may be a major contributor in some areas in the desert southwestern region of the country. Moreover, at the time of the last review, there were few data available from continuous PM<sub>10-2.5</sub> monitors to quantify the contribution of coarse PM to calculated light extinction.

<sup>3</sup> In estimating light extinction in the last review, the EPA did not consider it appropriate to include the term for hygroscopic sea salt in evaluating urban light extinction, given that sea salt is not a major contributor to light extinction in urban areas compared with more remote coastal locations. In particular, Pitchford (2010) estimated that the contribution of sea salt to PM<sub>2.5</sub> light extinction was generally well below 5% for PM<sub>2.5</sub> light extinction greater than 24 dv (U.S. EPA, 2010, p. 3-22; U.S. EPA, 2012, p. IV-5).

<sup>4</sup> At the time of the last review, the EPA considered the multiplier of 1.8 recommended by Pitchford et al. (2007) to convert OC to OM for use in the revised IMPROVE equation (Equation D-2 below) to be too high for urban environments. The composition of, and the mix of emission sources contributing to, PM<sub>2.5</sub> differ between urban and remote areas, and consequently, the light extinction may differ between urban and remote areas. Organic mass in urban areas is often from local and regional sources and would have a greater percentage of fresh emissions compared with aged emissions, which tend to be more prominent in rural areas, and a different PM mass to OC ratio than in urban areas. The EPA also considered the multiplier of 1.4 used with the original IMPROVE equation to be too low to adequately account for the contribution of OM to visibility impairment, particularly in urban areas where OM concentrations tend to be higher. Based on these considerations, along with an evaluation of the OC to OM relationship at CSN sites (2011 PA, Appendix F, section F.6), the EPA chose to use a multiplier of 1.6 to convert OC to OM in the light extinction calculations used in the last review (U.S. EPA, 2012, pages IV-5-IV-8).

Using 2008-2010 air quality data for 102 CSN network sites,<sup>5</sup> the 2012 analysis explored the relationship between the 3-year design values for the existing 24-hour PM<sub>2.5</sub> standard and values of the 3-year visibility metric.<sup>6</sup> The analysis indicated that increases in 24-hour PM<sub>2.5</sub> design values generally correspond to increases in the 3-year visibility metric values, and vice-versa (78 FR 3201, January 15, 2013). The analysis also found linear correlations between the 24-hour PM<sub>2.5</sub> design values and the 3-year visibility metric with an average  $r^2$  value of 0.75 across all of the sites (Kelly et al., 2012). A key implication of this analysis was that for the level proposed by the EPA for a visibility index-based standard, the 24-hour PM<sub>2.5</sub> standard of 35  $\mu\text{g}/\text{m}^3$  would be controlling in almost all or all instances (78 FR 3202, January 15, 2013).

## **D.2 ANALYSIS: METHODS AND INPUTS**

Consistent with the analyses conducted in the last review described above, we have conducted analyses examining the relationship between PM mass concentrations and estimated light extinction in terms of a PM visibility metric. These analyses are intended to inform our understanding of visibility impairment in the U.S. under recent air quality conditions, particularly those conditions that meet the current standards, and our understanding of the relative influence of various factors on light extinction. These analyses were conducted using three versions of the IMPROVE equation (Equations D-1 through D-3 below) to estimate light extinction to better understand the influence of variability in inputs across the three equations. This analysis included 67 monitoring sites that are geographically distributed across the U.S. in both urban and rural areas (see Figure D-1). The data set is comprised of sites with data for the 2015-2017 period that supported a valid 24-hour PM<sub>2.5</sub> design value<sup>7</sup> and met strict criteria for PM species. Light extinction at these 67 monitoring sites was calculated without the coarse fraction in the IMPROVE equations, consistent with the analyses conducted in the last review. For a subset of 20 of the 67 monitoring sites where PM<sub>10</sub> data were available and met completeness criteria, the coarse fraction was included when calculating light extinction to better characterize the influence of coarse PM on light extinction. Results for these two sets of analyses are presented in Figures 5-3 and 5-4 and discussed in section 5.2.1.2 of Chapter 5 and presented in Table D-7 and Table D-8 and Figure D-2 in section D.3 below.

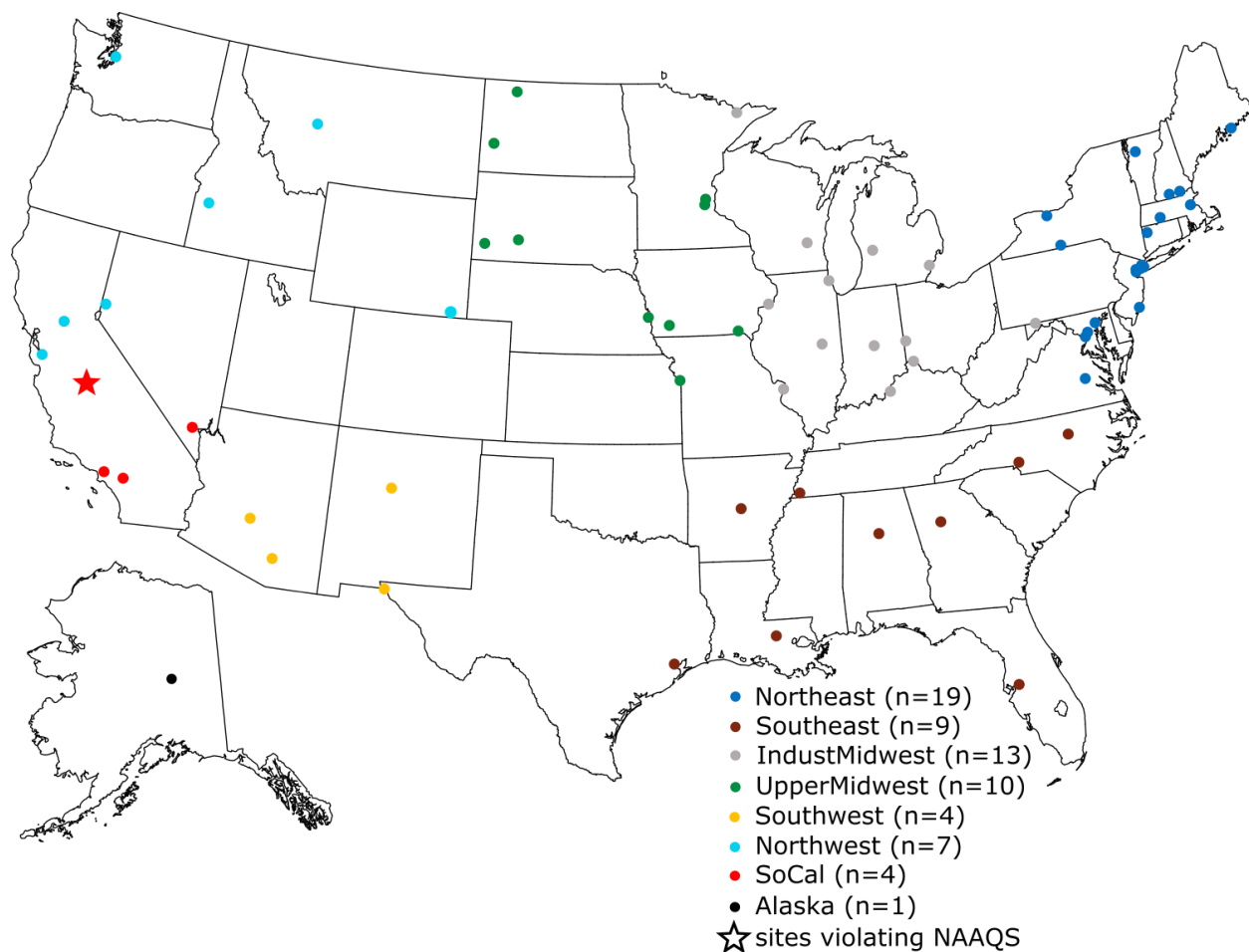
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<sup>5</sup> The 102 sites included in the Kelly et al. (2012) analysis were those sites that met the data completeness criteria used for that analysis (Kelly et al., 2012, p. 15).

<sup>6</sup> The EPA used monthly average relative humidity values rather than shorter-term (e.g., hourly) values to estimate light extinction in the last review in order to capture seasonal variability of relative humidity and its effects on visibility impairment. This was intended to focus more on the underlying aerosol contributions to visibility impairment and less on the day-to-day variations in humidity (U.S. EPA, 2012, p. IV-10).

<sup>7</sup> The design value (DV) for the standard is the metric used to determine whether areas meet or exceed the NAAQS. A design value is a statistic that describes the air quality status of a given area relative to the NAAQS.





**Figure D-1. Locations of monitoring sites with data for 2015-2017 with a valid  $PM_{2.5}$  design value and meeting completeness criteria for PM species.**

## **D.2.1 Data Sources for Inputs to Estimate Light Extinction**

### **D.2.1.1 Relative Humidity**

Relative humidity data were downloaded from the North American Regional Reanalysis (NARR). NARR is the National Centers for Environmental Prediction's (NCEP) high resolution combined model and assimilated meteorological dataset. NARR is an extension of the NCEP Global Reanalysis which is run over North American using the Eta Model (32 km) together with the Regional Data Assimilation System. Files for 3-hour average 10 m relative humidity data for 2015-2017 are available at <https://esrl.noaa.gov/psd/data/gridded/data.narr.html>.

Using NARR latitudes, relative humidity data were reassigned to each grid cell from coordinated universal time (UTC) to their closest time zone and the 3-hour relative humidity data were then averaged to 24-hour local time averages in order to approximate the 24-hour averaging

time (midnight-midnight) of the daily PM<sub>2.5</sub> measurements. The PM<sub>2.5</sub> and PM<sub>2.5</sub> component daily mass data (described in subsequent sections) were temporally and spatially matched with the closest 24-hour average relative humidity grid cell.

#### **D.2.1.2 PM<sub>2.5</sub> Concentrations**

The raw data for PM<sub>2.5</sub> site-level daily mass concentrations came from an Air Quality System (AQS)<sup>8</sup> query of the daily site-level concentrations. Data files used were for 24-hour average values from regulatory monitors for all sites in the U.S. for all available days (including potential exceptional events) for 2015-2017. When a single site had multiple monitors, the previously-determined primary monitor concentration was used. If the primary monitor value was missing, the average of the collocated monitors was used. These data were screened so that all days either had a valid filter-based 24-hour concentration measurement<sup>9</sup> or at least 18 valid hourly concentrations measurements.

#### **D.2.1.3 Coarse PM Concentrations**

The raw data for PM<sub>10-2.5</sub> monitor-level daily mass concentrations came from an AQS query of the daily monitor-level concentrations. Data files used were for 24-hour average concentrations from monitors mainly in the Interagency Monitoring of Protected Visual Environments (IMPROVE) network and NCore Multipollutant Monitoring Network. Data were included for sites with  $\geq 11$  valid days for each quarter of 2015-2017.

#### **D.2.1.4 PM<sub>2.5</sub> Component Concentrations**

The raw data for PM<sub>2.5</sub> component concentrations for the components listed in Table D-1 came from an AQS query of the daily monitor-level concentrations. Data files used were for filter-based, 24-hour average concentrations from monitors in the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, Chemical Speciation Network (CSN), and NCore Multipollutant Monitoring Network. Data were included for days with valid data for all chemical components listed in Table D-1 below and for sites with  $\geq 11$  valid days for each quarter of 2015-2017.

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<sup>8</sup> The Air Quality System is an EPA database of ambient air quality monitoring data (<https://www.epa.gov/aqs>).

<sup>9</sup> A valid filter-based 24-hour concentration measurement is one collected via FRM, and that has undergone laboratory equilibration (at least 24 hours at standardized conditions of 20-23°C and 30-40% relative humidity) prior to analysis (see Appendix L of 40 CFR Part 50 for the 2012 NAAQS for PM).

**Table D-1. PM<sub>2.5</sub> components from AQS used in IMPROVE equations.**

PM <sub>2.5</sub> Component Drawn from AQS	AQS Parameter Code
Sulfate	88403
Nitrate	88306
OC (TOR <sup>a</sup> )	88320, 88370
EC (TOR <sup>a</sup> )	88321, 88380
Aluminum (Al), Silica (Si), Calcium (Ca), Iron (Fe), Titanium (Ti)	88104 (Al), 88165 (Si), 88111 (Ca), 88126 (Fe), 88161 (Ti)
Chloride, Chlorine	88115 (Chlorine), 88203 (Chloride)
<sup>a</sup> OC and EC values are based on the thermal optical reflectance (TOR) analytical method, which replaced the NIOSH 5040-like thermal optical transmittance (TOT) method in the CSN network after 2009 (Spada and Hyslop, 2018).	

#### **D.2.1.5 24-Hour PM<sub>2.5</sub> Design Values**

Files for 24-hour PM<sub>2.5</sub> design values for 2015-2017 are located at <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2015-2017 PM<sub>2.5</sub> design values is described in Appendix N of 40 CFR Part 50 for the 2012 National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM).

#### **D.2.1.6 24-Hour PM<sub>10</sub> Design Values**

Files for 24-hour PM<sub>10</sub> design values for 2015-2017 are located at <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2015-2017 PM<sub>10</sub> design values is described in Appendix K of 40 CFR Part 50.

#### **D.2.1.7 Annual PM<sub>2.5</sub> Design Values**

Files for annual PM<sub>2.5</sub> design values for 2015-2017 are located at <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2015-2017 PM<sub>2.5</sub> design values is described in Appendix N of 40 CFR Part 50 for the 2012 National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM).

### **D.2.2 Calculating Light Extinction for Visibility Impairment Analyses**

For all days with a valid relative humidity value, PM<sub>2.5</sub> mass concentration, and all chemical components listed in Table D-1, daily light extinction was calculated using three versions of the IMPROVE equation, as shown below. Formulas for derivation of the equation variables from the AQS parameters are presented in Table D-6.

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**Original IMPROVE Equation (Malm et al., 1994):**

$$b_{ext} \cong 3f(RH)([AS] + [AN]) + 4[OM] + 10[EC] + 1[FS] + 0.6[CM] + 10$$

**Equation D-1**

where:

[AS] is concentration in  $\mu\text{g}/\text{m}^3$  of ammonium sulfate,

[AN] is concentration in  $\mu\text{g}/\text{m}^3$  of ammonium nitrate,

[OM] is concentration in  $\mu\text{g}/\text{m}^3$  of organic matter,

[EC] is concentration in  $\mu\text{g}/\text{m}^3$  of elemental carbon,

[FS] is concentration in  $\mu\text{g}/\text{m}^3$  of fine soil,

[CM] is concentrations in  $\mu\text{g}/\text{m}^3$  of coarse mass, and

f(RH) is the relative-humidity-dependent water growth function, assigned values as shown in Table D-2:

**Table D-2. Relatively-humidity-dependent water growth function for use in the original IMPROVE equation.**

RH (%)	1-36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56
f(RH)	1	1.02	1.04	1.06	1.08	1.1	1.13	1.15	1.18	1.2	1.23	1.26	1.28	1.31	1.34	1.37	1.41	1.44	1.47	1.51	1.54
RH (%)	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77
f(RH)	1.58	1.62	1.66	1.7	1.74	1.79	1.83	1.88	1.93	1.98	2.03	2.08	2.14	2.19	2.25	2.31	2.37	2.43	2.5	2.56	2.63
RH (%)	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98 <sup>a</sup>
f(RH)	2.7	2.78	2.86	2.94	3.03	3.12	3.22	3.33	3.45	3.58	3.74	3.93	4.16	4.45	4.84	5.37	6.16	7.4	9.59	14.1	26.4
Note: See fRHOriginalIMPROVE.csv file from <a href="http://vista.cira.colostate.edu/Improve/the-improve-algorithm/">http://vista.cira.colostate.edu/Improve/the-improve-algorithm/</a> (Malm et al., 1994).																					
<sup>a</sup> For our application, any relative humidity values greater than 98% were assigned the f(RH) value associated with 98%, the highest value available for the relative humidity function.																					

The various coefficients are the empirically derived extinction efficiency (mass scattering and absorption) coefficients, as originally specified by Malm et al. (1994).

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**Revised IMPROVE Equation (Pitchford et al., 2007):**

$$b_{ext} \cong 2.2f_S(RH)[small\ sulfate] + 4.8f_L(RH)[large\ sulfate] + 2.4f_S(RH)[small\ nitrate] \\ + 5.1f_L(RH)[large\ nitrate] + 2.8[small\ OM] + 6.1[large\ OM] + 10[EC] \\ + 1[FS] + 1.7f_{SS}(RH)[SS] + 0.6[CM] + 10$$

**Equation D-2**

where:

[small sulfate], [large sulfate], [small nitrate], [large nitrate], [small OM] and [large OM] are defined as follows in Table D-3:

**Table D-3. Values for use in the revised IMPROVE equation for small and large sulfate, nitrate, and organic matter concentrations.**

	If [ ] $\geq 20$	If [ ] $< 20$
<b>Large sulfate</b>	[AS]	[AS]÷20
<b>Small sulfate</b>	0	[AS] - ([AS]÷20)
<b>Large nitrate</b>	[AN]	[AN]÷20
<b>Small nitrate</b>	0	[AN] - ([AN]÷20)
<b>Large OM</b>	[OM]	[OM]÷20
<b>Small OM</b>	0	[OM] - ([OM]÷20)
Note: [AS], [AN] and [OM] are defined as for Equation D-1.		

[SS] is sea salt; and,

$f_{SS}(RH)$ ,  $f_S(RH)$ , and  $f_L(RH)$  are defined as shown in Table D-4:

**Table D-4. Relatively-humidity-dependent water growth function for sea salt, small particles, and large particles for use in the revised IMPROVE equation.**

RH (%)	1-36	37	38	39	40	41	42	43	44	45	46	47	48	49	50
f <sub>ss</sub> (RH)	1	1	1	1	1	1	1	1	1	1	1	2.3584	2.3799	2.4204	2.4488
f <sub>s</sub> (RH)	1	1.38	1.4	1.42	1.44	1.46	1.48	1.49	1.51	1.53	1.55	1.57	1.59	1.62	1.64
f <sub>L</sub> (RH)	1	1.31	1.32	1.34	1.35	1.36	1.38	1.39	1.41	1.42	1.44	1.45	1.47	1.49	1.5
RH (%)	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65
f <sub>ss</sub> (RH)	2.4848	2.5006	2.5052	2.5279	2.5614	2.5848	2.5888	2.616	2.6581	2.6866	2.7341	2.7834	2.8272	2.8287	2.8594
f <sub>s</sub> (RH)	1.66	1.68	1.71	1.73	1.76	1.78	1.81	1.83	1.86	1.89	1.92	1.95	1.99	2.02	2.06
f <sub>L</sub> (RH)	1.52	1.54	1.55	1.57	1.59	1.61	1.63	1.65	1.67	1.69	1.71	1.73	1.75	1.78	1.8
RH (%)	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80
f <sub>ss</sub> (RH)	2.8943	2.9105	2.9451	3.0105	3.0485	3.1269	3.1729	3.2055	3.2459	3.2673	3.3478	3.4174	3.5202	3.5744	3.6329
f <sub>s</sub> (RH)	2.09	2.13	2.17	2.22	2.26	2.31	2.36	2.41	2.47	2.54	2.6	2.67	2.75	2.84	2.93
f <sub>L</sub> (RH)	1.83	1.86	1.89	1.92	1.95	1.98	2.01	2.05	2.09	2.13	2.18	2.22	2.27	2.33	2.39
RH (%)	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95 <sup>a</sup>
f <sub>ss</sub> (RH)	3.6905	3.808	3.9505	4.0398	4.1127	4.2824	4.494	4.6078	4.8573	5.1165	5.3844	5.7457	6.1704	6.7178	7.3492
f <sub>s</sub> (RH)	3.03	3.15	3.27	3.42	3.58	3.76	3.98	4.23	4.53	4.9	5.35	5.93	6.71	7.78	9.34
f <sub>L</sub> (RH)	2.45	2.52	2.6	2.69	2.79	2.9	3.02	3.16	3.33	3.53	3.77	4.06	4.43	4.92	5.57
Note: See fRHRevisedIMPROVE.csv file from <a href="http://vista.cira.colostate.edu/improve/the-improve-algorithm/">http://vista.cira.colostate.edu/improve/the-improve-algorithm/</a> (Pitchford et al., 2007).															
<sup>a</sup> For our application, any relative humidity values greater than 95% were assigned the f(RH) value associated with 95%, the highest value available for the relative humidity function.															

and

[EC], [FS] and [CM] are defined as for Equation D-1.

This equation is generally dividing PM components into small and large particle sizes<sup>10</sup> with separate mass scattering efficiencies and hygroscopic growth functions for each size (included in the equation as f<sub>s</sub>(RH) for small particles, f<sub>L</sub>(RH) for large particles, and f<sub>ss</sub>(RH) for sea salt).

<sup>10</sup> The large mode for sulfate, nitrate, and OM represents aged and/or cloud processed particles, whereas the small mode represents freshly formed particles. These size modes are described by log-normal mass size distributions with geometric mean diameters and geometric standard deviations of 0.2  $\mu\text{m}$  and 2.2 for small mode and 0.5  $\mu\text{m}$  and 1.5 for the large mode, respectively.

**Lowenthal and Kumar (2016) Equation:**

$$\begin{aligned}
b_{ext} \cong & 2.2f_s(RH)[small\ sulfate] + 4.8f_L(RH)[large\ sulfate] + 2.4f_s(RH)[small\ nitrate] \\
& + 5.1f_L(RH)[large\ nitrate] + 2.8f_s(RH)_{OM}[small\ OM] \\
& + 6.1f_L(RH)_{OM}[large\ OM] + 10[EC] + 1[FS] + 1.7f_{SS}(RH)[SS] + 0.6[CM] \\
& + 10
\end{aligned}$$

**Equation D-3**

where:

$f_s(RH)_{OM}$  and  $f_L(RH)_{OM}$  are the relative-humidity-dependent water growth function for small and large organic matter, respectively, as defined in Table D-5 below.

**Table D-5. Relatively-humidity-dependent water growth function for small organic matter and large organic matter for use in the original IMPROVE equation.**

RH (%)	0-29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45
$f_s(RH)_{OM}$	1.000	1.321	1.325	1.329	1.333	1.337	1.340	1.343	1.346	1.349	1.352	1.354	1.356	1.358	1.360	1.362	1.364
$f_L(RH)_{OM}$	1.000	1.267	1.271	1.274	1.278	1.280	1.283	1.286	1.288	1.290	1.292	1.294	1.296	1.297	1.299	1.300	1.302
RH (%)	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62
$f_s(RH)_{OM}$	1.366	1.368	1.369	1.371	1.373	1.75	1.377	1.379	1.382	1.384	1.387	1.390	1.393	1.397	1.400	1.404	1.409
$f_s(RH)_{OM}$	1.303	1.305	1.306	1.308	1.309	1.311	1.306	1.308	1.309	1.311	1.313	1.314	1.316	1.318	1.320	1.323	1.325
RH (%)	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79
$f_s(RH)_{OM}$	1.413	1.419	1.424	1.430	1.437	1.444	1.452	1.460	1.469	1.478	1.489	1.500	1.511	1.524	1.537	1.51	1.566
$f_s(RH)_{OM}$	1.328	1.331	1.334	1.338	1.342	1.346	1.350	1.355	1.385	1.393	1.401	1.409	1.418	1.428	1.438	1.449	1.461
RH (%)	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95 <sup>a</sup>	
$f_s(RH)_{OM}$	1.582	1.599	1.617	1.637	1.657	1.679	1.703	1.727	1.754	1.782	1.812	1.843	1.877	1.912	1.950	1.989	
$f_s(RH)_{OM}$	1.473	1.486	1.500	1.515	1.531	1.548	1.566	1.585	1.605	1.626	1.648	1.672	1.696	1.722	1.750	1.779	

Note: See Table 1 in Lowenthal and Kumar (2016).

<sup>a</sup> For our application, any relative humidity values greater than 95% were assigned the  $f(RH)$  value associated with 95%, the highest value available for the relative humidity function.

and

[small sulfate], [large sulfate], [small nitrate], [large nitrate], [small OM], [large OM], [EC], [FS], [SS], [CM],  $f_s(RH)$ ,  $f_L(RH)$  and  $f_{SS}(RH)$  are defined as above for Equation D-2.

This equation updates the multiplier for estimating the concentration organic matter, [OM], from the concentration of organic carbon to 2.1 and incorporates  $f_s(RH)_{OM}$  and  $f_L(RH)_{OM}$  representing water absorption by soluble organic matter as a function of relative humidity for small and large organic matter, respectively.

Based on each equation, site-specific visibility metrics were derived for each site as follows. Daily light extinction values were derived for 2015, 2016, and 2017, the 90<sup>th</sup> percentile of daily values for each year was calculated, and the three years of values were averaged. The 3-year averages of the 90<sup>th</sup> percentiles of daily light extinction values were paired with the 2015-2017 PM<sub>2.5</sub> 24-hour design values for each site having valid data for both statistics.

**Table D-6. Derivation of equation variables from AQS PM<sub>2.5</sub> component concentrations.**

Equation Variable	How Calculated from AQS Parameter Values
Ammonium Sulfate	All three equations: $1.375 \times [\text{Sulfate}]^A$
Ammonium Nitrate	All three equations: $1.29 \times [\text{Nitrate}]^B$
Organic Matter	Original IMPROVE equation: $1.6 \times [\text{OC}]^C$ Revised IMPROVE equation: $1.6 \times [\text{OC}]^C$ Lowenthal and Kumar (2016) equation: $2.1 \times [\text{OC}]$
Elemental Carbon	$[\text{EC}]$
Fine Soil	All three equations: <sup>D</sup> $2.2 \times [\text{Al}] + 2.49 \times [\text{Si}] + 1.63 \times [\text{Ca}] + 2.42 \times [\text{Fe}] + 1.94 \times [\text{Ti}]$
Sea Salt	Revised IMPROVE and Lowenthal and Kumar, 2016 equations: <sup>D</sup> $1.8 \times [\text{Chloride}]$ $1.8 \times [\text{Chlorine}]$ (if chloride is missing)
<sup>A</sup> This formula is based on molar molecular weights of ammonium sulfate and sulfate (Malm et al., 1994). <sup>B</sup> This formula is based on molar molecular weights of ammonium nitrate and nitrate (Malm et al., 1994). <sup>C</sup> See footnote 4 earlier in this appendix. <sup>D</sup> This formula is documented in Malm et al. (1994).	

### D.3 SUMMARY OF RESULTS

Results for the visibility impairment analyses are discussed in section 5.2.1.2 of Chapter 5. Table D-7 presents the 24-hour PM<sub>2.5</sub>, 24-hour PM<sub>10</sub> design values, and 3-year visibility metrics based on light extinction calculations using the three versions of the IMPROVE equation with the coarse mass fraction excluded for the 67 monitoring sites included in the analyses. Table D-8 presents the 24-hour PM<sub>2.5</sub> and 24-hour PM<sub>10</sub> design values, along with the 3-year visibility metrics based on light extinction calculations using the three versions of the IMPROVE equation with and without the coarse mass fraction for the subset of 20 monitoring sites with coarse PM monitoring data that meet the completeness criteria as described above. Figure 5-3 and 5-4 in Chapter 5 show a comparison of the 3-year visibility metric and the 24-hour PM<sub>2.5</sub> design values for the 67 monitoring sites in the analyses where light extinction was calculated using the



original IMPROVE equation<sup>11</sup> and the Lowenthal and Kumar IMPROVE equation.<sup>12</sup> Figure D-2 below presents the 3-year visibility metric and the 24-hour PM<sub>2.5</sub> design values for the 67 monitoring sites with light extinction calculated using the revised IMPROVE equation.<sup>13</sup>

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<sup>11</sup> For this analysis, the original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM and to remove the coarse mass fraction from the light extinction calculation, consistent with the modifications in the last review.

<sup>12</sup> For this analysis, the Lowenthal and Kumar IMPROVE equation in Equation D-3 was modified to remove the coarse mass fraction from the light extinction calculation.

<sup>13</sup> For this analysis, the revised IMPROVE equation in Equation D-2 was modified to use a 1.6 multiplier to convert OC to OM and to remove the coarse mass fraction from the light extinction calculation, consistent with the modifications in the last review.

**Table D-7. Summary of 24-hour PM<sub>2.5</sub>, 24-hour PM<sub>10</sub>, and annual PM<sub>2.5</sub> design values, and 3-year visibility metrics at 67 monitoring sites (2015-2017).**

Monitor ID	State	Region	24-hour PM <sub>2.5</sub> Design Value (µg/m <sup>3</sup> ) <sup>A</sup>	24-hour PM <sub>10</sub> Design Value (number of exceedances) <sup>B</sup> <sub>C</sub>	Annual PM <sub>2.5</sub> Design Value (µg/m <sup>3</sup> ) <sup>D</sup>	3-year Visibility Metric (deciviews) <sup>E</sup>		
						Original IMPROVE Equation <sup>F</sup>	Revised IMPROVE Equation <sup>G</sup>	Lowenthal & Kumar IMPROVE Equation <sup>H</sup>
010730023	Alabama	Southeast	22	0	10.4	21	21	26
020900034	Alaska	Alaska	35	0	9.5	27	27	31
040139997	Arizona	Southwest	21	0.3	7.1	18	18	21
040191028	Arizona	Southwest	12		5.5	13	13	15
051190007	Arkansas	Southeast	19	0	9.4	20	20	24
060190011	California	SoCal	54	0.3	14	25	27	31
060371103	California	SoCal	32	0	12.1	24	25	27
060658001	California	SoCal	34	0	12.3	23	25	28
060670006	California	Northwest	34	0	9.6	24	25	30
060850005	California	Northwest	27	0	9.3	22	22	26
090050005	Connecticut	Northeast	13	0	4.6	17	16	18
110010043	District of Columbia	Northeast	21	0	9.2	23	22	25
120573002	Florida	Southeast	17	0	7.4	18	17	20
130890002	Georgia	Southeast	19	0	9.0	20	19	24
160010010	Idaho	Northwest	31		7.6	23	23	26
170191001	Illinois	IndustrialMidwest	17		7.6	21	20	21
170314201	Illinois	IndustrialMidwest	21	0	8.4	23	23	25
180970078	Indiana	IndustrialMidwest	21	0	9.1	23	23	26
191370002	Iowa	UpperMidwest	16		6.5	18	17	19
191630015	Iowa	IndustrialMidwest	20	0	8.2	22	21	23
191770006	Iowa	UpperMidwest	18	0	6.9	21	20	22

202090021	Kansas	UpperMidwest	21		8.8	21	21	24
211110067	Kentucky	IndustrialMidwest	19		8.6	22	21	24
220330009	Louisiana	Southeast	20	0	9.0	21	20	24
230090103	Maine	Northeast	12	0	4.1	18	16	19
240053001	Maryland	Northeast	23		8.9	23	23	26
240230002	Maryland	IndustrialMidwest	14		5.5	17	17	18
240330030	Maryland	Northeast	18	0	8.4	21	20	24
250130008	Massachusetts	Northeast	14		5.7	20	19	23
250250042	Massachusetts	Northeast	16	0	7.0	20	19	22
260810020	Michigan	IndustrialMidwest	23	0	8.5	23	23	25
261630001	Michigan	IndustrialMidwest	22	0	8.9	24	24	26
270031002	Minnesota	UpperMidwest	18	0	6.7	20	20	23
270530963	Minnesota	UpperMidwest	18		7.2	22	22	24
270750005	Minnesota	IndustrialMidwest	12		4.0	15	15	17
295100085	Missouri	IndustrialMidwest	20	0	8.9	22	21	24
300490004	Montana	Northwest	33		4.1	15	15	20
310550019	Nebraska	UpperMidwest	20	0	8.9	19	18	20
320030540	Nevada	SoCal	23	0.7	8.2	19	19	22
320310016	Nevada	Northwest	20	0	7.2	18	18	22
330115001	New Hampshire	Northeast	12		4.6	14	13	15
330150018	New Hampshire	Northeast	14		5.1	18	17	19
340010006	New Jersey	Northeast	15		6.8	19	19	20
340130003	New Jersey	Northeast	20	0	8.6	23	23	26
340390004	New Jersey	Northeast	23		9.7	24	24	27
350010023	New Mexico	Southwest	18	0	5.8	15	15	18
360050110	New York	Northeast	19		6.9	23	23	25
360551007	New York	Northeast	16		6.5	21	21	23
360610134	New York	Northeast	21		9.3	24	24	27
360810124	New York	Northeast	19		7.3	22	21	24
361010003	New York	Northeast	12		5.0	18	17	19
371190041	North Carolina	Southeast	17		8.5	19	19	23

371830014	North Carolina	Southeast	18		8.8	19	18	22
380070002	North Dakota	UpperMidwest	18	0	4.1	14	13	15
380130004	North Dakota	UpperMidwest	24	0	4.3	18	18	18
390610040	Ohio	IndustrialMidwest	20	0	8.9	23	22	24
391351001	Ohio	IndustrialMidwest	17		7.7	22	21	23
460330132	South Dakota	UpperMidwest	16	0	3.7	12	11	14
460710001	South Dakota	UpperMidwest	15	0	3.5	12	11	14
471570075	Tennessee	Southeast	15		7.6	19	18	21
481410044	Texas	Southwest	23		8.9	17	17	20
482011039	Texas	Southeast	20	0	8.6	21	21	24
500070007	Vermont	Northeast	10		3.2	16	15	17
510870014	Virginia	Northeast	16	0	7.4	20	19	24
530330080	Washington	Northwest	20		6.4	20	20	23
550270001	Wisconsin	IndustrialMidwest	18	0	6.8	22	22	24
560210100	Wyoming	Northwest	14		4.1	13	12	15

<sup>A</sup> The 24-hour PM<sub>2.5</sub> design value is the 3-year average of the 98<sup>th</sup> percentile of daily PM<sub>2.5</sub> mass concentrations. The current 24-hour PM<sub>2.5</sub> NAAQS is set at a level of 35 µg/m<sup>3</sup>.

<sup>B</sup> The 24-hour PM<sub>10</sub> design value is not to be exceeded more than once per year on average over three years. The current 24-hour PM<sub>10</sub> NAAQS is set at a level of 150 µg/m<sup>3</sup>.

<sup>C</sup> For some monitoring locations, PM<sub>10</sub> design values are not available because of a lack of collocated PM<sub>10</sub> monitoring at the site or insufficient data after applying completeness criteria for calculating PM<sub>10</sub> design values.

<sup>D</sup> The annual PM<sub>2.5</sub> design value is the annual mean, averaged over three years. The current secondary annual PM<sub>2.5</sub> NAAQS is set at a level of 15.0 µg/m<sup>3</sup>.

<sup>E</sup> The 3-year visibility metric is the 3-year average of the 90<sup>th</sup> percentile of daily light extinction. In the last review, the target level of protection identified for the 3-year visibility metric was 30 deciviews.

<sup>F</sup> The original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM and to remove the coarse mass fraction from the light extinction calculation, consistent with the modifications in the last review.

<sup>G</sup> The revised IMPROVE equation in Equation D-2 was modified to use a 1.6 multiplier to convert OC to OM and to remove the coarse mass fraction from the light extinction calculation, consistent with the modifications in the last review.

<sup>H</sup> The Lowenthal and Kumar IMPROVE equation in Equation D-3 was modified to remove the coarse mass fraction from the light extinction calculation.

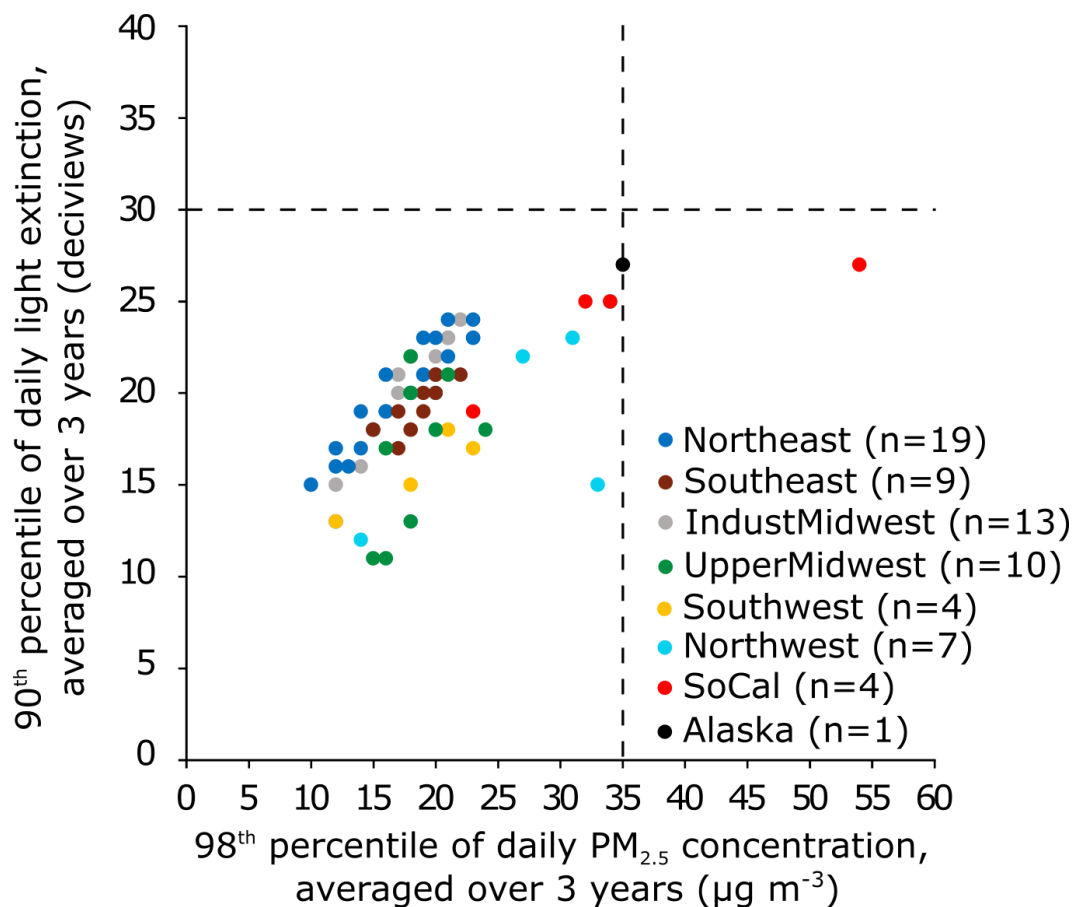
**Table D-8. Summary of 24-hour PM<sub>2.5</sub>, 24-hour PM<sub>10</sub> and annual PM<sub>2.5</sub> design values, and 3-year visibility metrics at 20 monitoring sites with collocated PM<sub>2.5</sub> and PM<sub>10</sub> monitoring data (2015-2017).**

Monitor ID	State	Region	24-hour PM <sub>2.5</sub> Design Value (µg/m <sup>3</sup> ) <sup>A</sup>	24-hour PM <sub>10</sub> Design Value (number of exceedances) <sup>B C</sup>	Annual PM <sub>2.5</sub> Design Value (µg/m <sup>3</sup> ) <sup>D</sup>	3-year Visibility Metric (deciviews) <sup>E</sup>					
						Original IMPROVE Equation <sup>F</sup>		Revised IMPROVE Equation <sup>G</sup>		Lowenthal & Kumar IMPROVE Equation	
						Without [CM] <sup>H</sup>	With [CM] <sup>I</sup>	Without [CM] <sup>H</sup>	With [CM] <sup>I</sup>	Without [CM] <sup>H</sup>	With [CM] <sup>I</sup>
051190007	Arkansas	Southeast	19	0	9.4	20	21	20	21	24	24
060670006	California	Northwest	34	0	9.6	24	25	25	25	30	29
060850005	California	Northwest	27	0	9.3	22	23	22	23	26	27
120573002	Florida	Southeast	17	0	7.4	18	19	17	18	20	20
160010010	Idaho	Northwest	31		7.6	23	22	23	23	26	25
180970078	Indiana	IndustrialMidwest	21	0	9.1	23	24	23	23	26	26
191630015	Iowa	IndustrialMidwest	20	0	8.2	22	22	21	22	23	24
211110067	Kentucky	IndustrialMidwest	19		8.6	22	22	21	22	24	24
230090103	Maine	Northeast	12	0	4.1	18	19	16	17	19	19
250250042	Massachusetts	Northeast	16	0	7.0	20	20	19	20	22	22
260810020	Michigan	IndustrialMidwest	23	0	8.5	23	23	23	23	25	26
261630001	Michigan	IndustrialMidwest	22	0	8.9	24	25	24	25	26	27
320310016	Nevada	Northwest	20	0	7.2	18	19	18	19	22	23
340130003	New Jersey	Northeast	20	0	8.6	23	24	23	24	22	26
390610040	Ohio	IndustrialMidwest	20	0	8.9	23	24	22	23	24	25
391351001	Ohio	IndustrialMidwest	17		7.7	22	22	21	21	23	23
471570075	Tennessee	Southeast	15		7.6	19	20	18	19	21	22
500070007	Vermont	Northeast	10		3.2	16	16	15	15	17	17
510870014	Virginia	Northeast	16	0	7.4	20	20	19	20	24	24
530330080	Washington	Northwest	20		6.4	20	21	20	20	23	25

<sup>A</sup> The 24-hour PM<sub>2.5</sub> design value is the 3-year average of the 98<sup>th</sup> percentile of daily PM<sub>2.5</sub> mass concentrations. The current secondary 24-hour PM<sub>2.5</sub> NAAQS is set at a level of 35 µg/m<sup>3</sup>.

<sup>B</sup> The 24-hour PM<sub>10</sub> design value is not to be exceeded more than once per year on average over three years. The current secondary 24-hour PM<sub>10</sub> NAAQS is set at a level of 150 µg/m<sup>3</sup>.

- <sup>C</sup> For some monitoring locations, PM<sub>10</sub> design values are not available because of a lack of collocated PM<sub>10</sub> monitoring at the site or insufficient data after applying completeness criteria for calculating PM<sub>10</sub> design values.
- <sup>D</sup> The annual PM<sub>2.5</sub> design value is the annual mean, averaged over three years. The current secondary annual PM<sub>2.5</sub> NAAQS is set at a level of 15.0 µg/m<sup>3</sup>.
- <sup>E</sup> The 3-year visibility metric is the 3-year average of the 90<sup>th</sup> percentile of daily light extinction. In the last review, the target level of protection identified for the 3-year visibility metric was 30 deciviews.
- <sup>F</sup> The original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM, consistent with the modifications in the last review.
- <sup>G</sup> The revised IMPROVE equation in Equation D-2 was modified to use a 1.6 multiplier to convert OC to OM, consistent with the modifications in the last review.
- <sup>H</sup> Light extinction was calculated with the coarse mass fraction removed from the equation.
- <sup>I</sup> Although the addition of coarse mass increases the daily extinction calculation, it is possible for the 90<sup>th</sup> percentile value to decrease due to a different set of days having valid measurements of both PM<sub>2.5</sub> chemical composition and PM<sub>10-2.5</sub>.



**Figure D-2. Comparison of 90<sup>th</sup> percentile of daily light extinction, averaged over three years, and 98<sup>th</sup> percentile of daily PM<sub>2.5</sub> concentrations, averaged over three years, for 2015-2017 using the revised IMPROVE equation.** (Note: Dashed lines indicate the level of current 24-hour PM<sub>2.5</sub> standard (35 µg/m<sup>3</sup>) and the target level of protection identified for the 3-year visibility metric (30 dv).)

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## **ATTACHMENT: SUMMARY OF VISIBILITY PREFERENCE STUDIES**

The preference studies available at the time of the last review were conducted in four urban areas. Three western preference studies were available, including one in Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009).<sup>14</sup> Study specific details for these preference studies are shown in Table D-9.

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<sup>14</sup> The replicate study with 26 participants was one test group of three included in Smith and Howell (2009). This study also included two additional test groups to assess varying light extinction conditions using the same scene as was used in the first test group. Study details in Table D-9 reflect all three test groups included in the study. However, for reasons described in section 2.5.2 of U.S. EPA (2010), results from the other two test groups were not included in the EPA's evaluation of levels of acceptable visibility impairment from the preference studies.

**Table D-9. Summary of visibility preference studies.** (Adapted from Table 9-2 in U.S. EPA, 2009).

	Denver, CO	Phoenix, AZ	Vancouver, British Columbia	Washington, DC	Washington, DC
Report Date	1991	2003	1996	2001	2009
Duration of session		45 minutes	50 minutes	2 hours	
Compensation	None	\$50	None	\$50	None
# focus group sessions	16 <sup>a</sup>	27 <sup>b</sup>	4	1	3 tests
# participants	214	385	180	9	64
Age range	Adults	18-65+	University students	27-58	Adults
Annual or seasonal	Wintertime	Annual	Summertime	Annual	Annual
# and type of scene presented	Single scene of downtown Denver with the mountains in the south in the background	Single scene of downtown Phoenix with the Estrella Mountains in the background, 42 km max. distance	Single scene from each of two suburbs in the lower Fraser River valley – Chilliwack and Abbotsford <sup>c</sup>	Single scene of Potomac River, Washington Mall and downtown Washington, DC, 8 km max. sight	Single scene of DC Mall and downtown, 8 km maximum sight
# total visibility conditions presented	20 conditions (+ 5 duplicates)	21 conditions (+ 4 duplicates)	20 conditions (10 from each city)	20 conditions (+ 5 duplicates)	22 conditions
Source of slides	Actual photos taken between 9am and 3pm	WinHaze	Actual photos taken at 1pm or 4pm	WinHaze	WinHaze
Medium of presentation	Slide projection	Slide projection	Slide projection	Slide projection	Slide projection
Ranking scale used	7 point scale	7 point scale	7 point scale	7 point scale	7 point scale
Visibility range presented (dv)	11-40	15-35	Chilliwack: 13-25 Abbotsford: 13.5-31.5	9-38	9-45
Health issue directions	Ignore potential health impacts; visibility only	Judge solely on visibility, do not consider health	Judge solely on visibility, do not consider health	Health never mentioned, "Focus only on visibility"	Health never mentioned, "Focus only on visibility"
Key questions asked	<ul style="list-style-type: none"> <li>•Rank VAQ (1-7 scale)</li> <li>•Is each slide "acceptable"</li> <li>•"How much haze is too much?"</li> </ul>	<ul style="list-style-type: none"> <li>•Rank VAQ (1-7 scale)</li> <li>•Is each slide "acceptable"</li> <li>•How many days a year would this picture be "acceptable"</li> </ul>	<ul style="list-style-type: none"> <li>•Rank VAQ (1-7 scale)</li> <li>•Is each slide "acceptable"</li> </ul>	<ul style="list-style-type: none"> <li>•Rank VAQ (1-7 scale)</li> <li>•Is each slide "acceptable"</li> <li>•If this hazy, how many hours would it be acceptable (3 slides only)</li> <li>•Valuation question</li> </ul>	<ul style="list-style-type: none"> <li>•Rank VAQ (1-7 scale)</li> <li>•Is each slide "acceptable"</li> </ul>
Mean dv found "acceptable"	20.3	23-25	Chilliwack: ~23 Abbotsford: ~19	~20 (range 20-25)	~30
<sup>a</sup> No preference data were collected at a 17 <sup>th</sup> focus group session due to a slide projector malfunction. <sup>b</sup> The 27 focus groups were conducted in 6 neighborhood locations in Phoenix, with 3 focus groups held in Spanish. <sup>c</sup> Chilliwack scene includes downtown buildings in the foreground with mountains in the background up to 65 km away. Abbotsford scene has fewer manmade objects in the foreground and is primarily a more rural scene with mountains in the background up to 55 km away.					

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United States  
Environmental Protection  
Agency

Office of Air Quality Planning and Standards  
Health and Environmental Impacts Division  
Research Triangle Park, NC

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October 22, 2019

The Honorable Andrew R. Wheeler  
Administrator  
U.S. Environmental Protection Agency  
1200 Pennsylvania Avenue, N.W.  
Washington, D.C. 20460

Ref: Docket ID No. EPA-HQ-OAR-2015-0072

Subject: Advice from the Independent Particulate Matter Review Panel (formerly EPA CASAC Particulate Matter Review Panel) on EPA's Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – September 2019)

Dear Administrator Wheeler:

We were members of the U.S. Environmental Protection Agency (EPA) Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel that was dismissed without notice by press release on October 10, 2018. After being disbanded, we formed the nongovernmental Independent Particulate Matter Review Panel (IPMRP, or “the Panel”). The Panel submitted comments to the CASAC on the draft PM Integrated Science Assessment (ISA) on December 10, 2018 and March 27, 2019. The IPMRP met on October 10-11, 2019, and October 18, 2019, to peer review EPA's Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – September 2019), hereafter referred to as the draft PA.

The roster of IPMRP members is given as **Attachment A**. Compared to the chartered CASAC, this IPMRP has more experts, covers more scientific disciplines, and has multiple experts who provide diversity of perspectives in many key disciplines, such as epidemiology, toxicology, and human clinical studies, among others. The IPMRP includes 20 members of the disbanded CASAC PM Review Panel, including seven members who have served on the chartered CASAC, three members who have chaired CASAC review panels, and one former CASAC chair. IPMRP members were subject to a good faith ethics review by the former director of the EPA Science Advisory Board Staff Office. The IPRMP meeting was conducted according to the same procedures as a CASAC meeting. Panelists were reimbursed by the Union of Concerned Scientists for travel to attend the October 10-11, 2019 meeting but did not accept honoraria or other compensation. The content of the meetings, this letter, and attachments were determined exclusively by the Panel, and reflect exclusively the Panel's deliberations.

The IPMRP's consensus responses to the agency's charge questions, and supplemental charge questions developed by us, are given in **Attachment B**. Individual review comments from members of the Panel are given in **Attachment C**. The history, membership criteria, and administrative procedures of the Panel are in **Attachment D**. Panel member biographies are in **Attachment E**. Major comments and recommendations are highlighted below and detailed in the consensus responses to charge questions, with additional details in individual comments.

## Summary

Based on scientific evidence, as detailed in Attachment B, the Panel finds that the current suite of primary fine particle (PM<sub>2.5</sub>) annual and 24-hour standards are not protective of public health. Both of these standards should be revised to new levels, while retaining their current indicators, averaging times, and forms. The annual standard should be revised to a range of 10 µg/m<sup>3</sup> to 8 µg/m<sup>3</sup>. The 24-hour standard should be revised to a range of 30 µg/m<sup>3</sup> to 25 µg/m<sup>3</sup>. These scientific findings are based on consistent epidemiological evidence from multiple multi-city

studies, augmented with evidence from single-city studies, at policy-relevant ambient concentrations in areas with design values at and below the levels of the current standards, and are supported by research from experimental models in animals and humans and by accountability studies.

The weight of evidence framework for causality determination that is applied by EPA is an appropriate and well-vetted tool for drawing causal conclusions. The epidemiologic evidence, supported by evidence from controlled human studies and toxicological studies, supports the 'causal' and 'likely to be causal' determinations for combinations of exposure duration, indicator, and health outcome that are the focus of the draft PA for the evidence- and risk-based approaches. The epidemiologic evidence provides strong scientific support for recommendations regarding current and alternative standard levels. Arguments offered in the draft PA for retaining the current primary PM<sub>2.5</sub> standards, which among other things, would require disregard of the epidemiological evidence, are not scientifically justified and are specious.

There is no new information that calls into question the current indicator, form, and averaging time for the coarse PM primary standard. The level of the coarse PM standard should be revised downward, consistent with the recommended downward revision of the 24-hour primary PM<sub>2.5</sub> standard, to at least maintain, if not increase, the current level of public health protection to coarse particles. A second draft of the PA should provide supporting analyses for this and other possible revised coarse PM standards.

The current annual secondary standard has no effect given that its level is higher than that of the current primary standard. Based on available evidence regarding visibility effects, and to be requisite to protect public welfare, the annual secondary standard should be revised to a level at least equal to that of the revised primary annual PM<sub>2.5</sub> standard. The current 24-hour secondary standard is also not adequate to protect against visibility effects. A second draft of the PA should analyze options for alternative secondary standards. The Panel offers detailed recommendations regarding alternative indicators, averaging times, forms, and levels that should be considered.

The Panel finds that background PM<sub>2.5</sub> levels are substantially below the levels of current and recommended alternative standards. Specific recommendations for areas of new research are provided.

A second draft of the ISA should be reviewed by CASAC and the public, and the ISA should be finalized, prior to release of a second external review draft of the PA. Although a smaller "pool" of consultants was recently appointed to support the CASAC, the pool is not focused on PM, did not review the draft PM ISA, interacts with the CASAC only in writing, and is not allowed to deliberate with the CASAC; therefore, the pool does not adequately or appropriately substitute for the disbanded CASAC PM Review Panel. The CASAC PM Review Panel should be reappointed to provide CASAC with the expertise it needs.

### **Unacceptable Process Changes Should be Documented and Corrected**

The Panel finds that the EPA staff in the Office of Air Quality Planning and Standards have undertaken a good faith effort to produce a first draft of the PA. This draft was produced under extenuating, unprecedented, and inappropriate constraints. The Panel commends the staff for this effort.

Chapter 1 should document all deviations to the CASAC and the National Ambient Air Quality Standards (NAAQS) review process for PM relative to the process outlined in the final 2016 PM Integrated Review Plan. Chapter 1 should cite and discuss the implications of the August 23,

2019 decision of the Court of Appeals for the District of Columbia Circuit in *Murray Energy v. EPA*.

Since 2017, the Panel finds that the EPA has made unwarranted changes to the CASAC and the NAAQS review process. At the least, these inappropriate changes should be mentioned in Chapter 1 in explaining the revised process used in this review, which differs so radically from that utilized in all prior reviews. Detailed recommendations to reverse the unwarranted changes are in the consensus responses.

## **Air Quality**

Depending on the location, either the annual or the daily standard may be controlling. New fine-spatial-scale modeling approaches (referred to in the draft PA as “hybrid” approaches) represent important and impressive scientific progress in the ability to quantify spatial variability in ambient concentrations. The performance of these approaches is sufficient to support their use in epidemiological studies and in risk assessment. In addition, the Panel recommends the development of Federal Reference Methods (FRMs) for measurement of Ultrafine Particles (UFP) and Black Carbon (BC), for which there is emerging evidence of health effects.

### **Primary Fine Particulate Matter Standards**

The evidence-based approach in the draft PA to reaching conclusions regarding the current and alternative primary PM<sub>2.5</sub> standards is a thoughtful and comprehensive synthesis of the epidemiological, controlled human exposure, and animal toxicological studies presented in the ISA, which strengthens the evidence since the last review. Given uncertainties, the risk assessment provides useful qualitative insights regarding risk and risk reduction. The Panel gives more weight to the evidence-based approach with the risk-based approach providing supporting information.

Limiting the evidence-based approach to assessment of associations and outcomes deemed as ‘causal’ or ‘likely causal’ is reasonable. The Panel recommends more extensive discussion and consideration of environmental justice with regard to disparities in health risk born by minority communities.

### Need for Both Annual and 24-hour Primary PM<sub>2.5</sub> Standards

The Panel concurs with the draft PA that there is compelling scientific evidence that the annual primary PM<sub>2.5</sub> standard is the ‘controlling’ standard in much of the U.S. and, if set at an appropriate level, can provide public health protection from both long- and short-term effects. However, the Panel finds, more strongly than is expressed in the draft PA, that the 24-hour standard is an important component of the suite of PM<sub>2.5</sub> standards. Specifically, the 24-hour standard, if set at an appropriate level, can provide needed public health protection not afforded by current or revised annual standards in locations for which the current or revised 24-hour standard is controlling.

### Current Fine Particulate Matter Primary Standards are Not Adequate to Protect Public Health

The weight of evidence framework for causality determination that is applied by EPA is an appropriate and well-vetted tool for drawing causal conclusions. The epidemiologic evidence, supported by evidence from controlled human studies and toxicological studies, supports the ‘causal’ and ‘likely to be causal’ determinations for combinations of exposure duration, indicator, and health outcome that are the focus of the draft PA for the evidence- and risk-based approaches. The epidemiologic evidence provides strong scientific support for recommendations regarding current and alternative standard levels. The existing strong and consistent epidemiological evidence was developed using accepted scientific methods, is peer-reviewed, and is coherent with peer-reviewed controlled human studies and toxicological



studies, which were also developed using accepted scientific methods. It would be irresponsible to dismiss any or all of the policy-relevant epidemiologic studies, as some on CASAC have suggested, merely because they have not been analyzed using emerging un-vetted advanced statistical methods that are still in their infancy for application to air pollution studies. The IPMRP notes that the epidemiologic evidence is extensive, particularly in terms of the large geographic domain and population sample size, and provides an overall consistent scientific basis for finding that the current primary PM<sub>2.5</sub> standards are not protective of public health. The epidemiologic evidence is scientifically valid and more than sufficient for informing recommendations regarding levels.

US multicity epidemiological studies, supported by consistent results from Canadian multicity epidemiologic studies, consistent results from accountability studies, and coherent results from animal toxicological and controlled human exposure studies, provide clear and compelling scientific evidence that the current PM<sub>2.5</sub> standards are not adequate to protect human health. The epidemiological evidence is based on different locations, study designs, and statistical approaches, which enhances its robustness. Of particular importance are the studies which continued to find health effects even when the air quality distribution was truncated to remove all days where annual PM<sub>2.5</sub> concentrations exceeded 12 µg/m<sup>3</sup> (the level of the current annual standard), and the pseudo-design value analyses which found health effects in areas likely to have design values of 12 µg/m<sup>3</sup> or less.

#### Retaining the Current Primary Standards is Not Scientifically Justifiable

Arguments offered in the draft PA for retaining the current standards are not scientifically justified and are specious. The revised PA should acknowledge the implausibility of these arguments or drop them altogether.

#### Revise the Annual Primary PM<sub>2.5</sub> Standard to a Level Between 10 µg/m<sup>3</sup> and 8 µg/m<sup>3</sup>

The Panel concurs with the draft PA that the current indicators, averaging times, and forms for the annual and 24-hour standards are suitable based on available scientific evidence, as detailed in Attachment B, and should be retained.

As detailed in Attachment B, based on the scientific evidence, the Panel finds that levels above 10 µg/m<sup>3</sup> for the annual standard are not protective of public health. An annual standard in the range of 10 µg/m<sup>3</sup> to 8 µg/m<sup>3</sup> would protect public health for the general public and for at-risk groups. However, even at the lower end of the range, risk is not reduced to zero. The margin of safety increases as the level of the standard is lowered within this range. The choice of margin of safety within this range is a policy judgment reserved for the Administrator. Based on the available scientific evidence, there is not a population threshold for annual concentration, within or below the recommended levels, at which the risk would drop to zero.

#### Revise the 24-hour Primary PM<sub>2.5</sub> Standard to a Level Between 30 µg/m<sup>3</sup> and 25 µg/m<sup>3</sup>

The Panel does not agree with the recommendation in the draft PA to leave the level of the 24-hour standard at 35 µg/m<sup>3</sup> if the annual standard is strengthened. Based on the scientific evidence, this would not provide an adequate level of public health protection in locations for which the 24-hour standard, and not the annual standard, would be controlling. Based on the scientific evidence and acknowledging that there is a continuum of adverse effects that decrease as the level of the standard decreases, the Panel recommends that the 24-hour standard be set between 30 µg/m<sup>3</sup> and 25 µg/m<sup>3</sup>. Lower levels within this range would provide an additional margin of safety. The choice of margin of safety within this range is a policy judgment reserved for the Administrator. Based on the available scientific evidence, there is not a population threshold for 24-hour exposure, within or below the recommended levels, at which the risk would drop to zero.

### **Primary Coarse Particulate Matter Standard: Maintain or Strengthen Level of Protection**

Although new evidence is available since the last review for a broader range of health outcomes associated with short- and long-term exposures to thoracic coarse particulate matter (PM<sub>10-2.5</sub>), this evidence is subject to considerable uncertainty. PM<sub>10-2.5</sub> can penetrate to the airways past the vocal cords, which should be acknowledged and discussed in the draft PA. While the Panel concurs that PM<sub>10</sub> is an appropriate choice at this time for the indicator for PM<sub>10-2.5</sub>, the Panel strongly recommends movement away from PM<sub>10</sub> and toward PM<sub>10-2.5</sub> as the indicator in the next review cycle. The Panel concurs with the draft PA that it is scientifically reasonable to retain at least *the level of protection* afforded by the current PM<sub>10</sub> standard. A second draft of the Policy Assessment should assess revision of the coarse particle standard downward coupled with a downward revision of the 24-hour fine particle standard, to at least maintain the current level of protection against exposure to coarse particles, as well as other recommendations from CASAC in the last review cycle for a range of alternative standards that would offer more protection.

### **Current Welfare Standards are Not Adequate; 2<sup>nd</sup> Draft PA Should Analyze Alternatives**

The Panel concurs with the draft PA that it is appropriate to focus quantitative assessments of welfare effects on visibility effects. Important scientific information regarding visibility effects has been omitted, perhaps inadvertently, from the draft ISA and should be included. Based on the scientific evidence, the Panel finds that the current welfare standards are not requisite to protect the public welfare from known and anticipated adverse effects from reduced visibility. The level of the secondary annual standard, which is higher than the level of the primary annual standard, is not requisite to protect against welfare effects and should be revised to at least match the level of the revised annual primary PM<sub>2.5</sub> standard. The draft PA fails to give due consideration to scientifically-justifiable alternatives for the indicator, averaging time, form, and level of possible alternative visibility-based welfare standards, particularly for the 24-hour standard. The combinations of indicator, averaging time, level and form recommended by CASAC in the past two NAAQS reviews are all considerably more protective than the current NAAQS. A second draft of the PA should systematically address these issues while taking into account the implications of revisions to the 24-hour PM<sub>2.5</sub> standard recommended by the Panel, which would have co-benefits with respect to visibility effects. The Panel concurs that the evidentiary basis for climate and materials effects are not sufficient to support quantitative assessment.

### **Areas for Future Research**

The Panel has identified numerous recommended areas for research to reduce uncertainties in support of the next NAAQS review for particulate matter. These recommendations focus on areas including air quality measurement, air quality modeling, health studies, analysis methods, and others. Examples of key recommendations include, but are not limited to, development and deployment of FRMs for UFP and BC, quantification of daily and sub-daily exposures and associations with adverse health effects for various PM sizes and compositions, development and application of improved approaches for accounting for confounding and effect modification in multipollutant models, and characterization of exposures and adverse effects for new health endpoints.

### **Status of the Integrated Science Assessment**

Scientific issues in the draft ISA should have been resolved prior to development and review of the PA. A second external review draft of the ISA should be made available to CASAC and the public, reviewed, and finalized, prior to release of a second draft of the Policy Assessment. The second draft of the Policy Assessment should be reviewed by CASAC and the public only after the ISA has been finalized. A summary of previous IPMRP comments on the draft ISA is given

at the end of the responses to charge questions. The Panel is concerned about the footnote to Table 3-1 in the draft PA indicates that final causality determinations for some endpoints are pending consideration of advice from CASAC. CASAC has already admitted, explicitly, that it is not qualified to offer these judgments, because it lacks the breadth, depth, and diversity of expertise for review of the PM NAAQS. Therefore, the CASAC PM Review Panel should be reappointed to augment CASAC during this review cycle before CASAC is asked to offer advice that it is not qualified to give.

Sincerely,

/signed/

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Chartered CASAC: Member 2008-2012, Chair 2012-2015  
CASAC PM Review Panel: Member 2007-2010, 2015-2018  
CASAC Ozone Review Panel: Member 2009-2012, Chair, 2012-2014  
CASAC Sulfur Oxides Review Panel: Member 2008-2009, 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2008-2009, Chair 2013-2015,  
Member 2015-2017  
CASAC Lead Review Panel: Chair 2011-2013  
SO<sub>x</sub>/NO<sub>x</sub> Secondary Standard Review Panel: Member 2009-2011  
CASAC Carbon Monoxide Review Panel: Member 2008-2010

/signed/

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Chartered CASAC: Member 2015-2018  
CASAC PM Review Panel: Member 2015-2018  
CASAC Secondary NAAQS for Oxides of Nitrogen and Sulfur Review Panel: Member 2015-2017  
CASAC Sulfur Oxides Review Panel: Member 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2016-2017  
CASAC Ambient Air Monitoring and Methods Subcommittee: Member 2004-2010  
CASAC Ambient Air Monitoring and Measurements Subcommittee: Member 2011-2018

/signed/

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CASAC PM Review Panel: Member 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2013-2017/signed/

/signed/

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CASAC PM Review Panel: Member 2015-2017

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CASAC PM Review Panel: Member 2015-2018

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Michigan State University  
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Chartered CASAC: Member 2012-2018  
CASAC PM Review Panel: Member 2015-2018  
CASAC Ozone Review Panel: Member 2009-2014  
CASAC Sulfur Oxides Review Panel: Member 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2013-2017

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CASAC PM Review Panel: Member 2015-2018  
CASAC CO Review Panel: Member 2009-2010  
CASAC Oxides of Nitrogen Review Panel: Member 2013-2017

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School of Public Health  
Boston University  
Boston, MA  
CASAC PM Review Panel: Member 2015-2018

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University of California, Irvine  
CASAC PM Review Panel: Member 2007-2010, 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2013-2015  
CASAC Carbon Monoxide Review Panel: Member 2008-2010

/signed/

Rob McConnell MD  
Professor of Preventive Medicine  
University of Southern California  
Los Angeles, CA  
CASAC PM Review Panel: Member 2015-2018

/signed/

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Consultant (formerly Air Quality Planner/ Planning Chief, Air Quality and Climate Division,  
Department of Environmental Conservation, VT Agency of Natural Resources, 1978-2015).  
Chartered CASAC: Member 2002-2007  
CASAC PM Review Panels: Member 2001-2006, 2008-2012, 2015-2018  
CASAC Ozone Review Panel: 2005-2008, 2010  
CASAC Lead Review Panels: Member 2006-2008, 2008-2013  
CASAC SO<sub>x</sub>/NO<sub>x</sub> Secondary Review Panels: Member 2008-2011, 2015-present  
CASAC Ambient Air Monitoring and Methods Subcommittee: Member 2004-2010

/signed/

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Rollins School of Public Health of Emory University  
Atlanta, GA  
CASAC PM Review Panel: Member 2015-2018  
CASAC Oxides of Nitrogen Review Panel: Member 2013-2015

/signed/

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Seattle, WA

Chartered CASAC: Member 2015-2018

CASAC PM Review Panel: Member 2015-2018

CASAC Ozone Review Panel: Member 2005-2008, 2010, 2011

CASAC Sulfur Oxides Review Panel: Member 2007-2010, 2014-2018

CASAC Oxides of Nitrogen Review Panel: Member 2007-2010, 2013-2017, Chair 2016-2017

/signed/

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Professor and Chair, Environmental Sciences and Engineering

Gillings School of Global Public Health

University of North Carolina at Chapel Hill

CASAC PM Review Panel: Member 2015-2018

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Retired, Electric Power Research Institute

Palo Alto, CA

Member, Independent Particulate Matter Review Panel

Chartered CASAC: Member 2012-2017

CASAC Ozone Review Panel: Member 2009-2014

CASAC Sulfur Oxides Review Panel: Member 2013-2018

CASAC Oxides of Nitrogen Review Panel: Member 2008-2010, 2013-2017

CASAC PM Review Panel: Member 2008-2011, Member 2015-2018

cc: Louis Anthony (Tony) Cox, Jr., Ph.D., Chair  
EPA Clean Air Scientific Advisory Committee

Docket ID No. EPA-HQ-OAR-2015-0072

[www.regulations.gov](http://www.regulations.gov)



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IPMRP members were subject to a good faith ethics review by the former director of the EPA Science Advisory Board Staff Office. The IPRMP meeting was conducted according to the same

The October 10-11, 2019 and October 18, 2019 meetings of the IPMRP were sponsored by the Union of Concerned Scientists, a 501(c)3 nonprofit organization. UCS does not take policy positions on NAAQS criteria and standards, other than to advocate that independent science advice be followed.<sup>1</sup> UCS is funded by individual members and private foundations and accepts no money from corporations or government entities.<sup>2</sup> Panelists were compensated for travel to attend the October 10-11, 2019 meeting but did not accept honoraria or other compensation for either meeting. The viewpoints and opinions of members of the IPMRP, and of the consensus of the IPMRP, are their own and do not represent any position of UCS. The content of the meetings, this letter, and attachments were determined exclusively by the Panel, and reflect exclusively the Panel's deliberations.

Any mention of trade names or commercial products does not constitute a recommendation for use.

The IPMRP reports are posted at [ucsusa.org/pmpanel](https://ucsusa.org/pmpanel).

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<sup>1</sup> Goldman, G.T. 2015. Union of Concerned Scientists. Comment on EPA-HQ-OAR-2008-0699-2472: Proposed Rule: National Ambient Air Quality Standards for Ozone. <https://www.regulations.gov/document?D=EPA-HQ-OAR-2008-0699-2472>

<sup>2</sup> Union of Concerned Scientists. 2018. Internal Revenue Service Form 990. <https://www.ucsusa.org/sites/default/files/attach/2019/03/ucs-fy18-990.pdf>

## **Independent Particulate Matter Review Panel**

### **CHAIR**

**Dr. H. Christopher Frey,\*\*** Glenn E. Futrell Distinguished University Professor, Department of Civil, Construction and Environmental Engineering, College of Engineering, North Carolina State University, Raleigh, NC and Adjunct Professor, Division of Environment and Sustainability, Hong Kong University of Science and Technology

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**Dr. John L. Adgate**, Professor and Chair, Department of Environmental and Occupational Health, Colorado School of Public Health, Aurora, CO

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**Dr. Kevin Boyle**, Professor, Agricutlural and Applied Economics and Willis Blackwood Director, Program in Real Estate, Virginia Tech, Blacksburg, VA

**Dr. Judith Chow,\*** Nazir and Mary Ansari Chair in Entrepreneurialism and Science and Research Professor, Division of Atmospheric Sciences, Desert Research Institute, Reno, NV

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**Dr. Jack Harkema,\*** Distinguished University Professor, Department of Pathobiology and Diagnostic Investigation, College of Veterinary Medicine, Michigan State University, East Lansing, MI

**Dr. Joel Kaufman**, Professor, Department of Environmental Health & Occupational Health, University of Washington, Seattle, WA

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**Dr. Rob McConnell**, Professor, Department of Preventative Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA

**Mr. Richard L. Poirot**,\* Independent Consultant, Burlington, VT

**Dr. Elizabeth A. (Lianne) Sheppard**,\* Professor of Biostatistics and Environmental & Occupational Health Sciences, School of Public Health, University of Washington, Seattle, WA

**Dr. Jeremy Sarnat**, Associate Professor of Environmental Health, Rollins School of Public Health, Emory University, Atlanta, GA

**Dr. Barbara Turpin**, Professor, Environmental Sciences and Engineering, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC

**Dr. Ronald Wyzga**,\* Retired, Electric Power Research Institute, Palo Alto, CA

**Independent Particulate Matter Review Panel Staff**

Mr. Chris Zarba, filling the role of a Designated Officer.

\* Denotes a former member of the chartered U.S. EPA Clean Air Scientific Advisory Committee

\*\* Denotes a former chair of the chartered U.S. EPA Clean Air Scientific Advisory Committee

## Attachment B

### Consensus Responses to Charge Questions on the EPA's Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – September 2019)

***EPA-1. Chapter 1 – Introduction: To what extent does the CASAC find that the information in Chapter 1 is clearly presented and that it provides useful context for the review?***

The Independent Particulate Matter Review Panel (IPMRP, or “the Panel”) finds that the staff in the U.S. Environmental Protection Agency’s (EPA’s) Office of Air Quality Planning and Standards have undertaken a good faith effort to produce a draft of the Policy Assessment (PA) under extenuating, unprecedented, and inappropriate constraints, as detailed below. The Panel commends the staff for this effort.

Chapter 1 clearly and concisely describes the purpose (Section 1.1), legislative requirements (Section 1.2), and history of National Ambient Air Quality Standard (NAAQS) reviews (Section 1.3). Its coverage of the current NAAQS review (Section 1.4) is inadequate and incomplete because it fails to document recent process changes. As detailed below, the Chapter omits mention of recent policy changes, including decisions and changes that affect the functioning of the review process and the timeline of the review. These are important parts of the peer review and public input process for the draft PA and the documents that feed into it. Section 1.4 also does not outline the process described in the final Integrated Review Plan (IRP) for Particulate Matter<sup>3</sup> or indicate how the current process is deviating from the PM IRP. Of particular concern, the draft PA is being reviewed before the Integrated Science Assessment (ISA) has been finalized, thus creating a blending of scientific and policy considerations. This sequence of events is not logical or appropriate.

Chapter 1 should clearly explain the difference between the sequences of draft documents indicated in the IRP versus the actual sequence of draft documents in this review. For example, contrary to the IRP, there is not a separate Risk and Exposure Assessment (REA) document in this review. To be consistent with the final IRP for this review, the text should state that EPA intended to make available to the U.S. EPA Clean Air Scientific Advisory Committee (CASAC) and the public two drafts of the REA. Furthermore, the IRP included a plan for two drafts of the ISA and two drafts of the PA. Although the scope of two drafts each of the ISA, REA, and PA were approved by CASAC in its 2016 review of the draft IRP,<sup>4</sup> the final IRP differed from the draft IRP<sup>5</sup> with regard to sequencing, as discussed further below. Thus, CASAC did not approve the sequence given in the final IRP.

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<sup>3</sup> EPA, “Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter,” EPA-452/R-16-005, U.S. Environmental Protection Agency, Research Triangle Park, NC, December 2016.  
<https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf>

<sup>4</sup> Diez Roux, A., “CASAC Review of the EPA’s Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – April 2016),” EPA-CASAC-16-003, Letter to Gina McCarthy, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, August 31, 2016.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/\\$File/EPA-CASAC+2016-003+unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/$File/EPA-CASAC+2016-003+unsigned.pdf)

<sup>5</sup> EPA, Draft Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter, EPA-452/D-16-001, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711.  
<https://www3.epa.gov/ttn/naaqs/standards/pm/data/201604-draft-integrated-review-plan-casac-review.pdf>

The first draft of the PA should not be released until the ISA has been finalized. CASAC, the IPMRP, and the public have recommended that there be a second draft of the ISA, which has been denied by the Administrator. Given that the ISA will go from first draft to final, but as of now has not been finalized, it is unclear what changes are pending for the final ISA and whether or how they will affect the content of the final PA. This is an unacceptable process deficiency that commingles policy considerations prior to finalization of the science assessment. This ‘puts the cart before the horse.’

Chapter 1 also fails to document the *ad hoc* changes to the NAAQS review process and to the CASAC that have been made since the final IRP was published in 2016. Compared to the final IRP, the following steps have been omitted in the current review: (a) no REA planning document(s); (b) no second external review draft of the ISA; (c) no external review drafts of the REAs; (c) no provision for a second draft of the PA; (d) no final REA as a separate document; and (e) no final ISA until after CASAC has completed its review of the draft PA. Although the IRP is cited on page 1-1, line 7, the deviations of the current review from the IRP are completely omitted. Both the omissions of the descriptions of these deviations, and the deviations themselves, are inappropriate and should be corrected. The chapter should enumerate all of the changes to the NAAQS review process, the CASAC, and the PM NAAQS review since 2016.

The final IRP scheduled that this review would end in 2022. Although the May 9, 2018 memo by then Administrator Pruitt<sup>6</sup> set a new end date of 2020, this is not consistent with the final IRP and there was no reference to the final IRP. While the five-year review schedule is a matter of law, it is also a matter of law that these must be science-based reviews. There are many factors in the review schedule that are in the control of EPA and not in the control of CASAC. The science review should not be sacrificed for the sake of expediency to play catch-up with the schedule. Deadlines do not excuse substantive deficiencies.

The following sections set forth detailed discussion reflecting the Panel’s profound concern with the process issues, and the Panel’s concern about science issues not being settled before the PA is drafted. The Panel makes consensus recommendations to reverse the numerous *ad hoc* changes to the CASAC and the NAAQS review process, that the draft PA be revised; that the second draft of the PA be reviewed by CASAC and the public after the ISA is finalized; that Chapter 1 document all deviations from the process outlined in the IRP; and that Chapter 1 cite and discuss the implications of the August 23, 2019 decision of the United States Court of Appeals for the District of Columbia Circuit in *Murray Energy v. EPA*.<sup>7</sup> Below are the Panel’s specific recommendations.

## **Process Issues**

Since 2017, numerous changes have been made to the scientific review process for the NAAQS, including changes that affect the membership and composition of the

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<sup>6</sup> Pruitt, S.E., “Back to Basics Process for Reviewing National Ambient Air Quality Standards,” Memorandum, U.S. Environmental Protection Agency, Washington, DC, May 9, 2018. <https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>

<sup>7</sup> *Murray Energy Corp. v. Environmental Protection Agency*, Respondent, and American Lung Association et al., Intervenor, No. 15-1385, United States Court of Appeals for the District of Columbia Circuit, Decided August 23, 2019. <https://cases.justia.com/federal/appellate-courts/cadc/15-1385/15-1385-2019-08-23.pdf?ts=1566572432>

CASAC.<sup>4,8,9,10,11,12</sup> These changes have been made without advance notice to, or input from, the CASAC, EPA staff, or the public. The changes include: (a) imposing non-scientific criteria for appointing CASAC members related to geographic diversity and affiliation with governments; (b) replacing the entire membership of the chartered CASAC in a period of one year; (c) banning nongovernmental recipients of EPA scientific research grants while allowing persons affiliated with regulated industries to be members of CASAC; (d) ignoring statutory requirements for the need for a thorough and accurate scientific review of the NAAQS in setting a review schedule; (e) reducing the number of drafts of a document for CASAC review irrespective of whether substantial revision of scientific content is needed; (f) commingling science and policy issues; (g) depriving CASAC of the needed breadth, depth, and diversity of scientific expertise for the PM NAAQS review by disbanding the CASAC PM Review Panel; (h) depriving CASAC of the needed breadth, depth, and diversity of scientific expertise for the ozone NAAQS review by refusing to form a CASAC Ozone Review Panel; and (i) creation of an ad hoc “pool” of consultants that fails to address the deficiencies created by disbanding the CASAC PM Review Panel and not forming a CASAC Ozone Review Panel. Each one of these changes harms the quality, credibility, and integrity of the NAAQS review for both PM and ozone.

The IPMRP recommends that EPA appoint members to CASAC and its review panels based on the need for breadth, depth, and diversity of scientific expertise, not geographic diversity and government affiliation, other than to meet the minimum requirement for the latter as required by the Clean Air Act. EPA should allow leading nongovernmental researchers who hold EPA scientific research grants to serve on CASAC and its augmented panels, consistent with existing Federal peer review guidance. EPA should appoint CASAC members to staggered overlapping terms to promote institutional memory and continuity. EPA should allow adequate time for scientific review by CASAC, including opportunities for public input. EPA should not combine assessment documents in a review unless this is consistent with a final Integrated Review Plan that has been agreed to by CASAC. EPA should develop NAAQS review schedules that allow for the likelihood that complex scientific and policy documents, such as an Integrated Science Assessment, a Risk and Exposure Assessment, and a Policy Assessment, may need substantial revision and re-review. EPA should better manage the timing of key milestones in the NAAQS review process so as not to selectively take time away from CASAC as a means to compensate for delays created by EPA elsewhere in the review. EPA should not be producing a Policy Assessment in advance of first finally determining what the science being assessed is – i.e. prior to finalizing the ISA. To do otherwise puts the cart before the horse. EPA should not introduce policy considerations until the scientific issues have been adequately settled. EPA

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<sup>8</sup> Pruitt, E.S., “Strengthening and Improving Membership on EPA Federal Advisory Committees,” Memorandum, U.S. Environmental Protection Agency, October 31, 2017. <https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>

<sup>9</sup> EPA, “Acting Administrator Wheeler Announces Science Advisors for Key Clean Air Act Committee Tasks Chartered Panel to Lead Review of Ozone & Particulate Matter Standards Under Reformed Process,” News Release, U.S. Environmental Protection Agency, Washington, DC, October 10, 2018, <https://www.epa.gov/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee>

<sup>10</sup> EPA, “Administrator Wheeler Announces New CASAC Member, Pool of NAAQS Subject Matter Experts,” News Release, U.S. Environmental Protection Agency, Washington, DC, September 13, 2019. <https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>

<sup>11</sup> GAO, EPA Advisory Committees: Improvements Needed for the Member Appointment Process, GAO-19-280, General Accountability Office, Washington, DC. <https://www.gao.gov/assets/710/700171.pdf>

<sup>12</sup> EPA, “Request for Nominations of Consultants To Support the Clean Air Scientific Advisory Committee (CASAC) for the Particulate Matter and Ozone Reviews,” *Federal Register*, 84(152):38625 (August 7, 2019). <https://www.govinfo.gov/content/pkg/FR-2019-08-07/pdf/2019-16913.pdf>

should continue to follow the successful practice, proven for four decades, of augmenting CASAC with the expertise it needs via review panels that deliberate interactively with members of the chartered CASAC. EPA should not make *ad hoc* changes to the NAAQS review process in the middle of a review. The changes since 2017 lead to a situation in which standards will not reflect air quality criteria — an “accurat[e] reflect[i]on [of] the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the present of [the] pollutant in the ambient air” (CAA section 108 (a)(2)) — since the CASAC and the process under which it is operating is incapable of properly assessing what that science is. If EPA wishes to make changes to the NAAQS review process, EPA should do so in a systematic manner similar to that employed in 2006, when EPA staff, CASAC, and others had an opportunity to provide input.<sup>13</sup>

Per its own statement in its letter of April 11, 2019, the current CASAC (or any CASAC, with only seven members, that is not augmented with a panel of experts) does not have adequate breadth, depth, and diversity of scientific expertise and experience needed to conduct thorough reviews based on the latest scientific knowledge of the kind and extent of scientific issues that pertain to the Particulate Matter NAAQS.<sup>14</sup> Thus, CASAC should be properly augmented, consistent with its charter with the U.S. Congress,<sup>15</sup> by reinstatement of the disbanded CASAC Particulate Matter Review Panel for the PM NAAQS Review.<sup>16</sup> Likewise, CASAC should be augmented with a properly constituted CASAC Ozone Review Panel.<sup>17</sup> Please see individual comments of Dr. H. Christopher Frey for more details on these points.

## **Scientific Issues Need to be Settled Before Formulating the Policy Assessment**

The lack of a second draft of the ISA is highly problematic, particularly because the draft Policy Assessment is based on scientific evidence from the ISA. In prior NAAQS reviews, it has been typical practice that CASAC reviews a second and sometimes third draft (as in the cases of the most recent lead and ozone reviews) of the ISA. It has been typical practice that CASAC has had the opportunity to review a draft Policy Assessment after it has completed reviews of draft ISAs. This sequence was by design. A key principle of the 2006 revisions to the NAAQS review

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<sup>13</sup> Peacock, M., “Process for Reviewing National Ambient Air Quality Standards,” Memorandum to George Gray and Bill Wehrum, U.S. Environmental Protection Agency, Washington, DC, December 7, 2006

<sup>14</sup> Cox, L.A. (2019), “CASAC Review of the EPA’s Integrated Science Assessment for Particulate Matter (External Review Draft – October 2018),” EPA-CASAC-19-002, Letter to A. Wheeler, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, April 11, 2019. [https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002+.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002+.pdf)

<sup>15</sup> United States Environmental Protection Agency Charter, Clean Air Scientific Advisory Committee, Filed with Congress, June 5, 2019, [https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/\\$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf)

<sup>16</sup> Yeow, A., Formation of the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel, Memorandum to C. Zarba, Science Advisory Board Staff Office, U.S. Environmental Protection Agency, Washington, DC, November 17, 2015, [https://yosemite.epa.gov/sab/sabproduct.nsf/0/EB862B233FBD0CDE85257DDA004FCB8C/\\$File/Determination%20memo-CASAC%20PM.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0/EB862B233FBD0CDE85257DDA004FCB8C/$File/Determination%20memo-CASAC%20PM.pdf)

<sup>17</sup> EPA, “Request for Nominations of Experts for the Clean Air Scientific Advisory Committee (CASAC) Ozone Review Panel,” *Federal Register*, 83(145): 35635- 35636 (July 27, 2018). <https://www.govinfo.gov/content/pkg/FR-2018-07-27/pdf/2018-16116.pdf>

process, which were modified in part in 2007 and 2009,<sup>18,19,20</sup> is that the scientific foundation of the review must be established before addressing policy issues. Failure to do this risks commingling policy issues prematurely before the science issues are adequately vetted and settled, which in turn creates the potential for policy choices to be made irrespective of the science. Thus, the integrity of the process is harmed when policy issues are addressed before the science issues are adequately settled. The IPMRP recommends that the ISA be finalized before a second version of the PA is provided and reviewed.

## **Chapter 1 Should Enumerate All of the Deviations from the Final Integrated Review Plan**

As detailed above, key steps have been omitted in the current review with respect to all key documents that provide the foundation for formulation of scientific advice. These omissions are inappropriate and have introduced deficiencies that undermine the scientific record regarding air pollutant criteria and upon which CASAC and the public may develop their advice to EPA. Chapter 1 should enumerate these changes and their impacts. See also detailed comments regarding process issues in the individual comments of Dr. H. Christopher Frey.

The schedule in the final IRP specified two drafts of each of the ISA, REA, and PA. However, the final IRP indicated that the drafts of the REA and PA would be concurrent. This differs from the schedule in the external review draft of the IRP that was reviewed by CASAC earlier in 2016. In the external review draft of the IRP, EPA had proposed to sequence the release of first drafts of the ISA, REAs, and PA such that CASAC would review them sequentially on a staggered schedule. Thus, under the initial proposed schedule, CASAC would have been able to provide its advice on the first draft of the REAs before receiving the first draft of the PA. The schedule in the draft IRP allowed for two drafts each of the ISA, REA, and PA.

The final IRP sequencing of the first drafts of the REA documents, such that they are released after receiving CASAC review of both the first draft of the ISA and of REA planning documents is appropriate. Since the REA builds upon information in the ISA, it is logical and appropriate that EPA consider CASAC's advice on the ISA before releasing a draft of the REA. Because the Policy Assessment is intended to integrate information from the ISA and the REA, it is generally not appropriate for a first draft of the PA to be released at the same time as the first draft of the REA. Simultaneous release of the first drafts of the REA and PA was done, for example, in the last review of the ozone NAAQS. As colleagues have pointed out (see November 26, 2018 letter to CASAC from former members of the 2009 to 2014 CASAC Ozone Review Panel),<sup>21</sup> the first

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<sup>18</sup> Peacock, M., "Process for Reviewing National Ambient Air Quality Standards," Memorandum to George Gray and Bill Wehrum, U.S. Environmental Protection Agency, Washington, DC, December 7, 2006

<sup>19</sup> Peacock, M., "Modifications to Process for Reviewing National Ambient Air Quality Standards," Memorandum, U.S. Environmental Protection Agency, Washington, DC, April 17, 2007

<sup>20</sup> Jackson, L., "Process for Reviewing National Ambient Air Quality Standards," Memorandum, U.S. Environmental Protection Agency, Washington, DC, May 21, 2009.

<https://www3.epa.gov/ttn/naaqs/pdfs/NAAQSReviewProcessMemo52109.pdf>

<sup>21</sup> Frey, H.C., J.M. Samet, A.V. Diez Roux, G. Allen, E.L. Avol, J. Brain, D.P. Chock, D.A. Grantz, J.R. Harkema, D.J. Jacob, D.M. Kenski, S.R. Kleeberger, F.J. Miller, H.S. Neufeld, A.G. Russell, H.H. Suh, J.S. Ultman, P.B. Woodbury, and R. Wyzga, "CASAC Advice on the EPA's Integrated Review Plan for the Ozone National Ambient Air Quality Standards (External Review Draft)," 24 page letter with 42 pages of attachments, submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA-HQ-OAR-2018-0279, November 26, 2018.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/0AC9E8672B0CA54985258351005BE54F/\\$File/Ozone+Letter+181126+Submitted-rev2.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0AC9E8672B0CA54985258351005BE54F/$File/Ozone+Letter+181126+Submitted-rev2.pdf)



draft of the PA in that review was very preliminary and required substantial revision. Transparency of the review process, and clear distinction of science and policy issues, is enhanced by obtaining CASAC's advice on the REA before submitting a first draft of the PA for CASAC review. However, in this review, there is no separate REA. The content of the REA has been incorporated into the draft PA. This is not appropriate since there are important scientific issues pertaining to the REA that should be reviewed and vetted prior to use in the PA.

The IPMRP recommends that Chapter 1 clearly explain the difference between the sequences of draft documents indicated in the IRP versus the actual sequence of draft documents in this review. Rather than multiple drafts of the ISA, REA, and PA, staggered so that science issues are vetted and settled before proceeding to policy issues, this review cycle has devolved into one draft of the ISA and one draft of the PA, with the drafts of the ISA and PA overlapping such that policy issues are inappropriately being addressed before the science issues are finalized.

## **Other Issues**

Given the importance of so-called wildfires as a source of ambient particulate matter, Chapter 1 could include more discussion of the rule regarding "Treatment of Data Influenced by Exceptional Events," (Federal Register, 81(191):68216-68282, October 3, 2016), particularly with respect to the role of events that are at least partly anthropogenic in origin and the case-by-case nature of the exception events rule. As noted elsewhere in this Panel's responses to charge questions, not all wildfires are purely natural in their ignition or extent. Whether and, if so, how wildfires might be appropriately considered is pertinent to the quantification of adverse health and welfare effects of such events, which in recent years are growing in frequency and magnitude, especially in some parts of the country. This topic might be appropriate for inclusion in Chapter 2 rather than Chapter 1.

***EPA-2. Chapter 2 – PM Air Quality: To what extent does the CASAC find that the information in Chapter 2 is clearly presented and that it provides useful context for the review?***

***SCQ-2.1 What are the Panel's views regarding whether the draft PA accurately reflects and communicates the air quality related information most relevant to its subsequent evidence-based assessment of the health and welfare effects studies, including uncertainties, as well as the development of the risk assessment for current and alternative standards? In particular, do the following sections accurately reflect and communicate current scientific understanding, including uncertainties, for: (a) relationships between annual and daily distributions of PM; (b) the review of hybrid modelling approaches used to estimate exposure in some studies and the risk assessment; and (c) information on background levels of various PM indicators?***

### **Relationships Between Annual and Daily Distributions of PM<sub>2.5</sub>**

Figure 2-11, page 2-26 shows several locations in the northwest U.S. and California that are below the annual primary PM<sub>2.5</sub> standard level of 12 µg/m<sup>3</sup> but above the 24-hour primary PM<sub>2.5</sub> standard level of 35 µg/m<sup>3</sup>. An extreme example of this is the Fairbanks (North Pole) valley site with a 2016-2018 24-hour-to-annual PM<sub>2.5</sub> design value ratio of 5.1 compared to the 2.9 ratio of 24-hour to annual primary PM<sub>2.5</sub> levels. The PA notes that, in the Northwestern US, daily and sub-daily (e.g., 2-hr average) concentrations (and the relationship between annual and daily concentrations) are heavily influenced by wildfire emissions in the summer/fall and stagnation in

the winter. Not reflected adequately here are the impacts of controllable emissions, including seasonal or episodic emissions on ambient concentrations. The text implies that these high concentrations are beyond our control. The episodic but substantial contribution of residential wood combustion for home heating is one of these anthropogenic sources. Currently, the inaccurate impression that is created regarding 24-hour and sub-daily concentrations is used to discount and exclude ambient measurements in the Northwest and California from the risk assessment and the consideration of whether the annual standard can adequately control for health effects associated with short-term exposures as discussed in Chapter 3.

The PA does not acknowledge that anthropogenic activities impact climate, which contributes to drought, and increased frequency and magnitude of fire, in the western U.S. (Abatzoglou and Williams, 2016; Barbero et al., 2015; Dennison et al., 2014; Littell et al., 2009; Miller and Safford, 2012; O'Dell et al., 2019). Based on 1.5 million government-recorded wildfires from 1992 to 2012, Balch et al. (2017) estimated that 84% of wildfires were human-caused, accounting for 44% of the total area burned. This study excluded prescribed burns for forest management that would add to the total of manmade fires.

The current 24-hour primary PM<sub>2.5</sub> standard, being based on a midnight-to-midnight 24-hour calendar day average, artificially divides a single overnight air quality event for smoke emitted from residential wood combustion into two separate days. As more monitoring sites transition to continuous PM<sub>2.5</sub> measurements that meet Federal Equivalent Method (FEM) performance requirements, the monitoring network will have the capability to support other averaging times for epidemiologic research and possible alternative forms of standards. For example, some exposure scenarios are less than 24-hours in duration, such as overnight peaks in ambient concentrations from residential wood smoke in some locations.

### **Hybrid Modeling Approaches**

In the context of this review of health-based standards, the air quality section on hybrid modeling approaches to PM<sub>2.5</sub> is important, since this is the area where substantial improvements in characterizing ambient PM<sub>2.5</sub> concentrations (exposures) over large areas have been made since the last PM NAAQS review. These methods clearly lead to improved ambient concentration estimates in locations without samplers. Impressively, some of the more sophisticated methods have n-fold cross validation coefficient of determination ( $R^2$ ) better than 80% and root-mean-square error (RMSE) of 2-3  $\mu\text{g}/\text{m}^3$  for daily PM<sub>2.5</sub> predictions. Approaches that account for high spatial resolution land-use features are better at capturing concentration gradients close to sources than are downscaling approaches based on 12 km by 12 km gridded air quality modeling predictions. The consistency of the regional concentration estimates across methods is remarkably good (Table 2-3).

The PA should explain why some methods work better than others. Larger spatial gradients, especially in the western U.S., are not well characterized by the 12-km downscaler models. The neural network (machine learning) 1-km model does better: Figure 2-28 (page 2-47) clearly shows the difference in resolution between the downscaler 12-km and neural network 1-km models. The Bayesian downscaler does not incorporate information about locations of primary PM<sub>2.5</sub> sources (i.e., surrogates such as land use variables), whereas several other methods, including the neural network, do. All these methods are designed to predict broad spatial PM<sub>2.5</sub> features, but the neural network and other methods including land use variables do a better job of capturing spatial gradients near sources. Ideally, the concentrations predicted across the US from the *best* performing methods should be used to conduct risk assessment for the entire country, rather than conducting the risk assessment for only a modest number of sites. The Bayesian downscaler is the worst of these methods (especially for the Northwest and

California), and yet it was the one selected for further analysis. The selection of the Bayesian downscaler likely leads to an underestimation of exposure and risk in the Northwest and California, assuming that populations are spatially collocated with sources. Although the Bayesian downscaler appears to have worse performance compared to the other methods, it is capable of providing reasonable estimates of spatially averaged concentrations even though it is not capable of capturing higher resolution variations. Thus, although it may not be the best choice for use in risk assessment, it is capable of supporting risk assessment at the urban scale as is done for 47 urban areas of the country in the risk assessment. See also the Panel's response to Supplemental Charge Question 3.4(c) for comments about the important features of exposure models for risk assessment versus epidemiologic inference.

Importantly, the text (e.g., p 2-41) is wrong as to the reason that there is less agreement between among these methods in the West. The reason is not because concentrations are low in the West; rather, it is because spatial concentration gradients are substantially greater in the West than in the East, where  $PM_{2.5}$  is more influenced by large secondary particle formation and more therefore regionally homogeneous. Models that are based on higher spatial resolution, and that account for localized spatial features, such as the machine learning-based method, are better at representing such gradients.

### **Background $PM_{2.5}$**

Background  $PM_{2.5}$  is low (10-20%) relative to the current annual NAAQS. However, the estimates of background  $PM_{2.5}$  concentrations provided in the draft PA are too high, because  $PM_{2.5}$  concentrations attributed as background are influenced, in part, by anthropogenic activity.

Wildfire, secondary organic aerosol (SOA), and dust are the major contributors to background  $PM_{2.5}$  concentration. However, some wildfire events are influenced by human activity. Hotter, drier western summers (driven in part by climate change) have resulted in increased major wildfire events in the western US and Canada over the last few years (see climate and wildfire references cited earlier on page B-7). Figure 2-2 of the draft PA shows estimated 2014 National Emission Inventory (NEI)  $PM_{2.5}$  emissions that include 32% from fires (mostly wild) and 18% from dust; these are surprisingly high. Page 2-50 (last line) says wildfire smoke is 10% to 20% of primary  $PM_{2.5}$  emissions; this difference compared to Figure 2-2 needs to be explained.

Background was estimated by assuming all biogenic SOA is natural, which provides an unacknowledged upper bound. Even though it is made from biogenic hydrocarbons, biogenic SOA is not necessarily purely natural, which should be acknowledged and discussed. There is substantial evidence that anthropogenic emissions impact the formation of SOA from biogenic VOCs. This was raised in comments from Dr. Turpin on the first draft of the ISA. A leading oxidation pathway of many biogenic VOCs is with ozone, which is clearly enhanced by anthropogenic emissions. Another important example is isoprene. Oxidation of isoprene leads to several gas phase products. A major SOA precursor is isoprene epoxydiol (IEPOX), which forms SOA when it reacts with wet acidic sulfate (anthropogenic). Thus, IEPOX SOA is formed as a result of reactions with anthropogenic emissions and, therefore, is controllable. Field studies measuring tracers of IEPOX SOA suggest that it is a major source of aerosol (roughly one-third of organic  $PM_{2.5}$ ) in the southeastern US in both rural and urban locations (Budisulistiorini et al., and in the draft ISA). As another example, model predictions by Carlton et al., suggest that more than 50% of biogenic SOA in the eastern U.S. could be controlled by reducing anthropogenic  $NO_x$  emissions. The draft PA should include a brief discussion regarding the challenges in attributing the share of natural origin of ambient particles and implications for determination of background ambient  $PM_{2.5}$  concentrations.

The thoracic size fraction of "dust" (coarse PM, the size range between 2.5 and 10  $\mu\text{m}$ ) is regulated as a component of  $\text{PM}_{10}$ . These are primary emissions from non-combustion sources, mostly from agricultural, construction, and road sources. These sources can also contribute smaller particles in the  $\text{PM}_{2.5}$  size range. A drier climate in parts of the U.S. could contribute to an increase in PM from these sources (Reich et al., 2018, Tong et al., 2017), so it may not be appropriate to consider all coarse PM as natural background. This is not discussed in the first draft of the PA.

## **Additional Comments on Chapter 2**

### **Issues with Federal Reference Method and Federal Equivalen Method $\text{PM}_{2.5}$ Monitor Comparisons.**

Monitoring agencies continue to struggle with getting their continuous FEM  $\text{PM}_{2.5}$  monitor performance within acceptable levels for them to be used to demonstrate compliance with the  $\text{PM}_{2.5}$  NAAQS. Of the ~900 FEMs in use, data from 40% of them cannot be used as "official" FEM measurements due to performance issues. This problem is caused by how filter-based Federal Reference Method (FRM) instruments are run as a benchmark for testing FEM performance compared to how FRMs are run in routine state and local monitoring networks. For FEM testing, FRM filters are removed and chilled immediately at the end of the 24-hour sampling period. For routine monitoring, FRM filters remain in the sampler at or somewhat above ambient temperatures for up to 6 days. FRM filters can lose up to 10% of their non-water mass over 24-96 hours if not removed from the sampler and chilled immediately. Therefore, in field comparisons of co-located FEM and FRM monitors, FEM measurements typically appear to be biased high compared to the FRM, when in reality this is an artifact of field sample handling for the FRM and not an actual limitation of the FEM. However, as a result of such comparisons, the FEM is often found (erroneously) to be deficient with respect to performance requirements for NAAQS compliance purposes. While changes could be made to either the way FEMs are tested or how FRMs are run in the field, neither of these approaches are currently practical in a regulatory context. There are approaches that could be implemented to make nearly all the existing FEM data of acceptable quality for comparison to the NAAQS based on data collected from co-located FRM and FEM  $\text{PM}_{2.5}$  monitors over the last several years, since nearly all FEMs produce 24-hour average  $\text{PM}_{2.5}$  concentrations that are well-correlated with FRM samples.

### **Federal Reference Methods Needed for Ultrafine Particles and Black Carbon**

The Panel recommends the development of FRMs for measurement of ultrafine particles (UFP) and black carbon (BC). UFP is classified as "likely to be causal" for long-term nervous system effects, and there is a growing body of literature on the health effects of BC. UFP is measured at some of the near-road network sites, and BC is measured at most of them, as well as at National Air Toxics Trends Stations (NATTS) sites. Both are good indicators of traffic-related air pollution and have substantial gradients away from the road. There is also a need for comprehensive measurements of UFP and BC that go beyond near-roadway monitoring. Chapter 2 mentions the history of development of the FRM for coarse particles. Likewise, an FRM for UFP should be developed, for similar reasons. Thus, Chapter 2 should note that there is not an UFP FRM. Such a statement is important because a future research need is to obtain more ambient monitoring data over space and time for UFP to support epidemiology based on UFP; the same goes for BC. Given that EPA has in the past established FRMs in anticipation of possible new indicators, it is appropriate to provide a rationale for establishing FRMs for UFP and BC. The rationale for development of an FRM for  $\text{PM}_{10-2.5}$  is on page 2-18, at the top of the page. This is a good example of the similar rationale for develop of new FRMs for UFP and BC.

UFP needs to be more clearly defined as particle number concentration with a low-end 50% response size of less than 10 nm; the low-end response particle size is an important parameter for UFP measurements.

### **Leverage Near-Road Monitoring Network**

A useful summary of the increase in  $PM_{2.5}$  at near-road sites is given, showing an average increment over urban background of less than  $1 \mu g/m^3$  with short-term (morning rush-hour) peaks of  $3 \mu g/m^3$  to  $4 \mu g/m^3$ . Briefly noted in Section 2.2.5 are other particle measurements at some of the near-road network sites, including BC and UFP concentration measurements. Although BC is being measured at many near-road sites, it is not required to be reported to EPA's Air Quality System (AQS) under current regulations, and some agencies still do not report it. Over the last several years, a network of approximately 75 near-road monitoring sites has been deployed to determine compliance with the hourly  $NO_2$  NAAQS. There is a large body of literature showing cardiovascular health effects from traffic-related air pollution (TRAP), presumably driven by particles and not  $NO_2$  or CO (see for example Jhun et al., 2019, and George Allen's individual comments). The existing near-road site infrastructure could be leveraged by adding additional particle measurements at a subset of sites with the largest traffic influence to inform future PM NAAQS reviews. In addition to robust UFP and BC measurements, EPA should consider augmenting some of the existing monitoring sites to measure lung-deposited surface area (using charge-based continuous methods), PM-coarse, on-line (hourly) total aerosol carbon (and OC by difference with BC), and on-line (hourly) elemental measurements using XRF (brake wear can produce particles containing iron, copper, and other aerosol fumes). Similar measurements could be added to the nearest NCore site in the same urban area. This paired network design would provide information on the elevated exposures (gradients) to these pollutants in the near-road environment.

### **Emissions and Air Quality Trends**

The summary of emission categories averaged nationally was of limited usefulness. It would be more useful to provide attributions of emissions to source categories for regions of the country that illustrate the variability among the sites included in the risk assessment. Figure 2-2 (page 2-5), emissions by source sector, is misleading; geographically stratified emissions would be preferred. There are differences in the quantified percentages given for emissions by source type between the draft ISA and draft PA; see Dr. Judith Chow's individual comments for more detail. These differences should be reconciled.

The national downward trend in  $PM_{2.5}$  ambient concentration over the last two decades, especially in the eastern US, has stopped and appears to have recently reversed (Figure 2-9, page 2-24). The draft PA should acknowledge and discuss this. For example, the recent change in the trend may be related to the end of substantial Electricity Generating Unit (EGU)  $SO_2$  emission reductions, which could be assessed in the second draft by evaluating evidence from speciation data. The draft PA notes recent increases in wildfire events, which could also be a factor in the recent change in the trend.

The discussion of UFP trends was weak and did not make use of available near-road UFP data in AQS. As noted above, establishing an FRM for UFP is a first step in expanding information needed for evaluating UFP trends and concentrations.

**EPA-3. Chapter 3 – Review of the Primary PM<sub>2.5</sub> Standards: What are the CASAC views on the approaches described in Chapter 3 to considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? What are the CASAC views regarding the rationales supporting the preliminary conclusions on the current and potential alternative primary PM<sub>2.5</sub> standards?**

**SCQ-3.1 Does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>2.5</sub> review? Are there additional policy-relevant questions that should be addressed?**

The questions posed in Chapter 3 appropriately reflect important policy-relevant issues for the PM<sub>2.5</sub> review.

**SCQ-3.2 What are the Panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?**

Together the evidence-based and the risk-based approaches show that the current PM<sub>2.5</sub> standard is not requisite to protect public health, with the evidence-based approach appropriately given more relative weight. Together these approaches, with more weight given to the evidence-based approach, provide a scientific evidentiary basis for recommending alternative levels for the annual and daily PM<sub>2.5</sub> standards. The Panel found that the PA evidence-based approach is a thoughtful and comprehensive synthesis of the observational (epidemiological) and experimental science (controlled human exposure and animal toxicological studies) presented in the ISA. The risk-based approach provides context for the scientific findings for current and alternative PM<sub>2.5</sub> standard levels in a large sample of the US population. The risk-based approach is limited in scope and would benefit from a clearer presentation of methods. The risk assessment is subject to uncertainty and is viewed as providing qualitative insight regarding magnitudes of, and relative differences in, risk. Nevertheless, the risk-based approach informs the scientific evaluation that risk would be reduced by alternative PM<sub>2.5</sub> standards. The Panel gives more weight to the evidence-based approach that documents the ambient levels at which adverse effects are observed, although no evidence was found for a discernable population threshold. Together, the complementary evidence-based and risk-based analyses, with more weight given to the evidence-based approach, provide strong support for drawing conclusions regarding current and alternative PM<sub>2.5</sub> standards.

**SCQ 3.3** *What are the Panel's views on the evidence-based approach, including:*

- a) The emphasis on health outcomes for which the draft ISA causality determinations are "causal" or "likely causal"?*
- b) The identification of potential at-risk populations?*
- c) Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?*
- d) Characterizing air quality in these key studies using two approaches: the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the "pseudo design value" reflecting a monitor with the highest levels in an area?*
- e) The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*
- f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?*
- g) Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?*

- a) The emphasis on health outcomes for which the draft ISA causality determinations are "causal" or "likely causal"?*

Limiting the evidence-based approach to assessment of associations and outcomes deemed as 'causal' or 'likely to be causal' is reasonable. However, specific attention should be given in future assessments to emerging evidence involving associations which, while less well-established, may provide more sensitive indicators of PM<sub>2.5</sub>-mediated risks. These include, for example, associations between various PM size fractions and corresponding neurological and metabolic effects.

- b) The identification of potential at-risk populations?*

The Panel felt that the more expansive identification of 'at-risk' populations employed in the draft PA is a positive change from the previous PA of the last review cycle. At-risk populations, as defined in the draft ISA and draft PA, include traditional definitions involving biological susceptibility, as well as those exposed to elevated PM due to social disparities. EPA staff deserves credit for thinking of risk in terms of sensitivity and vulnerability and for refining the approach to identification and assessment of at-risk populations in recent review cycles for other criteria pollutants and applying these concepts in the current PM review.

The Panel recommends more explicit discussion of environmental justice, including more depth regarding disparities in PM<sub>2.5</sub> risk borne disproportionately within African American and Hispanic communities. For example, the Di et al. (2017a) chronic mortality study presents a result of concern in this vein: the three times higher relative risk (hazard ratio) for African Americans compared to the general population.

- c) *Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?*

The Panel supports the decision to focus the evidence-based assessment on multicity epidemiologic studies. As stated in the draft PA, such studies examine potential associations over large geographic areas with diverse atmospheric conditions and population demographics. The Panel also supports and concurs with the choice in the draft PA to devote specific attention to recent studies conducted in cities with PM<sub>2.5</sub> levels well below current standards; these studies are compelling in showing excess risk at levels below the current standards. The Panel noted the strong concordance of findings among these observational studies, conducted throughout North America, in locations with varying exposure scenarios, using a range of exposure estimation and concentration-response modeling methods, which collectively provide strong evidence-based support for assessment of the adequacy of the current PM standards. Findings from toxicological, controlled human exposure, and accountability studies are coherent with these observational findings. Truncated distribution analysis, such those conducted by Di et al. (2017a&b), provides additional confidence of effects at levels below current standards.

The Panel notes that the draft PA focuses on U.S. and Canadian studies exclusively, and does not take into account that studies outside of North America (e.g., in Europe) could also be informative. The evidentiary basis from the U.S. and Canadian studies is sufficient to support findings regarding the adequacy (or lack thereof) of the current standards and alternative standards.

- d) *Characterizing air quality in these key studies using two approaches: the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the “pseudo design value” reflecting a monitor with the highest levels in an area?*

The Panel supports the approach described in the draft PA of focusing on the mean level of PM<sub>2.5</sub> in short- and long-term epidemiologic studies, especially for mean values at or below the level of the annual and 24-hour current standards. However, the Panel notes that there are scientifically valid and meaningful inferences to be made for other statistics of the PM<sub>2.5</sub> concentration distribution in epidemiological studies. While assessment of adverse effects at mean concentrations continues to be a suitable practice for quantifying threats to public health, the Panel notes that, as detailed in the attached individual comments by Dr. Douglas W. Dockery, statistical power is a function of exposure variance, not the mean. In this vein, the Panel finds that the evidence from epidemiologic studies over a continuum of observed concentrations, such as from the 25<sup>th</sup> to 75<sup>th</sup> percentiles, is also informative, and that evidence of adverse effects at levels below the mean observed concentrations provides information of value in assessing both the adequacy of the current standard and potential alternative levels.

The Panel finds that the pseudo design values (PDVs) are useful in providing a systematic basis for comparing individual studies (both single city and multicity) with the current and alternative standards. The PDVs essentially convert exposure metrics used in the observational studies (i.e., mean annual ambient concentrations) into values that are interpretable from a regulatory standpoint. Despite this, several Panel members felt that the PDVs were presented in a confusing manner in the draft PA, limiting their interpretability. Perhaps some of the detailed explanation in Appendix B of the draft PA could be included in the body of Chapter 3. Suggestions raised by Panel members for improving the PDV discussion include: adding a PDV column in Figure 3-3, which presents results from the multicity epidemiologic studies; remove the material within the PA describing the PDVs as reflecting health response; and provide comparisons between PDVs and conventional DVs.



EPA should also provide 98<sup>th</sup> percentile of PDVs for short-term (24-hour) studies to aid in the use of such studies to assess effects for the 24-hour standard at current and alternative levels. However, as noted in the draft PA, the PDVs are up to 10% higher than an actual design value, which should be taken into account when using the studies to support inferences related to actual design values.

- e) *The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principal means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*

While there was considerable debate among the Panel members concerning sub-populations not adequately protected by the annual standard (e.g., populations in the northwestern and northeastern US who may be exposed to episodic ambient PM<sub>2.5</sub> peak concentrations from residential wood combustion), there was consensus that the annual standard is appropriate as the principal means of protecting public health from PM exposures. The Panel concurs with prioritizing the annual standard based on the rationale outlined in the PM ISA from the prior NAAQS PM review cycle and noted that risks associated with long-term PM exposures are typically an order-of-magnitude larger than those associated with short-term exposures. However, the Panel notes that the annual standard is not the ‘controlling’ standard in all parts of the U.S., meaning, addressing the annual standard will not necessarily be protective of health effects in all parts of the country due to short-term exposures. In some parts of the U.S., the annual levels can be lower than the standard even though there are levels at or over the 24-hour standard. Thus, for some parts of the U.S., the 24-hour standard is controlling, or would be controlling under revised standards. Therefore, both the annual and 24-hour standards are needed to provide public health protection for situations in which one or the other would be controlling.

- f) *The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?*

Scientific findings since the last PM NAAQS review based on epidemiological and controlled exposure studies, relating to both short- and long-term exposure to PM<sub>2.5</sub> and corresponding acute and chronic effects, provide a robust foundation for assessing the adequacy of the current PM<sub>2.5</sub> standards. U.S. multicity epidemiologic studies, supported by Canadian multicity epidemiologic studies, coherent results from animal toxicology and controlled human exposure studies, and accountability studies that provide additional causal evidence, provide clear and compelling scientific evidence that the current PM<sub>2.5</sub> standards are not adequate to protect human health. The Panel agrees with and supports the assessment in the draft PA highlighting the U.S. and Canadian epidemiologic studies, specifically, those conducted in locations where study period PM<sub>2.5</sub> concentrations (and their PDVs) were clearly below the current annual and 24-hour standards. Most notable are an American study (Di et al., 2017a) and three Canadian studies (see Weichenthal et al., 2016b, 2016c and Pinault et al., 2016) that provide evidence of adverse health effects from long-term exposures and health; and two studies that examined risk from short-term exposures (Di et al., 2017b; Shi et al., 2016).

For example, the Di et al. (2017a) and Shi et al. (2016) studies are characterized by very large sample sizes, and Shi et al. (2016) has mean concentrations near 8 µg/m<sup>3</sup>. Even when data were truncated in Shi et al. (2016) such that air quality only under 12 µg/m<sup>3</sup> was considered, the effects were consistent. The Shi et al. (2016) study includes hybrid model-predicted concentrations that average just above 8 µg/m<sup>3</sup> and are well below 7 µg/m<sup>3</sup> at the 25<sup>th</sup> percentile of the distribution. The hybrid modeling approach is a substantial

advancement in exposure estimation that enables epidemiologic studies of large cohorts not served by the ambient monitoring network. Although the hybrid model air quality predictions are subject to some uncertainty, the performance of the hybrid models is quite good based on results described in Chapter 2 and serves as a valid basis for epidemiologic inference. The Canadian studies are informative in that they include notably low levels of exposure at which effects are observed, which provides consistency with the U.S.-based studies. These are groundbreaking studies that provide new results since the last review that are highly compelling.

Some discussion of PM<sub>2.5</sub> components other than based simply on particle diameter (i.e. ultrafine particles) is desirable. Such components typically include elemental carbon, organic carbon, nitrate, sulfate, mineral matter, and trace species, as well as black carbon. Although virtually all PM components have been shown to have some adverse health impacts, there is scientific evidence of some differences in toxicity among major components for both respiratory and cardiovascular endpoints. Although currently available scientific evidence is not sufficient to support development of standards related to differences among PM<sub>2.5</sub> components and variability in PM<sub>2.5</sub> composition, the limited available information about components is noteworthy and could help inform risk managers about the need to consider all major PM<sub>2.5</sub> components in achieving compliance.

- g) *Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?*

As noted above, the Panel finds that key uncertainties still exist concerning the adequacy of the existing standard, especially the daily standard, in protecting specific sub-populations, including those living in the areas affected by high PM<sub>2.5</sub> concentration episodes from residential wood combustion. The Panel recommends that additional analyses be conducted to assess the degree to which the current 24-hour standard is correlated with, or captures, sub-daily exposures occurring over a few hours.

Acute health effects associated with sub-daily exposures to PM<sub>2.5</sub> and UFP continue to be a key uncertainty in assessing PM health risk. While controlled human exposure and panel-based studies typically assess sub-daily exposures, endpoints used in these investigations are commonly sub-clinical, yielding important mechanistic rather than clinical insights. The Panel also agreed that uncertainties and limitations exist in using multi-pollutant models as a primary means of assessing confounding and robustness of PM health epidemiologic results, as is typically the case in the key studies noted above. However, such uncertainties and limitations were taken into account by the Panel in making expert scientific judgments that inferences from the studies were valid and robust, and in making scientific judgments based on these studies regarding the adequacy of existing and alternative standards. The scientific evidence supports robust inferences because of the consistency of epidemiological findings, and the coherence among multiple lines of scientific evidence from epidemiology, controlled human studies, and toxicology, and biological plausibility.

The draft PA should reframe the inference of policy-relevance of controlled human studies. While it is true that the controlled human studies in Table 3-2 for which effects were observed tend to have very short averaging time periods (e.g., sub-daily over a few hours), if the measured levels are averaged over 24 hours they are comparable to or below the level of the current 24-hour primary PM<sub>2.5</sub> standard. Thus, these studies represent 24-hour

concentrations that are policy relevant. Of course, there are challenges with interpretation of subclinical endpoints with respect to implications for clinical adverse effects. However, these studies provide indication of the potential importance of sub-daily exposures, including peak exposures. Therefore, the Panel recommends that the policy relevance of these studies receive more emphasis.

**SCQ 3.4 *What are the Panel's views on the quantitative risk assessment for PM<sub>2.5</sub> including:***

- a) *The choice of health outcomes and studies selected for developing concentration-response functions for long and short-term effects?***
- b) *The selection criteria for the 47 urban areas and PM<sub>2.5</sub> air quality scenarios analyzed?***
- c) *The hybrid modeling approach used for quantifying exposure surrogates across an area and adjusting air quality for alternative standard levels, as supplemented by interpolation/extrapolation?***
- d) *The characterization of variability and uncertainty in the risk assessment?***
- e) *The robustness and validity of the risk estimates?***

Overall the risk assessment has been thoughtfully and reasonably conducted given the compressed timeframe. However, as a procedural matter, and as noted earlier, it is a process deficiency and contrary to the final IRP that there was not a first draft of an REA to enable review of scientific issues in risk assessment prior to the use of risk assessment to support the PA. The risk assessment illustrates that there is more impact in terms of reduction in premature mortality from lowering the level of the annual standard, rather than the level of the 24-hour standard. However, there are nonetheless substantial risk reductions obtained by lowering the 24-hour standard, especially in locations for which the 24-hour, and not the annual, standard would be controlling. A second draft of the PA should include risk assessment analyses for combinations of alternative levels of the annual and 24-hour standards commensurate with the levels recommended by this Panel that are not already included: i.e., in the range of 10  $\mu\text{g}/\text{m}^3$  to 8  $\mu\text{g}/\text{m}^3$  for the annual standard combined with a range of 30  $\mu\text{g}/\text{m}^3$  to 25  $\mu\text{g}/\text{m}^3$  for the 24-hour standard. See also the Panel's discussion of SCQ-3.6.

The risk assessment indicates that there will be a large number of estimated premature deaths attributed to PM<sub>2.5</sub> for persons of age 30 or older for the 47 selected urban study areas based on simulation of air quality that just meets the current standard. The risk assessment accounts for approximately one-third of the U.S. population that is age 30 or older. Therefore, the risk estimates are based on a large population but underestimate the national total. Based on Table 3-5, the median estimated all-cause mortality from long-term exposure to PM<sub>2.5</sub>, based on 2015 air quality adjusted to just meet the existing standards, ranges from 13,500 based on Thurston et al. (2016) to 52,100 based on Pope et al. (2015). The estimated all-cause mortality from short-term exposure to PM<sub>2.5</sub> ranges from 1,200 based on Ito (2013) to 3,870 based on Zanobetti et al. (2014). The variability in these estimates account for two different air quality simulation approaches as well as different concentration-response functions from more than one study; most of the variability is due to the underlying study. While the specific estimates are uncertain, and should be interpreted qualitatively with regard to their magnitude, the draft PA risk assessment buttresses the conclusions based on the scientific evidence that at the levels of the current fine particle standards, the risk of premature mortality is unacceptably high.

The Panel has quite a few comments regarding the risk assessment, including: (a) the lack of clear rationale for the choice of health effect endpoints; (b) exclusion of some study areas that are of concern; (c) limitations of the Bayesian downscaler hybrid modeling approach and its

application; (d) the opportunity to improve the characterization of variability and uncertainty; and (e) robustness and validity of the risk assessment. Each of these are discussed in more detail below.

#### **a) Rationale for Health Endpoints**

The IPMRP agrees with the draft PA's focus on *health outcomes* that were judged in the ISA to be causal or likely causal. However, the risk assessment only focuses on three health outcomes (total mortality, ischemic heart disease mortality, and lung cancer mortality) and the rationale for this choice is not clearly articulated. Omitted are cardiovascular effects (long-term) other than IHD mortality, such as cerebrovascular (stroke); any short-term cardiovascular effects other than IHD mortality; respiratory effects at either long- or short-term time scales; cancer mortality other than lung cancer; and nervous system effects. Note that, by comparison, the Global Burden of Disease analyses have developed risk assessment estimates for mortality from All Causes, Ischemic Heart Disease (IHD), Cerebrovascular Events (Stroke), Lower Respiratory Infections (LRI), Chronic Obstructive Pulmonary Disease (COPD) and Lung Cancer. While the three selected endpoints are appropriate given their clear public health importance, the draft PA's characterization of risk is limited due to the focus on only a subset of endpoints. Nonetheless, the studies selected as the basis for quantification of exposure-response relationships in the risk assessment are large and well-designed; there is clear articulation of the criteria for selecting these studies and these are appropriate. Table C-1 is a succinct distillation of each of the selected studies with key information relevant to the risk assessment.

#### **b) Selection of Study Areas**

The individual *selection criteria* for the 47 urban areas are reasonable. They include PM<sub>2.5</sub> concentrations, availability of monitoring data, and geographic diversity. However, the manner in which these criteria are evaluated is not specifically and clearly explained. For example, the criterion related to "PM<sub>2.5</sub> air quality concentrations" is related to the need for adjustments of observed air quality to levels corresponding to current and alternative standards. The text does not clearly describe how the three criteria are assessed and or balanced in the process of decision-making regarding selection of study areas. Although the selected urban areas are reasonable, they do not adequately represent the range of geographic diversity that is needed, especially with respect to the 24-hour standard. For example, Figure 3-10 indicates that large parts of the central, northern, and western US were not included in the areas assessed. Fifty-six areas met the initial 10/30 (annual/24 hour) standard criteria for inclusion, but 9 (20%) were later excluded because of influence of wildfires (7 areas), high local conditions (Eugene, OR), and "uncertain" projections (Phoenix, AZ). The Panel is concerned that areas for which there are exposures to smoke from residential wood combustion are not represented. As noted earlier, the Panel is concerned that the draft PA is too easily dismissive of the fact that there have been a growing number of human-induced wildfires during the past two decades which have had evident adverse health and environmental effects. Based on 1.5 million government-recorded wildfires from 1992 to 2012, Balch et al. (2017) estimated that 84% of wildfires were human-caused, accounting for 44% of the total area burned. Nonetheless, the draft PA's approach is likely broad enough to provide a sufficient basis for making inferences regarding the potential for risk reduction from lowering standards given that nearly one-third of the U.S. population over the age of 30 is included and areas with large populations are included. The IPMRP suggests that EPA explore the feasibility of using the entire U.S. as an alternative to selecting only a subset represented by the 47 urban study areas, and expand the geographic scope of the risk assessment commensurate with data availability.

### c) Modeling Approach

The *hybrid modeling approach* relies on the Community Scale Air Quality (CMAQ) model predictions and a Bayesian downscaler method. The reductions in emissions needed to scale air quality to levels of current and alternative standards were specified based on adjustments in emissions from either primary PM<sub>2.5</sub> or secondary PM<sub>2.5</sub> precursors (specifically, SO<sub>2</sub> and NO<sub>x</sub>). Using two methods to estimate emissions allows better understanding of the sensitivity of the downscaling approach to the emissions estimates. Limitations include: (i) restricting the analysis to only one year, 2015, without adequate characterization of inter-annual variability; (ii) modeling at the 12-km grid level, which is relatively coarse with respect to spatial gradients found in some study areas; and (iii) the assumption of proportionate reductions scaled by fixed percentages.

While these decisions are justified and reasonable given the limited timeframe that EPA staff had to complete this risk assessment, a more complete analysis would evaluate the sensitivity to these assumptions. For example, the model could be run with data for multiple years to assess the robustness of the risk estimate to inter-annual variability. The risk modeling could be performed at a finer grid scale for at least a few representative choices among the study areas. Alternative assumptions regarding scaling and their impacts on spatial and temporal variability in predicted air quality and associated risk could be tested. Such analyses should be included in a second draft of the PA.

Nonetheless, the hybrid modeling approach is a practical and acceptable way of estimating effects that would occur over a range of current and alternative standards. The hybrid approach is a more realistic improvement over the rollback approaches employed in the previous NAAQS review cycle.

In a second draft of the PA the Panel would like to see more information to better understand the spatial scales, species specifics, and proportionate emissions reductions that ended up being used to meet the various PM concentration thresholds in the different urban areas. This information could be included in Appendix C, either as a tabular summary or for a few illustrative typical examples for cities in different regions of the U.S. These would show the spatial scales and absolute reductions (or increases) required of specific primary and secondary emissions species associated with the different PM thresholds evaluated.

The description and explanation of the 2015 downscaler is fairly cursory (Section C.1.4.5). While it is possible that this is justified given EPA's previous work (cited as U.S. EPA, 2018c), more details are warranted so that the PA can be a stand-alone self-explanatory document.

The risk assessment results are potentially very sensitive to the choice of the downscaler vs. one of the other "hybrid" models described in Chapter 2. For risk assessment, it is important that the model predict the same mean and capture the full variation of the distribution represented by the underlying concentration distribution in the area under consideration. While ground truth can only be approximated due to inherently limited monitoring data, it would be helpful to see a more direct assessment of the performance of the model for risk assessment purposes.

The linear interpolation approach to assessing additional standards represents a reasonable compromise to reduce EPA staff workload given the compressed timeframe for producing the Policy Assessment. However, the scientific quality of the work is compromised when not enough time is allowed. The IPMRP suggests modeling at least one more level in order to understand better whether the linear assumption is reasonable. (For further details on the above points, see Dr. Sheppard's individual comments.)

#### **d) Variability and Uncertainty**

The *characterization of variability and uncertainty* is generally appropriate, given the analyses that have been conducted, and is reasonably summarized in Section 3.3.2.4 with more detail provided in Appendix Section C.3. The draft PA appropriately references and utilizes the WHO multi-tiered approach to assessing uncertainty. By endpoint, the risk estimate results indicate that the most important factor influencing the estimated range of variability is the choice of underlying study from which the concentration-response function is selected. The draft PA has appropriately articulated this important source of variation by showing results based on multiple epidemiologic studies. Overall the IPMRP recommends a stronger discussion of the key features of the approach that affect variability and uncertainty of estimates produced, particularly for the sources discussed in the qualitative assessment section. As noted earlier, deadlines do not excuse substantive deficiencies. With more time to conduct the risk assessment it would be possible for the EPA to quantify at least some aspects of these qualitative sources and incorporate them into a second draft of the PA.

There has been incomplete consideration of uncertainties in the exposure estimates. The IPMRP recommends that EPA better articulate the analyses that could be conducted to reduce some of these sources of uncertainty in the revised PA, even if the schedule does not allow them to be conducted. This will be a valuable reference for future risk assessments. In particular, in the limitations section of Appendix C (Section C.1.4.7), some important limitations of the air quality projections are listed. These are important to consider because they could be a key source of uncertainty of the risk estimates. The IPMRP recommends adding:

- a. Reconsideration of reliance only on modeled 2015 concentrations, and not for multiple years, for which model performance was assessed at the national level (it appears), rather than with a focus on the 47 urban study areas.
- b. Additional assessment of whether the downscaler captures the full PM distribution within Core-Based Statistical Areas (CBSAs) (separately addressing spatial variation for long-term studies and temporal for short-term studies).
- c. Additional articulation of the performance of the hybrid models (most particularly the Bayesian downscaler). Model performance is not hampered by low concentrations but rather by strong spatial concentration gradients. Hybrid methods that include land use factors related to primary sources are better able to address spatial gradients. Regional secondary formation in the East means that spatial gradients are much smaller and the models perform better. For this reason, it makes sense that the neural network model would perform better than the Bayesian downscaler in the West. Thus, the uncertainty is larger for the Bayesian downscaler specifically in locations with large concentration gradients. In the West, more weight should be placed on the other hybrid models.

#### **e) Robustness and Validity**

The risk estimates appear to be *robust and valid* although they represent only a subset of at-risk individuals and health endpoints. The ability to assess the robustness and validity of the risk assessment is, however, hampered by the lack of needed clarity in the description of the approach and its application.

While Appendix C provides documentation of multiple aspects of the estimates, the text describing this process on page 3-83 is fairly brief. Although it points to Appendix C, it does not present the key findings or conclusions in a comprehensible way. The goals of this analysis need to be more clearly stated, and the text on the rationale for the different risk modeling approaches should be articulated up front. While the general approaches of upper bound

estimates and the use of sensitivity analysis are justified, as is the use of qualitative uncertainty assessment, several aspects are unclear. The process for selection of concentration-response functions should be explained more specifically. More specific explanation is needed regarding how sensitivity analysis was or will be conducted. The plausibility of the ranges of estimates values should be more completely described in the body of the PA.

The summary of associated premature mortality estimates under alternative standards and exposure reduction scenarios has results in the range that would be expected, although the process for obtaining them is hard to follow and the key features of the appendix tables cited are not well described. The lack of clarity in the descriptions of the approach hampers the ability to assess the robustness and validity of the risk assessment.

As noted, the primary factor that explains variability in the risk estimates for a specific air quality standard is the underlying concentration-response function from a published study. The IPMRP is concerned about whether the estimates are also sensitive to the use of the ambient concentration model, specifically the Bayesian downscaler versus one of the other national models presented in Chapter 2.

Nonetheless, considering all of the information about, and features of, the risk assessment approach, the robustness of the results is enhanced by key sources of variability and uncertainty that are taken into account. The risk estimates have been calculated across 47 urban areas that represent approximately one third of the U.S. population over age 30. They have been estimated using multiple underlying health studies, multiple endpoints classified as causal or likely causal in the ISA, and under different air quality standards and scenarios for downscaling estimates. Thus, the risk assessment is deemed to be adequate for its intended purpose, albeit there is opportunity for substantial improvement based on the recommendations offered here.

***SCQ-3.5 What are the Panel's views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?***

The draft PA reaches the preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards. The Panel concurs with the scientific rationale but recommends a stronger finding based on the scientific evidence: the current primary standards are unequivocally not adequately protective. The entire weight of scientific evidence supports more stringent standards. The Panel concludes that arguments offered in the draft PA for retaining the current standards are not scientifically justified. Both major points are elaborated below.

**Calling into question the adequacy of the current standards**

Overall, the IPMRP concurs with the draft PA's preliminary conclusion that the weight of scientific evidence from various study types and analyses calls into question the adequacy of the current standards to protect public health. This conclusion is based on scientific evidence from epidemiological, controlled human exposure, and animal toxicological studies. The evidence from both long-term and short-term studies supports this conclusion. There is also consistent support from policy-relevant accountability studies that allow more direct causal inferences. Overall, the results provide coherence from multiple scientific disciplines and biological plausibility. In this review there is new and compelling evidence that health effects are

occurring in areas that already meet the levels of the current primary PM<sub>2.5</sub> standards and that are at levels well below those of the current primary PM<sub>2.5</sub> standards. Similar to the prior review (e.g., see EPA-CASAC-10-015, Samet, 2010b), there is no evidence of an ambient concentration threshold for health effects. The concentration-response relationships are approximately linear. The epidemiologic evidence shows increased risks at the levels of the current standards and that there are at-risk groups that are disproportionately affected. The risk assessment is illustrative of a large magnitude of estimated premature mortality at the levels of the current standard. Thus, the scientific evidence in this review provides clear and compelling support of the conclusion, unanimously supported by this expert scientific Panel, that the current primary PM<sub>2.5</sub> standards do not protect public health. The risk-based approach provides additional support. The new scientific evidence in this review strengthens conclusions compared to previous reviews.

The weight-of-evidence framework for causality determination applied by EPA is appropriate and has been well-vetted over more than a decade by many previous CASAC reviews. The weight-of-evidence causal determination framework applied by EPA is an appropriate tool for drawing causal conclusions.

The existing strong and consistent epidemiological evidence was developed using accepted scientific methods, is peer-reviewed, and is coherent with peer-reviewed controlled human studies and toxicological studies that were also developed using accepted scientific methods. This combined body of evidence provides strong support for developing causal determinations. The existing epidemiological studies contain important insights, and, when taken together, provide a weight of evidence that is substantially stronger than any single study can provide alone. The IPMRP notes that the epidemiologic evidence is vast, particularly in terms of the geographic domain and number of subjects included, and provides an overall consistent scientific basis, supported by coherence with controlled human and toxicological studies, for finding that the current primary PM<sub>2.5</sub> standards are not protective of public health. The epidemiologic evidence is scientifically valid for informing recommendations regarding levels of alternative primary PM<sub>2.5</sub> standards.

There are recently emerging causal inference methods for the analysis of individual studies that members of the current CASAC have argued should be imposed as a condition of a study being considered in EPA's weight-of-evidence review. While it may be possible for EPA to integrate applications of emerging causal inference tools in future reviews, these emerging tools still require considerable development before they can be implemented in air pollution epidemiology studies (Carone et al., 2019). The existing epidemiologic evidence meaningfully contributes to the causal conclusions reached in the draft ISA and used in the draft PA. It would be irresponsible to dismiss any or all of these epidemiologic studies, which the Panel finds to be valid, merely because they have not been analyzed using emerging un-vetted advanced statistical methods that are still in their infancy for application to air pollution studies. The Clean Air Act requires EPA to act to protect public health in the presence of uncertainty. For this reason EPA's review and the Panel's advice rely upon the entire body of the scientific evidence.

The collective weight of the scientific evidence from the epidemiologic studies along with supporting experimental evidence from controlled human exposure studies and animal toxicology is unambiguous in showing serious human health effects of PM<sub>2.5</sub> at levels below the current primary standards. The overall strength of evidence from the longstanding body of evidence presented and reviewed in the 2009 ISA (EPA/600/AR-08/139F, U.S. EPA, 2009) has been further bolstered with new studies with a range of study designs. The strong evidence on mortality and morbidity endpoints, coupled with emerging evidence for less extensively studied health endpoints, such as nervous system effects, is scientifically credible. The expert scientific judgment of the IPMRP is that the evidence is credible even based on the epidemiologic studies



alone; other studies, including animal toxicology and human controlled exposure studies support and strengthen this evidence. In particular, the animal study evidence supports biologic plausibility for PM effects on the cardiovascular, respiratory, and nervous systems, as well as for cancer effects. The epidemiologic evidence includes multiple new epidemiologic studies in the U.S. and Canada not included in the 2009 review. These studies consider huge populations and report effects below the current standard, either by restriction of the cohort to individuals living in areas with lower exposures (Di et al., 2017a&b; Shi et al., 2016), or because the average cohort exposures are well below the annual standard (Weichenthal et al., 2016a&b; Pinault et al., 2016). The populations quantified in such recent studies are more than an order-of-magnitude larger than studies available in previous reviews, which has been made possible by scientific developments in the quantification of spatial variability in exposure concentrations using new modeling tools. The ambient air quality hybrid modeling tools are found to perform well and provide a solid foundation for including populations that are not well-served by the existing ambient monitoring network. Furthermore, these studies do not show any evidence of a threshold, including under a variety of statistical approaches and for analyses restricted to concentrations below the levels of the current primary PM<sub>2.5</sub> standards. Indeed, it is possible that the annual concentration-response relationship is steeper at lower exposures. For these reasons the conclusion that the existing standards are inadequate is warranted.

The draft PA considers potential at-risk populations and notes that older adults, populations at increased risk due to existing health conditions (e.g., existing cardiovascular and/or respiratory conditions), and populations with increased exposures (e.g., disadvantaged populations) are all sizable and represent a substantial portion of the U.S. population. These populations are at increased risk due to geographic location, proximity to sources, or population characteristics (specifically age and/or prior disease status) that increase their susceptibility. The conclusion that the existing standards do not provide an adequate margin of safety for these at-risk populations is warranted. There are environmental justice concerns associated with disparity in the distribution of risks which show that at least some minority groups are disproportionately affected. Given that spatial averaging, as described on page 3-102 of the draft PA, can result in disproportionate impacts in minority populations and populations with lower SES, it is appropriate to retain the approach of not using spatial averaging in the form of the standard.

In evaluating population exposures, the draft PA appropriately considers both epidemiologic and controlled human exposure studies. With respect to controlled human exposure studies, the IPMRP puts more weight than the draft PA on the significance of these exposures for informing the appropriateness of the current standard. While exposures are at levels higher than the 24-hour standard, the durations of exposures in these studies are short (typically 2 hours or less) meaning that when these two-hour exposures are averaged over 24 hours, their average levels can be below the 24-hour standard. Several of the controlled human studies indicate significant subclinical effects at high peak levels that are below the level of the current 24-hour standard when averaged over 24 hours.

In considering the epidemiologic studies, the draft PA looks at both the concentration means and lower (10<sup>th</sup> & 25<sup>th</sup>) percentiles in key studies, as well as pseudo-design values, to more directly address whether exposures in these studies would have occurred in areas which would attain the annual standard. The IPMRP concurs with the draft PA's conclusion that the epidemiological evidence for air quality scenarios that meet or are below the level of the current annual PM<sub>2.5</sub> primary standard is compelling, and that this evidence for effects at concentrations below the standard has been strengthened in the most recent review.

While the IPMRP concludes that the scientific evidence alone is sufficient to call into question the existing standards, the Panel finds that the risk assessment also supports this conclusion. As noted earlier (see response to SCG-3.4), the risk assessment indicates that there will be a

large number of estimated premature deaths attributed to PM<sub>2.5</sub> for persons of age 30 or older for the 47 selected urban areas based on simulation of air quality that just meets the current standard. The risk assessment accounts for approximately one-third of the U.S. population that is age 30 or older. Therefore, the risk estimates are based on a large population but underestimate the national total. Based on Table 3-5, the median estimated all-cause mortality from long-term exposure to PM<sub>2.5</sub>, based on 2015 air quality adjusted to just meet the existing standards, ranges from 13,500 based on Thurston et al. (2016) to 52,100 based on Pope et al. (2015). The median estimated all-cause mortality from short-term exposure to PM<sub>2.5</sub> ranges from 1,200 based on Ito et al. (2013) to 3,870 based on Zanobetti et al. (2014). Two different air quality simulation approaches are compared and contribute a smaller portion of variability to the risk estimates than the inter-study variability in concentration-response relationships. While the specific estimates are uncertain, and should be interpreted qualitatively with regard to their magnitude, the draft PA risk assessment buttresses the conclusions based on the scientific evidence that at the levels of the current fine particle standards, the risk of premature mortality is unacceptably high.

While the IPMRP strongly supports the conclusion in the draft PA that the current standards are inadequate, uncertainties remain, as discussed and taken into account in our consensus statements for both the evidence-based and risk-based approaches (SCG-3.3 and SCG-3.4, respectively). The IPMRP concludes that these uncertainties do not in any way overcome the strong weight of scientific evidence in support of lowering the levels of the annual and 24-hour standards.

### **Arguments for keeping the current standard are not justified**

The draft PA suggests a potential alternative argument for retaining the current standard, along with arguments that could be used to support alternative, more stringent standards. The Panel finds that the draft PA's alternative argument in favor of retaining the current standard is a scientifically unjustifiable interpretation of the evidence that over-emphasizes and inappropriately inflates the significance of uncertainties in biological pathways, inappropriately discounts the potential for public health improvements below the current NAAQS on the premise that accountability studies have not examined such levels yet, and inappropriately dismisses risk assessment as a tool. While the IPMRP acknowledges that there remain uncertainties in these realms, the Panel concludes that this is an extreme misinterpretation which runs counter to all reasonable scientific views of the available evidence. The IPMRP concludes that these arguments are not scientifically sound as outlined below.

To dispute the conclusion that the current PM<sub>2.5</sub> standards are not sufficiently protective, it would be necessary to discard the scientific findings from epidemiologic studies. A voluminous body of epidemiologic evidence, accumulated over more than three decades, has consistently shown adverse PM<sub>2.5</sub> health effects over a range of levels and averaging times. This includes hundreds of studies that quantitatively show an adverse effect of PM<sub>2.5</sub> exposure for mortality and multiple other health endpoints, have examined diverse populations and at-risk groups, have considered multiple exposure scenarios including natural experiments and accountability studies, have applied diverse designs, and have employed varied advanced analytic methods. Recent studies that are scientifically valid and policy relevant in this review provide new compelling evidence of effects at concentrations at and below the current primary PM<sub>2.5</sub> standards based on very large cohorts. It also would be necessary to inappropriately ignore conclusions drawn by EPA and CASAC multiple times since 1997 when an air quality standard for PM<sub>2.5</sub> was added. EPA concluded that there were serious health effects associated with PM<sub>2.5</sub> concentrations in areas that met the then (and now still) current PM<sub>10</sub> standard. Most recently in 2012, EPA again concluded the existing PM<sub>2.5</sub> standard was inadequate and thus strengthened the annual standard. The primary scientific evidence for these actions was

epidemiologic studies, supported by evidence from animal and controlled human studies. The current review is bolstered by ground-breaking new epidemiologic studies, based on far larger study populations, as a result of the emergence of new generation of models that quantify spatial variability in exposure concentrations and include populations that are not served by the existing monitoring network. These new studies reaffirm and substantially augment and strengthen the scientific evidence compared to the prior review. These new studies include multiple large U.S. cohort studies that show adverse effects of PM<sub>2.5</sub> on mortality. Several national policy-relevant cohort studies from Canada show mortality associations with long-term average exposures well below the current U.S. PM<sub>2.5</sub> standard. The IPMRP concludes that it is inappropriate to discard this voluminous and consistent body of epidemiologic evidence.

To dispute the conclusion that the current PM<sub>2.5</sub> standards are not sufficiently protective, it also would be necessary to discard the experimental evidence of the biological pathways and mechanisms of action for PM<sub>2.5</sub> health effects. Experimental evidence continues to accumulate that cardiovascular effects from exposure to PM<sub>2.5</sub> include endothelial dysfunction, arterial thrombosis, and arrhythmia. The strongest evidence is for endothelial dysfunction. Respiratory effects are supported by animal toxicological studies that suggest altered host defense, greater susceptibility to bacterial infection, and consistent evidence of respiratory irritant and inflammatory effects. For cancer, mechanisms include DNA damage, micronuclei formation, chromosomal abnormalities, differential expression of genes relevant to cancer pathogenesis and genomic instability. The IPMRP concludes that the growing body of animal and human controlled study scientific evidence since the last review augments and strengthens findings since the last review. Although uncertainties remain, the uncertainties do not outweigh robust inferences regarding biological pathways leading to PM<sub>2.5</sub> health effects based on the overall body of evidence.

To dispute the conclusion that the current PM<sub>2.5</sub> standards are not sufficiently protective, it also would be necessary to conclude that further decreases in PM<sub>2.5</sub> concentrations will not lead to beneficial public health impacts. It is a logical fallacy to claim that absence of evidence is evidence of absence. This fallacy underlies the proffered flawed rationale that because accountability studies have yet to be conducted at levels at or below the current standards, this is sufficient to call into question that there are benefits from reducing the current level of the standard. At levels somewhat higher than and overlapping with the current standard, existing accountability studies provide supporting evidence that there are increases in life expectancy and improvements in respiratory function in children associated with reductions in ambient PM<sub>2.5</sub>. The accountability studies listed in Table 3-3 of the draft PA are useful in supporting causality determinations of adverse effects of PM<sub>2.5</sub> at annual levels close to, and overlapping with, the current standard. Thus, they provide important insights related to risk reduction, even though they are not at low enough levels to serve as a basis for recommending alternative levels. While accountability studies have not yet been conducted in the range of the current or proposed alternative standards, the existing evidence that there is not a discernible threshold in PM<sub>2.5</sub> health effects supports a reasoned scientific judgment that there are public health benefits to lowering the current standard (as, of course, also shown in the numerous epidemiological studies showing health effects in areas with air quality distributions less than those allowed by the current annual and 24-hour standards). Such a judgment does not require that there must be policy-relevant accountability studies, even though they would be informative if they existed. Therefore, the IPMRP concludes that it is inappropriate to give weight to the lack of existing accountability studies below the current standard as a meaningful source of uncertainty in calling into question the current primary PM<sub>2.5</sub> standards.

To dispute the conclusion that the current PM<sub>2.5</sub> standards are not sufficiently protective, an implied flawed rationale is proffered on page 3-98 (lines 1-4) that uncertainties in the risk

assessment are so large as to render the risk assessment uninformative. As noted earlier, the Panel gives more weight to the evidence-based approach than to the risk-based approach in arriving at a finding that the current standards are not adequate to protect public health. The risk assessment provides support but is not necessary or essential to our finding. Nonetheless, taking uncertainties related to the risk assessment into due consideration, it is our expert scientific judgment that the risk assessment provides supporting information, as have risk assessments in past reviews. A claim that the risk assessment is not informative is only possible if one completely discards the epidemiologic evidence as irrelevant to estimating population risk, and/or disputes most of the methods used and assumptions made in the risk assessment. While the IPMRP believes that the risk assessment can be improved and has provided multiple suggestions in this regard, the Panel finds that the risk assessment approach is sound and the results are qualitatively informative for consideration of the adequacy of the current standard as a supplement to the findings based on the evidence-based approach. The Panel affirms that it is appropriate to base the risk assessment on the recent epidemiologic studies because these studies inform our understanding of population risk in the exposure range relevant to the current standards. The Panel also does not consider that the limitations of the risk assessment invalidate the qualitative conclusions that can be reached from its results, namely that the estimated magnitude of premature deaths attributed to PM-related mortality at the levels of the current primary PM<sub>2.5</sub> standards is unacceptably high. The IPMRP concludes that it is inappropriate to over-emphasize and inflate the significance of uncertainty in the risk assessment to the point of calling into question the key insights afforded by the assessment. However, the IPMRP also notes that, while the risk assessment is informative and supportive in providing the basis for qualitative insights regarding the magnitude of risk, more weight is given to the evidence-based approach in drawing conclusions.

Overall, the IPMRP concludes that in order to accept the current standards as adequate, multiple implausible and scientifically unjustifiable assumptions and conclusions are necessary. Applying Occam's razor – i.e., the more assumptions that are required, the more implausible the explanation – the IPMRP concludes that the arguments in favor of retaining the current standard are specious. The revised PA should acknowledge the implausibility of these assumptions or consider dropping them altogether.

**SCQ-3.6 What are the Panel's views on the conclusions in the draft PA regarding developing potential  $PM_{2.5}$  alternative standards with respect to:**

- a) The preliminary conclusion that the available information continues to support the  $PM_{2.5}$  mass-based indicator, remains too limited to support a distinct standard for any specific  $PM_{2.5}$  component or group of components, and remains too limited to support a distinct standard for the ultrafine fraction?**
- b) The preliminary conclusion to retain the annual and 24-hour averaging times?**
- c) The preliminary conclusion that it is appropriate to consider retaining the forms of the current annual and 24-hour  $PM_{2.5}$  standards, in conjunction with revised levels?**
- d) The preliminary conclusion that the range for alternative levels for the annual  $PM_{2.5}$  standard should begin below  $12 \mu\text{g}/\text{m}^3$  and extend as low as  $8 \mu\text{g}/\text{m}^3$ ?**
- e) The possible rationales for alternative annual  $PM_{2.5}$  levels of 12, 10, and  $8 \mu\text{g}/\text{m}^3$ ?**
- f) The preliminary conclusion that, in conjunction with a lower annual standard intended to protect against both short- and long-term exposures, the evidence does not support the need for a revised level for the  $PM_{2.5}$  24-hour standard?**
- g) The discussion of an alternative approach to lower the level of the 24 hour standard to  $30 \mu\text{g}/\text{m}^3$  to provide increased protection for both short- and long term exposures?**

The draft PA provides appropriate scientific rationales for retaining the current indicator, averaging time, and form for the primary  $PM_{2.5}$  standards. Based on the scientific evidence, as summarized in more detail in responses to SCG-3.3, SCG-3.4, and SCG-3.5, the Panel finds that annual levels above  $10 \mu\text{g}/\text{m}^3$  are not protective of public health. The draft PA provides an appropriate scientific rationale for annual levels between  $10 \mu\text{g}/\text{m}^3$  to  $8 \mu\text{g}/\text{m}^3$ . The Panel's scientific opinion regarding  $PM_{2.5}$  alternative standards is that an annual standard of 10 to  $8 \mu\text{g}/\text{m}^3$  and a 24-hour standard of  $30 \mu\text{g}/\text{m}^3$  to  $25 \mu\text{g}/\text{m}^3$  taken together as a suite of standards is appropriate, with the lower end of these ranges providing more protection against risk of premature mortality and other adverse effects due to exposure to  $PM_{2.5}$ .

**What are the Panel's views on the conclusions in the draft PA regarding developing potential  $PM_{2.5}$  alternative standards with respect to:**

- a) The preliminary conclusion that the available information continues to support the  $PM_{2.5}$  mass-based indicator, remains too limited to support a distinct standard for any specific  $PM_{2.5}$  component or group of components, and remains too limited to support a distinct standard for the ultrafine fraction?**

There is little new information since the last review to support consideration of changes to the indicator, form, or averaging times for the annual and daily NAAQS. Although there is not sufficient scientific evidence or analysis in the draft PA upon which to make a recommendation in this review cycle, a rolling 24-hour form would better reflect daily exposures than the current midnight to midnight 24-hour calendar day period, since some sources have a strong diel pattern, peaking overnight where a single ambient concentration high night is broken into two separate days under the current standard. This would require that nearly all monitoring sites

have valid continuous FEM PM<sub>2.5</sub> data, which is not currently the case; about 60% of the approximately ~900 PM<sub>2.5</sub> monitoring sites have valid FEM data. Thus, there is a need to improve the coverage of FEM monitors that measure continuous hourly ambient concentrations. It would be appropriate for UFP to be considered in the next review cycle as an additional indicator, contingent upon accumulation of additional quantitative evidence regarding exposure-response relationships, since it is described as “likely to be causal” for long-term nervous system effects. This would require development of an FRM for UFP and implementation of a UFP monitoring network which could be based upon the existing near-road network including pairing with existing nearby neighborhood or urban scale sites.

***b) The preliminary conclusion to retain the annual and 24-hour averaging times?***

The annual and 24-hour averaging times are appropriate and are supported by scientific studies of adverse health effects at these averaging times. The Panel concurs with the draft PA, page 3-101, lines 14-16, that “Epidemiologic studies continue to provide strong support for health effects associated with both long- and short-term PM<sub>2.5</sub> exposures based on annual (or multiyear) and 24-hour PM<sub>2.5</sub> average periods, respectively.”

There is limited evidence that suggests sub-daily PM exposures are important, but it is not sufficient to support a sub-daily averaging interval at this time. A sub-daily averaging time would require development of a reference and/or equivalent method for measurement of PM<sub>2.5</sub> unless the value of the 24-hour standard were reduced to protect against 4-hour to 12-hour exposures of concern. A rolling 24-hour form could provide additional protection against sub-daily exposures depending on the selected level. A 24-hour rolling average is typically more health protective than a 24-hour calendar average for a given level.

***c) The preliminary conclusion that it is appropriate to consider retaining the forms of the current annual and 24-hour PM<sub>2.5</sub> standards, in conjunction with revised levels?***

The forms of the current annual (3-year average) and 24-hour (98<sup>th</sup> percentile) primary PM<sub>2.5</sub> standards are appropriate in conjunction with revised levels. The Panel supports the rationale given in the draft PA for retaining these forms. Epidemiologic studies continue to provide strong scientific support for health effect associations with both long-term (annual, multiyear) and short-term (mostly 24-hour) PM<sub>2.5</sub> exposures. The form of the annual standard is appropriate for targeting protection against annual PM<sub>2.5</sub> exposures and offers protection in many areas of the country against 24-hour PM<sub>2.5</sub> exposures. Epidemiologic studies, with support from controlled human studies, provide scientific evidence of associated adverse effects at the 24-hour averaging time. The Panel concurs with the draft PA that “nothing in the evidence that has become available since the last review calls into question” the forms of the current standards. These forms are appropriate in conjunction with revised levels.

***d) The preliminary conclusion that the range for alternative levels for the annual PM<sub>2.5</sub> standard should begin below 12 µg/m<sup>3</sup> and extend as low as 8 µg/m<sup>3</sup>?***

The initial consideration in the draft PA of a range for an alternative annual primary PM<sub>2.5</sub> standard of 11 µg/m<sup>3</sup> to 8 µg/m<sup>3</sup> is a reasonable starting point given the robust new evidence of premature mortality down to at least 8 µg/m<sup>3</sup>, as covered in the draft ISA and this draft document (Pinault et al., 2016; Weichenthal et al., 2016a; Weichenthal et al., 2016b). However, as explained below, the scientific evidence supports 10 µg/m<sup>3</sup>, not 11 µg/m<sup>3</sup>, as the upper bound of the Panel’s recommended range. In determining the range of levels to be considered for a revised annual standard, the Panel concurred with the draft PA that it is appropriate to

consider the means of key epidemiologic studies, which is consistent with past practice in previous reviews. The Panel notes, however, that some studies have been re-analyzed based on truncated data (e.g., for ambient concentrations not exceeding the current standard) for which robust findings of adverse effect have been identified (Di et al., 2017a; Shi et al., 2016). Analyses based on “partial means” of truncated air quality distributions provide additional scientific support of adverse effects at levels below the current annual standard. The Panel also considered the scientific evidence from epidemiologic studies at ambient levels below the mean ambient level of the studies. For example, at the 25th percentile, or the 10th percentile, although the uncertainties are greater, there is variability in adverse effect with respect to variability in ambient concentration. Collectively, considering all of these factors, the Panel unanimously finds a scientific basis for  $8 \mu\text{g}/\text{m}^3$  as being the lower bound of annual ranges for which there is strong weight of scientific evidence of adverse effects. Although there is some evidence of adverse effects at levels below  $8 \mu\text{g}/\text{m}^3$ , the uncertainties at such lower levels become larger. The lower bound of  $8 \mu\text{g}/\text{m}^3$  for the annual primary  $\text{PM}_{2.5}$  standard is supported by U.S. based studies with additional support from Canadian studies. Multiple studies indicate that there may be risk below  $8 \mu\text{g}/\text{m}^3$ .

The Panel considered limitations of studies in arriving at these levels. Confounding by individual characteristics must be considered as an alternative explanation for observed associations in epidemiologic studies. In the key epidemiologic cohort studies, the estimated associations with  $\text{PM}_{2.5}$  are adjusted for individual life-style characteristics such as smoking, as in the Canadian (Pinault et al., 2016) and U.S. studies (Pope et al., 2015; Jerrett et al., 2016; Thurston et al., 2016; Turner et al., 2016). In national cohort studies where individual life-style characteristics are not available, indirect adjustment can be used drawing on other life-style surveys, such as in the CanCHEC study (Weichenthal et al., 2016a). In the U.S. Medicare cohort study (Di et al., 2017a) individual life-style characteristics were not available for the entire population. However, in a subset of the Medicare cohort, Di et al. showed that individual smoking and income levels were not associated with  $\text{PM}_{2.5}$  exposures, a necessary condition for confounding. The Panel found that mortality associations with long-term  $\text{PM}_{2.5}$  exposures were consistent after direct and indirect adjustment for individual life-style factors in all of these key U.S. and Canadian studies. Although not every study is able to control as well as possible for socioeconomic status at both the individual and neighborhood level, in those for which the data are available, the findings are robust to that adjustment. In studies of long-term exposure to particulate matter, there is neither rationale nor empirical support for concern over confounding by temperature. Consistency of results based on multiple studies that employ multipollutant models, among which there are differences in underlying factors such as the relative ambient mixtures of co-pollutants, population demographics, climatic zones, and distributions of housing characteristics, support the robustness of their results. Therefore, the expert scientific judgment of the Panel is that the available scientific evidence robustly supports the recommended range of levels.

***e) The possible rationales for alternative annual  $\text{PM}_{2.5}$  levels of 12, 10, and  $8 \mu\text{g}/\text{m}^3$ ?***

The Panel finds that  $10 \mu\text{g}/\text{m}^3$  is the upper bound of the recommended range for the annual primary  $\text{PM}_{2.5}$  standard based on the scientific evidence. At this level, there is a very high degree of scientific confidence in the relationship between exposure to fine particles and adverse effects, based on consistent epidemiological findings from multiple multi-city studies, augmented with findings from single-city studies, at policy-relevant ambient concentrations at or below the levels of the current standards and that are supported by research from experimental models in animals and humans. The overall body of evidence supports the causal determinations for adverse health effects of fine particulate matter as set forth in the draft Integrated Science Assessment. The Panel considered whether  $11 \mu\text{g}/\text{m}^3$  should be an upper

bound of its scientifically-recommend range. For example, a key study by Shi et al. (2016) has a pseudo-design value near 11  $\mu\text{g}/\text{m}^3$ . However, as noted elsewhere in the draft PA, the PDVs are up to 10% higher than an actual design value. The far more compelling scientific rationale for rejecting 11  $\mu\text{g}/\text{m}^3$  as an upper bound is the strong epidemiologic evidence of premature mortality at this annual concentration. An annual concentration of 11  $\mu\text{g}/\text{m}^3$  would not be protective of public health.

Thus, the Panel finds, based on the scientific evidence, that the annual standard should be revised within a range of annual average concentrations of 10  $\mu\text{g}/\text{m}^3$  to 8  $\mu\text{g}/\text{m}^3$ , while retaining the indicator, averaging time, and form of the current annual standard. The choice of level within this range is a policy judgment at the discretion of the Administrator. A choice toward the lower end of the range would provide additional health protection compared to a choice at the higher end of the range. Based on currently available evidence and inferences, the exposure-response relationship is approximately linear and there is no threshold within this range, nor is there evidence of a specific threshold below this range.

The draft Policy Assessment uses two approaches to assess the protection provided by alternative annual  $\text{PM}_{2.5}$  levels: the risk-based approach using 47 urban areas with downscaler rollback of ambient  $\text{PM}_{2.5}$  concentration to just meet alternative levels in each area for which health outcomes are predicted using BenMap, and the evidence-based (epidemiological study) approach where the risk of premature mortality is expressed as a hazard ratio for a 10  $\mu\text{g}/\text{m}^3$  increase in concentration. The Panel prefers the evidence-based approach for the reasons described under part (d). The evidence-based approach also demonstrates that certain sub-populations have different risk; in this case the Di et al. (2017a) chronic Medicare study shows that the relative risk for African Americans is three times higher than that of the entire population, with a hazard ratio of 1.21 per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ . If the primary  $\text{PM}_{2.5}$  standards are intended to provide protection to sensitive sub-groups and not just the population as a whole, this is important information that is not taken into account in the risk-based approach and is, therefore, not adequately taken into account in the draft Policy Assessment.

Taking the strengths and limitations of the risk assessment into account, including its uncertainties, the risk assessment is useful and scientifically robust in illustrating that reductions in the level of the annual standard will lead to proportional reductions in premature mortality. At the level of the current standard, the estimated magnitude of premature deaths for the populations that were included in the selected study areas is unacceptably high, as detailed in responses to SCG-3.4 and SCG-3.5. The risk is linear with no threshold below the current standard down to an annual level of 8  $\mu\text{g}/\text{m}^3$  or lower. The Thurston et al. (2016) (not 2015 as in some of the tables) AARP cohort shows lower mortality rates; this may be in part due to the AARP cohort having higher than average socio-economic status than the population as a whole, and being somewhat younger (starting at age 55, not 65) than the Medicare cohort. The risk assessment is useful for providing qualitative support to our finding that the current standard is not adequate, with the evidence-based approach being the more compelling source of scientific evidence.

The draft PA does not give sufficient emphasis in its discussion of the risk analysis with regard to study results and corresponding risk estimates below 8 to 9  $\mu\text{g}/\text{m}^3$  annual average concentration, even though results at such levels are shown in Figure 3-12. The draft PA claims that there is insufficient information from studies at those low concentrations. However, Figure 3-8 of the draft PA shows that the annual level of  $\text{PM}_{2.5}$  for 25% of the Di et al. (2017) chronic mortality Medicare study population was below 7  $\mu\text{g}/\text{m}^3$ . This represents 115 million person-years of follow-up, a very large sample size that results in relatively robust mortality estimates



even at levels below  $7 \mu\text{g}/\text{m}^3$  (Di et al., 2017a, Figure 3a). Thus, there is a very large population with current annual PM exposures less than  $8 \mu\text{g}/\text{m}^3$  for which effects have been found. While the effect is lower at these lower concentrations, there is a suggestion of a supralinearity of the CR curve below  $7 \mu\text{g}/\text{m}^3$  (higher risk per unit PM exposure increase), and the overall mortality is large in this group because of its size. These issues are not clearly or adequately addressed in the draft PA. Although the Panel gave consideration to whether the lower end of the recommended range for the revised annual primary  $\text{PM}_{2.5}$  standard might be at  $7 \mu\text{g}/\text{m}^3$ , the Panel finds that there is not sufficient scientific certainty at this low of a level to support such a recommendation.

***f) The preliminary conclusion that, in conjunction with a lower annual standard intended to protect against both short- and long-term exposures, the evidence does not support the need for a revised level for the  $\text{PM}_{2.5}$  24-hour standard?***

The Panel finds that the current  $\text{PM}_{2.5}$  24-hour standard is not adequate to protect public health, as explained above. The Panel concurs with the scientific rationale in the draft policy assessment for retaining the indicator, averaging time, and form of the current standard. Based on the scientific evidence, the Panel recommends that the level of the  $\text{PM}_{2.5}$  24-hour standard be revised to a range between  $30 \mu\text{g}/\text{m}^3$  to  $25 \mu\text{g}/\text{m}^3$ . In this regard, our scientific advice differs from that of the draft PA, with supporting details both above and below. In particular, the Panel notes that the 24-hour standard is controlling in some locations and, thus, in such locations provides health protection not adequately afforded by the annual standard alone.

When paired with an annual standard of  $10 \mu\text{g}/\text{m}^3$  or lower, the current  $\text{PM}_{2.5}$  24-hour standard of  $35 \mu\text{g}/\text{m}^3$  is not sufficient to provide adequate protection against short-term exposures in situations such as smoke from residential wood combustion in valleys, where  $\text{PM}_{2.5}$  is only elevated for part of the year. Exposures to smoke from residential wood combustion in several parts of the country may occur for 6 to 12 hours, typically overnight; high night-time  $\text{PM}_{2.5}$  concentrations are broken into separate days when calendar day (midnight to midnight) 24-hour averaging intervals are used.

The Panel notes that even at lower levels within its recommended range for the annual primary  $\text{PM}_{2.5}$  standard, the available scientific evidence indicates that the annual standard does not adequately protect against short-term exposures, including sub-daily exposures, in some parts of the U.S. These include locations with overnight exposures to smoke from residential wood combustion, as noted above. Furthermore, there are scientifically anticipated effects related to common exposure scenarios, such as short-term peaks in near-road exposures, especially during peak travel times, to particles across a range of sizes and chemical composition.

The Panel finds that the use of calendar-day 24-hour averages for the short-term standard may not be protective of public health, unless the level is set low enough to prevent potentially harmful peak exposures. Over time, a larger number of real-time FEM monitors have been placed in service that are capable of providing hourly-averaged  $\text{PM}_{2.5}$  concentration readings. Thus, the monitoring network has transformed such that it has the technical capability to support a 24-hour rolling average, calculated each hour. At a given level, a rolling average is typically more health protective than a calendar-day average. The Panel recommends that EPA conduct a comparative analysis of an hourly 24-hour rolling average versus the current 24-hour calendar-day average to assess the potential health protective benefits of a change in form. Without a supporting analysis, the Panel was unable to offer a recommendation for the rolling average form. Furthermore, the Panel recommends that data be collected and analyzed to

support consideration of sub-daily averaging times, with rolling average forms, in the next PM NAAQS review.

***g) The discussion of an alternative approach to lower the level of the 24 hour standard to 30 µg/m<sup>3</sup> to provide increased protection for both short- and long term exposures?***

For 24-hour exposures, there are numerous studies that find adverse effects at levels well below the current standard, within a range of 30 µg/m<sup>3</sup> to 25 µg/m<sup>3</sup> (Shi et al., 2016; Di et al., 2017b; Weichenthal et al., 2016a; Weichenthal et al., 2016b). The choice of the 47 urban areas does not include some areas of the country for which a 24-hour standard, rather than an annual standard, would be controlling. The draft Policy Assessment provides scientific support for a level of 30 µg/m<sup>3</sup> as an alternative to the current level of 35 µg/m<sup>3</sup> for the 24-hour primary PM<sub>2.5</sub> standard. Even with an annual level in the range of 10 µg/m<sup>3</sup> to 8 µg/m<sup>3</sup>, a 24-hour standard at 30 µg/m<sup>3</sup> may not be protective of acute health effects that could occur with sub-daily exposures, based on scientific evidence from controlled human studies. Furthermore, based on numerous epidemiologic studies for 24-hour average exposures, there is a continuum of adverse effects down to well below 25 µg/m<sup>3</sup>. Thus, 25 µg/m<sup>3</sup> is a 24-hour average level that is scientifically justifiable for consideration in setting a revised standard. However, there is no threshold for 24-hour daily average exposures; while a 24-hour level at 25 µg/m<sup>3</sup> would offer more protection than a 30 µg/m<sup>3</sup> level, it does not reduce risk to zero.

The choice of levels for the 24-hour standard is largely and predominately informed by multiple consistent epidemiologic studies of acute health effects based on daily metrics for exposure and health outcomes. However, the Panel notes that controlled human studies with high sub-daily exposures (2 hours at 24 to 300 µg/m<sup>3</sup> PM<sub>2.5</sub>) which exhibit subclinical effects are equivalent to 24-hour exposure concentrations that are policy relevant (Hemmingsen et al., 2015; Devlin et al., 2003; Gong et al., 2004; Tong et al., 2005). As such, these studies add support, but are not the primary factor informing, our expert scientific judgment that the current 24-hour average standard is not adequate to protect public health. PDVs should be calculated for the controlled human studies.

A secondary factor in identifying a range of alternative levels for the 24-hour standard is the argument that the annual standard is controlling and that the 24-hour standard is a backstop against acute adverse effects not otherwise controlled by the annual standard. In past reviews and in this review, there is an underlying notion that there is a typical mean ratio between annual and 24-hour levels. Thus, if the annual level is revised downward, the 24-hour level should be revised downward proportionally. A linearly proportional reduction in the 24-hour level implied by reducing the annual level, from 12 µg/m<sup>3</sup> to a range of 10 µg/m<sup>3</sup> to 8 µg/m<sup>3</sup>, would imply a range of 24-hour levels of 29 µg/m<sup>3</sup> to 23 µg/m<sup>3</sup>. However, the Panel views this as a secondary factor in the choice of levels, with more attention given to the scientific health-based rationale for choice of levels given above.

The Panel also considered a sub-daily averaging time, such as a 2 to 8 hour rolling average, calculated hourly. Such a standard would more directly protect against peak exposures such as near roadway or from residential wood combustion or so-called wildfires that are largely anthropogenic. A sub-daily standard could be based on the maximum daily X-hour average, where X is the selected averaging time, analogous to the current primary ozone standard. However, this is more appropriately a topic that should be seriously considered in the next review cycle, rather than this review cycle, given the lack of sufficient evidentiary support at this time upon which to make a recommendation.

**EPA-4. Chapter 4 – Review of the Primary PM<sub>10</sub> Standard: What are the CASAC views on the approach described in Chapter 4 to considering the PM<sub>10-2.5</sub> health effects evidence in order to inform preliminary conclusions on the primary PM<sub>10</sub> standard? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current primary PM<sub>10</sub> standard?**

**SCQ-4.1 To what extent does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>10</sub> NAAQS review? Are there additional policy-relevant questions that should be addressed?**

**SCQ-4.2 What are the Panel's views of the draft PA assessment of the currently available scientific evidence regarding the health effects associated with exposures to thoracic coarse particles, PM<sub>10-2.5</sub>?**

**SCQ-4.3 What are the Panel's views on the draft PA preliminary conclusion that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports consideration of retaining the current standard?**

Although new evidence is available since the last review for a broader range of health outcomes associated with short- and long-term exposures to thoracic coarse particulate matter (PM<sub>10-2.5</sub>), this evidence is subject to considerable uncertainty. The causality determinations in the draft ISA for PM<sub>10-2.5</sub> are no higher than “suggestive of, but not sufficient to infer, a causal relationship” for short-term respiratory, short-term cardiovascular, and short-term mortality effects, and “inadequate” to infer a causal relationship for other considered endpoints.

The draft PA appropriately discusses the strengths and limitations of the available scientific evidence regarding PM<sub>10-2.5</sub>. Multicity studies in Europe and Asia provide evidence of consistent associations between short-term exposure to PM<sub>10-2.5</sub> and premature mortality. However, more policy-relevant research is needed to better quantify the adverse effects of PM<sub>10-2.5</sub>. PM<sub>10-2.5</sub> can penetrate to the airways past the vocal cords, which should be acknowledged and discussed in the draft PA, and can help explain why there is some evidence attributing asthma exacerbation to PM<sub>10-2.5</sub> exposure.

The Panel concurs with the draft PA that PM<sub>10</sub> is an appropriate choice at this time for the indicator for PM<sub>10-2.5</sub>. However, PM<sub>10</sub> is an imperfect indicator of PM<sub>10-2.5</sub>. The Panel recommends movement away from PM<sub>10</sub> and toward PM<sub>10-2.5</sub> as the indicator. The use of PM<sub>10</sub> as an indicator for PM<sub>10-2.5</sub> dates to a time when there was not yet a reliable monitoring method specific to PM<sub>10-2.5</sub>. Nationwide, PM<sub>10-2.5</sub> sites are <20% of the ~1564 PM<sub>2.5</sub> sites, insufficient to capture the needed temporal and spatial variations.

EPA's lack of adequate support for PM<sub>10-2.5</sub> measurements (e.g., network design, ambient monitoring, and chemical speciation) hinders the assessment of the PM<sub>10-2.5</sub> relationships to health effects. Such measurements are essential to reduce uncertainties in causality determination (e.g., approaches to estimating PM<sub>10-2.5</sub>; measurement errors and lack of biological plausibility).

Since 2000, 24-hour PM<sub>10</sub> concentrations have decreased by ~30% with the majority of PM<sub>10</sub> sites measuring below 75 µg/m<sup>3</sup>. The 3-year average of annual 98<sup>th</sup> percentiles of 24-hour PM<sub>10-2.5</sub> concentrations for 2015-2017 are mostly less than 30 µg/m<sup>3</sup>, in line with the observed nationwide PM<sub>2.5</sub> to PM<sub>10</sub> ratios of 0.5-0.6.

The Panel concurs with the assessment in the draft PA that “the available evidence supports maintaining a PM<sub>10</sub> standard to provide some measure of protection against PM<sub>10-2.5</sub> exposures” (p 4-15, lines 9-10). The Panel concurs with the draft PA that it is scientifically reasonable to retain, without revision, at least the level of protection afforded by the current PM<sub>10</sub> standard. However, as noted below, this is not the same as retaining the current level of the standard.

The draft PA does not mention CASAC’s advice regarding the PM<sub>10</sub> standard in its 2010 ‘closure’ letter on the second external review draft of the Policy Assessment in the prior review (Samet, 2010b). At that time, EPA and CASAC considered a different form of the PM<sub>10</sub> standard based on the 98<sup>th</sup> percentile rather than the current one exceedance per year on average over three years. CASAC advised that “a 98th percentile level between 75 and 80 µg/m<sup>3</sup> is comparable in the degree of protection afforded to the current PM<sub>10</sub> standard.” CASAC further advised that “[w]hile recognizing scientific uncertainties, CASAC supports a lower level to provide enhanced protection, somewhere in the range of 75 – 65 µg/m<sup>3</sup>.” Thus, CASAC recommended consideration of a revised standard that would afford more health protection than the current standard. A second draft of the PA should acknowledge and discuss this prior advice. The Panel is supportive of consideration of ranges under the current form that have similar levels of protection as those recommended by CASAC in the last PM NAAQS review.

The draft PA does not address the impact of recommended reductions in the level of the 24-hour primary PM<sub>2.5</sub> standard with respect to the level of protection afforded by the primary PM<sub>10</sub> standard. Because PM<sub>2.5</sub> is a component of PM<sub>10</sub>, accounting for 50%-60% of PM<sub>10</sub> mass on a national average as noted above, a reduction in the level for the 24-hour primary standard for PM<sub>2.5</sub>, as recommended by this Panel, would lead to less protection from an unchanged primary PM<sub>10</sub> standard. This is because retaining the same primary PM<sub>10</sub> standard would allow proportionately more PM<sub>10-2.5</sub> mass as the primary PM<sub>2.5</sub> standard is revised downward. Thus, to retain the same public health protection, consideration should be given to revising the primary PM<sub>10</sub> standard downward.

***EPA-5. Chapter 5 – Review of the Secondary Standards: What are the CASAC views on the approach described in Chapter 5 to considering the evidence for PM-related welfare effects in order to inform preliminary conclusions on the secondary standards? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current secondary PM standards?***

The general approach employed in Chapter 5 begins by noting that relatively little new information is available on PM-related welfare effects on materials, climate and visibility. This disregards important new information on visibility preference indices (see response to SCQ-5.2(a) below). In developing a “rationale” for supporting conclusions on the current secondary standards, Chapter 5 begins with the 2012 Administrator’s observations that combining the most lenient end of the considered range with the most lenient end of the considered level of an alternative secondary NAAQS provided little added protection over the current NAAQS. It then presents these previous conclusions as if they represented the current state of the science. They do not, nor were they supported by CASAC advice provided during the 2012 review (see for example, Samet, 2010a&b).

***SCQ-5.1 To what extent does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the secondary PM standards? Are there additional policy-relevant questions that should be addressed?***

Generally, the questions posed in Chapter 5 reflect many of the important policy-relevant issues for secondary standards. One additional important question that should be raised is whether a single level of PM light extinction (or PM<sub>2.5</sub> mass) is appropriate for protecting visibility in all urban and rural areas in all regions of the country. Questions should also be raised about whether a 24-hour averaging time or a 90th percentile form are appropriate for protecting visibility. Several of these elements of the alternative 2012 secondary NAAQS considered and rejected by the Administrator were not consistent with current science or with CASAC advice in the two previous (2006 and 2012) PM NAAQS reviews (see for example: Hopke, 2004; Henderson, 2006; Samet, 2010a&b).

***SCQ-5.2 What are the Panel's views of the draft PA evaluation of the currently available scientific evidence with respect to the welfare effects of PM. Does the assessment appropriately account for any new information related to factors that influence:***

***a) Quantification of visibility impairment associated with PM<sub>2.5</sub> and examination of methods for characterizing visibility and its value to the public?***

The concept that there is a single level of “acceptable” visibility is flawed. Visibility preferences are likely to vary regionally, from one urban area to another and from urban to rural areas, depending on the nature of the scenes and landscape features typically viewed in those areas. While people in a given area may rate a certain level of visibility as acceptable, this does not imply that they would not realize a welfare gain from further improvements in visibility (Boyle et al., 2016; Haider et al., 2019; Yao et al., 2019). The relatively small number of currently available visibility preference studies have shown that there are different levels of “acceptable” visibility levels in different study areas when visibility impairment was expressed in terms of fixed levels of light extinction.

An important recent meta-analysis of these available visibility preference studies conducted by William Malm and colleagues (Malm et al., 2011, 2019; Malm, 2013, 2016; Molenaar and Malm, 2012) addresses the limitations with the concept that there is any specific level of light extinction that is universally acceptable. This important work was entirely omitted from the draft ISA and from the draft PA. Malm's recent work evaluated a large number of visibility preference indicators and found that the apparent contrast of distant, prominent but not necessarily dominant, scene elements was a much better and more consistent predictor of “acceptable” visibility, than any specific level of light extinction. Across all the currently available visibility preference studies, as the apparent contrast of distant, prominent scene elements approached an apparent contrast level of about -0.04 (i.e. very little contrast), 50% of respondents found the visibility unacceptable. In simpler terms, as the visual range approaches the distance of distant scenic elements, people everywhere find the visibility unacceptable. It would be a relatively straightforward GIS exercise to characterize typical average and/or maximal viewing distances across different urban/suburban/rural areas and regions. The Agency should include such calculations, along with associated extinction levels in the second drafts of the ISA and PA.

In addition to this recent work on visibility preference, the draft PA and draft ISA also neglect a relatively large body of recent (and historical) research on the economic effects of scenic views

on property values. A review of this literature could provide an additional approach for evaluating improvements (or degradation) in visibility regardless of any fixed definition of “acceptability”. See for example Jeong et al., 2019; Mittal and Byahut, 2017; Nicholls and Crompton, 2018; Walls et al., 2015; and others.

Regarding the quantification of visibility impairment associated with  $PM_{2.5}$ , the draft PA advocates 24-hour, filter-based, calculated light extinction as the preferred indicator of PM visibility effects. This is contrary to various CASAC recommendations during the 2012 NAAQS review (Samet, 2010a&b), which advised the Agency to consider:

- A. measuring PM light extinction directly and continuously to support an hourly or multi-hour daylight-only averaging time(s), or if the Agency still finds this unfeasible:
- B. using the relatively sparse PM speciation data to calculate seasonal (or monthly) regional species and  $f(RH)$  values to combine with the much denser continuous  $PM_{2.5}$  monitoring network to calculate hourly PM light extinction, or
- C. simply use the hourly  $PM_{2.5}$  as the basis for a sub-daily (hourly or multi-hour) daylight-only indicator, which would intentionally remove the variable influence of water from the regulatory metric.

In comments during the 2006 review, CASAC also concluded that the current  $35 \mu g/m^3$  daily standard was inadequate to protect visibility, and recommended a secondary NAAQS with a  $PM_{2.5}$  mass indicator, 4 to 8-hour daylight averaging time, 20 to  $30 \mu g/m^3$  level, and 92nd to 98th percentile form (Hopke, 2004; Henderson, 2006). Note also that CASAC comments during the 2012 review reiterated that the current NAAQS was inadequate for protecting visibility, observing that “the levels of the current  $PM_{2.5}$  and  $PM_{10}$  standards are too high, and their averaging times are too long, to guard against levels of visual air quality considered adverse over the short (hour or less) time periods during which changes in visual air quality are perceptible.” CASAC further noted that a form as lenient as the 90th (to 98th) percentile only be considered if the averaging time was for the single worst hour of the day, recommending the 95th to 98th percentile range if combined with multi-hour, sub-daily daylight averaging time (Samet, 2010a). The combinations of indicator, averaging time, level and form recommended by CASAC in the past two NAAQS reviews are all considerably more protective than the current NAAQS and the “most lenient possible combination” of elements considered and rejected in the 2012 review, and repeated again in the current draft PA. A second draft of the PA should systematically address these issues while taking into account the implications of revisions to the 24-hour  $PM_{2.5}$  standard recommended by the Panel, which may have co-benefits with respect to welfare effects.

#### ***b) The effects of $PM_{2.5}$ components on climate?***

The effects of the mix of PM species on climate remain complex, multi-directional and uncertain. It is not clear if a secondary standard would be the best way to address this issue.

#### ***c) The effects of fine and coarse particles on materials?***

Chapter 5 presents some interesting new work on adverse effects of PM deposition on the efficiency of solar panels, although this work may not yet lend itself to specific quantitative relationships with  $PM_{2.5}$  and or  $PM_{10}$  to support consideration of secondary standards.

***SCQ-5.3 What are the Panel's views of the draft PA preliminary conclusion that the currently available scientific evidence does not call into question the protection afforded by the current secondary PM standards against PM welfare effects and that it is appropriate to consider retaining the current secondary PM standards without revision?***

The Panel strongly disagrees with the draft PA preliminary conclusion that the currently available evidence supports retaining the current secondary standards without revision. As indicated above and with more detail in individual comments (see especially comments from Richard Poirot), the Panel finds that all elements -- indicator, averaging time, level and form -- have not been well-justified in the draft PA and are not consistent with current scientific evidence. Therefore, a second draft of the PA is needed that revisits these issues and provides sufficient supporting information for a reasonable range of alternatives to support formulation of advice by CASAC (if augmented with the appropriate expertise by reinstating the disbanded CASAC PM Review Panel) and the public, including the IPMRP.

***EPA-6. Chapters 3 to 5: What are the CASAC views regarding the areas for additional research identified in Chapters 3, 4 and 5? Are there additional areas that should be highlighted?***

The current review must be based upon existing information; however, there are several areas that could inform future reviews of the primary and secondary PM standards and help reduce some of the uncertainties associated with this process.

Future research needs include the following:

- Air quality monitoring and reporting for sub-daily and short-term levels of exposure for both near-roadway and more generic sites.
- Development of Federal Reference Methods for measurements of ultrafine particles and for black carbon.
- Development of an appropriate monitoring network for ultrafine particles and black carbon; the network should include near-roadway sites as well as other sites.
- To improve the scientific basis for the next review, EPA is urged to evaluate and expand the PM<sub>10-2.5</sub> network, along with speciation of PM<sub>10-2.5</sub> including multi-elements, major ions, carbon (including carbonate carbon), and bioaerosols.
- Characterize PM<sub>10-2.5</sub> in different health-relevant exposure environments (e.g., city center, suburban, roadside, agricultural, and rural areas) for mass, elements (including potential toxic species), carbonaceous materials (including selected organic compounds and carbonate), water-soluble ions, and bioaerosols (including endotoxins, 1,3 beta glucan, and total protein).
- More detailed monitoring for organic components of PM; there is also a need to develop less costly and more easily implemented ways of measuring the ambient levels of these components.
- Detailed examination of the distributions of short-term exposure levels over time.
- Research should continue to define in more detail the physiological bases for adverse health responses to PM and its components. Such research would help establish appropriate exposure averaging times for future consideration as well as indicate sub-clinical markers that could predict adverse health response. Particular attention needs to

be given to mechanisms that could explain relationships between PM exposures and neurological, metabolic, and autoimmune disease.

- Additional comparative toxicological studies designed to facilitate extrapolation from animal and cellular studies to humans.
- Alternative exposure metrics need to be explored in studies of health effects of PM. How important are peak exposures as opposed to average exposures in explaining observed health responses? This includes study of sub-daily exposure levels. What is the appropriate time average for peak exposures? Do current average measures adequately limit exposures to peak levels? The importance of relative changes in exposure in terms of risk reduction needs further research. How important are past exposures in explaining responses to current levels; indeed the correct question to ask is: what are the impacts of current exposures given past exposures? This is particularly important when health outcomes, such as cancer which develops over an extended period of time, and cross-sectional designs are considered. These designs compare exposures and health responses across geographic entities. Although there are changes in air quality over time, the relative ordering of air quality across geographic entities changes minimally. What is the latency of response? Tied to this is the issue of cumulative exposure, which should be examined.
- Better characterization of the performance of hybrid modeling approaches to estimate PM exposures over different averaging times, and evaluation of alternative modeling approaches.
- Additional health studies are needed of the effects of PM components on health. Greater attention needs to be given to organic components of PM and to trace metals. Additional focus on the impacts of near-road exposures are warranted. Studies are needed that further examine the role of PM from various sources on health responses.
- Appropriate epidemiological studies designed to look at the health impacts of ultrafine particle exposure are needed.
- Define efforts to better include the concept of pseudo-design values into epidemiological studies and controlled human studies.
- Greater consideration of the health impacts of the coarse fraction of PM, especially for asthmatic and respiratory responses.
- Research to quantify the acute and chronic health effects of particulate matter produced by combustion of biomass, including residential wood combustion and wildfire smoke.
- Studies regarding to what extent and how SOA from biogenic hydrocarbons are controllable (e.g., through effects of sulfate, nitrogen oxides on biogenic SOA formation).
- Health research tends to be focused on one pollutant at a time even when several pollutants are measured, but they are most often considered independently. Studies that facilitate the sorting out of response to the various components in a multi-pollutant study are needed. The potential impact of joint exposure to more than one pollutant is needed; this includes some examination of the importance of sequencing exposures to various pollutants. This research should also include further efforts to understand the impacts of differential exposure error.
- People spend more of their time in indoor environments. Indoor PM levels can be high in these environments. How important are these? If they are not as important, why?



What is the health impact of joint indoor and outdoor exposures? Are health responses to outdoor PM levels greater when indoor levels are high?

- The use of microenvironmental exposure modeling to account for infiltration of ambient particles to enclosed environments, and implications for explaining variability in concentration-response functions between cities should be explored.
- PM clearly impacts visibility, which can influence emotional well-being. Studies to examine this association are needed.
- Additional support is needed to enhance photo-based air quality visualization tools (for example to add additional urban areas and clouds to the WinHaze model). Support is also needed to conduct visibility preference studies, using consistent, best practices, over a wide range of urban and suburban areas throughout the country.
- The Panel notes that the recent emergence of newer causal methods for controlling for confounding may be appropriate for PM health effects modelling, and recommends future development of models designed to assess effect modification by PM co-pollutants and joint exposures to address this area of uncertainty.

### **Additional Consensus Statement: Draft Integrated Science Assessment**

In addition to responding to the charge questions on the draft Policy Assessment, the concerns of the Panel regarding the draft Integrated Science Assessment are summarized here.

In our December 10, 2018 letter to CASAC and the EPA docket for the draft Integrated Science Assessment (Frey et al., 2018), the Panel offered consensus advice on numerous issues related to the draft ISA. The failure of EPA to provide a second external review draft of the ISA compromises the credibility and integrity of the NAAQS review process. This is because there were many important scientific issues raised regarding the first external review draft that require revision and iteration prior to their application in risk and exposure assessment and prior to their interpretation in the policy assessment. Although the Panel found that the draft ISA was a comprehensive scientific document, the Panel identified numerous areas for which refinement or revision was needed as detailed in our December 10, 2018 letter to CASAC. These areas include low cost sensors, air quality, contrasts between PM<sub>2.5</sub> and UFP, coarse PM, PM components, onroad and near-road microenvironments, mixtures and copollutants, study selection, transparent application of the causal framework, more in-depth treatment of specific issues related to PM<sub>2.5</sub> and mortality, more explanation and possible reconsideration of the causal determination for short-term exposure to coarse PM and respiratory adverse effects, more explanation and possible reconsideration of the causal determination for long-term exposure to UFP and central nervous system effects, and reconsideration of the at-risk causal finding for populations with pre-existing cardiovascular or respiratory disease. Members of the IPMRP also provided extensive individual comments that were attached to the December 10, 2018 letter from the Panel.

In our March 27, 2019 letter to CASAC (Frey et al., 2019), the Panel noted that “the framework for causal determination, including terminology, and the overall plan for development of the ISA, was reviewed by CASAC in 2016.” The Panel noted that “the various considerations in developing causal determinations are explained in the Preamble to the ISAs and have been considered already in CASAC’s review of the Draft Integrated Review Plan.” The Panel further noted that “[w]hile there may be opportunities for EPA staff to improve the clarity and transparency of the explanations of the inferences it makes and the conclusions it draws, this is not a fundamental limitation of the underlying framework but rather a matter of routine scientific review and iteration to improve the clarity and transparency of the final document.”

Normally, in prior review cycles, there is a second external review draft of the ISA concurrent with a first review draft of the Risk and Exposure Assessment (REA). In this review cycle for PM, EPA has not produced a separate draft REA, but instead has subsumed the REA into the draft PA. Typically, in a normal review cycle, the draft PA would not be released until after EPA has finalized the ISA and completed a second draft of the REA. The typical sequence in a normal review cycle was intended to protect the science assessments from being commingled with the policy assessment, so that the scientific basis could be established irrespective of later policy interpretations. In the current review cycle, the fact that the ISA is not completed prior to external review of the draft PA provides EPA leadership with the opportunity to change the ISA to support pre-determined policy outcomes in the final PA. This is a completely unacceptable situation.

The draft PA has elected to retain the causality determination framework for health effects attributed to exposures of varying durations to particular indicators, and to retain the causality framework for at-risk populations. The Panel concurs.

The Panel expresses its concern regarding the footnote to Table 3-1, on page 3-18 of the draft Policy Assessment, to the effect that “we recognize that the final ISA will reflect the EPA’s consideration of CASAC advice and that, based on CASAC advice, some or all of these causality determinations could differ in the final ISA. The final PA will reflect these updates.” This footnote is inappropriate in a draft PA because the scientific issues should have been resolved prior to development of the draft PA. CASAC has already admitted, explicitly, that it is not qualified to offer these judgments, because it lacks the breadth, depth, and diversity of expertise for review of the PM NAAQS (see the April 11, 2019 letter from CASAC to the Administrator). Expert scientific judgment must be conditioned on appropriate domain knowledge (see Dr. H. Christopher Frey’s individual comments for more details) which is lacking in the CASAC.

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## Appendix C

**Individual Comments by  
Independent Particulate Matter Review Panel Members  
on the  
EPA’s Policy Assessment for the Review  
of the  
National Ambient Air Quality Standards  
for  
Particulate Matter  
(External Review Draft – September 2019)**

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## Dr. Peter Adams

### **EPA-2. Chapter 2 – PM Air Quality: To what extent does the CASAC find that the information in Chapter 2 is clearly presented and that it provides useful context for the review?**

SCQ-2.1 What are the Panel's views regarding whether the draft PA accurately reflects and communicates the air quality related information most relevant to its subsequent evidence-based assessment of the health and welfare effects studies, including uncertainties, as well as the development of the risk assessment for current and alternative standards? In particular, do the following sections accurately reflect and communicate current scientific understanding, including uncertainties, for: (a) relationships between annual and daily distributions of PM; (b) the review of hybrid modelling approaches used to estimate exposure in some studies and the risk assessment; and (c) information on background levels of various PM indicators?

Overall, I found that Chapter 2 was clearly presented, provided useful context for the review, and accurately summarized and communicated relevant knowledge of the atmospheric behavior of PM. In particular, I found that Figures 2-10 and 2-11 and associated discussion provided useful and relevant evidence about the relationship between annual and daily PM levels. Similarly, Section 2.3.3 provided a good overview of the strengths and weaknesses of hybrid modeling approaches for exposure assessment.

I note that information about ultrafine PM was very sparse in this report. I urge EPA to consider dedicating more resources to modeling, monitoring, and exposure assessment for ultrafine PM.

Regarding background levels of PM, this is a somewhat harder question. While useful information was presented, I noted a tendency to label some kinds of PM as natural and/or background when it might, in fact, be a mix of natural and anthropogenic. This includes wildfires and biogenic SOA. More detailed notes on this are below.

Page 2-3: Wildland fires are partly natural sources but partly anthropogenic as well, depending on the origin of the fire. This becomes relevant again in Section 2.4 on Background PM.

Page 2-3: Similarly, it is not straightforward to say whether biogenic SOA is natural or anthropogenic. The VOC precursor is natural (well, even this is debatable for any managed land). But there is a literature of work pointing out that biogenic SOA levels are higher due to human activity for at least two reasons: 1) ozone is enhanced by anthropogenic activities and is a key oxidant for many biogenic VOCs and 2) some SOA yield are NO<sub>x</sub>-dependent and most NO<sub>x</sub> is anthropogenic. Hence, separating natural from anthropogenic biogenic SOA is non-trivial. This becomes relevant again in Section 2.4 on Background PM.

Section 2.4.3: The text describes the measured organic matter at IMPROVE sites in the Southeast as an "upper bound" of natural biogenic aerosol, and it is indeed an upper bound. The fact that these IMPROVE sites have all



demonstrated significant decreases in organic matter concentrations strongly suggests that much of the organic matter is controllable. It strikes me as highly unlikely that additional emissions controls would not result in further decreases even in biogenic SOA for the reasons described above.

Otherwise, I present some more minor notes of statements that could be revised or clarified but do not substantially hinder the overall success of the document.

Page 2-9: "Anthropogenic SO<sub>2</sub> and NO<sub>x</sub> are the predominant precursor gases in the formation of secondary PM<sub>2.5</sub>, and ammonia also plays an important role in the formation of nitrate PM by neutralizing sulfuric acid and nitric acid."

I think it is wrong, or at least an over-simplification, to call SO<sub>2</sub> and NO<sub>x</sub> "predominant" and relegate ammonia and VOCs to supporting roles. In many US locations, there is more organics (mostly SOA) in PM<sub>2.5</sub> than sulfate. Hence, VOCs are important. Sulfate has declined in importance over the past 10-15 years – and in some locations has not been important for a while. NO<sub>x</sub>/nitrate are very important in some locations, really not important in others. The current text acknowledges an "important role" for ammonia, but by many measures, PM<sub>2.5</sub> concentrations are more sensitive to ammonia than NO<sub>x</sub> emissions.

Page 2-18: Section 2.2.5 mentions particle count measurements but does not elaborate to the same degree as the section does for other measurements (aetholometer, EC/OC).

Pages 2-21 and 2-22: The text gives a somewhat too simple view of PM<sub>2.5</sub> concentrations (highest in west, lower in east). Except for a few locations in the west (CA's central valley, LA, and others), the west is cleaner than the east. There are more people breathing air just below the annual-average NAAQS (i.e. in the 10-12 µg/m<sup>3</sup> range) in the east than in the west.

Page 2-29: "The draft ISA describes a two-peaked diurnal pattern in urban areas, with morning peaks attributed to rush-hour traffic and afternoon peaks attributed to a combination of rush hour traffic, decreasing atmospheric dilution, and nucleation (U.S. EPA, 2018, section 2.5.2.3, Figure 2-32)."

I cannot believe that nucleation has any impact on PM<sub>2.5</sub> mass concentrations. Rather, the draft probably means to say efficient oxidation in the afternoon of precursor gases, which condense (rather than nucleate) onto existing particles.

**EPA-5. Chapter 5 – Review of the Secondary Standards: What are the CASAC views on the approach described in Chapter 5 to considering the evidence for PM-related welfare effects in order to inform preliminary conclusions on the secondary standards? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current secondary PM standards?**

I found the approach and rationale EPA took in reaching the preliminary conclusions to be reasonable.

SCQ-5.1 To what extent does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the secondary PM standards? Are there additional policy-relevant questions that should be addressed?

I found the questions to be sufficient and relevant.

SCQ-5.2 What are the Panel's views of the draft PA evaluation of the currently available scientific evidence with respect to the welfare effects of PM. Does the assessment appropriately account for any new information related to factors that influence:

- a) Quantification of visibility impairment associated with PM<sub>2.5</sub> and examination of methods for characterizing visibility and its value to the public?
- b) The effects of PM<sub>2.5</sub> components on climate?
- c) The effects of fine and coarse particles on materials?

I found that the draft PA did a good job of summarizing the state of knowledge at the time of the last NAAQS review, now, and the new information that has become available in between. I found this to be the case for visibility and climate effects but note that I do not consider myself an expert on material damage.

SCQ-5.3 What are the Panel's views of the draft PA preliminary conclusion that the currently available scientific evidence does not call into question the protection afforded by the current secondary PM standards against PM welfare effects and that it is appropriate to consider retaining the current secondary PM standards without revision?

I found the draft PA preliminary conclusion to be appropriate and well supported.

Lastly, I note some minor issues that could be revised and clarified in the PA but do not substantially impair it from serving its purpose.

Page 5-5: "In addition, at the time of the proposal, the Administrator recognized that suitable equipment and performance-based verification procedures did not then exist for direct measurement of light extinction and could not be developed within the time frame of the review (77 FR 38980-38981, June 29, 2012)."

This statement confuses me since nephelometers and aetholometers exist and could do the job. This also seems to contradict statements made on the bottom of page 5-11 about available measurement methods.

Page 5-25: "The IPCC AR5, taking into account both model simulations and satellite observations, reports a radiative forcing from aerosol-radiation interactions (RFari) from anthropogenic PM of  $-0.35 \pm 0.5$  watts per square meter (Wm<sup>-2</sup>) (Boucher, 2013), which is slightly reduced compared to AR4."

Here “reduced” is confusing. The effect is reduced in absolute magnitude but increased from -0.5 to -0.35 W/m<sup>2</sup> from AR4 to AR5. This could be revised for clarification.

Page 5-26: “While research on PM-related effects on climate has expanded since the last review, there are still significant uncertainties associated with the accurate measurement of PM contributions to the direct and indirect effects of PM on climate.”

I think it’s more appropriate to say “accurate estimation” given the number of modeling studies involved.

Page 5-29: “Such uncertainties include those related to our understanding of: • The magnitude of PM radiative forcing and the portion of that associated with anthropogenic emissions; and,”

Although the term “radiative forcing” can sometimes be used slightly different ways, the most common and general definition is the difference in the Earth’s energy balance due to the presence (versus absence) of anthropogenic emissions. Hence, radiative forcing is, by definition, anthropogenic. In contrast, it’s common to say “radiative effect” when one means the net result of anthropogenic and natural aerosols. A similar statement is made on page 5-40 and should be remedied there.

## Dr. John L. Adgate

### **EPA-1. Chapter 1 – Introduction: To what extent does the CASAC find that the information in Chapter 1 is clearly presented and that it provides useful context for the review?**

The information in Chapter 1 of the draft Policy Assessment (PA) is clearly presented for the most part: it addresses the implications of the available scientific evidence and provides some useful context, including the purpose, legislative requirements, history, and key elements and case law related to the Clean Air Act that govern the development of NAAQS. The review leaves out elements of the recent policy changes, the functioning of the review process, and timeline of the review that are important parts of the peer review process for the PA and the documents that feed into it. The PA document would be strengthened if it provided a summary of the timeline of the overall review in contrast to past reviews, and stated whether important related documents, such as the draft Independent Science Assessment (ISA) and earlier planning documents (e.g., the REA), will be released in final peer reviewed form prior to the finalization of the PA. These documents were part of previous comprehensive reviews prior to the changes implemented by the Administrator in 2017 and 2018. Outlining those changes and their rationale would make section 1.4 of the PA complete and the overall timeline clearer.

### **EPA-3. Chapter 3 – Review of the Primary PM<sub>2.5</sub> Standards: What are the CASAC views on the approaches described in Chapter 3 to considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? What are the CASAC views regarding the rationales supporting the preliminary conclusions on the current and potential alternative primary PM<sub>2.5</sub> standards?**

Chapter 3 is well written and addresses the charge questions on the Primary PM<sub>2.5</sub> Standards, and summarize the policy-related issues and the most important weight of evidence findings identified in the draft Independent Science Review. Table 1 on page 3-18 is a useful summary, though footnote 15 implies that conclusions on the 3 “likely to be causal” endpoints may be reversed or disregarded in the final PA based on CASAC’s commentary on the validity of these determinations. Given that CASAC itself has asked for additional scientific expertise on particulate matter health studies, some CASAC members have called for a reinstatement of the PM subcommittee, and recent CASAC communications indicate lack of consensus on a number of scientific and science policy issues, the language of the footnote indicating that the PA would be finalized based on advice from CASAC seems imponderable.

#### **SCQ-3.1 Does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>2.5</sub> review? Are there additional policy-relevant questions that should be addressed?**

The policy questions posed in the chapter address the central questions on adequacy of the current annual and 24 hour PM<sub>2.5</sub> standards and related issues, such as what is known, not known, and key scientific issues and uncertainties. The chapter context

could be strengthened by noting health impacts on vulnerable and/or sensitive subpopulations,

Comments on Figures/Tables:

Many of the figures/tables do not stand alone as comprehensible units as they have undefined acronyms and in some cases incomplete titles or other descriptors that are not clear without extensive review of the text elsewhere in the document (e.g., “hybrid model” in Figure 3-8, “pseudo-design” in Figure 3-9). The document would be more readable if all graphics were at the high image quality.

Table 3-2 on controlled human exposure studies should include the number of exposed/unexposed individuals in each study.

**SCQ 3.4 What are the Panel’s views on the quantitative risk assessment for PM<sub>2.5</sub> including:**

In general this section is less clearly written than other parts of the PA. It is also jargon dense, often without defining key terms used multiple times and does not concisely summarize the key features and conclusions from the text and tables that make up Appendix C.

**f) The choice of health outcomes and studies selected for developing concentration-response functions for long and short-term effects?**

The choice of the three health outcomes presented in this section is not clearly articulated. Nonetheless, the studies selected for developing the C-R response functions are based on large well designed studies included in prior analyses and present epidemiological evidence for total mortality, ischemic heart disease mortality, and lung cancer mortality. The first two endpoints have more extensive evidence of causality per the summary in this review and were vetted in prior reviews, while the evidence for lung cancer mortality is less robust and the designation of “likely to be causal” based on judgment of the less robust findings related to individual study power and other factors, such as latency. Inclusion of this less robust endpoint that likely has greater uncertainty in its C-R function estimate provides an opportunity to assess the effect of using endpoints with weight of evidence determinations that are more uncertain.

**g) The selection criteria for the 47 urban areas and PM<sub>2.5</sub> air quality scenarios analyzed?**

The selection criteria for the 47 urban areas are based on availability of monitoring data and geographical diversity is reasonable given the range of health outcomes assessed in large studies and the observed differences in response in different locations inside the US. The third criteria, “PM<sub>2.5</sub> air quality concentrations” is unclear as the text describes the need for adjustment, but doesn’t clearly describe how these three criteria are assessed and or balanced in the process of decision-making. Nonetheless, this approach is likely broad enough to provide a representative risk assessment based on the population, though even a cursory glance of Figure 3-10 indicates that large parts of the central, northern, and western US were not included in the areas assessed. In the end the approach appears to be sufficiently broad and include areas with

large populations, so it will allow for examination of estimated effects below the existing standards as well as the examination of the shape of the C-R response curve for long and short-term health endpoints.

- h) The hybrid modeling approach used for quantifying exposure surrogates across an area and adjusting air quality for alternative standard levels, as supplemented by interpolation/extrapolation?**
- i) The characterization of variability and uncertainty in the risk assessment? &**
- j) The robustness and validity of the risk estimates?**

**[text below responds to both d and e]**

The text describing this process on 3-83 is fairly brief and points to Appendix C but does not present the key findings or conclusions in a comprehensible way. The goals of this analysis need to be more clearly stated, and text on the rationale for the different risk modelling approaches articulated up front. While the general approach of upper bound estimates and use of sensitivity analysis are justified, as is the use of qualitative assessment, the process of selecting concentration-response functions, how the sensitivity analysis will be conducted and the range of plausible values is incompletely described in the body of the PA and thus the quality of this analysis is unclear. The subsequent summary of associated mortality under alternative standards and exposure reduction scenarios has results in the range that would be expected, though the process is hard to follow and key features of the appendix tables cited are not well described. In the end the lack of clarity in the approach here reduces confidence in the validity of the results presented.

**SCQ-3.5 What are the Panel's views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?**

Overall, the preliminary conclusion that the weight of evidence from various study types and analyses presented support questioning whether the current standards are sufficiently protective of public health. The overall strength of evidence from a longstanding body of evidence has been further bolstered with new studies from a range of disciplines. This strong evidence on mortality and morbidity endpoints, coupled with emerging evidence for less extensively studied health endpoints, such as nervous system effects, and observation of health effects at or below current standards are scientifically credible. Furthermore, since it is likely that some populations are at increased risk due to geographic location, proximity to sources, or population characteristics (such as age or prior disease status) that increase their susceptibility, the conclusion that the existing standards may not provide an adequate margin of safety is warranted.

**Mr. George Allen**

General comment. It remains unclear how EPA will address the CASAC's April 11, 2019 comments on the draft PM ISA in the final ISA. These comments assume there will not be any substantial changes to the causal findings as presented in the draft ISA that would result in how the draft ISA findings are used in this draft PA.

## Chapter 2, Air Quality.

### Hybrid Modeling.

In the context of this review of health based standards, the air quality section on hybrid modeling approaches to PM<sub>2.5</sub> is the most important, since this is the area where substantial improvements in characterizing ambient PM<sub>2.5</sub> exposures over large areas have been made since the last PM NAAQS review. The performance of four different approaches are summarized, with the Bayesian downscaler 12 km model and the machine learning 1 km model having better overall performance. All models had degraded performance at low PM concentrations and in rural areas, although for use in health effect studies, uncertainties in annual average concentrations below ~ 6 to 7 µg/m<sup>3</sup> are less important.

Of particular relevance for this review is the performance of the machine learning approach for daily PM<sub>2.5</sub> with a 1 km grid used by Di et al. from the Harvard-Chan School of Public Health, since this was used in the pair of Di et al. chronic and acute mortality papers from 2017. The ability to predict PM<sub>2.5</sub> at the 1 km scale provides improved estimates in urban areas, which is important since much of the US population is urban and PM<sub>2.5</sub> tends to be higher in urban areas.

### Near-road PM.

A useful summary of the increase in PM<sub>2.5</sub> at near-road sites is given, showing an average increment over urban background of less than 1 µg/m<sup>3</sup>. Briefly noted in section 2.2.5 are other particle measurements at some of the near-road network sites, including black carbon (BC) and ultra-fine particle concentration measurements. It is worth noting that although BC is being measured at many near-road sites, it is not required to be reported to AQS under current regulations, and some agencies still do not report it.

### Re-purposing the near-road network from NO<sub>2</sub> to PM.

There are approximately 75 near-road monitoring sites that were originally deployed with NO<sub>2</sub> as the primary pollutant of interest. That turned out to be unnecessary, since there are no near-road sites even close to being out of compliance with the NO<sub>2</sub> NAAQS. Even exceedances of the 1-h 100 ppb standard are unusual. This doesn't mean there is no issue with near-road pollution health effects though, with particles being the most likely driver of the observed increase in several different health endpoints including cardio-vascular effects. EPA should reconsider how to use the existing near-road monitoring network infrastructure in the context of characterizing a range of on-line particle metrics at a subset of near-road sites, including UFP, lung-deposited surface area (using charge-based measurements), black carbon and total aerosol carbon (and OC by difference), and speciation of tire and brake wear emissions (including iron and copper) using 1 to 2-hour automated XRF methods. Coarse PM is elevated in the near-road environment and should also be measured using continuous methods. To quantify the increase of these pollutants relative to urban background, matching measurements could be made at NCore sites in the same urban area, preferably within a few km of the near-road site.

Relationship between annual and daily PM<sub>2.5</sub> design values.

This is an important analysis, given that EPA continues to recommend that the daily PM<sub>2.5</sub> NAAQS not be changed and continued to be used only as a backstop, with the annual PM<sub>2.5</sub> NAAQS as the primary control mechanism. While it is true that most sites that are in compliance with the current annual NAAQS of 12 have daily design values less than 35, there is a subset of sites where the daily NAAQS DV is greater than 35 and the annual is less than 12. A common driver of this situation is winter woodsmoke from residential space heating, where elevated levels of PM<sub>2.5</sub> occur only during the heating season. An extreme example of this scenario is the North Pole (Fairbanks) AK valley monitoring site, in severe non-compliance for PM<sub>2.5</sub> because of winter woodsmoke. The ratio of the 2016-2018 daily DV to annual average is 5.1, substantially larger than the 35/12 ratio of 2.9. For the annual NAAQS to provide equivalent protection of the daily NAAQS at this location, it would have to be 7 µg/m<sup>3</sup>. If the annual PM NAAQS is reduced, the daily should not be left unchanged unless an annual NAAQS of less than 8 µg/m<sup>3</sup> is chosen.

Issues with FRM and FEM PM<sub>2.5</sub> monitor comparisons.

Monitoring agencies continue to struggle with getting their continuous FEM PM<sub>2.5</sub> monitor performance within acceptable levels for them to be used to demonstrate compliance with the PM<sub>2.5</sub> NAAQS. This problem goes back to how the FRM is run for FEM testing requirements; it is well known that FRM filters can lose up to 10% of their non-water mass over the 177 hours allowed before post-sampling weighings are done. Dirk Felton described this issue in 2009 in an AWMA Environmental Manager article “Is It Time to Upgrade the PM<sub>2.5</sub> Federal Reference Method?”, available at <http://pubs.awma.org/gsearch/em/2009/2/felton.pdf>. From a programmatic perspective it is unlikely that the FRM or FEM certification process will be changed to resolve this performance difference. EPA could allow instrument specific correction factors to reduce the bias relative to the FRM of most of the more than 900 FEM sites to the point where current data, and to some extent historical data, would be of sufficient quality for comparison to the 24-hour NAAQS. This becomes important for consideration of a change to the averaging interval of the daily standard to a rolling 24-hour average, similar to how the ozone NAAQS is an 8-hour daily maximum value.

Background PM.

This section covers sources of background (non-anthropogenic, domestic) PM well, with estimates of background PM from 0.5 to 3 µg/m<sup>3</sup>, with the upper end of that range probably driven by secondary organic aerosol (SOA). Other than wind-blown dust, SOA is the largest source, especially in the southeast from the reaction of photochemical oxidants with biogenic hydrocarbons (isoprene, terpenes). This document treats all of this source as natural, but since some of the photo-oxidant load is anthropogenic, perhaps some of the SOA should be considered that as well. Smoke from wildfires, especially in western states, could be considered anthropogenic to some extent, since human activity accounts for some portion of wildfire events. This could include climate change-related effects of drier and hotter weather, as well as ignition events from power transmission lines. The 2018 Camp fire in California is a good example of this kind of event.

#### Chapter 3, Section 3.4.2, Potential PM<sub>2.5</sub> alternative standards

There is little new information since the last review to support serious consideration of changes to the indicator, form, or averaging times for the annual and daily NAAQS. There is some



discussion of UFP as an additional indicator since it is described as Likely to be causal for long-term nervous system effects, but it is unclear if this association is independent of PM<sub>2.5</sub> which is also Likely to be causal. As noted in the draft PA, there is a very large body of research showing PM<sub>2.5</sub> mortality effects since the last PM review. The most robust work is the pair of chronic and acute studies of the Medicare population by Di et al. from the Harvard-Chan School of Public Health. In addition to having a 61 million person cohort with a median follow-up of 7 years and hybrid-modeled daily PM<sub>2.5</sub> 1x1 km exposure estimates for the entire continental US, the combination of chronic and acute mortality analysis on the same data set provides increased confidence that the analytical methods used are robust, since potential confounders for the chronic and acute analysis are different. These studies justify serious consideration of annual PM<sub>2.5</sub> values down to 8 µg/m<sup>3</sup>. While these studies are an important part of EPA's analysis, the agency is still using the "study area" approach for the REA. When you have robust exposure and mortality estimates for the entire country, this approach seems too limited.

The draft PA looks at a range of annual PM<sub>2.5</sub> between 8 and less than 12 (e.g., 11), and performs risk assessments at 11, 10, and 9 µg/m<sup>3</sup> (Table 3-7, page 3-88). Table 3-8 presents % risk reduction for these concentrations relative to 12. Since the CR curve is assumed to be linear within this range, the reductions are not large: 21 to 27% across all table categories. The Di and Pope all-cause mortality estimates for the 47 urban study areas are ~ 50,000/year - a very large number from a public health perspective. Reducing this by ~ 25% is still a very large number, and does not reflect mortality on a national scale; the 47 urban study areas represent about 1/3 of the total population (Table C-2).

The risk analysis mostly ignores or de-emphasizes study data below 8 to 9 µg/m<sup>3</sup>, saying there is insufficient information from studies at those low concentrations. However, figure 3-8 shows that average pm<sub>2.5</sub> for 25% of the Di et al. chronic mortality study population was below 7 µg/m<sup>3</sup>. This represents 115 million person-years of follow-up, a very large sample size that results in relatively robust mortality estimates even at levels below 7 µg/m<sup>3</sup> (see Di et al., NEJM 2017 Figure 3a). There is a very large population with current annual PM exposures less than 8 µg/m<sup>3</sup>, and while the effect is lower with lower concentrations and there is a suggestion of flattening of the CR curve below 7 µg/m<sup>3</sup>, the overall mortality is large in this group because of its size. This issues is not clearly addressed in the draft PA.

Figure 2 of the Di et al. 2017 NEJM chronic mortality study presents another measure of concern: the three times higher risk for African Americans compared to the general population.

This is not addressed in the risk assessment. If standards are set for what we think is appropriate for the general population, the 13% of the over 65 population that is black will be at substantially elevated risk relative to the general population.

#### Daily PM2.5 NAAQS.

There is no reasonable rationale to leave the daily PM2.5 NAAQS unchanged if the annual is reduced to 10 µg/m<sup>3</sup> or lower. Yes, it is appropriate to have the annual NAAQS be the primary control, but in addition to providing protection for short-term sub-daily peak exposures, one reason to keep the daily NAAQS at least somewhat relevant is that EPA's PM2.5 health messaging (AQI) is based only on the daily standard. Other than for wildfire events, at 35 µg/m<sup>3</sup> health messaging is almost never more than yellow/moderate. That messaging communicates little to no risk at concentrations that EPA says causes more than 50,000 premature deaths annually. Health messaging should not excessively discourage exercise, and as long as PM2.5 health messaging doesn't routinely communicate code orange (unhealthy/sensitive groups, at the level of the daily standard) or red (unhealthy, substantially above the daily standard), this should not be an issue.

Typo: Thurston 2015 in many Chapter 3 tables should be 2016.

#### Black Carbon (BC) health effects.

The 2018 Draft Integrated Science Assessment for PM mentions BC in the context of both short-term respiratory and cardiovascular effects. It is not mentioned in any of the long-term exposure categories, and unlike UFP for the first time, does not rise to the level of inclusion in any of the tables of causality. This is surprising since there is a growing body of literature that suggests BC is a good indicator of traffic-related air pollution (TRAP) health effects, if not causal of the cardio-vascular health effects observed in the near-road environment. BC can serve as a delivery vehicle for semi-volatile components of mobile-source exhaust since it is small enough to penetrate deep into the lung. BC particles can have a coating of fresh semi-volatile organic carbon material on their surface. They have a large surface area relative to their mass since the size of fresh tailpipe BC [~ 0.25 µm] is about where surface area distributions peak. A partial list of literature on BC health effects since the 2009 ISA is included below; none of these are included in the 2018 draft PM ISA. BC should be included in future tables of causality, since it would seem to be at least "somewhat suggestive" of having a causal health effect. Vermeulen et al. (2013) is included here since it used EC (a similar metric to BC) as the indicator for diesel engine exhaust, and points to a large body of literature linking cancer to EC or BC long-term exposures.

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## Dr. John Balmes

### Charge Question SCQ-3.1

*Does the Panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>2.5</sub> review? Are there additional policy-relevant questions that should be addressed?*

In general, the questions posed in the chapter capture most of the policy-relevant issues. One area that deserves more attention is the relatively greater exposure to PM<sub>2.5</sub> of communities of color and low socioeconomic status (SES) for which there is considerable evidence. These communities also tend to have greater vulnerability to adverse health effects of PM<sub>2.5</sub> exposure. The chapter briefly alludes to the greater exposure and vulnerability of poor people of color when spatial averaging is discussed, but the need to protect the health of this population deserves greater attention in the draft PA.

### Charge Question SCQ-3.2

*What are the Panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?*

The evidence-based approach to whether the current and alternative PM<sub>2.5</sub> standards protect public health using the air quality distributions of the epidemiological studies that demonstrate associations between exposures to PM<sub>2.5</sub> and adverse health effects is appropriate. The second approach using “pseudo-design values” to determine whether PM<sub>2.5</sub> concentrations in epidemiological study areas would have exceeded the current or alternative standards also adds to the assessment.

The description of the risk-based approach is more difficult to follow, especially regarding the adjustments that were made for areas “requiring either a downward adjustment to air quality or a relatively modest upward adjustment.” The method by which exposure reductions based on a hybrid approach using both measured concentrations and modeled estimates are developed both for the current and alternative standards is again somewhat difficult to follow.

The evidence-based approach deserves more weight, but the fact that the risk-based approach produces similar information is reassuring.

### Charge Question SCQ-3.3

*What are the Panel's views on the evidence-based approach, including:*

*a) The emphasis on health outcomes for which the draft ISA causality determinations are “causal” or “likely causal”?*

The emphasis on health outcomes that the draft ISA identified as “causal” or “likely causal” is appropriate, although the lack of treatment of respiratory outcomes and long-term exposures with the risk-based approach is disappointing.

*b) The identification of potential at-risk populations?*

Again, people of color and low SES should also be identified as a potential at-risk population.

*c) Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?*

European multi-city epidemiological studies should also be considered.

*d) Characterizing air quality in these key studies using two approaches: the overall mean and 25th/75th percentiles of the distribution and the “pseudo design value” reflecting a monitor with the highest levels in an area?*

Mean PM<sub>2.5</sub> concentration may not be the best way to characterize the exposure of the populations in epidemiological studies that demonstrate associations with adverse health effects. Some of the statements about pseudo-design values are hard to understand such as “For studies with 25th percentiles  $\leq 12.0 \mu\text{g}/\text{m}^3$ , at least 25% of the study area population lived in locations likely to have met the current annual standard over the study period (i.e., in at least 25% of health events occurred in such locations”. How do we know this?

*e) The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principal means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*

The argument for the use of an annual standard as the primary approach to protecting public health is logical and well-stated. That said, high short-term exposures to PM<sub>2.5</sub> from catastrophic wildfires remain a major driver of health impacts even if these are not regulated by EPA.

*f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?*

These conclusions are appropriate based on the review of the health effects literature in the draft ISA.

*g) Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?*

While the discussion of Chapter 3 accurately reflects the currently available health effects evidence, communication of important uncertainties, such as the impacts of high peak sub-24-hour exposures, is not always clear. High sub-24-hour peak exposures are increasingly occurring as a result of wildfires in the Mountain West.

**Charge Question SCG-3.5**

*What are the Panel’s views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?*

The preliminary conclusion that the current may not be adequate to protect the public health with a sufficient margin of safety is reasonable given the evidence reviewed in the draft ISA.

## Dr. Kevin J. Boyle

Here, I refer to the charge questions for Chapter 5 of the report.

PA-5. Chapter 5 – Review of the Secondary Standards: What are the CASAC views on the approach described in Chapter 5 to considering the evidence for PM-related welfare effects in order to inform preliminary conclusions on the secondary standards? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current secondary PM standards?

SCQ-5.1 To what extent does the panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the secondary PM standards? Are there additional policy-relevant questions that should be addressed?

**Comment:** I think that it is good that additional attention was given to urban areas where the largest share of the populace resides without overlooking rural residents (p. 5-14, lines 1-6) Consideration of regional variation is also important (p. 5-14, 5-15).

However, there are important missing components to adequately consider public welfare that I outline below.

SCQ-5.2 What are the Panel's views of the draft PA evaluation of the currently available scientific evidence with respect to the welfare effects of PM. Does the assessment appropriately account for any new information related to factors that influence:

- a) Quantification of visibility impairment associated with PM<sub>2.5</sub> and examination of methods for characterizing visibility and its value to the public?

**Comment:** The use of “*acceptable*” visibility is a fundamentally flawed policy concept (p.15, line 25 – p. 17, line 9). What is acceptable in an urban area with a certain baseline visibility may not be acceptable in a rural area with a higher baseline of visibility. This is not just a dichotomy between urban and rural residents. Urban residents may expect greater visibility when they travel to a rural area for vacation, and rural residents may consider urban visibility a forgone condition. An additional question is whether the visibility standard should be higher in some locations such as already the case in Class I visibility areas, national parks and wilderness areas.

The more concerning element is that while people may rate a certain level of visibility as acceptable, this does not imply that they would not realize a welfare gain from further improvements in visibility (Boyle et al., 2016; Haider et al., 2019; Yao, 2019). Compromised visibility can also affect property values (Walls, Kousky and Chu, 2015). In short, the question is never posed or answered to consider if there are net public benefits, improved welfare, for enhancing visibility beyond the acceptable level. Further, the acceptability studies were focus groups with small numbers of participants.

- Ely et al. (1991) conducted 17 focus groups of members of civic organizations in Denver, CO for a total of 214 participants (about 12-13 people per group).
- BBC Consulting (2002) conducted 27 focus groups in Phoenix, AZ for a total of 385 participants ( about 14 people per group).



- Pryor (1996) conducted four classroom exercises in British Columbia, CAN with 180 university students (about 45 students per class).
- Abt (2001) conducted a single focus group in Washington< DC with nine participants.

The Ely and BBC studies represent initial research that would be conducted at the beginning of a well-designed national preference study with one exception. The focus groups would be conducted at several locations around the U.S., not in single cities. The Pryor study presents an interesting investigation to learn about preferences for visibility, but is not indicative of national preferences in the U.S. Finally, the Abt study represents the first step in study design from which no firm policy implications could be drawn. Johnston et al. (2017) discuss best practices in the conduct of an economic preference study to evaluate public welfare gains and losses and the use of focus groups in the design of such studies. The American Association for Public Opinion Research's Best practices for Survey Research include the recommendation that "(a)ll questions should be pretested to ensure that questions are understood by respondents, can be properly administered by interviewers or rendered by web survey software and do not adversely affect survey cooperation" (<https://www.aapor.org/Standards-Ethics/Best-Practices.aspx#best6>, accessed September 23, 2019). The conduct of focus groups is a key step in this process to learn how best to present visibility images and query subjects about visibility in the implementation of a national visibility preference study. Thus, the above studies present evidence of the importance of visibility but do not present enough information to support national policy decisions.

The report states that the "... preliminary conclusions for the Administrator's consideration is that it is appropriate to consider retaining the current secondary PM standards, without revision. In so concluding, we recognize, as noted above, that the final decision on this review of the secondary PM standards to be made by the Administrator is largely a public welfare judgment, based on his judgment as to the requisite protection of the public welfare from any known or anticipated adverse effects." (p. 39, lines 21-26) This conclusion is based on flawed logic because an implicit premise of the report is that there are no societal benefits beyond what some small and incomplete studies found as acceptable.

- b) The effects of PM<sub>2.5</sub> components on climate?

**Comment:** The report concludes that "(w)hile evidence in this review suggests that PM influenced temperature trends across the southern and eastern U.S. in the 20th 26 century, uncertainties continue to exist and further research is needed to better characterize the effects of PM on regional climate in the U.S." (p. 28, lines 25-28). It seems questionable to me to treat ecological effects and climate separately, which has been done by partitioning ecological impacts to a separate assessment. While this is not my area of expertise, it seems logical to ask if induced changes in climate over time will have ecological impacts that are not observed today.

- c) The effects of fine and coarse particles on materials?

**Comment:** The report concludes that “(w)hile some new evidence is available with 21 respect to PM-attributable materials effects, the data are insufficient to conduct quantitative analyses for PM effects on materials in the current review” (p. 5-35, line 20-22). The report is unclear on what literature was reviewed and there is evidence outside of the U.S. on the cost of soiling from air pollution (e.g., Besson, 2017; Grøntoft, 2019

SCQ-5.3 What are the Panel’s views of the draft PA preliminary conclusion that the currently available scientific evidence does not call into question the protection afforded by the current secondary PM standards against PM welfare effects and that it is appropriate to consider retaining the current secondary PM standards without revision?

**Comment:** I have several major concerns.

First, the framing of the policy from a welfare perspective using “acceptable”, by default, leads to the conclusion that no further protection is required. From a welfare perspective, the question is never posed to ask if welfare would be enhanced if protection was increased.

Second, given the uncertainties in the current state of knowledge the question is never posed to inquire if further protection is warranted until the uncertainties are resolved. The “what if nothing is done” question is never explored in any substantial manner to explore how large or small the consequences might be from holding the current standard.

Finally, in addition to advocating for a “better characterization” of the scientific knowledge, it would be appropriate to recommend a precautionary principle in setting policy until the visibility impacts and resulting welfare impacts are better understood (Kiebel et al., 2001). A safe minimum standard would call greater emphasis on protection of the environment, visibility here, so long as the social costs of doing so are not unreasonable (Bishop, 1978).

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## Dr. Judith Chow

### **EPA-2. Chapter 2 – PM Air Quality: To what extent does the CASAC find that the information in Chapter 2 is clearly presented and that it provides useful context for the review?**

SCQ-2.1 *What are the Panel's views regarding whether the draft Policy Assessment accurately reflects and communicates the air quality related information most relevant to its subsequent evidence-based assessment of the health and welfare effects studies, including uncertainties, as well as the development of the risk assessment for current and alternative standards? In particular, do the following sections accurately reflect and communicate current scientific understanding, including uncertainties, for: (a) relationships between annual and daily distributions of PM; (b) the review of hybrid modelling approaches used to estimate exposure in some studies and the risk assessment; and (c) information on background level of various measures of PM?*

Chapter 2 documents particulate matter (PM) emission sources, ambient monitoring methods and networks, as well as ambient air urban and non-urban PM concentrations. The chapter provides useful information, but several key areas deserve additional discussion including: 1) clarification of discrepancies in source types and percent contributions to gaseous precursors (i.e., SO<sub>2</sub>, NO<sub>x</sub>, NH<sub>3</sub>, and VOCs) and primary PM emissions; 2) documentation of the zones of representation of ambient monitoring sites for PM exposure assessments; 3) specification of the relationship between annual average and 98<sup>th</sup> percentile 24-hour PM<sub>2.5</sub> concentrations; and 4) exclusion of exceptional events in the PM<sub>10</sub> analysis.

- **Sources of PM Emissions (Section 2.1.1)**

Total PM<sub>2.5</sub> emissions are estimated at ~5.4 million tons/year (similar to the <5400 Ktons/year in the draft ISA with different units), but the aggregation of the seven source types in the draft PA (U.S. EPA, 2019) varies from that in the draft ISA (U.S. EPA, 2018a); both are based on the 2014 National Emissions Inventory (NEI, U.S. EPA, 2018b). Figure 2-2 (page 2-5) shows that the “Dust” source (including agriculture, construction, and road dust) and “Agriculture” (tilling) source each account for 18% of the total PM<sub>2.5</sub> emissions in the PA, which differs from the 13% “Unpaved Road Dust” and 19% “Agriculture- Crops & Livestock Dust”) emissions in the ISA. As agricultural tilling results in suspended PM dust, it should be part of the agricultural dust. The rationale to assign agricultural dust to the “Dust” source and agricultural tilling to the

“Agriculture” source needs to be explained.

Aggregation of different dust types or subtypes should be documented. Separation of “Dust” source emissions into paved and unpaved road dust and construction dust provides insight on the magnitude of suspended PM for each source subtype. This information is useful to evaluate source contributions by receptor modeling source apportionment and has been applied in the development of State Implementation Plans (SIPs).

Table 1 compares the percent contributions of seven source types between the draft PA and ISA for both annual PM<sub>2.5</sub> and PM<sub>10</sub> emissions. It shows the inconsistency in definition of source types and source subtypes between the PA and ISA. Similar discrepancies are found for the percent distribution of PM<sub>10</sub> emissions. Given that ~75% of the PM<sub>10</sub> emissions are attributed to “Dust” and “Agriculture” sources, it would be helpful to illustrate the source subtype contributions.

As PM<sub>10</sub> consists of PM<sub>2.5</sub>, the percent distribution of major emission sources to PM<sub>10-2.5</sub> should be given to provide some perspective on major source contributions to the coarse particle size fraction. It should also be noted that fugitive dust emission estimates are highly inaccurate and do not agree with source apportionment contributions at receptors (Watson and Chow, 2000). Emissions of precursor gases (i.e., SO<sub>2</sub>, NO<sub>x</sub>, NH<sub>3</sub>, and VOCs) also differ between the draft PA and ISA. For SO<sub>2</sub>, the 79% “Stationary Fuel Combustion” source in Figure 2-5a (page 2-10) is 6% higher than the 73% “Fuel Combustion” source (sum of Electric Generation and Industrial Boilers in Figure 2-4 [page 2-15] of the draft ISA); for NO<sub>x</sub>, the 58% “Mobile” source in Figure 2-5b is 4% higher than the 54% in the draft ISA (Figure 2-4b); and for NH<sub>3</sub>, the 80% “Agriculture” source (Figure 2-5c) is 22% higher than the 58% “Agriculture- Livestock Waste” source in the draft ISA (Figure 2-4c).

The most confusing discrepancies concern VOC emissions. The naming convention changes from “VOC” in the ISA to “Anthropogenic VOCs” in the PA. Both documents report annual average VOC emissions of 17 million tons per year (page 2-9 of draft PA and page 2-13 of draft ISA). Figure 2-5d of the PA attributes 24% of VOC to “Mobile” sources, this is four times higher than the 6% in the ISA (Figure 2-4d). The 71% of VOCs attributed to the “Biogenics-Vegetation and Soil” source type in the draft ISA is not included in the draft PA. Discrepancies between the two EPA reports need to be resolved.

Since these emission estimates serve as input to air quality models, consistent source types and emission estimates should be used. Reasons for different percent contributions of precursor gases and PM emissions, based on the same 2014 NEI, should be clarified.

- **Ambient PM Monitoring Methods and Networks (Section 2.2)**

Discussions of the spatial scales and monitors that characterize mobile and stationary source emissions (pages 2-12 and 2-13) are not consistent with the community monitoring zones (CMZ) defined by the U.S. EPA (1998) network design document. Zones of representation are defined as: microscale (<100 m), middle scale (~100-500 m), neighborhood scale (0.5-4 km), and urban scale (4-50 km) (40 CFR, Part 58, Appendix D). The statement for PM<sub>10</sub> monitoring that “...the network design criteria emphasize monitoring at middle and neighborhood scales to effectively characterize the emissions from both mobile and stationary sources...” from pages 2-12 and 2-13 is misleading as most of the PM<sub>10</sub> sites represent urban-scale community exposures. Only the near-road PM<sub>2.5</sub> or PM<sub>10</sub> sites can represent micro- and middle-scale monitoring.

The zone of representation for each monitor is important for exposure assessment and epidemiological studies that use data from compliance monitoring stations. Emission source zones of influence and receptor site zones of representation need to be defined for exposure assessment.

It appears that network-wide annual PM<sub>2.5</sub> concentrations have been reduced from 8.6 µg/m<sup>3</sup> during 2013-2015 (Table 2-4, pages 2-48 of ISA) to 8.0 µg/m<sup>3</sup> during 2015-2017 (page 2-24 of PA). Apparently, PM<sub>2.5</sub> concentrations have continuously declined nationwide with a ~30% reduction since 2000. It would be helpful to provide statistics on the number of sites included in each concentration bracket for the annual and 98<sup>th</sup> percentile 24-hour PM<sub>2.5</sub> concentrations in Figure 2-8 (page 2-23), especially for locations with annual averages between 8-10 µg/m<sup>3</sup> and 10-12 µg/m<sup>3</sup>.

Not much information is given to illustrate relationships between annual and daily PM<sub>2.5</sub> distributions. It is not clear why most sites exhibit high correlation coefficients between the trends in annual average PM<sub>2.5</sub> concentrations and trends in 98<sup>th</sup> percentile of 24-hour PM<sub>2.5</sub> concentrations at individual sites (Figure 2-10, page 2-25). The implications of these high correlations, especially for eastern U.S. and in coastal California sites, need to be explained. The 24 hour PM<sub>10</sub> NAAQS is 150 µg/m<sup>3</sup>, not to be exceeded more than once per year averaged over three years. However, only the average second highest 24-hour PM<sub>10</sub> concentrations during 2015-2017 (Figure 2-16, page 2-33) and 2000-2017 national trends (Figure 2-17, page 2-34) are presented. As many western sites exceeded the 150 µg/m<sup>3</sup> PM<sub>10</sub> NAAQS, days with exceptional events should be excluded in these presentations to provide a better perspective of potential areas with elevated PM<sub>10</sub> concentrations. As elevated PM<sub>10</sub> concentrations occur episodically (e.g., wildfires and dust storms), a summary of PM<sub>10</sub> levels on standard exceedance days should be given. Prolonged biomass burning can result in adverse health effects, sampling periods, and locations with elevated PM<sub>10</sub> concentrations should be specified. Although it appears that the majority of the PM<sub>10</sub> sites showed levels <75 µg/m<sup>3</sup> during 2015-2017, maximum (instead of second highest) 24-hour PM<sub>10</sub> concentrations should be given to provide information on sites and locations with potential exceedances of 24-hour PM<sub>10</sub> NAAQS over the three year period.

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Table 1  
Comparison of percent source type contributions to total PM<sub>2.5</sub> and PM<sub>10</sub> emissions between draft PA<sup>a</sup> and ISA<sup>b</sup>

Total PM<sub>2.5</sub> Emissions (5.4 million tons/year)

<b>Source Type</b>	<b>Draft PA (U.S. EPA 2019)<sup>a</sup></b>	<b>Source Type</b>	<b>Draft ISA (U.S. EPA 2018)<sup>b</sup></b>	<b>Difference (PA minus ISA)</b>
Fires	32%	Wildfires	17%	--
		Prescribed Fires	15%	--
Dust	18%	Unpaved Road Dust	13%	+5%
		Agriculture- Crops & Livestock		
Agriculture (Tilling)	18%	Dust	19%	-1%
Stationary Fuel				
Combustion	14%	Fuel Comb- Residential Wood	6%	+8%
Industrial Processes	5%		0%	+5%
Mobile Sources	7%		0%	+7%
	0%	Waste Disposal	4%	-4%
Misc.	6%	Other	26%	-20%

Total PM<sub>10</sub> Emissions (13 million tons/year)

<b>Source Type</b>	<b>Draft PA (U.S. EPA 2019)<sup>a</sup></b>	<b>Source Type</b>	<b>Draft ISA (U.S. EPA 2018)<sup>b</sup></b>	<b>Difference (PA minus ISA)</b>
Fires	11%	Wildfires	6%	--
		Prescribed Fires	5%	--
Dust	47%	Unpaved Road Dust	39%	-8%
	0%	Paved Road Dust	5%	-5%
Agriculture (Tilling)	28%	Agriculture- Crops & Livestock Dust	30%	+2%
Stationary Fuel Combustion	5%	Fuel Comb- Residential Wood	0%	+5%
Industrial Processes	4%		0%	+4%
Mobile Sources	3%		0%	+3%
Misc.	2%	Other	15%	-13%

<sup>a</sup>U.S. EPA (2019) Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft. U.S. Environmental Protection Agency, Research Triangle Park, NC, USA; based on Figures 2-2 and 2-3.

<sup>b</sup>U.S. EPA (2018) Integrated Science Assessment for Particulate Matter, External Review Draft. U.S. Environmental Protection Agency, Research Triangle Park, NC, USA; based on Figures 2-2 and 2-6



**EPA-4. Chapter 4 – Review of the Primary PM<sub>10</sub> Standard: What are the CASAC views on the approach described in Chapter 4 to considering the PM<sub>10-2.5</sub> health effects evidence in order to inform preliminary conclusions on the primary PM<sub>10</sub> standard? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current primary PM<sub>10</sub> standard?**

SCQ-4.01 *To what extent does the Panel find that the key policy questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>10</sub> NAAQS review? Are there additional policy-relevant questions that should be addressed?*

Little information is given in Chapters 2 and 4 to evaluate the adequacy of the 24-hour PM<sub>10</sub> NAAQS to protect public health and welfare. Little progress has been made since the previous ISA (U.S. EPA, 2009). Equal weight and effort should be dedicated to each criteria pollutant in evaluating the NAAQS. However, different approaches are used for PM<sub>10-2.5</sub> as compared to PM<sub>2.5</sub> for causality determination. It is not clear why the draft PA did not include evaluations of PM<sub>10</sub> distributions in locations with epidemiological studies; comparison of experimental exposures with ambient air quality; or quantitative assessments of PM<sub>10-2.5</sub> health risks. As PM<sub>10</sub> includes PM<sub>2.5</sub>, the key policy questions should reflect the policy-relevant issue for PM<sub>10-2.5</sub> that highlights different properties in the PM<sub>2.5</sub> and PM<sub>10-2.5</sub> size fractions.

SCQ-4.02 *What are the Panel's views of the draft PA assessment of the currently available scientific evidence regarding the health effects associated with exposures to thoracic coarse particles, PM<sub>10-2.5</sub>?*

Although only a few new short-term PM<sub>10-2.5</sub> exposure studies were presented in the draft ISA (Table 11-9 on pages 11-100 to 101), these demonstrate consistent positive associations with total (nonaccidental) mortality (U.S. EPA, 2018). The long-term exposure to PM<sub>10-2.5</sub> and mortality (Table 11-11 on pages 11-119 to 120 of ISA) resulted in inconsistent outcomes. The lack of available scientific evidence is mainly due to a lack of PM<sub>10</sub> and PM<sub>10-2.5</sub> monitoring. Nationwide, there are 391 FRM and 365 FEM PM<sub>10</sub> sites as compared to 624 FRM and 579 FEM PM<sub>2.5</sub> sites for integrated 24-hour and hourly PM concentrations, respectively. In addition, there are 361 PM<sub>2.5</sub> monitors, not approved as FEMs, operated to report the AQI. Therefore, the total number of PM<sub>10</sub> sites is less than 50% of the PM<sub>2.5</sub> sites. This results in a dearth of PM<sub>10</sub> data, and is therefore, PM<sub>10-2.5</sub> (coarse) concentrations.

Although a PM<sub>10-2.5</sub> FRM was specified in the 2006 PM NAAQS review, little effort has been made over the last decade to better understand the temporal and spatial variations or the composition of PM<sub>10-2.5</sub>. As of 2018, there are only 279 PM<sub>10-2.5</sub> sites in the AQS database, less than 20% of the PM<sub>2.5</sub> sites. In addition to the commonly measured multielements, major ions (e.g., nitrate, sulfate, and ammonium), and organic and elemental carbon, speciation of PM<sub>10-2.5</sub> components should also include carbonate carbon and bioaerosols (e.g., endotoxin, 1,3-β-glucan, and total protein), prominent in PM<sub>10-2.5</sub> fractions (e.g., Chow et al., 2015) that may be associated with health effects.

SCQ-4.03 *What are the Panel's views on the draft PA preliminary conclusion that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports consideration of retaining the current standard?*

Given the lack of measurements and resources, it is not surprising that the same key uncertainties (e.g., approaches to estimating PM<sub>10-2.5</sub>; measurement errors; potential for confounding by co-pollutant; and lack of biological plausibility) in causality determination are given in the previous (U.S. EPA, 2009) and current (U.S. EPA, 2018) ISA assessments. Figure 2-16 (page 2-33) shows that the average second highest 24-hour PM<sub>10</sub> concentration during 2015-2017 was 56 µg/m<sup>3</sup> (ranging 18-173 µg/m<sup>3</sup>). The majority of the sites measured below 75 µg/m<sup>3</sup>, with the exception of those in the southwestern U.S. The annual second highest 24-hour PM<sub>10</sub> concentrations in Figure 2-17 (page 2-34) show a downward trend of ~30% from 2000-2017, and are <75 µg/m<sup>3</sup> after 2007. The 98<sup>th</sup> percentile PM<sub>10-2.5</sub> concentrations for 2015-2017 (Figure 2-20, page 2-36) are mostly less than 30 µg/m<sup>3</sup>, consistent with nationwide PM<sub>2.5</sub> to PM<sub>10</sub> ratios of 0.5-0.6 for the second highest PM<sub>10</sub> concentrations during 2015-2017 (Figure 2-19, page 2-35). Therefore, 24-hour average PM<sub>10</sub> concentration of 60-75 µg/m<sup>3</sup> with a 24-hour PM<sub>10-2.5</sub> of 30 µg/m<sup>3</sup> most represents community exposure.

Given that 24-hr PM<sub>10</sub> concentrations have decreased by ~30% since 2000 and a positive association between PM<sub>10</sub> and health effects is still present, it is hard to justify retaining the 24-hour PM<sub>10</sub> NAAQS at the current level (150 µg/m<sup>3</sup>) and form (not to be exceeded more than once per year on average over a three-year period), which has not been revised since 1987 (see Table 1-1, pages 1-6).

More analyses are needed to test the association of lower (e.g., 60-75 µg/m<sup>3</sup>) 24-hour PM<sub>10</sub> concentrations with health effects and to demonstrate that the 24-hour PM<sub>10</sub> NAAQS of 150 µg/m<sup>3</sup> promulgated over 30 years ago is still adequate to protect public health.

**EPA-5. Chapter 5 – Review of the Secondary Standards: What are the CASAC views on the approach described in Chapter 5 to considering the evidence for PM-related welfare effects in order to inform preliminary conclusions on the secondary standards? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current secondary PM standards?**

SCQ-5.01 *To what extent does the Panel find that the key policy questions posed in this chapter appropriately reflect the important policy-relevant issues for the secondary PM standards? Are there additional policy-relevant questions that should be addressed?*

SCQ-5.02 *What are the Panel's views of the draft PA evaluation of the currently available scientific evidence with respect to the welfare effects of PM. Does the assessment appropriately account for any new information related to factors that influence:*

d) *Quantification of visibility impairment associated with PM<sub>2.5</sub> and examination of methods for characterizing visibility and its value to the public?*

- e) *The variable effects of PM<sub>2.5</sub> and its light absorbing and scattering components on climate?*
- f) *The effects of fine and coarse particles on materials?*

SCQ-5.03 *What are the Panel's views of the draft PA preliminary conclusion that the currently available scientific evidence does not call into question the protection afforded by the current secondary PM standards against PM welfare effects and that it is appropriate to consider retaining the current secondary PM standards without revision?*

- **Visibility Effects (Section 5.2.1)**

The analysis of visibility effects is mainly based on outdated (2005-2008 vs. 2011-2014) data and doesn't provide new information that might influence evaluation of light extinction and visibility. To achieve consistent and objective quantification of regional haze, the Regional Haze Rule (Section 308 of Protection of Visibility, 40 CFR Part 51, Subpart P, Sections 51.300-51.309) uses PM<sub>2.5</sub> chemical components to estimate particle light extinction (Watson 2002). Information on spatial interpolation of average light extinction by major chemical component for the most recent period (e.g., 2015-2017) should be compared with that from the last review to provide some perspective on overall changes.

As shown in Hand et al (2019), the organic mass (OM) to OC ratio increased across the network after 2011, highest in the east during summer, unrelated to the influence of particle bound water. The effects of visibility from changes in PM<sub>2.5</sub> composition over the past decade needs to be addressed. The reanalysis of three versions of IMPROVE light extinction algorithms (Malm et al., 1994; Pitchford et al., 2007; Lowenthal and Kumar, 2016) should provide IMPROVE 2015-2017 reconstructed light extinction coefficients ( $b_{ext}$ , Mm<sup>-1</sup>) by chemical components with monthly average PM<sub>2.5</sub> concentrations, to compare with those of 2005-2008 period. The revised IMPROVE algorithm (Pitchford et al, 2007) uses different scattering coefficients for the large and small sulfate, nitrate, and OM concentrations. The 20 µg/m<sup>3</sup> cut-off was selected to separate the large vs. small components. Owing to the nationwide reduction in PM<sub>2.5</sub> mass and sulfate concentrations, the "20 µg/m<sup>3</sup>" cut-off in the revised IMPROVE algorithms (Pitchford et al., 2007; Lowenthal and Kumar, 2016) may no longer be applicable. A reexamination with concentration levels more relevant to current air quality should be used to develop a more representative IMPROVE light extinction algorithm.

The draft PA suggests expanding the number and geographic coverage of "Preference" studies in urban, rural, and Class I areas to account for differences in population preference based on the scenic views. The "magnitude of scenic values" or the "ability of the public perception on visibility degradation" is judgmental and qualitative at best. Efforts should be put on science-based visibility estimates.

- **Key Uncertainties and Areas for Future Research (Section 5.4)**

New measurement techniques that can be used to estimate the radiation balance or climate change should be discussed. The newly developed multiwavelength (e.g., 405, 532, and 870 nm) Photoacoustic Extinctionmeter (PAX) provides high resolution aerosol optical measurements (Droplet Measurement Technologies, Boulder, CO) and is more advanced than the teleradiometers and telephotometers listed in the draft PA. Both the photoacoustic system and the dual and seven wavelength aethalometers (AE22 [370 and 880 nm] and AE33 [370 to 950 nm], Magee Scientific, Berkeley, CA, USA) can be used to estimate brown carbon (BrC),

organic carbon that absorbs light at a low wavelength (~300-400 nm). Estimates of BrC are included in the most recently released report by the Intergovernmental Panel on Climate Change (IPCC, 2019)

Starting with PM<sub>2.5</sub> filter samples from January 2016, the IMPROVE network reports seven wavelength (i.e., 405-980 nm) optical measurements along with the OC and EC analysis (e.g., Chen et al, 2015; Chow et al, 2015; 2018; 2019) that demonstrate the impact of BrC during fire episode. These data can be used to address changes in OM/OC ratios; develop revised IMPROVE algorithm; improve emissions inventory estimates; and provide data for climate assessment.

These data are also useful for determining natural visibility conditions related to the U.S. Regional Haze Rule; examining the effectiveness of emission reduction strategies for wood burning; and identifying exceptional events that cause exceedances of air quality standards. The draft PA should most represent state-of-the-art measurement techniques.

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**Dr. Douglas W. Dockery**

**SCQ-3.2 What are the panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?**

Section 3.2 provides a well-structured and clearly presented synthesis of the evidence for the health effects of PM exposures. There is no evidence for a discernable population threshold. Two approaches are used to attempt to draw out information relevant to recommending or evaluating current and alternative PM<sub>2.5</sub> standards.

In the first approach, the PM<sub>2.5</sub> air quality distributions over which epidemiologic studies support health effect associations and the degree to which such distributions are likely to occur in areas meeting the current (or alternative) standards are evaluated. Key studies are characterized based on the intuitive notion that the measures of association (exposure-response functions) are most precise at the mean of the exposure distribution. This misses the point that power is a function of the variance of exposure, not the mean. However, evaluating studies based on mean does show evidence for PM<sub>2.5</sub> effects for studies with mean exposures below the current PM<sub>2.5</sub> standards.

In the second approach, PM<sub>2.5</sub> design values ("pseudo-design values") are calculated where possible for epidemiologic study sites. These calculations attempt to determine if these epidemiologic study areas would have met or violated the current or alternative standards during study periods. It is an interesting to examine whether the PM<sub>2.5</sub> exposure measures used in the epidemiologic studies (whether directly measured or estimated) would differ from the observed PM<sub>2.5</sub> from regulatory monitoring. Indeed, it is clear that regulatory monitoring by targeting compliance will produce values higher than monitoring or hybrid methods targeted on estimating population exposures. Ultimately, this approach also provides evidence for PM<sub>2.5</sub> effects in communities not violating the current standard.

Section 3.3 is a risk assessment that estimates population-level health risks associated with PM<sub>2.5</sub> air quality "requisite" to protect the public health, that is "just meeting" the current standards. Given the evidence based conclusions of effect below the current standards from the epidemiology, risks associated with PM<sub>2.5</sub> air quality adjusted to simulate "just meeting" alternative annual and 24-hour standards with lower levels are estimated. Although characterized as representative of the US population, this risk assessment is limited to 47 urban areas with monitored PM<sub>2.5</sub> above or marginally below the current NAAQS. Multiple urban areas affected by "special" circumstances such as wildfires, seasonal local wood smoke, and "uncertain" measurements are excluded. A multistep process is used to estimate exposure reductions for each monitoring site to achieve targeted alternative based on a hybrid model of monitored and CMAQ model surfaces. The observed exposure response functions for a limited set of health outcomes ("causal" and "likely to be causal") are applied using BENMAP to estimate expected numbers of events. There is some quantitative, but largely qualitative

assessment of uncertainty. While this risk assessment is limited in scope, and not clearly described, the approach is sound and the numbers of preventable deaths at the current standard or alternative levels of the standard are informative. In particular, the risk assessment shows there are substantial numbers of deaths even in these limited analyses due to existing PM<sub>2.5</sub> exposures at the current standard. There would be substantial numbers of deaths prevented if stricter alternative levels of the PM<sub>2.5</sub> standard were in place. Moreover, the numbers of preventable deaths attributable to the annual standard are much larger than those attributable to the 24-hour standard. This supports the notion that the annual standard is the controlling limit.

While the evidence-based approach synthesizes the scientific evidence for adverse effects of PM<sub>2.5</sub> across the full range of exposures, the risk-based approach provides context for exposures and expected benefits from the current and alternative levels of the PM<sub>2.5</sub> standards. The consistency and coherence of the results of these two approaches is important in showing the PM<sub>2.5</sub> current standards are not protecting the public health adequately, and in providing guidance on possible alternative levels.

**SCQ 3.3 What are the panel's views on the evidence-based approach, including:**

**a) *The emphasis on health outcomes for which the draft ISA causality determinations are “causal” or “likely causal”?***

The focus on the health outcomes which are “causal” or “likely causal” is entirely appropriate, and well supported by the synthesis of the evidence in the ISA. (Note that the risk assessment approach only considers a subset of these health outcomes, see SCQ 3.4 a).

**b) *The identification of potential at-risk populations?***

Section 3.2.2 (page 3-42) on “Potential At-Risk Populations” is remarkably succinct. It would be helpful to structure this discussion around the multiple pathways that people could be at risk because of exposure, susceptibility, ameliorating personal characteristics, and community context.

The evidence continues to be that the young, the old, and those with pre-existing chronic conditions have increased susceptibility. In addition, minority and economically disadvantaged populations have higher exposures and less ability to modify their exposure, to obtain appropriate health care, or to modify lifestyle (e.g. moving or improving nutrition) to ameliorate response. The conclusion is correct that “*the groups at risk of PM<sub>2.5</sub>-related health effects represent a substantial portion of the total U.S. population*” (page 3-43, line 19).

**c) *Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?***

While there have been many important and informative epidemiologic studies from Europe and Asia as well as from North America, since the last review. All of these studies are important in defining the scientific basis for the adverse effects of PM<sub>2.5</sub>

exposures. However, the evidence from the multicity US and Canadian epidemiologic studies is adequate (and compelling) to assess the health effects of PM<sub>2.5</sub>. The US studies are the most informative in assessing relevant PM<sub>2.5</sub> exposures for standard setting. The Canadian studies are particularly informative in showing adverse effects at PM<sub>2.5</sub> exposures below the current US standards. One might ask about the relevance of the Canadian studies. The population in Canada tend to be in the southern provinces which are further south than many US cities. While the 49th parallel is often thought of as the border between the US and Canada, the vast majority of Canadians (roughly 70%) live below it.

**d) Characterizing air quality in these key studies using two approaches: the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the “pseudo design value” reflecting a monitor with the highest levels in an area?**

It is commendable to examine the distribution of the underlying PM<sub>2.5</sub> exposure data used in epidemiologic studies (page 3-51). Indeed there is useful information to be gained, particularly in considering how informative these studies are in the lower exposures ranges. However, characterizing these studies based on the mean exposure is based on a mis-understanding of the statistics.

The statement that epidemiologic studies provide the strongest support for reported health effect associations over the part of the distribution corresponding to the bulk of the underlying data (page 3-51, line 2-3) has some intuitive validity. However, extending that to say the associations are “strongest” at the mean is flawed. Figure 3-2 (page 3-52) from Lepeule et al (2012) is used to show that the confidence intervals are smallest at the center of the distribution of exposures (where there is the most data), and widest at the extremes. This is true, but this does not mean that the association is strongest (or alternatively has the smallest confidence interval) at the center of the distribution. The plotted confidence intervals show the uncertainty around the expected hazard ratio at each exposure, and indeed these are larger where there is less data (or less exposure measures). In simple statistics, the error of the expected value is inversely proportional to the square root of the number of data points. Thus confidence intervals are wider where there is less data. However, the association is determined by the slope of the fitted line (not the expected value at any given point). In linear regression, the uncertainty (confidence interval) of the slope is inversely proportional to the square root of the number of data point times the standard deviation of the exposure. Thus the important characteristics is not the mean of the exposure distribution but the standard deviation or heterogeneity of the exposures. Studies with large differences in exposures are more precise than studies with little variation in exposure. A study with large numbers but no variation in exposure would produce a very precise estimate of the health indicator, but provide no information on the slope or association with exposure. Thus the parameter that should be examined in Figures 3-3, 3-4, 3-5, 3-6 and Table 3-3 is the variance or other index of heterogeneity (e.g. IQR) of exposure. Likewise for Figure 3-7 and 3-8. Here it is positive that 25<sup>th</sup> and 10<sup>th</sup> percentiles are considered when available as well as the mean or median. Indeed these percentiles would be a much more informative statistic to use in this risk assessment, but only a handful of these percentiles are available. Note that in these two figures, the arithmetic



means are compared for the short and long term studies. They show similar means for both types of exposures. However, the variances and therefore the 10<sup>th</sup> and 25<sup>th</sup> percentiles should be very different, and cannot be directly compared. For the short-term studies variance is between daily PM<sub>2.5</sub> concentrations, and the number of data point is number of days. For long-term studies variance is between annual mean PM<sub>2.5</sub> concentrations, and the number of data points is the number of cities or spatial locations. Thus the short term studies will tend to have much larger variances than the long term studies.

There is a significant logical misinterpretation of the “pseudo design values”.

Throughout the PA there are statements such as “50% of the study area populations lived in locations with pseudo-design values below these concentrations, or 50% of the health events occurred in such locations.” This would appear to state that 50% of the population experiences such pseudo-design values, and equivalently 50% of the health events occur in these locations. Neither of these interpretations can be supported by the data. These statements ignore the base populations associated with each exposure.

**e) The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?**

The logic for this has not changed since the 2009 review. There is some additional discussion of this issue in the PA, which concludes there is no reason to modify this approach. However, the increased frequency of wildfires and associated acute exposures to anomalously high, short term episodes of PM<sub>2.5</sub> raises the importance of examining these effects in the evidence based analyses. (Note the risk assessment analyses explicitly exclude these events from consideration.)

**f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?**

What are the changes in the evidence since the last review?

- Experimental studies (both controlled human and animal toxicology) providing evidence of causal pathways. Notably, some of these examine the same physiological and clinical indicators as in the epidemiologic studies. Exposures/doses in these experimental studies are higher than typically experienced as ambient exposures by populations in the community, requiring extrapolation. On the other hand, these exposures are now much closer (within an order of magnitude) of ambient 24 hour exposures. Note in particular that controlled human exposures are limited to a few hours. When these short, high exposure periods are extrapolated to 24-hour averages, the net exposure is often comparable to commonly observed ambient levels.
- The hybrid methods combining information from stationary monitors, land use regression, chemical transport model and remote sensing data to estimate exposures have allowed the epidemiology studies to examine not only populations

living near a fixed monitoring station, but across larger regions or across the entire country. Thus, studies now include complete samples of the population, not just those in urban areas where there were networks of air pollution monitors. Importantly rural populations which were previously unmonitored are included. These rural populations tend to have lower exposures to PM<sub>2.5</sub>, and therefore extend the range of observations to levels substantially below those included in the 2009 review. The national Canadian cohort studies have been particularly informative about effects at low PM<sub>2.5</sub> levels. However, the national cohort studies in the US have been able to examine associations restricting to communities with exposures below the current annual NAAQS.

- The hybrid methods have also improved the spatial resolution of the PM<sub>2.5</sub> estimates for epidemiologic analyses. These improved PM<sub>2.5</sub> exposure estimates have reduced exposure misclassification, increased the effective sample size, and provided stronger, more precise associations.

All of these advances have strengthened the evidence for health effects of PM<sub>2.5</sub> exposures.

**g) *Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?***

The evidence for effects of sub-daily peak exposures to PM<sub>2.5</sub> are in my opinion undervalued in the PA and the ISA. The PA concludes that there is insufficient evidence for consideration of averaging times less than 24-hours. However, this lack of evidence is largely driven by the current form of the PM<sub>2.5</sub> NAAQS based on annual and 24-hour averages. The epidemiologic studies are largely based on exposure measurement methods that follow the EPA FRMs and NAAQS. Thus, few studies have considered sub-daily exposures. Consideration of peak versus 24-hour mean is not equivalent to examining PM<sub>2.5</sub> associations in the previous hour(s). As the Integrated Science Assessment notes, there are a limited number of studies which show increased risk of cardiovascular events (myocardial infarctions and arrhythmias) associated with PM<sub>2.5</sub> exposures in the previous hours. Likewise, controlled human exposure studies show changes in clinical cardiac indicators after PM<sub>2.5</sub> exposures of only an hour or less. Wildfire exposures while lasting multiple days, are usually brief (sub-daily) but intense in a given location because of shifting winds and moving sources. Understanding the effects of these specific short, intense exposures is challenging, but increasingly important.

**SCQ 3.4 *What are the panel's views on the quantitative risk assessment for PM<sub>2.5</sub> including:***

- a) *The choice of health outcomes and studies selected for developing concentration-response functions for long and short-term effects?***

The risk assessment was nominally based health outcomes determined to be “causal” or “likely to be causal”. As determined in the ISA there were “causal” associations for PM<sub>2.5</sub> with Mortality

and Cardiovascular Effects, and “likely causal” for Respiratory, Cancer, and Nervous System effects. However, only a subset of these are included in the risk assessment calculations (see table below).

Health Outcome	Exposure Duration	Causal Determination	Risk Assessment
Mortality	Long & Short	Causal	All Cause Mortality (Long & Short)
Cardiovascular Effects	Long & Short	Causal	Ischemic Heart Disease Death (Long only)
Respiratory Effects	Long & Short	Likely to be Causal	
Cancer	Long	Likely to be Causal	Lung Cancer Deaths (Long only)
Nervous System Effects	Long	Likely to be Causal	

Notably not included:

- Cardiovascular effects (long term) other than IHD mortality, such as cerebrovascular (stroke).
- Any short-term cardiovascular effects (short term), other than IHD mortality
- Respiratory effects either long or short term; mortality or morbidity
- Cancer mortality other than lung cancer
- Nervous system effects (morbidity)

Compare this to the Global Burden of Disease analyses which have developed risk assessment estimates for mortality from All Causes, Ischemic Heart Disease (IHD), Cerebrovascular Events (Stroke), Lower Respiratory Infections (LRI), Chronic Obstructive Pulmonary Disease (COPD) and Lung Cancer.

Presumably because of the time and resource constraints, the risk assessment is limited to a subset of the relevant health end-points. This implies that any findings of increased risk will be an underestimate of the true net risk.

***b) The selection criteria for the 47 urban areas and PM<sub>2.5</sub> air quality scenarios analyzed?***

Urban areas were selected for the risk assessment to be in some sense a representative sample of the US population. Three criteria are given for the selection of the 47 urban areas:

- *Available ambient monitors: “areas with relatively dense ambient monitoring networks”* This is not defined.
- *Geographical Diversity: “areas that represent a variety of regions across the U.S. and that include a substantial portion of the U.S. population”* Again not defined and there is not evidence that this actually was used as selection criteria. Some regions

were (e.g. northwest) were explicitly excluded. The population (>30 yrs) of these areas ranges from ~12 million to ~0.1 million. Thus, while a substantial fraction of the US population (~1/3) is included in these risk estimates, the sample is skewed towards large urban areas.

- *PM<sub>2.5</sub> air quality concentrations: “areas requiring either a downward adjustment to air quality or a relatively modest upward adjustment (i.e., no more than 2.0 µg/m<sup>3</sup> for the annual standard and 5 µg/m<sup>3</sup> for the 24-hour standard). In addition, ... we excluded several areas that appeared to be strongly influenced by exceptional events.”* In other words, areas with PM<sub>2.5</sub> above or modestly below the NAAQS were included in the initial screen (10/30 criteria). There were multiple adjustments to the air quality data for apparent non-representative values. 56 areas met the initial 10/30 criteria, but 9 (20%) were excluded for influence of wildfires (7 areas), one for anomalous local conditions (Eugene, OR), and another “uncertain” projections (Phoenix, AZ).

Overall, these selection criteria are ill defined with post-hoc adjustments that undermine the basis describing these 47 urban areas as representative of the US population. Nevertheless, these urban areas do provide a basis for this risk assessment and do not invalidate the results. By explicitly excluding consideration of impact of wildfires, and local and seasonal sources (wood burning), these risk assessments will underestimate the total net health burden from PM<sub>2.5</sub>.

c) **The hybrid modeling approach used for quantifying exposure surrogates across an area and adjusting air quality for alternative standard levels, as supplemented by interpolation/extrapolation?**

The objective was to provide scaling factors to bring the values at the highest monitor in selected urban areas into compliance with current or proposed alternative standards. In this case, the chemical transport model calculations were matched to regulatory monitoring to estimate the degree adjustment needed to meet current or alternative standards. Frankly, following the logic and process for this modeling was almost impossible, either in the text or the appendix. A more detailed flow chart in the text may have been helpful. While the overall approach appears to be sound, not being able to understand the details of the method does not provide confidence in the calculations.

Note that the “hybrid” model used here for assessing regulatory compliance is not in any sense comparable to the “hybrid” models used for exposure estimation in the epidemiology studies. It would be beneficial in the PA not to describe these very different approaches as “hybrid models”.

d) **The characterization of variability and uncertainty in the risk assessment?**

The characterization of the uncertainties and variability of the risk assessment is ad hoc. Alternative exposure response functions were considered, including their individual

confidence intervals. However, generally the highest value was cited, with no assessment of a central value or range of values across alternative exposure-response functions. Alternative approaches for achieving standards (PM primary and PM secondary) were considered, but effectively no consideration of uncertainties in exposure estimates. Recall also that only a subset of health outcomes found to be “causal” or “likely to be causal” are considered, so estimated numbers will be a subset of expected health numbers. This does not diminish the conclusion that there are substantial numbers of premature deaths in the United States among populations exposed to PM<sub>2.5</sub> at or below the current standards.

**e) The robustness and validity of the risk estimates?**

The risk assessment was limited in scope, only a fraction of the US population living in urban areas was examined, and the description of the methods was difficult to follow. Nevertheless, the approach was sound and the calculated numbers of premature deaths is a conservative (that is underestimate) of the true expected numbers of deaths and other adverse health events.

**SCQ-3.5 What are the panel’s views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?**

The scientific evidence from the epidemiologic studies with supporting experimental evidence from controlled human exposure studies and animal toxicology is unambiguous in showing effects of PM<sub>2.5</sub> at levels below the current primary standards.

The air quality analysis of mean values and distributions of PM<sub>2.5</sub> values in the key epidemiologic studies, comparing to design values, and examination of “pseudo-design values” addresses some secondary questions in extrapolating from the epidemiologic studies to practical control issues. These analyses confirm that the epidemiologic studies are showing health effects at PM<sub>2.5</sub> levels defined either by the epidemiologic exposure measures or appropriate design values which are at or below the current annual and 24-hour standards.

The risk assessment approach was appropriate although not clearly presented. The risk assessment itself was limited to a subset of the “causal” and “likely to be causal” health effects, and limited to a subset of the US population with PM<sub>2.5</sub> ambient concentrations above or slightly below the current standards. Thus, the risk assessment, which is built on the epidemiologic evidence and the air quality analyses, provides conservative (that is underestimates) of likely net numbers of adverse health events attributable to PM<sub>2.5</sub> levels around the current standard. The risk assessment findings of substantial number of PM<sub>2.5</sub> attributable deaths provides important context for the evidence-based analyses.

Together these approaches provide coherent and consistent evidence that the current PM<sub>2.5</sub> annual and 24-hour standards do not provide adequate protection of the public health.

**EPA-6. Chapters 3 to 5: What are the CASAC views regarding the areas for additional research identified in Chapters 3, 4 and 5? Are there additional areas that should be highlighted?**

Sub-Daily PM<sub>2.5</sub> Exposures: The PA concludes that there is insufficient evidence for consideration of averaging times less than 24-hours. However, this lack of evidence is largely driven by the current form of the PM<sub>2.5</sub> NAAQS based on annual and 24-hour averages. Epidemiologic studies are largely based on exposure measurement methods which follow the EPA FRMs and NAAQS. Thus, few studies have considered sub-daily exposures. Consideration of peak versus 24-hour mean is not equivalent to examining PM<sub>2.5</sub> associations in the previous hour(s). As the Integrated Science Assessment notes, there are a limited number of studies which show increased risk of cardiovascular events (myocardial infarctions and arrhythmias) associated with PM<sub>2.5</sub> exposures in the previous hours. Likewise, controlled human exposure studies show changes in clinical cardiac indicators after PM<sub>2.5</sub> exposures of only an hour or less. As continuous PM data becomes available, it is important to examine associations with these sub-daily exposures. Note that wildfire exposures are usually brief (sub-daily) but intense, so understanding the effects of these specific exposures is challenging, but increasingly important.

## **Mr. Henry (Dirk) Felton**

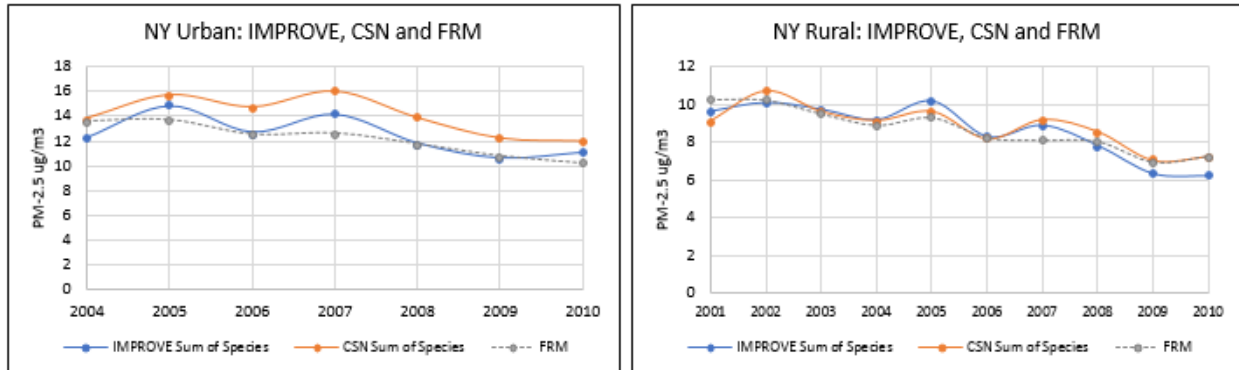
### **SCQ 2.1**

The draft PA does not provide a clear and concise summary of air quality. When data from different monitoring programs are discussed, inconsistent date ranges are used. The PM data are presented as design values from 2015-2017, ultrafine data are presented for 2014-2015, IMPROVE data are presented from 2004 and 2016 and the analysis on background PM used 2016 IMPROVE data. These data sets from different time periods were then compared to model results for 2011 and source categories from the 2014 NEI. Taking data from different date ranges reduces the validity of the conclusions that can be drawn. For instance, 2016 was a year that included the Fort McMurray wildfire in Alberta Canada. That year should not have been singled out as a representative year to look at background PM. The number of acres burned varies from year to year so a longer dataset should be used.

The plots used to show the NEI for PM-2.5 and PM-10 were not very helpful. Pie charts showing national average emissions don't provide information specific to regions, urban or rural regions or for areas with high or low ambient concentrations.

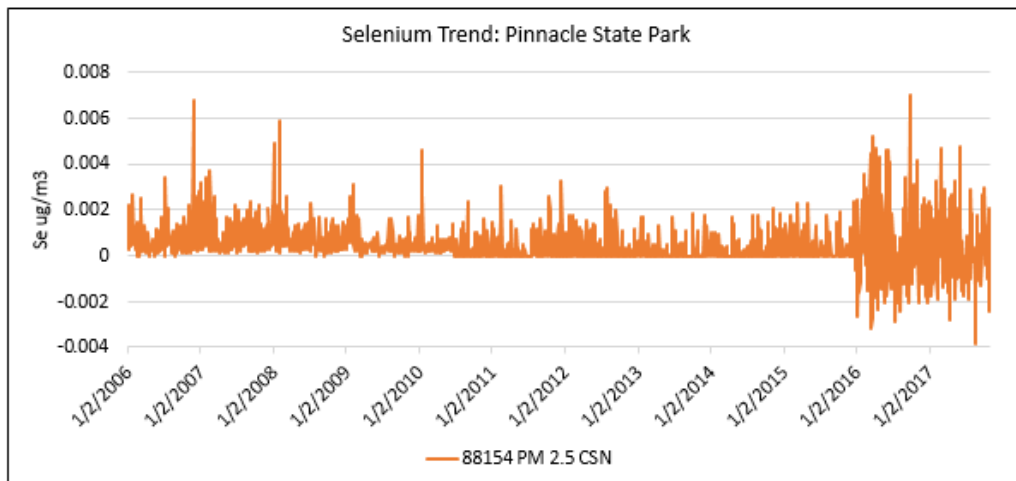
The draft PA also provided very little information about the components of PM-2.5. The National plots only included four species and no elemental data and no mass balance analysis was provided.

The draft PA's summary of air quality should address the shortcomings of the CSN program. This program was originally designed with six objectives linked to assessing PM-2.5 components over time so States could develop and track SIPs and related control programs. One objective included comparing the mostly urban CSN data with the mostly rural IMPROVE program. Over time, the CSN sampling protocol and the analysis methods for some of the species have been changed to more closely align with the equipment and methods used in the IMPROVE program. These changes have been to the detriment of the State Agencies who need this data to align as closely as possible with the equipment and protocols related to the PM-2.5 FRM. NYSDEC operated collocated CSN and IMPROVE sites to assess the differences between the programs. The two methods were in better agreement at the rural site where volatile species including OC were lower. At the urban site where the accuracy of the species data were critically important for source attribution, SIP development and control strategy tracking, the CSN results were too high in comparison to the PM-2.5 FRM.



Data from the South Bronx and from Pinnacle State Park

The CSN program has also been impacted by contractual changes. In late 2015, the CSN laboratory contract was awarded to a different laboratory and this has negatively impacted many of the elemental results. Some low concentration elements have been useful because they can be linked to specific source categories. This data has been used to identify local and out of State source impacts so they can be addressed appropriately. In the plot below, Selenium which has been used to identify coal combustion does not have a useful trend after the change in laboratories.



Selenium CSN Data from Pinnacle State Park

The CSN program is a valuable resource but it has been compromised by competing interests and as a result correction factors have to be applied to various species and some of the elemental data can no longer be used to detect trends. This program needs to be redesigned to make it more representative of the PM-2.5 in urban areas where ambient concentrations are likely to be closer to the primary NAAQS.

### SCQ 3.3

**d)** Setting a health-based standard that only attempts to limit detrimental health effects for the population within the 25<sup>th</sup> and the 75<sup>th</sup> percentile of annual PM concentrations does not represent an adequate margin of safety for at least one quarter of the population. In fact, the admission that the level based on this analysis would only protect a portion of the population against “an array of serious health effects, including premature mortality and increased hospitalizations for cardiovascular and respiratory



effects” shows that little attention is paid to susceptible populations and no protections are afforded for health effects short of hospitalization and mortality.

### **SCQ 3.5**

The draft PA addresses each element of the NAAQS individually: indicator, averaging time, form and level. The problem is that the analyses of PM concentration and health effects that accompany each element do not examine each element in isolation. The analyses that accompany the discussion about levels only examine studies that conform to either the averaging time and form of the annual or 24-hour standard. No effort was made to examine health effects resulting from data collected using other averaging times or forms. With this kind of limit: “blindness” on analyses, there is no opportunity to demonstrate the need for a sub daily or alternate form of the standards.

### **SCQ 3.6**

**a)** The current PM-10 and PM-2.5 standards are set to protect against respiratory and circulatory system health impacts. Ultrafine particles (UFP) have an additional central nervous system (CNS) health exposure pathway that is not controlled by a standard. A new standard should be set to reduce exposures to higher UFP levels. Some of the largest sources of UFP are combustion sources including stationary and motor vehicles. Motor vehicle emissions can be high from HDD vehicles that have damaged or poorly maintained emission control systems. Vehicle brake and tire wear are also sources that impact most of the population. Setting a UFP standard with a short averaging time would help drive improved controls on sources including HDD vehicles and would reduce exposures in near road communities.

**b)** The averaging times of the existing PM standards do not adequately protect populations exposed to elevated PM concentrations (UFP, PM-2.5, PM-10) typically found near roadways during weekday morning commuting hours. These impacts are often the highest exposures in many communities and are more evident near roadways with a higher proportion of HDD vehicles.

The beginning and end times for the averaging time of the 24-hr standard are also not adequate to protect against residential heating and recreational wood smoke impacts. The occurrences of these emissions typically begin in the evening and end in the early morning. The midnight to midnight form of the 24-hour standard effectively cuts these impacts into two which in many cases ends up reducing the regulatory impact by averaging additional cleaner hours of two days. Monitoring data have shown that exceedances of the 24-hour standard would be more frequent if the standard were based on noon to noon or on a rolling 24-hour average basis.

**f)** A lower annual standard does not do enough to reduce the impact from short-term or sporadic sources such as wood smoke from building heating, agricultural burning or industrial activity. Impacts from these sources can have very significant impacts on smaller scales in urban or rural communities. These emissions must be controlled if they impact fewer people just as much as the sources that impact larger scales. Another disadvantage of lowering just the annual standard is that it may increase the number of times when there is a  $10 \mu\text{g}/\text{m}^3$  change in concentration. Health effects have been found to occur when there are  $10 \mu\text{g}/\text{m}^3$  changes in air quality in relatively clean and in relatively polluted cities. To prevent these harmful swings in air quality, the daily standard must be lowered in conjunction with or prior to lowering the annual standard.

**GC-4**

Peaks in background PM are often the result of wildfire emissions or dust storms. These sporadic emissions should not be included in a discussion of peak background PM relative to a NAAQS because these emissions can be excluded from attainment consideration using the exceptional events policy.

Peaks in concentrations resulting from anthropogenic emissions do need to be included in NAAQS data assessments. In urban areas where PM-2.5 concentrations are closer to current NAAQS, contributions from background PM sources are smaller and less relevant.

## H. Christopher Frey

These comments build upon written comments that I submitted to CASAC and EPA as an attachment to a consensus letter from the Independent Particulate Matter Review Panel (IPMRP) on December 10, 2018,<sup>22</sup> as individual comments to CASAC and EPA on March 26, 2019,<sup>23</sup> and as part of a consensus letter from the IPMRP on March 27, 2019.<sup>24</sup>

### Process Issues

Since 2017, numerous changes have been made to the scientific review process for the National Ambient Air Quality Standards (NAAQS), including changes that affect the membership and composition of the EPA Clean Air Scientific Advisory Committee (CASAC). These changes have been made without advance notice to, or input from, the full chartered CASAC, EPA staff, or the public. The changes include: (a) imposing non-scientific criteria for appointing CASAC members related to geographic diversity and affiliation with governments; (b) replacing the entire membership of the chartered CASAC in a period of one year; (c) banning recipients of scientific research grants while allowing persons affiliated with regulated industries to be members of CASAC; (d) ignoring statutory requirements for the need for a thorough and accurate scientific review of the NAAQS in setting a review schedule; (e) reducing the number of drafts of a document for CASAC review irrespective of whether substantial revision of scientific content is needed; (f) commingling science and policy issues; (g) depriving CASAC of the needed breadth, depth, and diversity of scientific expertise for the PM NAAQS review by disbanding the CASAC PM Review Panel; (h) depriving CASAC of the needed breadth, depth, and diversity of scientific expertise for the ozone NAAQS review by refusing to form a CASAC Ozone Review Panel; and (i) creation of an ad hoc “pool” of consultants that fails to address the deficiencies created by disbanding the CASAC PM Review Panel and not forming a CASAC Ozone Review Panel. Each one of these changes harms the quality, credibility, and integrity of the NAAQS review for both PM and ozone.

EPA should appoint members to CASAC and its review panels based on the need for breadth, depth, and diversity of scientific expertise, not geographic diversity and government affiliation. Consistent with Federal peer review guidance, EPA should allow leading researchers who hold EPA scientific research grants to serve, subject to previously existing requirements that such

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<sup>22</sup> Frey, H.C., A.V. Diez Roux, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “CASAC Review of EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 34 page letter and 100 pages of attachments submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA–HQ–ORD–2014–0859, December 10, 2018.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/\\$File/PMRP+Letter+to+CASAC+181210+Final+181210.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/$File/PMRP+Letter+to+CASAC+181210+Final+181210.pdf)

<sup>23</sup> Frey, H.C., “Public Comment: Deficiencies of Procedure and Expertise Must Be Corrected,” Written Comment to the Clean Air Scientific Advisory Committee,” U.S. Environmental Protection Agency, Washington, DC, March 26, 2018,  
[https://yosemite.epa.gov/sab/sabproduct.nsf/46BBA443B9D953A9852583C9004F1F00/\\$File/Frey+Written+Public+Comments+to+CASAC+190326+Final.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/46BBA443B9D953A9852583C9004F1F00/$File/Frey+Written+Public+Comments+to+CASAC+190326+Final.pdf)

<sup>24</sup> Frey, H.C., A.V. Diez Roux, P. Adams, G. Allen, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R. McConnell, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “03-07-19 Draft CASAC Review of EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 19 page letter submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, March 27, 2019.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/A491FD482BB83BEE852583CA006A2548/\\$File/Written+Comments+from+17+Members+of+the+CASAC+PM+Review+Panel+that+was+Disbanded+on+October+11+2018+rev.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/A491FD482BB83BEE852583CA006A2548/$File/Written+Comments+from+17+Members+of+the+CASAC+PM+Review+Panel+that+was+Disbanded+on+October+11+2018+rev.pdf)

persons do not deliberate on their own work. EPA should recognize that there is a learning curve to service on CASAC and, therefore, value in appointing members to staggered terms and reappointing members to a second three-year term. EPA should allow adequate time for the scientific review. EPA should not combine assessment documents in a review unless this is consistent with a final Integrated Review Plan that has been agreed to by CASAC. EPA should allow for the likelihood that complex scientific and policy documents such as an Integrated Science Assessment, Risk and Exposure Assessment, and Policy Assessment may need substantial revision and re-review. EPA should better manage the timing of key milestones in the NAAQS review process so as not to selectively take time away from CASAC as a means to compensate for delays created by EPA elsewhere in the review. EPA should not introduce policy considerations until the scientific issues have been adequately settled. EPA should continue to follow the successful practice, proven for four decades, of augmenting CASAC with the expertise it needs via review panels that deliberate interactively with members of the chartered CASAC. EPA should not make ad hoc changes to the NAAQS review process in the middle of a review. If EPA wishes to make changes to the NAAQS review process, it should do so in a systematic manner similar to that employed in 2006, when EPA staff, CASAC, and others had an opportunity to provide input.

CASAC does not have adequate breadth, depth, and diversity of scientific expertise and experience needed to conduct thorough reviews based on the latest scientific knowledge of the kind and extent of scientific issues that pertain to the Particulate Matter NAAQS.

**Emphasis has been placed on geographic diversity, not scientific expertise**, in appointing members of CASAC, per an October 31, 2017 memorandum by former Administrator Scott Pruitt.<sup>25</sup> This policy has been implemented by Administrator Scott Wheeler in appointing members to CASAC on October 31, 2017 and by Administrator Andrew Wheeler in appointing five members to CASAC on October 10, 2018.<sup>26</sup> In revising criteria for membership on EPA Federal Advisory Committees, the October 31, 2017 memorandum from former Administrator Pruitt, EPA should have recognized that such committees may serve different purposes, and should have acknowledged Federal guidance on peer review. The membership criteria for a scientific review committee should not be the same as the membership criteria for a stakeholder committee.

**Emphasis has been placed on affiliation with state, local, and tribal governments, not scientific expertise**, in appointing members of CASAC, per October 31, 2017 memorandum by former Administrator Scott Pruitt. Although by law CASAC must have at least “one person representing State air pollution control agencies,” CASAC must also have sufficient expertise to do its job. As of October 10, 2018, with the new appointments by Administrator Wheeler, CASAC had four members from state agencies (Georgia, Texas, Alabama, and Utah) and had another appointee who was affiliated with a Federal agency. Having four members from state agencies does not make CASAC four times better. CASAC is less scientifically qualified than it would otherwise have been had the appointments been made, instead, based on selecting the best scientists.

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<sup>25</sup> Pruitt, E.S., “Strengthening and Improving Membership on EPA Federal Advisory Committees,” Memorandum, U.S. Environmental Protection Agency, October 31, 2017. <https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>

<sup>26</sup> EPA, “Acting Administrator Wheeler Announces Science Advisors for Key Clean Air Act Committee Tasks Chartered Panel to Lead Review of Ozone & Particulate Matter Standards Under Reformed Process,” News Release, U.S. Environmental Protection Agency, Washington, DC, October 10, 2018, <https://www.epa.gov/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee>

**A policy to have more member turnover** on CASAC, per the October 31, 2017 memorandum by former Administrator Scott Pruitt, has led to 100% turnover in just one year. In his October 10, 2018 appointments to CASAC, Administrator Wheeler replaced five CASAC members with five people who had never served on the chartered CASAC. Coupled with the appointments a year earlier by Administrator Pruitt of a chair and a member with no prior CASAC experience, as of October 2018 the chair and members of the chartered CASAC had a grand total of two person-years of experience on the CASAC, and little to no institutional memory of how CASAC operates. The new policy to enhance member turnover fails to acknowledge that there are benefits of continuity and knowledge provided by having some previous members continue to serve. Under this new policy, **well-qualified scientists have been “rotated” off of the CASAC, in favor of new members without needed subject matter expertise and without prior experience** on CASAC or CASAC review panels, selected instead for their affiliation or geographic location. CASAC is now the most inexperienced and unqualified that it has been in its history.

**Banning recipients of EPA research grants from serving on CASAC**, per the October 31, 2017 memorandum by former Administrator Scott Pruitt, is clearly intended to keep top academic researchers from serving on CASAC. The memorandum states that “no member of an EPA federal advisory committee currently receive EPA grants,” but that this “principle should not apply to state, tribal, or local government agency recipients of EPA grants.” This is inconsistent with the Federal Advisory Committee Act and inappropriate for four reasons. One is the obvious inconsistency of implying that receiving a grant creates a conflict of interest for one but not another class of persons. The second is the longstanding recognition that **receipt of a peer-reviewed scientific research grant, for which the Agency does not manage the work nor control the output, is not a conflict of interest**. Per the Office of Management and Budget (OMB): “When an agency awards grants through a competitive process that includes peer review, the agency’s potential to influence the scientist’s research is limited. As such, when a scientist is awarded a government research grant through an investigator-initiated, peer-reviewed competition, there generally should be no question as to that scientist’s ability to offer independent scientific advice to the agency on other projects.”<sup>27</sup> A 2013 report by the EPA Office of Inspector General reaffirmed that receipt of an EPA research grant is not a conflict of interest.<sup>28</sup> However, there can be situations in which a member of an advisory committee should recuse themselves from discussions that might pertain to their own work. Thus, third, the CASAC has had recusal policies in place for dealing with this issue and situations in which a member’s work may come up for deliberation. Fourth, **the memorandum does not acknowledge that persons with financial or professional ties to regulated industries have, at the very least, the appearance of conflict of interest**.

**Former EPA Administrator Pruitt signed a memorandum on May 9, 2018 that made major changes to the scientific review process for the NAAQS.**<sup>29</sup> The memo is replete with

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<sup>27</sup> Office of Management and Budget, “Final Information Quality Bulletin for Peer Review,” *Federal Register*, 70(10):2664-2677 (January 14, 2005), <https://www.govinfo.gov/content/pkg/FR-2005-01-14/pdf/05-769.pdf>

<sup>28</sup> EPA, “EPA Can Better Document Resolution of Ethics and Partiality Concerns in Managing Clean Air Federal Advisory Committees,” Report No. 13-P-0387, Office of Inspector General, U.S. Environmental Protection Agency, Washington, DC, September 11, 2013. <https://www.epa.gov/sites/production/files/2015-09/documents/20130911-13-p-0387.pdf>

<sup>29</sup> Pruitt, S.E., “Back to Basics Process for Reviewing National Ambient Air Quality Standards,” Memorandum, U.S. Environmental Protection Agency, Washington, DC, May 9, 2018. <https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>

cherry-picking of incomplete information that fails to accurately characterize the previously existing NAAQS review process, including its strengths. The memorandum emphasizes that the Clean Air Act requires that NAAQS be reviewed every five years, but fails to emphasize the statutory mandate for a thorough and accurate scientific review. For those NAAQS reviews for which EPA entered into a consent decree or was under court order to complete a review, the court-supervised schedules have taken into account the need for EPA staff to develop assessment documents and for CASAC to review the documents and advise the Administrator. Thus, the memorandum fails to acknowledge that courts have recognized that the time needed for a thorough and accurate scientific review can be taken into account in setting schedules that go beyond the five year time frame. Instead, EPA is self-imposing a schedule that compromises the quality, credibility, and integrity of the scientific review and is doing so in a manner beyond what courts have historically imposed.

The memorandum gives the misleading impression that delays in the review process are attributed to CASAC. Based on analysis that I submitted as part of my individual member comments attached to the IPMRP's December 10, 2018 letter to CASAC, I showed that the duration of CASAC activities in a NAAQS review cycle is far less than the total duration of the review cycle. A key factor that increases the duration of CASAC's involvement in a review cycle is delay in EPA providing CASAC with assessment documents for review. Furthermore, the memorandum omits any discussion of the more salient factors that have led to delays in the NAAQS review process related to decisions made by the EPA, not CASAC, as detailed below. EPA should not impose a reduced duration schedule for the scientific review that compromises the scope and quality of the scientific review. The duration of a review cycle is dependent on the following:

- (1) EPA controls the duration of time between the conclusion of a prior review cycle and the initiation of the subsequent review cycle;
- (2) EPA decides the allocation of resources for development of assessment reports by EPA staff that are part of the scientific review process;
- (3) EPA decides when to release a draft document for CASAC review;
- (4) EPA has been responsible for delays in providing draft assessments to the CASAC for review;
- (5) Whether a draft EPA document requires further iteration depends on its initial scientific quality; and
- (6) EPA has control over the timing of the NAAQS review process from the time that it receives closure on advice from CASAC until it promulgates a final decision.

Although the May 9, 2018 memorandum gives some attention to the last point in the list above, it fails to account the first five listed EPA-driven factors that lead to delays in review cycles.

**Based on incomplete and erroneous diagnosis of leading causes of delay**, and without due consideration for statutory requirements as described above, including the need for a "thorough review" based on the "latest scientific knowledge" of the "kind and extent of... effects," **the May 9, 2018 memorandum inappropriately targets measures to reduce the duration of CASAC's engagement in the review process.**

The late 2020 deadline for completing the particulate matter review given in the May 9, 2018 memorandum is contrary to EPA's own final Integrated Review Plan for the PM NAAQS

review<sup>30</sup> and does not provide sufficient time to complete the “thorough review” of the “latest scientific information” of the “kind and extent” of “all identifiable effects” mandated by the Clean Air Act for the review of NAAQS, even if the CASAC were supported by a robust panel of experts in the multiple disciplines involved. Furthermore, the quality and credibility of the review depends on whether CASAC is augmented with an appropriately constituted PM Review Panel.

On October 10, 2018, then acting EPA Administrator Wheeler eliminated the CASAC PM Review Panel by press release,<sup>31</sup> with a follow-up email from the SAB office on October 11, 2018. This was done without advance notice and without prior consultation with the panel or the CASAC. There is no precedent for disbanding a review panel in the middle of a review cycle.

The actual reason as to why Administrator Wheeler disbanded the PM Review Panel and refused to form an Ozone review panel has likely not yet been publicly disclosed. Two general talking points have emerged from EPA leadership regarding the elimination of review panels for PM and ozone. One is that the CASAC is the sole advisory body charged with advising EPA per the Clean Air Act. The other is that the panels needed to be eliminated to ‘streamline’ the review process. Both of these talking points are specious.

The talking point that only CASAC should advise the Administrator is specious because in fact it has only been the CASAC that has advised the Administrator throughout the history of CASAC. Per CASAC’s charter with the U.S. Congress:<sup>32</sup>

“EPA, or CASAC with the Agency’s approval, may form subcommittees or workgroups for any purpose consistent with this charter. Such subcommittees or workgroups may not work independently of the chartered committee and must report their recommendations and advice to the chartered CASAC for full deliberation and discussion. Subcommittees or workgroups have no authority to make decisions on behalf of the chartered committee, nor can they report directly to the EPA.”

Thus, **it has always been the chartered CASAC, not its panels, that advise the EPA.** It has been long-standing practice since the 1970s to augment the 7-member CASAC with additional independent experts, so as to have the breadth and depth of expertise required to conduct a “thorough review” based on the “latest scientific knowledge,” consistent with requirements of the Clean Air Act, as detailed in my individual comments attached to the IPMRP letter to CASAC dated December 10, 2018. **It is not sufficient, as the Administrator suggested, to state that the 7 member committee meets the minimum requirements of the law.**

The talking point that panels must be eliminated to streamline the review process is specious because, **without the panels, CASAC does not have the breadth, depth, and diversity of**

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<sup>30</sup> EPA, “Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter,” EPA-452/R-16-005, U.S. Environmental Protection Agency, Research Triangle Park, NC, December 2016.  
<https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf>

<sup>31</sup> EPA, “Acting Administrator Wheeler Announces Science Advisors for Key Clean Air Act Committee Tasks Chartered Panel to Lead Review of Ozone & Particulate Matter Standards Under Reformed Process,” News Release, U.S. Environmental Protection Agency, Washington, DC, October 10, 2018,  
<https://www.epa.gov/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee>

<sup>32</sup> United States Environmental Protection Agency Charter, Clean Air Scientific Advisory Committee, Filed with Congress, June 5, 2019,  
[https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/\\$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf)

**expertise to conduct scientific review consistent with the Clean Air Act requirements for being accurate and thorough.** Thus, the panels are essential. Secondly, the panels do not slow down CASAC's review time. They work in parallel and concurrently with the chartered CASAC.

The EPA released the external review draft of the Integrated Science Assessment (ISA) on October 15, 2018, five days after disbanding the CASAC PM Review Panel.<sup>33</sup> The Federal Register notice announcing that the draft ISA was available for public review was dated October 16, 2018 and published on October 23, 2018.<sup>34</sup>

Compared to the chartered CASAC, the PM review panel had more experts, covered more scientific disciplines, and had multiple experts who provide diversity of perspectives in many key disciplines, such as epidemiology, toxicology, and human clinical studies, among others.

After receiving public comments at its December 2018 and March 2019 public meetings on the draft ISA, CASAC requested in its April 11, 2019 letter to the Administrator that it review a second draft of the Integrated Science Assessment for Particulate Matter, and that it be augmented with the expertise necessary for such a review by either reappointing the disbanded PM review panel or appointing a similar panel.<sup>35</sup> In a July 25, 2019 letter to CASAC, the Administrator refused these requests.<sup>36</sup> **The Administrator stated that there will not be a second external review draft of the ISA.** The Administrator did not directly address any rationale for why he did not reappoint the disbanded panel or form a similar panel. Instead, the Administrator decided to appoint a "pool" of "subject matter" consultants. As described below, **the "pool" of consultants does not address deficiencies created by the same Administrator when he disbanded the PM review panel.**

The lack of a second draft of the ISA is highly problematic, particularly because the draft Policy Assessment is based on scientific evidence from the ISA. In prior NAAQS reviews, it has been typical practice that CASAC reviews a second and sometimes third draft (as in the cases of the most recent lead and ozone reviews) of the ISA. It has been typical practice that CASAC has had the opportunity to review a draft Policy Assessment **AFTER** it has completed reviews of draft ISAs. This sequence was by design. A key principle of the 2006 revisions to the NAAQS review process, which were modified in part in 2007 and 2009, is that the scientific foundation of

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<sup>33</sup> EPA, "Integrated Science Assessment for Particulate Matter (External Review Draft)," EPA/600/R-18/179, U.S. Environmental Protection Agency, Research Triangle Park, NC, October 2018.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/0/932D1DF8C2A9043F852581000048170D/\\$File/PM-1STERD-OCT2018.PDF](https://yosemite.epa.gov/sab/sabproduct.nsf/0/932D1DF8C2A9043F852581000048170D/$File/PM-1STERD-OCT2018.PDF)

<sup>34</sup> EPA, "Integrated Science Assessment for Particulate Matter (External Review Draft)," *Federal Register*, 83(205):53471-53472 (October 23, 2019). <https://www.govinfo.gov/content/pkg/FR-2018-10-23/pdf/2018-23125.pdf>

<sup>35</sup> Cox, L.A. (2019), "CASAC Review of the EPA's Integrated Science Assessment for Particulate Matter (External Review Draft – October 2018)," EPA-CASAC-19-002, Letter to A. Wheeler, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, April 11, 2019.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002+.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002+.pdf)

<sup>36</sup> Wheeler, A.R. (2019), Letter to L.A. Cox, EPA Clean Air Scientific Advisory Committee, from Administrator, U.S. Environmental Protection Agency, Washington, DC, July 25, 2019,  
[https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002\\_Response.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002_Response.pdf)



the review must be established before addressing policy issues.<sup>37,38,39</sup> Failure to do this risks commingling policy issues prematurely before the science issues are adequately vetted and settled, which in turn creates the potential for policy choices to be made irrespective of the science. Thus, **the integrity of the process is harmed when policy issues are addressed before the science issues are adequately settled.**

In this review cycle for PM, there are significant areas of indicated need for revision for the draft ISA based on comments from the Independent Particulate Matter Review Panel and members of the public. Thus, neither CASAC nor the public will have an opportunity to see how unresolved issues in the draft ISA that might have impacted the PA will be handled in a final version of the ISA. The final version of the ISA will not be available until after this EPA forces CASAC to conclude its involvement in this review cycle.

The Administrator announced a “pool” of 12 subject matter experts in an EPA press release on September 13, 2019.<sup>40</sup> The pool of 12 are intended to respond to written questions from the chartered CASAC for both the PM and ozone NAAQS reviews. In contrast, the disbanded PM review panel had 20 experts in addition to the chartered CASAC. At the same time that the Administrator disbanded the CASAC PM Review Panel on October 10, 2018, he also announced that he would not form a CASAC Ozone Review Panel. This was despite the fact that EPA had requested nominations for a CASAC Ozone Review Panel in a Federal Register notice on July 27, 2018.<sup>41</sup> In the prior ozone NAAQS review, which was completed in 2015, the CASAC was augmented with 15 additional experts to form an ozone review panel. Thus, **the total number of augmented experts for the prior ozone review and the current PM review through 2018 was 35. Twelve people is not an adequate number to cover the breadth, depth, and diversity of scientific expertise and experience needed for review of both ozone and PM.**

**The use of a “pool of subject matter experts” rather than a review panel to augment the chartered CASAC is unprecedented.** Review Panels augment and report through the chartered CASAC, working in parallel and in collaboration with the members of the chartered CASAC. Members of review panels are nominated by the public and the nominations are subject to public comment. The SAB staff office reviews, vets, and appoints members of review panels. Members of review panels participate in meetings with members of the chartered CASAC, and deliberate interactively with members of the chartered CASAC on complex subject matter. The chartered CASAC is ultimately responsible for the content of advice sent to the Administrator, but the formulation of that advice is informed based on deliberations with panelists who provide the breadth, depth, and diversity of needed scientific expertise.

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<sup>37</sup> Peacock, M., “Process for Reviewing National Ambient Air Quality Standards,” Memorandum to George Gray and Bill Wehrum, U.S. Environmental Protection Agency, Washington, DC, December 7, 2006.

<sup>38</sup> Peacock, M., “Modifications to Process for Reviewing National Ambient Air Quality Standards,” Memorandum, U.S. Environmental Protection Agency, Washington, DC, April 17, 2007

<sup>39</sup> Jackson, L., “Process for Reviewing National Ambient Air Quality Standards,” Memorandum, U.S. Environmental Protection Agency, Washington, DC, May 21, 2009.  
<https://www3.epa.gov/ttn/naaqs/pdfs/NAAQSReviewProcessMemo52109.pdf>

<sup>40</sup> EPA, “Administrator Wheeler Announces New CASAC Member, Pool of NAAQS Subject Matter Experts,” News Release, U.S. Environmental Protection Agency, Washington, DC, September 13, 2019.  
<https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>.

<sup>41</sup> EPA, “Request for Nominations of Experts for the Clean Air Scientific Advisory Committee (CASAC) Ozone Review Panel,” *Federal Register*, 83(145): 35635- 35636 (July 27, 2018). <https://www.govinfo.gov/content/pkg/FR-2018-07-27/pdf/2018-16116.pdf>

In contrast, **there was no opportunity for public comment on the nominees for the pool of subject matter experts.** The decision regarding appointments of ad hoc consultants to serve as subject matter experts was made by the Administrator, not by the SAB Staff Office. The General Accountability Office has documented irregularities in the process since 2017 by which appointments have been made to EPA advisory committees, including the CASAC.<sup>42</sup>

**Appointments made directly by the Administrator are subject to political considerations** and can disregard input from EPA career staff in the Science Advisory Board Staff Office regarding scientific considerations in selecting members and consultants. All interactions between CASAC and the subject matter experts are done only in writing. **Subject matter experts are not allowed to participate in deliberative meetings with CASAC.** For example, subject matter experts are not allowed to, unless invited in writing by the chair or designees of the chair, respond to all charge questions that might be of interest to the consultant. If a member of the pool of experts offers written comments that are inaccurate, are out of scope, or have other problems, there is not an effective mechanism for interaction that might have led to more relevant and refined input. Moreover, the composition of the pool of consultants does not provide CASAC the breadth, depth, and diversity of expertise needed for review of either the ozone or the PM NAAQS. **The appointment of consultants by the Administrator is not correcting the deficiencies in CASAC's ability to conduct a thorough review that have resulted from disbanding the PM Review Panel.**

**EPA should reinstate the disbanded PM review panel and appoint an ozone review panel. These panels should be appointed by the director of the SAB staff office, not by the Administrator, per established procedures in place prior to interference by the current EPA Administrator.**

In attempting to alter the NAAQS review process, if any changes are warranted, EPA should have followed the kind of open and transparent process undertaken in 2006, which included input from EPA career staff, the chartered CASAC, and members of the public. Such a process would lead to a better understanding of the key needs and challenges of NAAQS review and perhaps effective ideas for reviews which are more timely.

As a result of the many deleterious, unprecedented, and unwarranted changes to the CASAC described above, CASAC has transitioned from a committee of nationally and internationally recognized researchers at the leading edge of their fields toward a committee composed predominantly of stakeholders chosen based on geographic location and affiliation with state government, rather than scientific expertise first and foremost. **CASAC does not have adequate breadth, depth, and diversity of scientific expertise and experience needed to conduct thorough reviews based on the latest scientific knowledge of the kind and extent of scientific issues that pertain to the Particulate Matter NAAQS.** This is generally true given that CASAC is comprised of only seven members, whereas these reviews require multiple experts in each of many scientific disciplines. This is even more true given that the current CASAC was appointed based primarily on geography and affiliation, and not by scientific discipline, in accordance with the October 31, 2017 memo by former Administrator Pruitt. According to November 7, 2018 "determination" memorandum from the EPA SAB office, the CASAC has no epidemiologists,<sup>43</sup> even though epidemiology is a key scientific discipline related

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<sup>42</sup> GAO, EPA Advisory Committees: Improvements Needed for the Member Appointment Process, GAO-19-280, General Accountability Office, Washington, DC. <https://www.gao.gov/assets/710/700171.pdf>

<sup>43</sup> Yeow, A., "Determinations Associated with the Clean Air Scientific Advisory Committee (CASAC) Review of the Particulate Matter (PM) National Ambient Air Quality Standards (NAAQS)," Memorandum to T.H. Brennan, Science

to both the ozone and PM reviews. The CASAC lacks adequate coverage of many other disciplines, such as exposure assessment, welfare effects, and other areas, and lacks depth in areas for which CASAC has historically and necessarily engaged multiple experts, such as toxicology and controlled human studies.

**The Administrator should reinstate the disbanded CASAC PM Review Panel or should form a similar panel to augment CASAC for the current review of the PM NAAQS. The Administrator should form a CASAC Ozone Review panel to augment the CASAC for the current review of the ozone standard. The EPA should reaffirm and continue the established and successful practice, demonstrated for four decades, of augmenting CASAC with expert panels for each NAAQS review.**

To promote transparency of the review and opportunity for public input consistent with long-standing practice, the CASAC should have a longer time frame for its deliberations, consistent with historic practice in the last decade, and should not have the public meeting process truncated to meet shortened deadlines that resulted from EPA delays in starting the current review. The current self-imposed review schedule for the PM NAAQS review is contrary to the final PM IRP. It has fewer public meetings of CASAC and, therefore, fewer opportunities for public comment. For the ozone NAAQS review, the EPA is planning that CASAC will have only one face-to-face meeting to simultaneously review the draft ISA and draft PA, which even more severely limits opportunities for public comment compared to prior review cycles.

EPA's focus on rushing the scientific review of both the PM and Ozone NAAQS is clearly hypocritical. Although the Administrator has emphasized the need to meet the five year statutory mandate of the Clean Air Act for NAAQS review, not only has the Administrator not acknowledged that courts have allowed adequate time for scientific review when EPA has missed such deadlines, but the Administrator has been silent regarding the timing of reviews for carbon monoxide, lead, nitrogen dioxide, and sulfur oxides. For example, the most recent review of the carbon monoxide NAAQS concluded on August 31, 2011. The most recent lead review concluded on October 18, 2016. The most recent nitrogen dioxide review concluded on April 6, 2018. Why has the EPA not started new review cycles for these pollutants? **Delays by EPA in starting review cycles or developing assessment documents should not infringe on the duration of review and comment activities by CASAC and the public.**

### **Decision Context for NAAQS Review May Not Be Redefined by CASAC**

CASAC may not redefine the policy and decision context of NAAQS review. This context is set forth by Congress in the Clean Air Act, including but not limited to the following excerpts. From Section 108:

The NAAQS must address "air pollution which may reasonably be anticipated to endanger public health or welfare"

"Air quality criteria for an air pollutant shall accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the

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Advisory Board, U.S. Environmental Protection Agency, Washington, DC, November 7, 2018.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebProjectsCurrentCASAC/64C246444C9CC319852584430045E365/\\$File/Determination%20memo-Chartered%20CASAC%20PM-110718.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebProjectsCurrentCASAC/64C246444C9CC319852584430045E365/$File/Determination%20memo-Chartered%20CASAC%20PM-110718.pdf)

ambient air, in varying quantities.” and “any known or anticipated adverse effects on welfare”

And from Section 109:

The Administrator “shall complete a thorough review of the criteria” published under Section 108.

“National primary ambient air quality standards, prescribed under subsection (a) shall be ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”

Note that nowhere does the Clean Air Act state that EPA should take a risk-neutral or risk-seeking attitude toward risk, nor that EPA should limit its assessment only to those studies that individually can demonstrate manipulative causality consistent with particular quantitative causal tests and inference methods. The language of the Clean Air Act means that EPA cannot throw out studies according to arbitrary “quality” criteria if that would compromise the ability to conduct a thorough review and account for the full scope of review as mandated in the Act.

### **The Role of Expert Judgment in Scientific Review of the NAAQS**

In the current review process the Administrator has arbitrarily and capriciously done away with the CASAC PM Review Panel. **Given the important role of expert judgment in CASAC’s work, it is essential that CASAC be augmented with additional experts in the multiple scientific disciplines needed for this review.** Furthermore, there must be multiple experts in key areas, such as air quality physics and chemistry, exposure assessment, toxicology, controlled human studies, epidemiology, and others, to have a diversity of perspectives to assure that judgment is based on the large body of relevant scientific evidence using accepted inference methods. For four decades, CASAC has been augmented with expert panels as documented by Frey et al. (2018) and others<sup>44,4546</sup>. Augmented panels advise the CASAC and supplement it with the expertise it needs. Absent such augmented expertise, the chartered CASAC is scientifically unqualified to conduct a review consistent with language in the Clean Air Act.

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<sup>44</sup> Frey, H.C., A.V. Diez Roux, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “CASAC Review of EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 34 page letter and 100 pages of attachments submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA–HQ–ORD–2014-0859, December 10, 2018, Appendix E. [https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/\\$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf)

<sup>45</sup> Bloomer, L., and J. Goffman, “The Legal Consequences of EPA’s Disruption of the NAAQS Process,” Environmental and Energy Law Program, Harvard Law School,” undated, <http://eelp.law.harvard.edu/wp-content/uploads/Legal-Consequences-of-NAAQS-Changes.pdf>, accessed 10/7/19

<sup>46</sup> Bachmann, J., “Written Statement for the Public Meeting of the EPA Chartered Science Advisory Board, Re: 5/31 SAB Discussions about EPA Planned Actions and their Supporting Science,” Environmental Protection Network, May 29, 2018, <http://www.scientificintegrityinstitute.org/EPATransBachmann052918.pdf>

**Expert judgment requires judgment by domain experts.<sup>47,48</sup> Given that this CASAC lacks experts in the appropriate scientific domains, it is unqualified to offer such judgments.**

Given that this CASAC lacks expertise in many key disciplinary areas, especially epidemiology, and that EPA arbitrarily and capriciously disbanded the CASAC PM Review Panel a few days before the Draft ISA was released, thereby depriving CASAC of the needed expertise, this CASAC is not in a credible position to offer judgments regarding causal determinations.

Expert judgment should be based on conditioning of available evidence and inference methods. The conditioning step is substantially more credible when it is based on a group of experts with breadth and depth of expertise, and diversity of perspectives. EPA had such a group in the form of the CASAC PM Review Panel and yet arbitrarily and capriciously dismissed that panel without prior notice and without public consultations with CASAC.

There are well known biases in expert elicitation, some of which are cognitive and some of which are motivational. An example of a motivational bias is the so-called “expert bias,” which is when people who are not the relevant experts pretend that they are to make themselves appear to be important experts. Another well-known motivational bias is when an “expert” wants to influence the outcome of a scientific review process to achieve a particular policy or regulatory outcome. Such biases might be indicated, for example, when members of a scientific review committee earn their living based on funding from regulated industries, and offer opinions that are consistent with policy outcomes of interest to their funders. Motivational biases also arise when an expert has taken strongly stated public positions previously, as a result of which it becomes more difficult for that person to change their views.

Biases can be counter-acted. The approach to counter-act “expert” bias is to engage experts who have relevant expertise and to make sure that there is breadth and depth of needed expertise, as well multiple experts in key scientific disciplines who have diverse opinions. In contrast, if the goal is to undermine the science review process, efforts could be made to promote and enhance “expert” bias. This can be done, for example, by doing away with a group of domain experts, as EPA has done by eliminating the CASAC PM Review Panel, and instead placing the review in the hands of a group that lacks the breadth and depth of expertise, and diversity of perspectives, to properly condition the review. A corollary is that “true” experts are usually the first to admit that they are not qualified to undertake a particular review and to call for the inclusion of additional experts. Persons who are over-confident of their own expertise or who seek to be perceived as an expert in an area for which they are not are unlikely to want to cede their position to experts.

An example of over-confidence is the inability of a person to admit to any limitations of methodologies that they advocate while emphasizing only limitations but not strengths of other methodologies. For example, advocates of new quantitative methods should acknowledge limitations related to problem selection, data selection, limitations of the methodology itself, and challenges with interpretation of results. As a simple example, consider the use of statistical methods to making inferences regarding a statistic. There is judgment regarding how to structure the analysis, what data to select (including geographic area, time period, spatial and

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<sup>47</sup> EPA, Expert Elicitation Task Force White Paper, Science and Technology Policy Council, U.S. Environmental Protection Agency, Washington, DC, August 2011. [https://www.fwspubs.org/doi/suppl/10.3996/052017-JFWM-041/suppl\\_file/10.3996052017-jfwm-041.s7.pdf](https://www.fwspubs.org/doi/suppl/10.3996/052017-JFWM-041/suppl_file/10.3996052017-jfwm-041.s7.pdf)

<sup>48</sup> Morgan, M.G., and M. Henrion, Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis, Cambridge University Press, 1990.

temporal resolution, and so on), what analysis methods to use, what criteria to use in hypothesis testing, and how to interpret the results.

One way to counter-act motivational biases related to experts who want to influence the outcome is, preferably, to not include persons with clear conflicts of interest as part of an expert advisory committee, especially in a regulatory context. This would typically exclude people with financial ties to regulated industries who have a vested interest in the outcome of the review process, and would also include people who have strongly stated prior positions that imply pre-judgment of the policy-relevant outcomes and people who work at agencies with publicly stated perspectives on issues under deliberation for which there is also a close reporting and line of management relationship. Such persons could still participate in the process as stakeholders via public comments.

In contrast, if the goal is to undermine the science review process, efforts could be made to promote and enhance motivational bias. A way to promote and enhance motivational biases is to have fewer experts and include among them persons who are susceptible to such biases. This is what EPA has done in doing away with the CASAC PM Review Panel and with recent changes to the composition of the CASAC.

**It is evident that the recent changes to the NAAQS review process have undermined prior measures that were in place to avoid or mitigate motivational biases. Changes to the NAAQS review process and to the CASAC since 2017 clearly produce bias.**

### **History of CASAC Advice on the Framework for Causal Determinations**

CASAC has reviewed the Framework for Causal Determinations in each NAAQS review cycle for a decade. Early work on development of the framework is evident in CASAC's comments on the second external review draft of the Integrated Science Assessment for Oxides of Nitrogen in 2008 (Henderson, 2008):

In regard to the Agency's approach to synthesis of the evidence and causal inference, an extensive Annex has been prepared that reviews a number of relevant frameworks. The background is a useful foundation for informing the selected approach for assessing available evidence and should be extended to justify the adopted framework. Based on this Annex, the Agency has made changes in Chapter 1 that are responsive to prior critiques. In particular, there is a description of literature selection; an approach to evaluating evidence for inferring causality is provided; and a reasonable set of descriptors of strength of evidence for causation is offered.

The CASAC made recommendations for improvement in the framework, such as to include consideration of publication bias, model selection bias, concentrations relevant to ambient levels, and common-causes (Henderson, 2008a).

Similarly, in 2008, the CASAC, augmented by subject-matter-experts to form the CASAC Sulfur Oxides Primary NAAQS Review Panel, likewise found that an early version of the framework in the first draft of the Sulfur Oxides ISA was promising but needed revisions (Henderson, 2008b):

The hierarchy of causal claims used in Chapter 5 is appropriate, but the criteria used to satisfy each of the categories of causal strength are not well specified and in some cases do not comport with best scientific practice. This aspect of the chapter can be improved, especially with

respect to criteria of coherence of evidence and robustness of conclusions. A complete description of the approach to causal inference should be provided in a revised ISA.

In its review of the second draft of the Sulfur Oxides ISA, CASAC found that (Henderson, 2008c):

*Chapter 1 has been improved, particularly by drawing on recent reports that offer models of approaches for causal inference and classification schemes for the weight of evidence for inferring causation. The ISA utilizes a five-level hierarchy for causal determination to be consistent with the Guidelines for Carcinogen Risk Assessment (EPA, 2005). We concur with using the five levels but recommend that the descriptions be changed to better reflect the level of certainty or confidence in the classification of the level of evidence.*

CASAC further advised that EPA “should avoid using statistical significance as a criterion for evidence interpretation,” and should improve “the presentation of the epidemiological concepts of effect modification and confounding that are particularly challenging in the face of multi-pollutant mixtures.”

In 2009, CASAC offered the following endorsement of the framework in its review of the first external review draft of the ISA for particulate matter (Samet, 2009a):

*The evidence is thoughtfully synthesized in a transparent fashion; the framework for classifying the strength of evidence has continued to evolve, and it provides transparency in documenting how determinations were made with regard to causation. The CASAC is particularly pleased that the Agency has adopted a uniform descriptive language for various levels of confidence in making causality determinations. We support the five-level hierarchy developed for causal determinations, and recommend it as the model for future ISAs.*

The CASAC went on to further state (Samet, 2009a): “The CASAC regards the framework for causal determination and judging the weight of evidence, as presented in Chapter 1, to be appropriate.”

In its review the second external review draft of the PM ISA, CASAC further stated (Samet, 2009b): “CASAC also commends EPA for the continued evolution of the process for evidence evaluation. The five-level classification of strength of evidence for causal inference has been systematically applied; this approach has provided transparency and a clear statement of the level of confidence with regard to causation, and we recommend its continued use in future ISAs.”

In 2009 the CASAC CO Review Panel advised EPA “as EPA receives comments on this framework when reviewed by various panels of CASAC, EPA should strive for consistency across documents” (Brain and Samet, 2009).

In 2010, the CASAC CO Review Panel found that (Brain and Samet, 2010): “EPA Framework for Causal Determination, now incorporates a detailed description of the criteria for causal determination. The introductory sentence to Section 1.6.3 clearly describes the process of moving from association to causation, requiring the elimination of alternative explanations for the association”. The CASAC went on to recommend more detail regarding confounding and

effect modification, and improved presentation of epidemiologic concepts include related to “available methods to control for confounding in the design and analysis phase of a study.”

In 2011, the Clean Air Scientific Advisory Committee (CASAC) augmented with additional experts to form the Ozone Review Panel reviewed the 1st draft of the Ozone ISA and stated (Samet, 2011):

*The CASAC continues to support the use of the EPA’s framework for causal determination that was first used in the ISA for particulate matter. This framework provides a comprehensive and transparent approach for evaluating causality. Based on long-standing approaches in public health, as brought together in a recent National Academy of Sciences (NAS) Institute of Medicine (IOM) report, the framework employs a two-step approach that first determines the weight of evidence in support of causation and then characterizes its strength in a standard scheme for causal classification. The second step further evaluates the available quantitative evidence regarding concentration-response relationships and the duration, level and types of exposures at which effects are documented. The EPA’s adoption of this framework has greatly improved the consistency and transparency of its assessment as compared to the approach seen in past reviews.*

The CASAC went on to further state “Panel members were largely satisfied with the framework for causal determination” while offering recommendations for further improvements pertaining to terminology, use of the “so-called Hill criteria” as a “guide to thinking about the data to ensure that relevant aspects of the data are adequately considered and taken as a whole rather than used as a checklist,” and that the “criteria not be ranked in any way; their relative importance will depend on the specific context and specific issue under consideration.”

In its review of the 2nd draft Ozone ISA, the CASAC augmented with additional experts had less to say about the framework itself, instead offering comments pertaining more to the explanation and application of the framework (Samet, 2012), thus indicating that the framework itself was mature and useful. CASAC called for EPA to provide a third draft of the ISA to address numerous other issues.

Likewise, in its review of the 1st draft ISA for Lead, the CASAC augmented with additional experts to form the Lead Review Panel also advised that “The framework for causal determination should be applied consistently and transparently,” thus affirming the utility of the framework itself but calling for improved explanation of its application to specific combinations of exposure duration and adverse outcome (Frey and Samet, 2011). The CASAC found that the 2nd draft ISA for Lead also had an “incomplete application of causal determination criteria outlined in the ISA’s preamble” and required further revision (Samet and Frey, 2012). In its review of the 3rd draft ISA for Lead, CASAC found that “the application of the causal framework is clearer and better documented” (Frey, 2013). One of the key issues in the lead review was to group health endpoints by major organ systems that share common modes of action.

In its review of the 3rd draft Ozone ISA, the CASAC found that the framework was well-developed and useful, leading to a recommendation to EPA staff to “consider developing the discussion of the causality framework into a manuscript for submission to a journal” (Frey and Samet, 2013).



In its review of the 1st draft of the ISA for Oxides of Nitrogen in 2014, the CASAC expressed concern that the framework was not “applied with sufficient transparency,” and advising that “there needs to be better substantiation and better documentation of the evidence and lines of reasoning for the causal determinations,” and offered specific recommendations for achieving improved transparency (Frey, 2014). CASAC found that the 2nd draft of the ISA for Oxides of Nitrogen “is a much improved document and is very responsive to the CASAC’s comments,” although offering specific suggestions for further improvements in the explanation of particular causal determinations (Diex Roux and Frey, 2015).

Given that CASAC comments pertaining to the framework for causal determination shifted over time from the formulation of the framework to its transparent application, the framework itself matured and remained unchanged in the most recent review cycle. The framework had been reviewed, improved, and endorsed by CASAC as a result of repeated review cycles, including the 2007 to 2010 review of oxides of nitrogen, 2007 to 2010 review of sulfur oxides, 2008 to 2013 review of particulate matter, 2009 to 2014 review of ozone, 2011 to 2013 review of lead, and 2013 to 2017 review of oxides of nitrogen. These review panels involved 66 different scientific experts. The review process further involved receipt of public comment at 14 public meetings for the review of each of the ISA drafts. Thus, the framework for causal determination has been extensively reviewed. Because the framework is generally applicable to reviews of each criteria pollutant, the framework is now described in a separate document, Preamble to the Integrated Science Assessments (EPA, 2015). The framework is also described in a journal publication by Owen et al. (2017).

In its review of the 1st draft ISA for oxides of sulfur, CASAC had extensive comments on specific causal determinations but did not have comments on the framework itself (Diex Roux, 2016). The CASAC review of the 2nd draft of the ISA for oxides of sulfur found that the causal determinations were appropriate (Diex Roux, 2017). The most recent sulfur oxides review panel included eight experts who had not served on previous panels that review the framework. Thus, the framework and its application has been evaluated by 74 experts over multiple panels and review cycles.

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## **History of Augmented Review Panels**

**The previous four particulate matter review panels have been comprised of members of the chartered CASAC augmented with additional expert consultants.** Based on the December 1982 EPA report on Air Quality Criteria for Particulate Matter and Sulfur Oxides (EPA-600/8-82-029a), CASAC was augmented with consultants. The CASAC Subcommittee on Health Effects of Particulate Matter and Sulfur Oxides included six consultants in addition to members of the chartered CASAC. The CASAC Subcommittee on Welfare Effects of Particulate Matter and Sulfur Oxides included five consultants in addition to members of the chartered CASAC. The consultants were different for these two review activities. Thus, there were 11 consultants who augmented the chartered CASAC for this review cycle. For the 1994 to 1996 PM review, there were 6 members of the chartered CASAC and 15 additional experts on the review panel. For the 2001 to 2006 scientific review, and for the 2008 to 2010 scientific review, there were 7 members of the chartered CASAC and 15 additional experts. From 2015

to 2018, the CASAC Particulate Review Panel had 6 members of the chartered CASAC and 20 additional experts. Thus, the use of augmented ad hoc review panels for particulate matter dates back more than 35 years.

**Table 1 summarizes data regarding ad hoc review panels for review of primary standards for all six criteria, based on review of the CASAC reports to the EPA administrator for each review cycle for each pollutant.** For many of the earlier review cycles in the late 1970s and in the 1980s, the letter reports from CASAC do not list the members of the chartered CASAC or consultants who augmented CASAC. Thus, it was not possible to compile data for every CASAC review of a primary or secondary standard. However, data are available for 20 CASAC reviews of primary standards dating to as early as 1987.

**Table 1. Number of CASAC Members and Consultants for NAAQS Review Panels by Topic and Dates<sup>a</sup>**

Review	Primary or Secondary	Years	CASAC Members	Consultants	Total
CO Review	P	1999 to 2000	7	5	12
CO Review	P	1991 to 1992	6	5	11
CO Review Panel	P	2008 to 2010	3	13	16
Lead Review Committee	P,S	1986 to 1990	7	12	19
Lead Review Panel	P,S	2006 to 2008	7	17	24
Lead Review Panel	P,S	2011 to 2013	2	18	20
NOx and Sox Secondary Review Panel	S	2008 to 2011	4	12	16
NOx and Sox Secondary Review Panel	S	2013 to present	1	21	22
Oxides of Nitrogen Review Panel	P	2007 to 2009	7	17	24
Oxides of Nitrogen Review Panel	P	2013 to 2017	4	13	17
Ozone Review Committee	PS	1987 to 1992	7	12	19
Ozone Review Panel	P,S	1995 to 1996	6	10	16
Ozone Review Panel	P,S	2005 to 2008	7	18	25
Ozone Review Panel	P,S	2010 to 2014	7	13	20
PM Review Panel	PS	1994 to 1996	6	15	21
PM Review Panel	PS	2001 to 2006	7	15	22
PM Review Panel	PS	2008 to 2010	7	15	22
PM Review Panel	PS	2016 to 2018	6	20	26
Sulfur Oxides Panel	P	2007 to 2010	7	17	24
Sulfur Oxides Panel	P	2013 to 2018	6	16	22

<sup>a</sup>All of this information was obtained from [www.epa.gov/casac](http://www.epa.gov/casac) by reviewing CASAC reports posted online.

**Table 2. Summary of Primary NAAQS Review Panels By Number of Consultants<sup>a</sup>**

Description	Number
Consultants: 16 to 20	8
Consultants: 12 to 15	9
Consultants: 5 to 10	3
Total	20

<sup>a</sup>All of this information was obtained from [www.epa.gov/casac](http://www.epa.gov/casac) by reviewing CASAC reports posted online.

As shown in Table 1, although there are a few panels with only 5 to 10 additional expert consultants, it has been more typical that the chartered CASAC has been augmented with 12 or more additional experts in a given review cycle for a given criteria pollutant. **The average number of consultants for these 20 panels is 14, and the average size of the augmented ad hoc review panels is 20 members. The averages for ozone and PM review panels are 15 consulting experts and panels with a total of 21 members.**

As shown in Table 2, of 20 panels for which data could be characterized regarding the number of consultants who comprised review panels, 3 had 5 to 10 consultants, 9 had 12 to 15 consultants, and 8 had 16 to 20 consultants.

The use of augmented panels or subcommittees dates at least to the late 1970s. On October 9, 1979, the Subcommittee on Carbon Monoxide of the CASAC issued its “findings, recommendations and comments.” However, a list was not included of members of that subcommittee. Based on the December 1982 EPA report on Air Quality Criteria for Particulate Matter and Sulfur Oxides (EPA-600/8-82-029a), CASAC was augmented with consultants. There were 11 consultants who augmented the chartered CASAC for this review cycle. The dates on which these subcommittees met are not readily available, however.

Therefore, although there are not as many details available in the public record to quantify the membership or meeting dates of either subcommittees or augmented panels prior to 1987, there is evidence in the public record that **augmentation of CASAC with additional experts has been a routine practice for four decades.**

### **Integrated Science Assessment**

In our December 10, 2018 letter to CASAC and the EPA docket for the draft Integrated Science Assessment, we offered consensus advice on numerous issues related to the draft ISA. The failure of EPA to provide a second external review draft of the ISA compromises the credibility and integrity of the NAAQS review process. This is because there were many important scientific issues raised regarding the first external review draft that require revision and iteration prior to their application in risk and exposure assessment and prior to their interpretation in the policy assessment. Although we found that the draft ISA was a comprehensive scientific document, we identified numerous areas for which refinement or revision was needed as detailed in our December 10, 2018 letter to CASAC. These areas include low cost sensors, air quality, contrasts between PM<sub>2.5</sub> and UFP, coarse PM, PM components, onroad and near-road microenvironments, mixtures and copollutants, study selection, transparent application of the causal framework, more in-depth treatment of specific issues related to PM<sub>2.5</sub> and mortality,

more explanation and possible reconsideration of the causal determination for short-term exposure to coarse PM and respiratory adverse effects, more explanation and possible reconsideration of the causal determination for long-term exposure to UFP and central nervous system effects, and reconsideration of the at-risk causal finding for populations with pre-existing cardiovascular or respiratory disease. Members of the IPMRP also provided extensive individual comments that were attached to the December 10, 2018 letter from the panel.

In our March 27, 2019 letter to CASAC, we noted that “the framework for causal determination, including terminology, and the overall plan for development of the ISA, was reviewed by CASAC in 2016.” However, we strongly disagreed with statements in CASAC’s draft letter to the Administrator “that the Draft ISA lacks explicitly stated principles for drawing conclusions or lacks operational definitions.” We noted that “the various considerations in developing causal determinations are explained in the Preamble to the ISAs and have been considered already in CASAC’s review of the Draft Integrated Review Plan.” We further noted that “[w]hile there may be opportunities for EPA staff to improve the clarity and transparency of the explanations of the inferences it makes and the conclusions it draws, this is not a fundamental limitation of the underlying framework but rather a matter of routine scientific review and iteration to improve the clarity and transparency of the final document.”

The chartered CASAC developed comments that in many cases appeared to exclusively focus on doubt-raising without acknowledgment of inferences that can be supported by the scientific evidence. In our March 27, 2019 letter, the IPMRP stated that “it is inappropriate to over-emphasize or exclusively focus on discordant results and ignore the overall preponderance of the evidence when making inferences.”

The IPMRP further stated that the draft ISA “follows methods previously reviewed by CASAC, including the approach to literature review, the causal determination framework, the framework for assessing at-risk populations and life stages, and assessment of concentration-response functions, consistent with the Preamble to the ISAs and the 2016 Integrated Review Plan for the current review cycle.” Consistent with our December 10, 2018 comments, we noted on March 27, 2018 that “the ISA takes into account poverty, temperature, and season, including lags related to temperature, and makes inferences regarding whether ambient PM concentration independently causes adverse effects and whether concentration and response relationships are either confounded or modified by other variables. Some of these inferences could be explained more clearly or in more detail.”

The draft PA appears to accept the draft ISA as it was prior to external review by CASAC and the public, including the IPMRP. There is no summary in the draft PA of any changes that are being made to the draft ISA as a result of comments from CASAC and the public, including the IPMRP. Normally, in prior review cycles, there is a second external review draft of the ISA concurrent with a first review draft of the Risk and Exposure Assessment (REA). In this review cycle for PM, EPA has not produced a separate draft REA, but instead has subsumed the REA into the draft PA. Typically, in a normal review cycle, the draft PA would not be released until after EPA has finalized the ISA and completed a second draft of the REA. The typical sequence in a normal review cycle was intended to protect the science assessments from being commingled with the policy assessment, so that the scientific basis could be established irrespective of later policy interpretations. In the current review cycle, the fact that the ISA is not completed prior to external review of the draft PA provides EPA leadership with the opportunity to change the ISA to support pre-determined policy outcomes in the final PA. This is a completely unacceptable situation.

Based on the content of the draft PA, it is clear that EPA staff have elected to retain the causal determination framework for health effects attributed to exposures of varying durations to particular indicators, and to retain the causal framework for at-risk populations. This is an appropriate choice. Although the chair of CASAC has aggressively advocated that EPA adopt quantitative causal tests for individual studies based on the chair's own work, such methods have not been adequately vetted and are not ready for widespread use at this time. The merits of such proposals could be a research topic that may be informative in future review cycles. It is certainly the case that leading edge research in the field of air pollution epidemiology is concerned with potential threats to validity of making inferences as well as adoption of improved techniques that better account for confounding and modification and that help support inferences regarding causality. However, because CASAC does not have epidemiologists among its seven members, and does not have access to a sufficient number of epidemiologists with breadth, depth, and diversity of expertise and experience, this CASAC is hardly an appropriate authority on the state of epidemiological practice and science and the directions it should go.

***EPA-1. Chapter 1 – Introduction: To what extent does the CASAC find that the information in Chapter 1 is clearly presented and that it provides useful context for the review?***

The draft PA, Chapter 1, fails to document the ad hoc changes to the NAAQS review process and to the CASAC that have been made since the final Integrated Review Plan (IRP) was published in 2016.<sup>49</sup> Table 1-3 of the final IRP laid out the following schedule for the review of the PM NAAQS:

- Fall 2017: Release of first external review draft of the ISA  
Release Risk and Exposure Assessment (REA) planning document(s)
- Winter 2018: CASAC Review of First Draft ISA, REA Planning Documents
- Fall 2018: Release of second external review draft of the ISA  
Release of First Draft REAs  
Release of First Draft PA
- Winter 2019: CASAC Review of Second Draft ISA, First Draft REAs, and First Draft PA
- Fall 2019: Release Final ISA  
Release of Second Draft REAs  
Release of Second Draft PA
- Winter 2020: CASAC Review of Second Draft REAs, Second Draft PA
- Fall 2020: Final REAs, Final PA
- 2021 Proposed Rule
- 2022 Final Rule

Compared to the IRP, the following steps have been omitted in the current review: (a) no REA planning document(s); (b) no second external review draft of the ISA; (c) no external review drafts of the REAs; (c) no provision for a second draft of the PA; (d) no final REA as a separate document; and (e) no final ISA until after CASAC has completed its review of the draft PA.

Although the IRP is cited on page 1-1, line 7, the deviations of the current review from the IRP are complete omitted. This is inappropriate and should be corrected. The chapter should enumerate all of the changes to the NAAQS review process, the CASAC, and the PM NAAQS review since 2016. See my detailed comments above on process issues.

The schedule in the final IRP specified two drafts of each of the ISA, REA, and PA. However, the final IRP indicated that the drafts of the REA and PA would be concurrent. This differs from

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<sup>49</sup> See Reference 9.

the schedule in the external review draft of the IRP that was reviewed by CASAC earlier in 2016.<sup>50,51</sup> In the external review draft of the IRP, EPA had proposed to sequence the release of first drafts of the ISA, REAs, and PA such that CASAC would review them sequentially on a staggered schedule. Thus, under the initial proposed schedule, CASAC would have been able to provide its advice on the first draft of the REAs before receiving the first draft of the PA. The schedule in the draft IRP allowed for two drafts of each of the ISA, REA, and PA.

The final IRP sequencing of the first drafts of the REA documents such that they are released after receiving CASAC review of both the first draft of the ISA and of REA planning documents is appropriate. Since the REAs build upon information in the ISA, it is logical and appropriate that EPA consider CASAC's advice on the ISA before releasing a draft of the REAs.

Because the Policy Assessment is intended to integrate information from the ISA and the REAs, it is generally not appropriate for a first draft of the PA to be released at the same time as the first draft of the REAs. Simultaneous release of the first draft of the REAs and PA was done, for example, in the last review of the ozone NAAQS. As colleagues have pointed out (see November 26, 2016 letter to CASAC from former members of the 2009 to 2014 CASAC Ozone Review Panel), the first draft of the PA in that review was very preliminary and required substantial revision.<sup>52</sup> Transparency of the review process and clear distinction of science and policy issues is enhanced by obtaining CASAC's advice on the REAs before submitting a first draft of the PA for CASAC review.

However, in this review, there is no separate REA. The content of the REA has been incorporated into the draft PA. This is not appropriate since there are important scientific issues pertaining to the REA that should be reviewed and vetted prior to use in the PA.

Chapter 1 should clearly explain the difference between the sequence of draft documents indicated in the IRP versus the actual sequence of draft documents in this review. Rather than multiple drafts of the ISA, REA, and PA, staggered so that science issues are vetted and settled before proceeding to policy issues, this review cycle has devolved into one draft of the ISA and one draft of the PA.

The draft of the PA is being reviewed before the ISA has been finalized. Whether or how issues raised by CASAC and the public regarding the draft ISA will be resolved, if at all, are unknown. What changes, if any, are in progress for the draft ISA, and which of these changes affect content of the draft PA? For example, the draft PA argues that focus should be given to health effects causal determinations that are "causal" or "likely to be causal" in assessing the adequacy of the current primary standards with regard to protection of public health and in assessing

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<sup>50</sup> EPA, Draft Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter, EPA-452/D-16-001, U. S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711. <https://www3.epa.gov/ttn/naaqs/standards/pm/data/201604-draft-integrated-review-plan-casac-review.pdf>

<sup>51</sup> Diez Roux, A., "CASAC Review of the EPA's Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – April 2016)," EPA-CASAC-16-003, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, August 31, 2016. <https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/%24File/EPA-CASAC+2016-003+unsigned.pdf>

<sup>52</sup> Frey, H.C., J.M. Samet, A.V. Diez Roux, G. Allen, E.L. Avol, J. Brain, D.P. Chock, D.A. Grantz, J.R. Harkema, D.J. Jacob, D.M. Kenski, S.R. Kleeberger, F.J. Miller, H.S. Neufeld, A.G. Russell, H.H. Suh, J.S. Ultman, P.B. Woodbury, and R. Wyzga, "CASAC Advice on the EPA's Integrated Review Plan for the Ozone National Ambient Air Quality Standards (External Review Draft)," 24 page letter with 42 pages of attachments, submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA-HQ-OAR-2018-0279, November 26, 2018, [https://yosemite.epa.gov/sab/sabproduct.nsf/0AC9E8672B0CA54985258351005BE54F/\\$File/Ozone+Letter+181126+Submitted-rev2.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0AC9E8672B0CA54985258351005BE54F/$File/Ozone+Letter+181126+Submitted-rev2.pdf)



possible revised or new standards. The draft ISA posits a determination of “likely to be causal” for long-term exposure to UFP and central nervous system effects. Yet, it seems that this finding is not adequately addressed in the draft PA. Is this because the finding may be revised downward in the final ISA? Or, is the finding in the final ISA to later be revised downward to match a pre-determined policy outcome from the PA? The commingling of science and policy by having so much overlap in the timing between the draft PA and draft ISA, at a minimum, creates the perception that the final ISA may be tailored to match policy outcomes in the final PA that were determined before the ISA was completed.

As noted on page 1-1, line 25, the role of the PA is to “bridge the gap” between the scientific assessments, which include not just the ISA but also REAs, and judgments required of the Administrator. The fact that the science has not been appropriately vetted prior to the release of the draft PA is problematic, as noted above.

Page 1-2, lines 9-11. Should also acknowledge that CASAC is to advise on background levels and research needs.

Page 1-2, lines 12-13: There is not a separate Risk and Exposure Assessment (REA) document in this review. To be consistent with the final IRP for this review, the text should state that EPA intended to make available to CASAC and the public two drafts of the REA. The most appropriate sequence of documents is to have the first draft of the ISA reviewed and revised prior to a first draft of the REA. The first draft of an REA should be made available and reviewed before a first draft of the PA is released. This was the situation in the most recent prior review of the PM NAAQS, for which there was a separate health risk and exposure assessment (HREA) and a welfare risk and exposure assessment (WREA).<sup>5354</sup> The latter was focused on visibility. In a few cases, the REA (HREA, WREA, or both) has been combined into the PA, such as for the most recent lead NAAQS review.<sup>55</sup> However, in such cases, this is because there were no substantial updates to the REA compared to the prior review cycle. In the case of the current PM NAAQS review, there are clearly substantial updates that have led to an entirely new REA in this review. This draft PA is not based on a reinterpretation of the REA from the prior review cycle. Instead, a new REA for health effects is included in the draft PA appendices. However, the REA should have been provided separately from the draft PA. The draft REA should have been provided for review after considering CASAC and public comments on the draft ISA and before releasing a draft PA.

Page 1-3, lines 9-11: Given that CASAC has been populated with members appointed based on geographic location and government affiliation, and that CASAC has been deprived of a duly appointed CASAC PM Review Panel, CASAC is not qualified to advise the EPA in a manner

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<sup>53</sup> Samet, J.M., CASAC Review of Quantitative Health Risk Assessment for Particulate Matter – Second External Review Draft (February 2010), EPA-CASAC-10-008, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, April 15, 2010.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/BC4F6E77B6385155852577070002F09F/\\$File/EPA-CASAC-10-008-unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/BC4F6E77B6385155852577070002F09F/$File/EPA-CASAC-10-008-unsigned.pdf)

<sup>54</sup> Samet, J.M., CASAC Review of Particulate Matter Urban-Focused Visibility Assessment – Second External Review Draft (January 2010), EPA-CASAC-10-009, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, April 20, 2010.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/0D5CB76AFE7FA77C8525770D004EED55/\\$File/EPA-CASAC-10-009-unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0D5CB76AFE7FA77C8525770D004EED55/$File/EPA-CASAC-10-009-unsigned.pdf)

<sup>55</sup> Frey, H.C., CASAC Review of the EPA’s Policy Assessment for the Review of the Lead National Ambient Air Quality Standards (External Review Draft – January 2013), EPA-CASAC-13-005, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, June 4, 2013,  
[https://yosemite.epa.gov/sab/sabproduct.nsf/E2554E264EEF8CCB85257B80006B3014/\\$File/EPA-CASAC-13-005+unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/E2554E264EEF8CCB85257B80006B3014/$File/EPA-CASAC-13-005+unsigned.pdf)

that accurately reflects that latest scientific knowledge of the kind and extent of salient issues that must be considered.

Page 1-3, lines 23-24. The text should also cite the recent *Murray Energy v. EPA* decision of the Court of Appeals for the District of Columbia Circuit.<sup>56</sup> As stated in the court's decision, "[i]ndustry Petitioners also point to section 109(d)(2)(C) of the Act, which requires CASAC to advise EPA 'of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance' of revised NAAQS. 42 U.S.C. § 7409(d)(2)(C). According to Petitioners, the fact that CASAC is required to supply information to EPA about the 'social, economic, or energy effects' of the revised NAAQS implies that EPA is obliged to consider that information in setting the NAAQS." However, contrary to the petition, this decision reaffirms that "this provision was intended to 'enable the [EPA] to assist the States in carrying out their statutory role as primary implementers of the NAAQS,' but had 'no bearing upon whether cost considerations are to be taken into account in formulating the [NAAQS].'"

Page 1-4, lines 17-18: Per *Murray Energy v. EPA* (2019), background is simply irrelevant in setting the level of the NAAQS. The level of the NAAQS must be set based on health effects. Proximity to background may be an issue for implementation.

Page 1-4, lines 28-29: Given that CASAC lacks the breadth, depth, and diversity of expertise necessary for this review, which was embodied in the disbanded CASAC PM Review Panel, CASAC is poorly positioned to offer advice on "recent advanced in scientific knowledge on the effects of the pollutant on public health and welfare."

Page 1-5, lines 1-17. See also CASAC's charter with the U.S. Congress, which should be cited.

Page 1-10, lines 8-10: the text here regarding the establishment of a Federal Reference Method for measurement of ambient coarse PM sets an important precedent. EPA should establish a FRM for measurement of UFP.

Page 1-11, line 6. The NAAQS review process was revised in 2006 and then again in 2008 and again in 2009. The 2006 revision was the major revision. The revisions in 2008 and 2009 were incremental changes of the process established as a result of the 2006 revision. The text should be rewritten to more accurately convey this sequence of events, with citations.

Page 1-12, lines 15-19. Although the IRP has been followed in part, there have been substantial deviations from the IRP. The deviations from the IRP should be specifically enumerated and discussed. See my comments above on this point.

Page 1-12, lines 20-22. This memorandum contradicted EPA's own IRP for this review. See comments above.

Page 1-12, line 23. Should note that on October 10, 2018, the CASAC PM Review Panel was disbanded by Acting Administrator Wheeler. The draft ISA was released on October 15, 2018.

Page 1-12, lines 24-25. Please give the dates of the meetings.

Page 1-12, line 33. What changes are being made to the draft ISA in response to comments from CASAC and the public. How will changes in the ISA be incorporated into the draft PA? What is the rationale for depriving CASAC and the public of the opportunity to see a revised draft ISA before the PA is finalized? Related to this issue, is EPA under a court order or a consent decree to complete the PM NAAQS review by 2020?

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<sup>56</sup> <https://law.justia.com/cases/federal/appellate-courts/cadc/15-1385/15-1385-2019-08-23.html>

***EPA-2. Chapter 2 – PM Air Quality: To what extent does the CASAC find that the information in Chapter 2 is clearly presented and that it provides useful context for the review?***

Specific comments on Chapter 2:

Page 2-3, line 17: text should be more clear if this is specifically about primary PM emissions. Aside from stationary and mobile sources, should mention area sources and fugitive emissions. At an appropriate place, should more systematically also address sources of secondary PM precursors.

Page 2-3, line 23, should add NO<sub>x</sub> and NH<sub>3</sub> to the parenthetical note about SO<sub>2</sub>).

Page 2-7: the definitions of and distinctions between elemental carbon and black carbon should be discussed. Given that this is a topic that probably has no end, EPA could acknowledge that there are differences of opinion about the use of these terms and offer an operational definition for use here. Also related to this page, a figure that apportions PM<sub>2.5</sub> to the components of section 2.1.1.3 would be useful, such as based on a typical average for a selected year. This would help put into context information in Figure 2.5 and elsewhere... e.g., how much do EC and OC each contribute to PM<sub>2.5</sub> mass on average, and what is the variability in this contribution (e.g., inter-city? Inter-monitor? Inter-annual?) Inter-daily?).

Page 2-9, lines 9-11. To be more clear, what is meant by “or can form new particles”? Is this via condensation?

Page 2-9, lines 16-17: This text appears to be correct but may give a misleading impression. EGUs appear to be responsible for 69% of national SO<sub>2</sub> emissions in 2014, not 80%. The reader might interpret that “nearly all” of the 80% is from EGUs, which appears not to be the case. 69% is not “nearly all” of 80%.

Page 2-9, line 19: According to the emissions trend data reported by EPA,<sup>57</sup> the total NO<sub>x</sub> emitted in 2014 was 12.589 million tons, not 14.4 million tons. Please check the number and correct as appropriate.

Page 2-9, line 24: it would help to give some quantitative idea of what “significantly” means... i.e. more than X%? Between Y% and Z%?

Page 2-9: related to the content here, it would be useful to either have similar content regarding components of UFP, PM<sub>10</sub>, and PM<sub>10-2.5</sub> or some explanation of the lack of such data. This could be a paragraph on each.

Page 2-11, line 12: What is a “robust” national network? How is “robust” defined, quantified, and assessed?

Related to Page 2-11: A statement should be made that there is not a Federal Reference Method for Ultrafine Particles. Such a statement is important because a future research need is to obtain more ambient monitoring data over space and time for UFPs to support epidemiology based on UFP. Given that EPA has in the past established FRMs in anticipation of possible new indicators, it will be appropriate to provide a rationale for establishing a FRM for UFP.

Page 2-12, Figure 2-6. What are the values on the vertical axis? Are these the number of stations? Axes should be explicitly defined with axes labels.

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<sup>57</sup> <https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data>

Page 2-5, top of the page. Please add a paragraph regarding the precision and accuracy of FRM and FEM monitors for PM<sub>2.5</sub>, particularly for annual averages down to 8 µg/m<sup>3</sup> and perhaps as low as 5 µg/m<sup>3</sup>.

Page 2-18, top of page. This example of the development of an FRM for PM<sub>10-2.5</sub> is a good. An FRM should similarly be developed for UFP.

Page 2-19, line 7: I think this probably is supposed to be “country” rather than “county”.

Page 2-19: monitoring methods related to ultrafines should also be briefly summarized.

Page 2-20, top of page. What are the demonstrated uses of sensor technologies for improved spatial resolution of ambient concentration or exposure concentrations, if any, for UFP, PM<sub>2.5</sub>, PM<sub>10</sub>?

Page 2-24, 4<sup>th</sup> line from the bottom (there are no line numbers): I could not find the “design value ratio line” in Figure 2-11.

Page 2-28, bottom paragraph, continued to next page – this is very useful information. Agree that there are decreasing trends in near road PM<sub>2.5</sub> increments related to fleet turnover of heavy duty diesel trucks that is leading to increased diffusion of diesel particle filters into the onroad fleet.

Page 2-38: the text refers to the accuracy and precision of publicly available data without any quantification. It would help to say something more on this topic, earlier (see comment above about the precision and accuracy for annual average concentrations down to 5-8 µg/m<sup>3</sup>.)

Page 2-41: the discussion and treatment of this material regarding the performance of alternative hybrid modeling methods seems appropriate. The text points out that the hybrid model performance tends to be worse in parts of the western U.S and attributes this, in part to “low concentrations.” Please see Dr. Barbara J. Turpin’s comments on this issue. The performance of the modeling approaches is perhaps more related to how well the models represent spatial gradients as opposed to how well they can represent “low concentrations.” In areas with stronger spatial gradients, finer resolution models perform better, including at low concentration, whereas in areas with little to no spatial gradients, models at fine and coarse scale may have comparable performance.

Page 2-42, line 30: The text here seems a bit superficial and could be supported with more specifics.

Page 2-43, line 8: What is the interpretation/implication/significance of information given in Table 2-3? Or, if the text immediately above is in reference to this table, then the table should be cited earlier in this paragraph.

Page 2-44, line 8. What is meant by spatial “texture”? Avoid metaphors in formal technical writing. Perhaps this is referring to a spatial ‘distribution’?

Page 2-44, lines 11-14: This is a good summary of comparisons, but what is the assessment based on this information? Which of these results are more plausible?

Page 2-45, line 7: Coefficient of variation of what? And for what averaging time? In general, always indicate averaging time when reporting concentrations or concentration-derived metrics.

Page 2-49: It appears that the assessment of background PM is largely based on results from the prior review. Is there anything new that can be learned from the hybrid modeling work that could inform some of this discussion?

Page 2-49, lines 33-35: it would be useful to mention some of the dynamics of UFP that are mentioned in the draft ISA – e.g., that they are more dynamic and have spatial gradients near

sources, in part because they agglomerate to larger size ranges and thus are transformed out of the UFP size range. This has implications for the characterization of UFP background, which could be discussed.

Also, the background discussion should differentiate based on averaging times, notably daily average and annual average.

Page 2-52: what about transboundary PM precursors, such as SO<sub>2</sub>, NO<sub>x</sub>, NH<sub>3</sub>, and VOCs? Although there is some mention of a few of these, these could be treated more systematically in the text.

Minor comment: change “like” to “such as” – e.g., page 2-2, line 8.

***EPA-6. Chapters 3 to 5: What are the CASAC views regarding the areas for additional research identified in Chapters 3, 4 and 5? Are there additional areas that should be highlighted?***

This charge question should have also included reference to Chapter 2. EPA should develop a Federal Reference Method for Ultrafine Particles. There is need for ongoing comprehensive characterization of the performance of modeled ambient concentration fields estimated using hybrid modeling methods.

## Dr. Terry Gordon

SCQ-3.2 What are the Panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?

The draft PA appropriately gives the evidence-based approach the deserving amount of weight to using those studies that "demonstrate a causal or a likely to be causal relationship with PM exposures" in the risk estimates. The choice and presentation of health outcomes was logical and well written. Similarly, the risk-based approach was clearly written and well-balanced, thus permitting the logic and presentation of the conclusions and recommendations in a fair and balanced setting. In particular, the weight of the different categories of evidence was well delineated between the studies with new evidence to suggest adverse health outcomes at levels below the current standards.

SCQ 3.3 What are the Panel's views on the evidence-based approach, including:

- a) The emphasis on health outcomes for which the draft ISA causality determinations are "causal" or "likely causal"?

The emphasis on causal and likely causal health outcomes was very appropriate. The designation of nervous system effects to a likely causal level was well described. The designation of birth outcomes/reproduction as "suggestive", however, is puzzling given the large amount of epidemiologic studies that show associations between these outcomes and ambient PM. Admittedly, this field is rapidly expanding and perhaps the ISA needs updating.

- b) The identification of potential at-risk populations?

The at-risk populations are appropriate as identified.

- c) Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?

This reviewer agrees that the reliance on US and Canadian epidemiology studies is the correct approach given the potential for different PM composition and sources among continents/countries.

- d) Characterizing air quality in these key studies using two approaches: the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the "pseudo design value" reflecting a monitor with the highest levels in an area?

These approaches seemed appropriate and balanced.

- e) The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?

This preference was presented in a logical fashion and is correct.

- f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?

This reviewer agrees that the current scientific evidence strengthens the conclusions of the last review and, in particular, provides new epidemiological evidence of adverse health outcomes at or below the current standards.

- g) Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?

These issues were appropriately discussed and communicate.

SCQ-4.1 To what extent does the panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>10</sub> NAAQS review? Are there additional policy-relevant questions that should be addressed?

This chapter did an excellent job of presenting the important policy-relevant issues. This reviewer can think of no other policy-relevant questions.

SCQ-4.2 What are the Panel's views of the draft PA assessment of the currently available scientific evidence regarding the health effects associated with exposures to thoracic coarse particles, PM<sub>10-2.5</sub>?

Based upon the currently available evidence, as stated in the draft ISA, the draft PA presents a reasonable assessment.

SCQ-4.3 What are the Panel's views on the draft PA preliminary conclusion that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports consideration of retaining the current standard?

This reviewer agrees that based upon the available evidence, there is not need to question the adequacy and the evidence does support that the Administer consider retaining the current PM<sub>10-2.5</sub> standard.

EPA-6 Chapters 3 to 5: What are the CASAC views regarding the areas for additional research identified in Chapters 3, 4 and 5? Are there additional areas that should be highlighted?

The designated areas are excellent although, even as an inhalation toxicologist, to be honest, it is unclear how much mechanistic studies will impact this or future PM NAAQS. It would be more impactful to emphasize research on associations of individual sources with adverse health outcomes, so states/regions could perhaps focus on the 'worst' polluters. In particular, more research is needed on traffic (i.e., pollution vs. noise/stress; environmental justice), coal emissions, and wildfire contributions to adverse health effects.

## Dr. Jack R. Harkema

### General Comments

Overall, Chapters 3 and 4 are well written and address the charge questions mandated for this PA. The authors have provided the needed policy-related assessments that are based on the key findings provided by studies identified in the ISA.

SCQ-3.3. I agree with the EPA's evidence-based approach including the emphasis on health outcomes deemed *causal* or *likely to be causal*.

SCQ-3.5. I agree with the draft PA preliminary conclusion that, *taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards*.

SQ-3.6. *I would agree that new PM<sub>2.5</sub> alternative standards need to be developed*. First, the levels above 10 µg/m<sup>3</sup> for the annual standard are not protective of public health, and an annual standard in the range of 10 to 8 µg/m<sup>3</sup> would provide more protection, but will not eliminate substantial premature mortality effects, especially in susceptible population subgroups. Second, a 24-hour standard in the range of 30 to 25 µg/m<sup>3</sup> would provide additional protection. Third, the EPA should work towards developing a rolling 4-hour standard, instead of a midnight to midnight average interval. Fourth, based on substantial scientific evidence of the health effects of ultrafine particles near roadways, the EPA should develop Federal Reference Methods for these specific particulate pollutants.

A few specific comments and suggestions are listed below that are intended to strengthen the document for the administrator.

### Specific Comments.

#### Chapter 3.2

Table 3-1 (p 3-18). The footnote for this table is quite unusual and raises questions and concerns. I suggest deleting this footnote in the final PA. As currently written, it implies that CASAC did not provide comments and suggestions to the EPA authors in a timely manner so that they could fully refine this part of the PA. Since there will be no additional review of the ISA document there will be adequate time for the authors to thoroughly evaluate and respond to the CASAC's additional comments/suggestions on the causality determinations stated in this table. I suggest the authors continue to base their causality determinations on the weight of the scientific evidence. To this reader, all the causality determinations are appropriately defended in the text but could be better summarized in the table (see below).

Table 3-1. This table would be improved with a column for key determinates (rationale points) for each causality. This would nicely reiterate and summarize the discussion in the text.

#### Chapter 3.3 Risk Based Considerations



The initial subsections (e.g., approach) of this part of Chapter 3 contain technical risk assessment jargon that could be eliminated or carefully defined for the lay person (non-risk assessor).

A summary table for the suggested changes or no changes to the PM<sub>2.5</sub> standards (including indicator, averaging time, form, and level) this section would complement the text and help the reader understand the authors' conclusions and rationales.

#### Chapter 3.5

I would suggest adding the following future areas of research

- More state-of-the-art comparative toxicological studies (in vivo and in vitro) that are designed to determine 1) the similarities and differences in human and animal sensitivity to comparable concentrations/doses of PM exposure (species-dependent responses, the animal may have a greater or lesser response to the same dose of inhaled PM) and 2) the cellular and molecular mechanisms underlying the adverse health effect. This will enhance our ability to translate animal toxicology findings to human health concerns and provide plausible and advanced biologic mechanisms for epidemiological associations.
- Studies to better understand PM exposure-related associations with neurological, metabolic and autoimmune diseases (e.g., autism, depression, diabetes, pre-diabetic disorders, systemic lupus erythematosus).

#### Chapter 4.1-4.3

No additional comments.

## **Dr. Patrick Kinney**

### **Section 3.2 Evidence-Based Considerations**

Overall this section is well done. However, I do have a serious concern about the footnote to Table 3-1 on page 3-18. The table lists causality determinations in the 2009 PM ISA and 2018 draft ISA. These provide a central foundation for the entire chapter on primary NAAQS recommendations. The footnote says that the table does not reflect CASAC advice on the draft ISA and that “some or all of these causality determinations could differ in the final ISA.” If interpreted literally, this clause opens the door for a complete revision to the evidence on causality which then feeds into the discussions and recommendations regarding the primary NAAQS. This seems like a sort of poison pill for the entire section, which as I said is very well done.

Page 3-61, line 9, and elsewhere in this section. The statements about PM concentrations “around”, i.e., “somewhat below to somewhat above” the overall mean observed in the key long-term epidemiology studies is rather vague. I am pleased to see that this notion is made more explicit on the following page, line 7, where there is a suggestion to use the 10<sup>th</sup> or 25<sup>th</sup> percentile of the health or concentration distribution to define the lower bound of the data region in which epi results are most precise. These are then plotted in figures 3-7 and 3-8, which is very helpful.

The pseudo-design value analysis starting on page 3-70 provides a useful complement to the previous sections.

### **Section 3.3 Risk-based considerations**

Again, this section is well done, incorporating an appropriate set of inputs and assumptions to examine health outcomes which might occur under a range of assumptions regarding the primary NAAQS.

**SCQ-3.2 What are the Panel’s views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?**

Both sets of evidence are given appropriate weight in the draft PA.

### **Section 3.4 Preliminary Conclusions on the Primary PM<sub>2.5</sub> Standards**

This section accurately recaps and summarizes the evidence, analyses and arguments that were presented in sections 3.2 and 3.3. The draft PA reaches the following appropriate conclusions starting at the bottom of page 3-97.

- There is a long-standing body of strong health evidence demonstrating relationships between long- or short-term PM<sub>2.5</sub> exposures and a variety of outcomes, including mortality and serious morbidity effects. Studies published since the last review have reduced key uncertainties and broadened our understanding of the health effects 1 that can result from exposures to PM<sub>2.5</sub>.

- Recent U.S. and Canadian epidemiologic studies provide support for generally positive and statistically significant health effect associations across a broad range of ambient PM<sub>2.5</sub> concentrations, including for air quality distributions with overall mean concentrations lower than in the last review and for distributions likely to be allowed by the current primary PM<sub>2.5</sub> standards.
- Analyses of PM<sub>2.5</sub> pseudo-design values additionally support the occurrence of positive and statistically significant health effect associations based largely on air quality likely to have met the current annual and 24-hour primary standards.
- The risk assessment estimates that the current primary PM<sub>2.5</sub> standards could allow a substantial number of PM<sub>2.5</sub>-associated deaths in the U.S. The large majority of these estimated deaths are associated with the annual average PM<sub>2.5</sub> concentrations near (and above in some cases) the average concentrations in key epidemiologic studies reporting positive and statistically significant health effect associations.

When taken together, we reach the preliminary conclusion that the available scientific evidence, air quality analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the combination of the current annual and 24-hour primary PM<sub>2.5</sub> standards.

This material is then followed by a section that presents an alternative, more skeptical, interpretation of the evidence, highlighting uncertainties in biological pathways, potential for public health improvements below the current NAAQS (because accountability studies haven't examined those levels yet), and in risk assessment as a tool. This is a rather extreme interpretation that runs counter to most current scientific views of the available evidence. However, it does provide the administrator considerable scope in evaluating the primary PM<sub>2.5</sub> NAAQS.

**Sections 4.1-4.3 regarding the PM<sub>10</sub> standard.**

**Sections 5.1-5.3 regarding the secondary standard.**

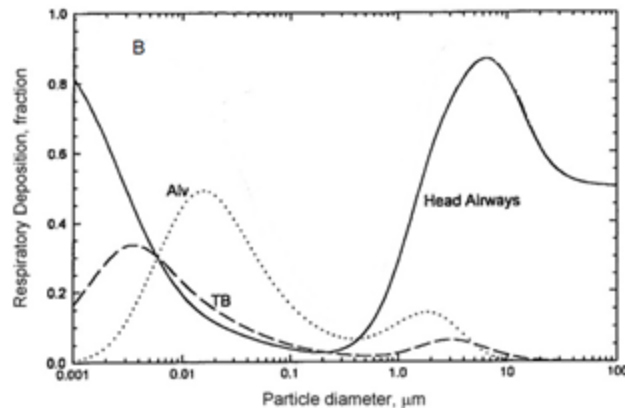
I reviewed both sections and found both to be well done and to have reached reasonable conclusions. I note that I am not an expert on this literature, so was not in a position to independently evaluate the underlying evidence.

## Dr. Michael Kleinman

### EPA-1. Chapter 1 – Introduction: To what extent does the CASAC find that the information in Chapter 1 is clearly presented and that it provides useful context for the review?

Chapter 1 provides a useful starting point.

The depiction of particle sizes in Figure 2-1 does not provide information with regard to why particle size might be important. A discussion of the role of particle size on lung deposition would be appropriate and would provide context for the later discussion of health effects of PM as a function of size. A diagram would be useful and could be discussed later as one talks about the differences between coarse and fine PM.



### EPA-2. Chapter 2 – PM Air Quality: To what extent does the CASAC find that the information in Chapter 2 is clearly presented and that it provides useful context for the review?

SCQ-2.1 *What are the Panel's views regarding whether the draft PA accurately reflects and communicates the air quality related information most relevant to its subsequent evidence-based assessment of the health and welfare effects studies, including uncertainties, as well as the development of the risk assessment for current and alternative standards? In particular, do the following sections accurately reflect and communicate current scientific understanding, including uncertainties, for: (a) relationships between annual and daily distributions of PM; (b) the review of hybrid modelling approaches used to estimate exposure in some studies and the risk assessment; and (c) information on background levels of various PM indicators?*

The discussion of the relationships between daily and annual distributions of PM would have benefitted from some integration with potential mechanisms of toxicity. Many of the disease-causing or exacerbating processes induced by PM exposures is related to formation of free radicals and the development of oxidative stress and inflammation. While in healthy individuals there are innate defenses against oxidative stress, One reason to be concerned with short term peak exposures is that normal defenses can be overwhelmed (i.e antioxidants can be consumed faster than they can be replenished) and the un-neutralized free radicals can injure tissues and organs. In the California Bay Area there were 13 occasions for which the

daily average was below the NAAQS of 35 µg/m<sup>3</sup> but there were 1 hr peak concentrations greater than 3 times the NAAQS, ranging from 113 TO 415 µg/m<sup>3</sup> (mean concentration = 197 ± 102). These days were distributed over various stations in the Bay area and the interval was Feb to November 2018. Thus, on days when the 24hr concentration was within the NAAQS people were exposed for at least 1 hr to PM<sub>2.5</sub> concentrations that were equivalent to the levels used in controlled human studies, documented in Table 3-1 from the PA. Note that November 2018 was a severe fire month and there were several days above the NAAQS 24 hr standard and 1 hr concentrations exceeding 105 µg/m<sup>3</sup>, but the other months with high 1 hr peak exposures were most likely not fire-related.

**Table 3-2. Summary of information from PM<sub>2.5</sub> controlled human exposure studies.**

Study	Population	Exposure Details (average concentration; duration)	Results
Bräuner et al., 2008	Healthy adults	10.5 µg/m <sup>3</sup> PM <sub>2.5</sub> (unfiltered) vs below detection (filtered); 24 h	No significant effect on markers of vascular function
Hemmingsen et al., 2015a, Hemmingsen et al., 2015b	Healthy, overweight older adults	24 µg/m <sup>3</sup> (unfiltered) vs 3.0 µg/m <sup>3</sup> (filtered) Copenhagen PM; 5 h	Impaired vascular function and altered heart rate variability; no significant changes in blood pressure or markers of inflammation or oxidative stress
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	64 µg/m <sup>3</sup> CAP (lower exposure); 2 h	No significant change in blood markers of inflammation or oxidative stress
Huang et al., 2012	Healthy adults	90 µg/m <sup>3</sup> CAP; 2 h	No significant changes in heart rate variability
Devlin et al., 2003	Healthy older adults	99 µg/m <sup>3</sup> CAP <sup>28</sup> ; 2 h	Decreased heart rate variability
Hazucha et al., 2013	Adult current and former smokers	109 µg/m <sup>3</sup> CAP; 2 h	No significant changes in markers of inflammation or coagulation
Ghio et al., 2000	Healthy young adults	120 µg/m <sup>3</sup> CAP; 2 h	Increased fibrinogen (coagulation)
Ghio et al., 2003	Healthy young adults	120 µg/m <sup>3</sup> CAP; 2 h	Increased fibrinogen; no significant effect on markers of inflammation
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	140 µg/m <sup>3</sup> CAP (higher exposure); 2 h	Increased blood inflammatory markers
Brook et al., 2009	Healthy adults	149 µg/m <sup>3</sup> CAP; 2 h	Impaired vascular function, increased blood pressure; no significant change in markers of inflammation (compared to filtered air)
Ramanathan et al., 2016	Healthy adults	149 µg/m <sup>3</sup> CAP; 2 h	Decreased anti-oxidant/anti-inflammatory capacity when baseline capacity was low

Sivagangabalan et al., 2011	Healthy adults	150 µg/m <sup>3</sup> CAP; 2 h	Increase in indicator of possible arrhythmia; no significant effect on heart rate
Kusha et al., 2012	Healthy adults	154 µg/m <sup>3</sup> CAP; 2 h	No significant effect on indicator of possible arrhythmia
Gong et al., 2003	Adults with and without asthma	174 µg/m <sup>3</sup> CAP; 2 h	Increased heart rate; No significant effect on indicators of arrhythmia, inflammation, coagulation; inconsistent effects on blood pressure
Gong et al., 2004	Older adults with and without COPD	200 µg/m <sup>3</sup> CAP; 2 h	Decreased heart rate variability, increase in markers of inflammation (without COPD only); inconsistent effect on arrhythmia; no significant effect on markers of blood coagulation
Liu et al., 2015	Healthy adults	238 µg/m <sup>3</sup> CAP; 130 min	Increase in urinary markers of oxidative stress and vascular dysfunction; no significant effect on blood markers of oxidative stress, vascular function, or inflammation
Bellavia et al., 2013	Healthy adults	~242 µg/m <sup>3</sup> CAP; 130 min	Increased blood pressure
Behbod et al., 2013	Healthy adults	~250 µg/m <sup>3</sup> CAP; 130 min	Increase in markers of inflammation
Tong et al., 2015	Healthy older adults	253 µg/m <sup>3</sup> CAP; 2 h	Impaired vascular function and increased blood pressure; no significant change in markers of inflammation or coagulation
Lucking et al., 2011	Healthy young men	320 µg/m <sup>3</sup> (unfiltered) vs 7.2 µg/m <sup>3</sup> (filtered); 1 h	Impaired vascular function and increased potential for coagulation; no significant effect on blood pressure, markers of inflammation, or arterial stiffness
Vieira et al., 2016a, Vieira et al., 2016b	Healthy adults; Heart failure patients	325 µg/m <sup>3</sup> (unfiltered) vs 25 µg/m <sup>3</sup> (filtered) diesel exhaust; 21-min	Increase in marker of potential impairment in heart function, impaired vascular function (heart failure patients); no significant effect on blood pressure, heart rate or heart rate variability, markers of inflammation, markers of coagulation, or arterial stiffness

**EPA-3. Chapter 3 – Review of the Primary PM<sub>2.5</sub> Standards: What are the CASAC views on the approaches described in Chapter 3 to considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? What are the CASAC views regarding the rationales supporting the preliminary conclusions on the current and potential alternative primary PM<sub>2.5</sub> standards?**

*SCQ-3.1 Does the panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>2.5</sub> review? Are there additional policy-relevant questions that should be addressed?*

The question of the importance of short term standards is one that deserves additional consideration. In fact the human controlled

exposures suggest that a shorter term (1 hr ?) acute standard might have some protective value.

Based on the discussion for 2.1, the controlled human studies, which found significant cardiovascular effects should be considered as relevant to actual exposures and taken into stronger consideration with respect evaluating the adequacy of the current NAAQS levels.

*SCQ 3.3 What are the Panel's views on the evidence-based approach, including:*

*The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*

The use of the annual standard to protect against short and long term exposure health effects may not be the best approach, from the standpoint of biological mechanisms. As stated earlier, many of PM's health effects are subsequent to formation and release of free radicals leading to oxidative stress and inflammation. These are hallmarks of heart diseases, lung diseases, cancer and degenerative nerve diseases. While in healthy individuals there are innate defenses against oxidative stress, short term peak exposures can overwhelm the normal immunological defenses (i.e antioxidants can be consumed faster than they can be replenished) and the un-neutralized free radicals can injure tissues and organs. This could be especially true in people with impaired immunity, people with pre-existing diseases, the very young and the elderly.

*SCQ-3.5 What are the Panel's views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?*

The evidence and discussion consistently demonstrate that the current standards do not provide an adequate margin of safety to prevent health effects. It should be noted that while the weight of evidence for PM's effects cardiovascular disease causation is stronger than that for pulmonary disease, having an impaired pulmonary system will put significant extra load on the cardiac system and could be a contributing factor to the ultimate cause of death, i.e. cardiac-related disease.

*GC-1. What scientific evidence has been developed since the last review to indicate if the current primary and/or secondary NAAQS need to be revised or if an alternative level or form of these standards is needed to protect public health and/or public welfare? Please recommend to the Administrator any new NAAQS or revisions of existing criteria and standards as may be appropriate. In providing advice, please consider a range of options for standard setting, in terms of indicators, averaging times, form, and levels for any alternative standards, along with a description of the alternative underlying interpretations of the scientific evidence and risk/exposure information that might support such alternative standards and that could be considered by the Administrator in making NAAQS decisions. Shorter averaging times (1 hr ?) to protect against acute health effects (sudden cardiac death, acute asthma attacks)*

*GC-2. Do key studies, analyses, and assessments which may inform the Administrator's decision to revise the NAAQS properly address or characterize uncertainty and*

*causality? Are there appropriate criteria to ensure transparency in the evaluation, assessment, and characterization of key scientific evidence for this review?*

There are appropriate criteria that are relevant to any scientific endeavor. Thorough documentation of methods and approaches, documentation of quality control and quality assurance, rigorous, objective analysis of the data are all necessary. The studies that were discussed in the documents were evaluated and selected because they were quality science.

- GC-3. *Are there areas in which additional knowledge is required to appraise the adequacy and basis of existing, new, or revised NAAQS? Please describe the research efforts necessary to provide the required information.*

New areas of health effects studies and new assessment methods are continuing to evolve. Evaluation and characterization of “hot spots” of high exposure, especially where those areas can be identified with impacts from local sources are needed.

- GC-4. *What is the relative contribution to air pollution concentrations of natural as well as anthropogenic activity? In providing advice on any recommended NAAQS levels, please discuss relative proximity to peak background levels.*

Recent laboratory studies have demonstrated that natural organic vapors when combined with atmospheric photochemical processes and anthropomorphic combustion gases (NO<sub>x</sub>) form particles that are more toxic than secondary organic particles formed in the absences of the human pollutants. Some future attention to these could be warranted.



## Dr. Rob McConnell

**EPA-3. Chapter 3 – Review of the Primary PM<sub>2.5</sub> Standards: What are the CASAC views on the approaches described in Chapter 3 to considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? What are the CASAC views regarding the rationales supporting the preliminary conclusions on the current and potential alternative primary PM<sub>2.5</sub> standards?**

*Overall, this is a very solid review and synthesis of literature and policy alternatives and implications.*

SCQ-3.2 What are the Panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e. draft PA, section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM<sub>2.5</sub> standards?

*There is appropriate focus on the evidence that has emerged since the last PM review for the key outcomes, including mortality and cardiovascular disease. Evidence is well summarized incl cross discipline, low level effects and accountability studies. The risk-based approach provides complementary information relevant to policy.*

*The summary of the changing conclusions regarding causality in Table 3-1 largely reflects the emerging scientific consensus based on a stronger evidence base. However, I am puzzled that there was not further consideration of likely causal relationships with premature birth and low birth weight. There is also rapidly emerging evidence from epidemiological and toxicological studies indicating that PM<sub>2.5</sub> exposure causes insulin resistance, impairs beta-cell function and causes Type 2 diabetes. The criteria for the conclusions that these were not likely causal might be explained in more detail.*

*One disturbing feature of Table 3-1 is the footnote indicating that the CASAC that reviewed the ISA found that the evidence that evidence was not sufficient to conclude that the relationship was likely causal between PM<sub>2.5</sub> exposure and nervous system effects; between long-term ultrafine particulate (UFP) exposure and nervous system effects; or between long-term PM<sub>2.5</sub> exposure and cancer". While it is within the purview of the CASAC to make such a determination, did not the CASAC itself acknowledged that it lacked the expertise to do so?*

SCQ 3.3 What are the Panel's views on the evidence-based approach, including:

- a) The emphasis on health outcomes for which the draft ISA causality determinations are "causal" or "likely causal"?

*This is a reasonable approach. See also response to SCQ 3.2*

- b) The identification of potential at-risk populations?

*The PA acknowledges susceptibility of children, the elderly, the poor and ethnic and racial minorities based on increased exposure, people with pre-existing conditions, in short a large proportion of the population. There is voluminous data on exposure and environmental justice that was not reviewed in any detail. Also, there was little discussion of genetic susceptibility and the implications for causal inference. Where variants in*

*pathways predicted to be targeted by exposure modify effects, these results can provide a very strong argument for causality.*

- c) Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?

*These are the most relevant to exposures to the U.S. population. Although it might be argued that the composition of European PM<sub>2.5</sub> is different than in the U.S., PM<sub>2.5</sub> composition also differs across the U.S. and Canada, and there is strong evidence of health effects from the ESCAPE studies, for example, and other European studies (as well as elsewhere). The approach should not preclude review of selected studies from elsewhere that provide compelling evidence based on novel design or relevance to questions of interest to the PA.*

- d) Characterizing air quality in these key studies using two approaches: is the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the “pseudo design value” reflecting a monitor with the highest levels in an area?

*I look forward to the committee discussion of this question.*

- e) The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?

*The PA makes a credible argument for this approach.*

- f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?

*The PA makes a strong case that health effects are occurring at concentrations below the current long-term standard, based on studies showing effects among populations exposed at levels at or below the standard, and the supportive evidence from the design and pseudo-design values. The PA provides rationales for a lower alternative standard to levels around 10 µg/m<sup>3</sup>, levels below 10 (to as low as 8 µg/m<sup>3</sup>), and levels between 10 and 12.*

- g) Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?

*There is appropriate consideration of the uncertainties.*

**EPA-4. Chapter 4 – Review of the Primary PM<sub>10</sub> Standard: What are the CASAC views on the approach described in Chapter 4 to considering the PM<sub>10-2.5</sub> health effects evidence in order to inform preliminary conclusions on the primary PM<sub>10</sub> standard? What are the CASAC views regarding the rationale supporting the preliminary conclusions on the current primary PM<sub>10</sub> standard?**

- SCQ-4.1 To what extent does the panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>10</sub>

NAAQS review? Are there additional policy-relevant questions that should be addressed?

SCQ-4.2 What are the Panel's views of the draft PA assessment of the currently available scientific evidence regarding the health effects associated with exposures to thoracic coarse particles, PM<sub>10-2.5</sub>?

SCQ-4.3 What are the Panel's views on the draft PA preliminary conclusion that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM<sub>10</sub> standard and that evidence supports consideration of retaining the current standard?

*The PA makes a case that, in spite of additional epidemiological studies, key uncertainties in the evidence that precluded a determination of causal role for PM<sub>10-2.5</sub> by itself or a justification for considering alternative standards for PM<sub>10</sub> in the last PM review. Additional research is needed: toxicological effects of coarse-thoracic PM; inhalation challenge studies to characterize acute effects and pathways and subclinical effects; studies of susceptible populations, especially asthmatics. Appropriate methods for exposure assessment of PM<sub>2.5</sub> and for analysis of exposure assigned using different methods, and of co-pollutant effects, are acutely in need of further investigation. This is not to say that current levels of exposure to PM<sub>10-2.5</sub> are safe, rather that there is not enough evidence to make a determination.*

## Mr. Richard Poirot

### Chapter 2

Overall, chapter 2 accurately reflects and clearly communicates air quality information relevant to conducting evidence-based assessments of health and welfare effects, and for conducting risk assessments for evaluating effects of current and alternative NAAQS. Relationships between recent daily and annual PM exposures are clearly presented (for example Figure 2-11). The different hybrid modeling approaches used to estimate exposures are logically derived and clearly described, and information on so-called “background” PM levels are more or less clearly explained. Some interesting results from recent near-road monitoring efforts were presented, although it wasn’t clear how/if these results were folded into the hybrid modeling analyses.

Regarding background PM, it’s not clear [how](#) this information is or will be useful in reviewing and potentially revising the NAAQS. Clearly it could be useful in the implementation phase of the NAAQS (identifying/ getting exemptions for “exceptional” and/or “natural” events). As illustrated, influence of some of these background contributions may be relatively easy to identify and quantify (especially episodes), but I assume we’re not saying these background influences don’t contribute to health effects (right?). In addition, I think there are likely complex interactions between so-called background influences and “jurisdictionally-controllable” anthropogenic sources (see p. 2-49 comment below).

An additional comment on Chapter 2 is that it would be helpful to see some graphic depictions (a few maps and perhaps a time series like Figure 2.6 but for recent years) showing the locations and numbers of the various different PM<sub>2.5</sub> monitoring techniques/networks (filter FRMs, filter CSN, IMPROVE, continuous (FEM & non-FEM), near-road, etc. A few national maps on this would be useful, as well as a few zoomed-in urban area examples - from some of the cities used in the Risk Assessment (maybe underlain by the hybrid modeling grid).

**p. 2-2, lines 20-22:** I don’t think its correct that only a small fraction of coarse mode mass occurs in particles > 10 microns. Much of the coarse mode mass is often > 10  $\mu\text{m}$ . See for example Brook et al. (1997), who noted that averaged across 19 long-term Canadian NAAPS sites “PM<sub>25</sub> accounted for 49% of the PM<sub>10</sub>, and PM<sub>10</sub> accounted for 44% of the TSP”. This would leave > 4 times more coarse mass in particles >10  $\mu\text{m}$  than in PM<sub>10-2.5</sub>. I think maybe the authors meant to say something like “small fractions of [inhalable](#) coarse mode mass can be ... greater than 10  $\mu\text{m}$  in diameter”.

**p. 2-7, lines 7-8:** You could also mention ammonium as an important component of PM<sub>2.5</sub>.

**p. 2-9, lines 4-8 (similar to above comment):** Why not add ammonia to your list of important precursor gasses, instead of just indicating that it “also contributes”.

**p. 2-23, Figure 2-8:** Figure 2-8 shows recent 2015-2017 average concentrations, along with some much longer-term 2000-2017 trends. I think in late 2019 that 2018 data have been available for a while and that these charts could be updated.

**p. 2-23, Figure 2.9:** As in previous comment, this could be updated through 2018. I’ve also noted from EPA’s Air quality trends website that the long-term 41% improvement you cite 2000 through 2017 decreases to 38% when carried through one more year to 2018 (U.S. EPA (2019). Zooming in to more recent 2010 to 2018 data, I note that there was steady progress each year.

Both the national mean and 90th percentile PM<sub>2.5</sub> decreased from 2010 to 2011, from 2011 to 2012, etc. all the way through 2016. After 2016, both the national mean and the 90th percentile concentrations increase from 2016 to 2017 and increase again from 2017 to 2018. It would be informative to explore this recent reversal of long-term progress, and provide explanations of possible causal factors, along with estimates of future trends.

**p. 2-49, section 2.4:** Here and/or elsewhere when you discuss “natural” vs. “anthropogenic” aerosols, you could add some discussion of PM that results from combinations of natural & manmade sources. For example, emissions from a “natural” dust storm may be enhanced by human actions such as cattle grazing, desert recreation activities, or climate change. “Wildfires” may be started by a careless match, electrical transmission lines, enhanced by historical forest & fire management practices, climate change, etc. “Natural” VOCs may be converted to SOA by reactions with manmade oxidants or through reactions catalyzed by acidic (sulfate) aerosols. Natural sea salt or dust reacts with manmade nitric or sulfuric acids, etc. Sea salt emissions are projected to increase due to climate-driven increases in surface wind speeds. Historical and continuing US emissions represent the largest contribution of any country to the cumulative buildup of global climate-forcing greenhouse gases. Thus, a fraction of transcontinental dust and smoke (considered both “natural” and “non-US”) PM reaching the US may have been enhanced by effects our own anthropogenic GHG emissions

## **Chapter 3**

### **SCQ 3-4**

This is not my area of expertise and I defer to other panelists for their thoughts on the quantitative risk assessment. Overall, I found the choices of health outcomes and studies selected for developing long-term and short-term CR functions reasonable and clearly justified. The selection criteria for included urban areas appear to be similarly logical and clearly described. Variability and uncertainty are clearly characterized, and the results appear to be valid and robust.

I support the hybrid modeling approach as a way of estimating effects that would occur over a range of current and alternative standards (a more realistic improvement over the statistical “quadratic rollback” approach employed several NAAQS review cycles ago). I was somewhat surprised to note that the relative mortality benefits generally appeared to be somewhat greater for meeting the different annual standards for the secondary PM reductions than for the primary PM reductions (Table 3-8, for example). I might have guessed the opposite - assuming the secondary PM reduction would have been more uniform across each urban study area, while the primary PM reductions would have shown more local influence and variability (residential space heating, roadway emissions, industrial sources) within each urban area. I wonder what the reasons are for this general pattern? Could some discussion be provided? Perhaps you could provide some high resolution images of sections of a few individual urban areas contrasting spatial patterns of differences between the primary & secondary control concentrations, underlain by the hybrid model grid.

## **Chapter 5**

Welfare effects considered in Chapter 5 include those on climate, materials and visibility. Some new information is available on climate effects, and while these remain complex, mixed, and uncertain for various PM species, I think a reasonable argument could probably be developed in

support of climate-related reductions in black (& brown) carbon concentrations, although a secondary standard may not be an appropriate mechanism. Some interesting new work quantifying PM materials (soiling) effects on efficiency of solar panels is presented, but does not seem (yet) to lend itself to setting a quantitative secondary NAAQS. Relatively little new information is available on visibility effects (although I think some useful recent information on visibility preference indices has been overlooked in the ISA and PAD (more on this below).

#### **SCQ-5.1**

The policy questions raised in Chapter 5 relate primarily to visibility. These questions essentially begin with the assumptions that the indicator, level, averaging time and form of the visibility-related PM NAAQS considered (and rejected) in 2012 - are all appropriate, state of the science, and need no further justification or reconsideration. The PAD furthermore jumps immediately to the weakest end (30 DV) of the previously considered 20 to 30 dv range, combined with the weakest (90th percentile) end of the previously recommended 90th to 98th percentile range when considering possible future benefits (of which - Surprise! - there are none). Some additional modeling of reconstructed extinction using a slightly modified equation is conducted in Appendix D, and while this shows somewhat higher light extinction levels, there still appears to be minimal exceedance of the 30 dv, 90th percentile threshold. I think all 4 elements of the secondary PM NAAQS considered in the 2012 review need to be reconsidered, justified (if possible), compared to alternatives, and, if warranted, revised.

#### **SCQ-5.2 What are the Panel's views of the draft PA evaluation of the currently available scientific evidence with respect to the welfare effects of PM. Does the assessment appropriately account for any new information related to factors that influence:**

##### **Quantification of visibility impairment associated with PM<sub>2.5</sub> and examination of methods for characterizing visibility and its value to the public?**

Regarding charge question GC-1, I have concerns with all 4 elements ((indicator, averaging time, level and form) of the secondary PM NAAQS presented for consideration in the Draft PAD document (and rubber-stamped from the 2012 review), and these relate in several cases to information not considered in the ISA.

##### **Indicator (reconstructed PM light extinction from 2012 review)**

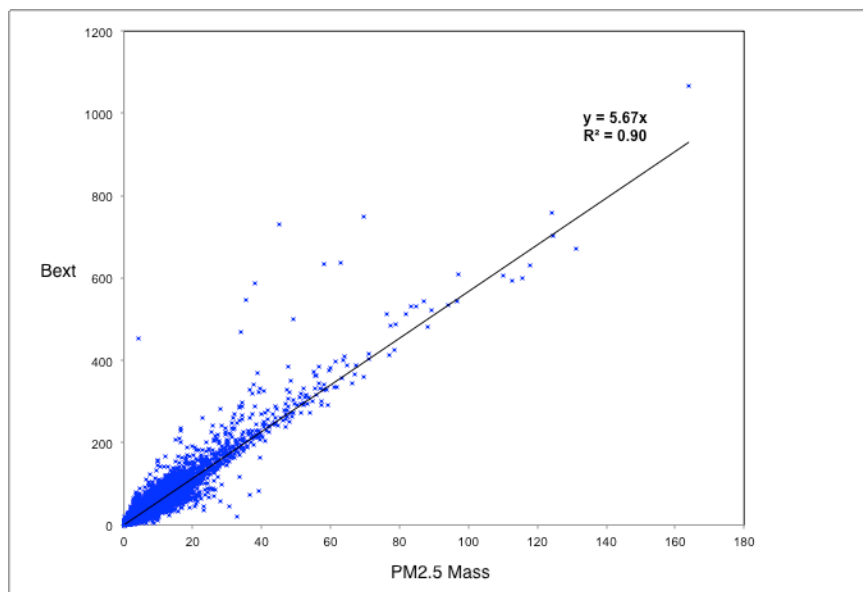
The first PM NAAQS established in 1970 included a separate secondary standard with a PM mass-based indicator (TSP). In subsequent NAAQS reviews completed in 1987, 1997 and 2006, EPA considered, with CASAC support, setting separate, visibility-related secondary PM NAAQS, in each case with a PM<sub>2.5</sub> mass indicator (although separate secondary standards were not set after those reviews). In the last review completed in 2012, EPA staff, with CASAC support, considered a different indicator: PM light extinction. During much of that recent review, it was assumed that PM light extinction (or PM<sub>2.5</sub> light extinction) could and would be directly measured by available continuous methods, such as nephelometer and Aethalometer.

Late in the review, it became clear that the Agency had no intention (resources, will, etc.) of establishing a new national monitoring network, and an inferior fallback methodology was employed to calculate PM light extinction from 1-in-3-day 24-hour filters collected in the EPA STN network and at similar state-sponsored speciation sites using the revised (II) IMPROVE algorithm. This approach takes into consideration the differential densities, size distributions,

light scattering and absorption properties and water retention characteristics of different aerosol species. This is basically the method employed to define visibility impairment and track (very) long-term progress toward improving it in remote Class I National Parks and Wilderness Areas under the Regional Haze Rule. It is not, however, necessarily any better (or as good as) a much simpler PM<sub>2.5</sub> mass indicator, [especially compared to the benefits of using the data from the existing continuous PM<sub>2.5</sub> monitors](#) in urban/suburban sites.

- The continuous PM<sub>2.5</sub> network includes 6 times as many sites as the CSN network, providing [much](#) better spatial coverage. Note that the modeled 2015-2017 reconstruction extinction in Appendix D is based on only 67 sites meeting data completeness criteria (see Figure D-1).
- The CSN network samples only every 3rd day, at best, leaving 2/3 of days unmonitored, compared to hourly sampling, every day in the continuous PM<sub>2.5</sub> network, providing 72x more temporal information - at 6x more sites.
- Filter-based CSN monitoring allows only “24-hour average extinction” estimates. This is [not](#) the averaging time over which people perceive impairment. Shorter hourly or 4 to 8-hour (daylight) averaging times would be [much](#) more appropriate, especially in urban/suburban areas where light pollution and other factors render night-time PM visibility impairment much less important. Focusing on daylight or mid-day hours would also minimize the importance of RH & speciation, leading to even tighter relationships between actual short-term visibility effects and PM<sub>2.5</sub> mass data (which are pretty good already). See CASAC recommendations on this from the 2006 review (Hopke et al., 2005, Henderson et al., 2006).
- While the IMPROVE algorithm - perhaps as enhanced by changes such as suggested by Lowenthal and Kumar (2016) - is “state of the science”, it still requires assumptions which are not always well met (the degree of sulfate ammoniation, chemical form(s) of nitrate, the varying relationships between measured OC and POM mass, etc.) See for example Hand et al., (2019); Preni et al. (2019). Use of 24-hr data also inflates the influence of higher nighttime RH (when urban visibility is least important).
- The filter-based algorithm itself has problems (which appear to be getting worse over time) in reproducing light extinction measured by nephelometry. Conversely, nephelometers have been successfully deployed as PM<sub>2.5</sub> monitors.
- A good argument can also be made that influence of (naturally) varying RH should be removed from the regulatory metric. Water influence would be minimized by focusing on the (more important) daytime hours. You could also use a fixed, long-term average RH to remove the natural variability from the regulation, or you could impose an RH screen (say eliminating hours with RH < 70%) on the PM data (as is done with urban visibility standards in Phoenix and Denver). Water effects are also decreasing over time as sulfate, nitrate and secondary semi-volatile organics decrease. I don’t think you really want the most extreme events driven by extreme uncontrollable variations in RH.
- Use of hourly data would allow eliminating hours with natural impairment (rain, snow, fog, natural dust storms, forest fires).
- Continuous PM data would allow extinction estimates or multi-hour averaged PM<sub>2.5</sub> values to be publicly reported in near-real-time, rather than waiting for months for the filter data results. (Note that the most recently available CSN data employed in Appendix D were from 2017).
- Use of the continuous PM data for secondary NAAQS regulatory purposes would lead to (needed) closer scrutiny, improved QA and better data quality.
- Light extinction from coarse particles is relatively unimportant in most regions and seasons, and when/where it is important (Southwest, spring), it’s often primarily due to natural sources. Alternatively, you could require added use of colocated continuous PM<sub>10</sub> samplers in areas like the Southwest where coarse particle scattering is important, or set a fine particle NAAQS this time and add a coarse PM component next time.

The figure below is based on [all](#) the (unscreened) IMPROVE data from all sites for the 3-year period 2015-2017, limited to sample days when both PM<sub>2.5</sub> mass and filter-based light extinction estimates are available (about 50,000 sample days).



Similar high correlations have also been observed between continuous PM<sub>2.5</sub> mass-based PM<sub>2.5</sub> monitors and nephelometers (or when the continuous nephelometer results are aggregated to 24-hr means - for comparison to filter PM<sub>2.5</sub>). See for example Chung et al. (2001), Chow et al (2006), Puget Sound (2001), Snider et al., 2015, etc. Note also that the slope of this scatterplot implies a generic extinction to mass ratio of about 6 m<sup>2</sup>/g. This is a bit higher than the expected dry PM<sub>2.5</sub> scattering efficiency (about 4 m<sup>2</sup>/g), as it includes influence from water, light absorption and coarse mass. The average scattering efficiency of coarse particles is about a factor of 10 lower (0.6 m<sup>2</sup>/g), and while this is generally a minor contributor, it can be important in certain regions and seasons (Southwest, spring). Given the above, reasonable estimates of total PM light extinction might be approximated by something like  $6 \times (PM_{2.5} + PM_{10-25}/10)$ .

The bottom line is that fine mass is a very good indicator of visibility effects, and the small amount of information gained by using speciation filter-based estimates is way more than offset by the spatial, temporal information and visibility relevance that would be gained using continuous PM<sub>2.5</sub> monitors and a sub-daily daytime averaging time. Please note the CASAC comments on secondary NAAQS from the review completed in 2006, for example: Hopke et al. (2004), page 9 and pages B9-B26, Henderson et al. (2006).

If you really want to keep the light extinction indicator, use the filter-based speciation data to calculate regional monthly or seasonal species composition + f(RH) factors to adjust the continuous PM<sub>2.5</sub> data to (slightly) better extinction estimates - which could then be considered on a sub-daily basis, much more relevant to human perception, and could be publicly reported from a much larger network in near-real time. Please note the CASAC recommendations (Samet et al., 2010) on various options for secondary PM indicators averaging times, forms, etc. in comments on the 1st draft (March, 2010) draft PM PAD. These comments came at a point



when it was still assumed PM light extinction would be continuously and directly measured, but also supported using seasonal & regional speciation and RH data to develop modifications to the hourly PM<sub>2.5</sub> data - needed to support the recommended sub-daily averaging times. I think a simple sub-daily PM<sub>2.5</sub> mass indicator which intentionally limits the influence of naturally varying RH on the regulatory metric is a better choice for an indicator. If the Agency wants to persist in advocating continued use of the every 3rd day 24-hour, filter-based reconstructed light extinction indicator, it needs to justify why it thinks it has a superior indicator. I don't think it can.

### **Averaging Time (24 Hours from 2012 PM NAAQS)**

As indicated above, once the decision was made that the PM light extinction indicator introduced in the 2012 review would not be measured directly and continuously, a fallback method was proposed to calculate PM light extinction based on every 3rd day 24-hour filter sampling. (This, in my opinion, was the point where the 2012 review ceased to represent any advancement of the science and became notably inferior to the sub-daily PM<sub>2.5</sub> secondary standard considered in the 2006 review). Filters limit the averaging time to no shorter than 24 hours, which is not the time frame over which visibility impairment is perceived. It's also especially inappropriate in urban areas where visibility during daylight hours is much more important (and is also characterized by lower RH levels - reducing the small difference between PM mass and light extinction).

EPA's Final Staff Paper (U.S. EPA, 2005) from the 2006 PM NAAQS review (which recommended a sub-daily PM<sub>2.5</sub> mass indicator in the range of 20 to 30 ug/m<sup>3</sup> for a secondary PM NAAQS) stated:

In considering appropriate averaging times for a standard to address visibility impairment, staff has considered averaging times that range from 24 to 4 hours, as discussed in section 6.2.3. Within this range, as noted above, correlations between PM<sub>2.5</sub> concentrations and RE [reconstructed extinction] are generally less influenced by relative humidity and more consistent across regions as the averaging time gets shorter. Based on the regional and national average statistics considered in this analysis, staff observes that in the 4-hour time period between 12:00 and 4:00 p.m., the slope of the correlation between PM<sub>2.5</sub> concentrations and hourly RE is lowest and most consistent across regions than for any other 4-hour or longer time period within a day (Chapter 6, Figure 6-4). Staff also recognizes that these advantages remain in looking at a somewhat wider time period, from approximately 10:00 am to 6:00 pm. Staff concludes that an averaging time from 4 to 8 hours, generally within the time period from 10:00 am to 6:00 pm, should be considered for a standard to address visibility impairment.

It can also be noted that the quality of the continuous PM<sub>2.5</sub> mass data has improved considerably over the past 20+ years, providing greater confidence in its accuracy over shorter averaging times. The Agency should consider the many benefits of using the continuous PM<sub>2.5</sub> data as the measurement basis for a sub daily 4 to 8 hour daylight averaging time. This could be combined with a continuous PM<sub>2.5</sub> mass indicator, or regional & seasonal generic species composition and f(RH) factors could be developed to convert the mass to estimated extinction (if you need to stick with a b<sub>ext</sub> indicator). Either way, the data could be reported in near-real time, and would relate more directly to the human perception of impaired visibility.

## **Level (20 to 30 dv from 2012 review)**

While the previous 2011 PAD recommended a range of 20 to 30 dv as an appropriate level, the Administrator (sort of) picked the upper end, before concluding that such a NAAQS wouldn't do much good. The current PAD simply starts with this upper end (30 dv) as if this were a logical, technically-supported absolute definition of "acceptable visibility" or adverse effects. It's not.

In a previous review of the draft ISA, I noted that the ISA had neglected an important recent meta-analysis of visibility preference studies by Bill Malm (Malm et al. 2011, 2019 and Malm, 2013 and 2016) which could support an alternative way of defining adversity based on geographical differences in distant landscape features. My earlier comments on this omission in the ISA are pasted below:

A second general criticism of this brief summary - as well as with the more detailed Chapter 13 discussion of visibility - is the absence of discussion of recent work on visibility preference indicators developed by William Malm over the past several years (Malm et al. 2011, 2019 and Malm, 2016). His meta analysis of multiple available visibility preference studies (in many different kinds of locations) noted that "unacceptable" levels of visibility impairment occurred at different extinction levels in different areas, but that in any area, when the more-distant visible landscape features nearly disappear - which occurs at apparent contrast levels of about 0.02–0.05 - the haze level became unacceptable to about half of the participants in each study area. This has important implications for the potential setting of PM visibility standards at nationally consistent contrast levels which are geographically variable with changing distant landscape features. It would be a relatively straightforward GIS exercise to characterize distances to prominent landscape features in population centers throughout the country and then use PM<sub>2.5</sub>-based extinction estimates to calculate contrast levels for those landscape objects to determine the extent to which visual air quality is (or is not) considered acceptable in each of those areas.

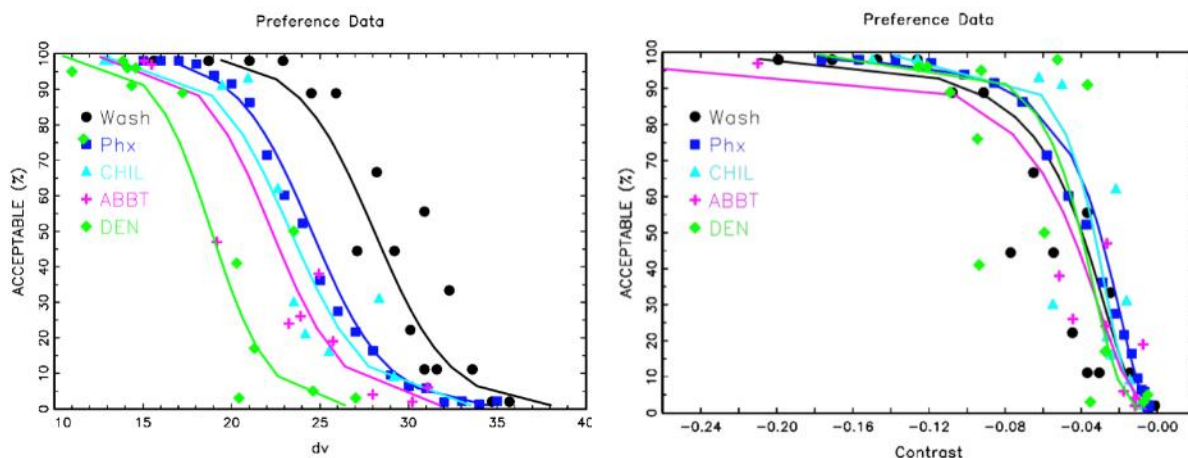
There appears to be a reference to Malm's work in the executive summary: "There have been no recent visibility preference studies; however, a recent meta-analysis demonstrates that scene-dependent haze metrics better account for preference compared to only using the deciview scale as a metric." However, any discussion of this recent work seems to be missing from the Integrated Synthesis or Chapter 13. Section 13.2.5 on "human perception of haze and landscape features" heavily emphasizes the divergent results in different visibility preference studies in areas with (or using photographs showing) different landscape features, when visual air quality is expressed as light extinction (deciviews). It concludes with:

"There is little new published information regarding preference levels in the U.S. The single new study by Smith (2013) was an investigation of "framing bias" in preference studies that can potentially occur because preference levels are chosen in part based on experimental variables such as number of photographs shown or range of the range of dv levels participants are shown when asked to state a preference about whether scenes in photographs are acceptable."

This disregards important new work in this area, which clearly shows a convergence of results across many different urban areas when the visual air quality is expressed in terms of the contrast of the most distant landscape features. Another important recent

related technological development is the ability to incorporate clouds into the Winhaze model - developed by John Molenar (Molenar and Malm, 2012). For cities in relatively flat terrain which lack distant landscape features, clouds often are the most distant scenic attribute. As they begin to disappear, viewers tend to find the degradation of visibility unacceptable, at lower levels of light extinction than they would viewing cloud-free scenes. Some discussion of this work, implications and potential future applications is warranted in chapter 13.

The figures pasted below are from a 2013 status report Malm presented on this work (Malm 2013) Please see Figures 4 and 5 from Malm et al., 2019, for updated versions of these figures and more detailed descriptions of the methods. Both figures plot percent acceptability levels from 5 urban visibility preference studies. The figure on the left (similar to Figure 5.2 in the PAD) plots percent acceptability against absolute light extinction in  $dv$ . Note that at the 50% acceptability levels in all 5 studies are bounded by a range of extinction between about 20 and 30  $dv$ . This was the basis for suggesting this range in the 2012 review, although the current PAD starts at 30, a level which is clearly unacceptable to the majority of respondents in all 5 study areas.



In the figure on the right, Malm plots percent acceptability results from the same studies against the apparent contrast of “a distant, prevalent, but not necessarily dominant, feature”, which shows a remarkable consistency at a contrast of about -0,04 across many diverse types of study areas. This contrast threshold of about -0.04 basically occurs as the visual range nears the distance of prominent distant scenic elements. People everywhere tend to find decreased visibility unacceptable as prominent, distant landscape features begin to disappear.

If this kind of approach were applied across multiple urban/suburban areas throughout the country, it would be clear that people in many diverse regions would likely find visibility impairment of 30  $dv$  to be unacceptable. The Agency should consider using this apparent contrast threshold as a basis for setting a consistent national standard which could vary geographically depending on local scene characteristics. I think it would be a relatively straightforward GIS exercise to determine regional scene characteristics across the US. This would be a similar concept to what the Agency considered in the last review of secondary SOx

+ NO<sub>x</sub> NAAQS, in which the varying biogeochemical features of local eco-regions were incorporated into the proposed standard.

#### **Form (90th Percentile from the 2012 review)**

The 90th percentile is not supported in the PAD or ISA. Its just repeated from the last review cycle (where it was never justified either). It was simply a way for a secondary NAAQS - considered at the most lenient end of the staff-recommended 20 to 30 dv range - would have little to no benefit over the primary standard. The forms of the various secondary standards that have been considered/ recommended by EPA staff and/or CASAC over the years has varied widely: not to be exceeded more than 1 day/year (1971), 3-month seasonal mean averaged spatially over multiple years (1987), 98th percentile averaged over 3 years (1997), 92nd to 98th percentile, 3-year average (2006), and 90th to 98th percentile, 3 year average (2012).

With the exception of 1971, when a separate secondary PM standard was set, the secondary NAAQS considered in all subsequent reviews were rejected for various different “reasons” (see: Poirot, 2011). In the 2012 review, the Administrator selected the (most lenient) 90th percentile combined with the weakest level (30dv) before concluding that this combination really wouldn’t have much incremental benefit over the primary. The only stated justification was that the Regional Haze Rule is focused on the haziest 20% days, and that the 90th percentile - the midpoint of the haze range - would be consistent. (Although the average of haziest 20% days is closer to the 92 percentile - considered as the low bound in 2006 for that stated reason).

More importantly, this is a [false](#) equivalency. The focus in the Haze Rule is specifically on [improving](#) conditions on these worst days. The use of a similar percentile as a NAAQS form has exactly the opposite effect - of completely [ignoring](#) the worst visibility days, exculpating them from any consideration of improvement. Visibility could be worse, or much worse than 30 dv on 36 days each year, but people only find it objectionable when this happens 37 or more days per year (averaged over 3 years). This is not logical, and no other justification is provided in the PAD or ISA.

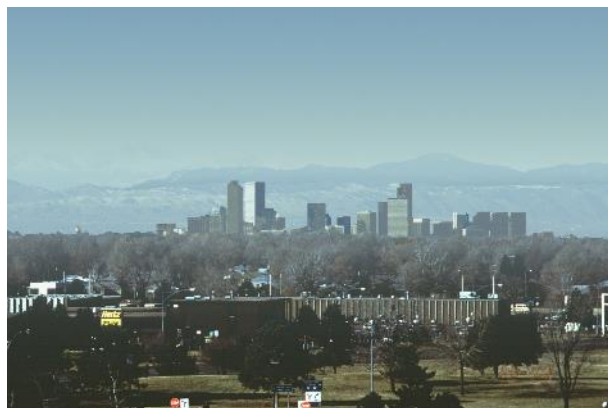
#### **SCQ-5.3 What are the Panel’s views of the draft PA preliminary conclusion that the currently available scientific evidence does not call into question the protection afforded by the current secondary PM standards against PM welfare effects and that it is appropriate to consider retaining the current secondary PM standards without revision?**

As indicated above, I have criticisms of all elements of the secondary NAAQS which was considered (but ultimately rejected) by the Administrator in 2012. I also don’t think current secondary PM NAAQS provide protection against adverse visibility effects on public welfare. The combination of daily average, 90th percentile, 30 dv, filter-based reconstructed PM light extinction is a substantially weaker secondary standard than those considered by EPA staff and supported by CASAC in all previous (1987, 1997, 2006 and 2012) PM NAAQS reviews.

To illustrate the visual AQ effects of the current 24-hour NAAQS, the images below show a clear day view from Denver which has been modified by a model called WinHaze developed by John Molenaar at Air Resource Specialists and now available on-line at: <http://vista.cira.colostate.edu/Improve/winhaze/> . See Poirot (2011) for added details on visual effects of alternative NAAQS.

The figure on the right models the visual air quality effects of 35 ug/m<sup>3</sup> of PM<sub>2.5</sub> (composed of equal parts organic matter, ammonium sulfate and ammonium nitrate at 50% RH). It may be noted that this mix of pollutants at the level of the current daily PM<sub>2.5</sub> NAAQS results in light extinction of 202.71 Mm<sup>-1</sup> - or 30.09 dv - basically the upper end of the 20 to 30 dv range suggested in the final 2011 PM PAD and rubber stamped in the current PAD. So clearly, PM light extinction at 30 dv (90th percentile) offers no protection beyond that provided by the current NAAQS.

The question is does anyone really believe this is an adequate level of visibility protection?



Similarly, the current annual secondary PM<sub>2.5</sub> NAAQS at 15 ug/m<sup>3</sup> is weaker than the primary, and therefore protects nothing, since the primary standard must be attained within a fixed period of time while a secondary standard has no time requirement. Nor has any scientific justification been provided for this irrational selection. The modeled image on the left shows a similar mix of PM<sub>2.5</sub> species at 15 ug/m<sup>3</sup>. Coincidentally this results in visibility impairment of 20.15 dv - the low end of the range considered in the 2012 review. Is this acceptable annual average visibility?

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## Dr. Jeremy A. Sarnat

*General Comments:* The EPA staff who prepared the draft Policy Assessment for the PM NAAQS reviews have done a commendable job summarizing the scientific evidence presented in the PM ISA. Broadly, I find the document to be clearly written and well-justified, and presents a justifiable set of approaches for outlining the policy implications contained in the ISA. Most of the comments below address recommendation to changes in interpretation or emphasis and are not criticisms of the substantive approach for conducting this PA. In addition, I included a couple of comments that relate to the ISA, but appear not to have been addressed and could affect policy decisions within the PA.

I did find it notable, with exceptions, that much of the process and base assumptions presented in this PA, while reasonable, is largely incremental, building heavily on well-established understandings of PM exposure and health, and mainly avoiding emerging evidence, especially as it relates to susceptibility and biological mechanisms. I do appreciate this approach and recommend only that more be added, to the future directions portion of the assessment, preparing staff for what I feel are imminent larger changes to how we understand PM toxicity and regulate its presence in the environment.

The specific comments below relate to the charge questions for SCQ 3.3:

### ***‘What are the Panel’s views on the evidence-based approach, including:’***

- a) *The emphasis on health outcomes for which the draft ISA causality determinations are “causal” or “likely causal”?*

The practice of basing evidence-based policy exclusively on outcomes where ‘causal’ and ‘likely causal’ determination exist, is a common practice within the NAAQS Policy Assessment process and reasonable based on weight-of-evidence rationale. In some ways, however, this follows a proverbial ‘looking under the lamppost’ approach and may, for some pollutants, represent a less conservative element within the current PA (i.e., a practice being less protective of human health). With caution taken to avoid false comparisons among pollutants and the respective processes that govern their regulation, chlorpyrifos comes to mind as an example of this. With chlorpyrifos, traditional, well-established pathways and endpoints were used in regulatory decision making, when novel, perhaps slightly less established, endpoints were not adequately considered.

For PM, specifically, it is possible and even likely that the pace of discovery into molecular mechanisms and its modes of toxicity will lead to new insights into more relevant (or sensitive) outcomes that may inform the standard. This is a major current direction of the health effects work being conducted and is rightly acknowledged in section 3.5 (‘Areas for Future Research’) of Chapter 3. Of particular note are the numerous investigations using high-throughput and omics-based methods. These, and future studies should contribute towards the identification of novel modes of PM toxicity and also specific groups of individuals who may be especially susceptible to PM exposures, particularly those with metabolic syndrome. I believe this is a point that should also be mentioned earlier in the chapter when discussing the current decision to emphasize the more studied and established exposure-outcome associations.



b) *The identification of potential at-risk populations?*

The identification of 'at-risk' to include those beyond traditional definitions centering around biological susceptibility is a substantive (non-incremental) change from the previous PA. EPA staff deserve credit for thinking about elevated exposures that may arise from societal disparities, as another factor conferring risk.

c) *Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM<sub>2.5</sub> levels associated with health effects?*

Even though I strongly believe that single-city studies offer important insights into both acute and chronic health effects associated with PM, I support the decision to conduct the evidence-based assessment using the multicity studies.

That said, while I appreciate the rationale, the decision to exclude high-quality multicity studies from other parts of the world may be a bit restrictive. For long-term exposures, for example, it would have been reasonable to include the numerous published findings from the European ESCAPE study, specifically. Given the relatively large number of US and Canadian cities included in the analysis, however, I am generally comfortable with the current approach. From these multi-city studies, I think the PA appropriately draws attention to findings showing adverse health occurring at levels currently below the NAAQS (both with the mean and distributional data and the pseudo design values). Among the most important of these studies are three Canadian analyses (Weichenthal et al., 2016b, 2016c and Pinault et al., 2016) where significant effects following long- and short-term exposures were observed well below the current NAAQS, and > 75% of the study populations in these analyses were living in areas above the pseudo design values. As an aside, from an exposure perspective, it's worth speculating about the observed rate/odds ratios reported in these studies and whether they may actually be attenuated relative to some of the other multi-city study results presented. It could be that exposure to ambient PM in these Canadian cities is actually lower than US cities due to lower ambient PM infiltration arising from more tightly sealed homes in colder climates. This would mean that the risks from exposure to PM<sub>2.5</sub> in these studies is actually higher than reported. (It is also possible, though, that the milder warm seasons may mean that Canadians use less central AC (leading to higher exposures to ambient origin PM)).

d) *Characterizing air quality in these key studies using two approaches: the overall mean and 25<sup>th</sup>/75<sup>th</sup> percentiles of the distribution and the "pseudo design value" reflecting a monitor with the highest levels in an area?*

Characterizing exposures and corresponding health response using distributional and pseudo design values reflects a point worth reiterating and not often directly acknowledged or addressed within the regulatory community; namely, that a single PM standard likely does not reflect the same level of population exposure, nor protective of corresponding population health for all locations, or for even a single location during different times of the year. I believe the approaches used by EPA to generalize the findings from the multi-city studies is appropriate and the evidence-based conclusions drawn from these studies also seems reasonable.

- e) *The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*

While the bulk of my research frequently targets sub-daily exposure and acute response to PM, I agree with decision to use longer averaging times as a principle means of protecting health. While it may be necessary to reconsider averaging times and indicators in future assessments, I still believe the rationale used in the 2012 ISA for lowering the annual standard still makes sense.

- f) *The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached in the last review on the health effects of PM<sub>2.5</sub>?*

Differentiating causal determination for both short- and long-term PM<sub>2.5</sub> exposure and corresponding cardiovascular and respiratory health effects seems arbitrary. I have noted in previous comments to the ISA that, to date, hundreds of observational and controlled results suggest casual links between PM<sub>2.5</sub> and adverse acute and chronic respiratory response. It's extremely difficult to discern meaningful differences in the weight-of-evidence collected for the PM-respiratory link, with that presented for PM-cardiovascular effects, which has been determined to be causal. Moreover, to retain this status determination, effectively places the weight-of-evidence for these health endpoints on a similar level as those presented for adverse chronic neurological effects; which I don't believe is warranted.

- g) *Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties, as characterized in the ISA?*

I have a long-standing concern regarding the use of multi-pollutant models as a primary means of assessing confounding and robustness in the ISA and now draft PA. There are serious limitations in assessing potential confounding through this approach and I believe this discussion deserves greater attention. Briefly, there are several sources of uncertainty and potential bias in using linear multi-pollutant regressions as the sole or predominant means of assessing potential confounding. The use of linear expressions, within a co-pollutant setting, to control for confounding of non-linearly correlated co-pollutants could lead to imprecision and/or bias; an appearance of effects associated with either PM or one of its correlated co-pollutants, where they do not exist. Related to this issue is that the vast majority of the co-pollutant models focus on the issue of confounding solely (i.e., what is the effect estimate of PM, while controlling for another pollutant), rather than the potential for joint effects or effect modification. These latter scenarios appear to me to be equally plausible in characterizing PM-related health effects, and that PM, including a complex suite of particulate components and other pollutant gases, may elicit response via inflammation-mediated pathways.

## **Dr. Elizabeth A. (Lianne) Sheppard**

Note: I only retained in my final comments points from my preliminary comments that I do not consider fully covered in the IPMRP consensus comments. The remaining preliminary comments have been edited and additional thoughts added.

### **Risk assessment comments**

1. The hybrid modeling approach relies on CMAQ predictions, a Bayesian downscaler, and is restricted to year 2015. The reductions were based on emissions from either primary or secondary PM; using two methods allows better understanding of the sensitivity to the downscaling approach. There are important limitations to the approach including restriction to 2015, working at the 12 km grid level, and assuming proportionate reductions scaled by fixed percentages. Specific comments:
  - a. The air quality modeling assessment section (C.1.4.3) should make it clear what time scale the evaluation is being considered. Is it daily? Similarly the N in table C-6 is not defined. I'm guessing it is the number of observations, which is the sum of days across AQS monitors. If so, the number of monitors should also be included (e.g., in parentheses).
  - b. It is a limitation that only 2015 was used. The choice is reasonable, appropriately justified, and acceptable given the compressed timeframe EPA was working under.
  - c. The performance of the 2015 CMAQ model doesn't look particularly good to me (Table C-6). Air quality modeling experts are not concerned about this performance and I note that this concern may not be particularly important for the risk assessment.
  - d. The scientifically important features of exposure models are different when the purpose is epidemiology vs. risk assessment. Exposure predictions are often much less variable than the full range of the underlying exposure. This is OK for epidemiologic inference but a weakness in a comprehensive risk assessment. For risk assessment, it is important that the model predict the same mean and capture the full variation of the distribution represented by the underlying concentration distribution in the area under consideration. While ground truth can only be approximated due to inherently limited monitoring data, it would be helpful to see a more direct assessment of the performance of the downscaler model for the risk assessment purpose. Also it would be worth considering additional exposure models in the risk assessment as the risk assessment results may be particularly sensitive to the choice of exposure model.
  - e. The treatment of the 2015 downscaler is fairly cursory (Section C.1.4.5). I think more details are warranted. For instance, the cross-validation should be more clearly described (e.g. how were the 10% of withheld locations selected? On what time scale is Table C-8?). Also a useful assessment would be where the 47 urban areas are withheld and these are evaluated.
  - f. The linear interpolation approach to assessing additional standards represents a reasonable compromise to meaningfully reduce EPA's workload given the compressed timeframe for producing the PA. Were additional time

available I would suggest modeling at least one more level in order to understand better whether the linear assumption is reasonable.

- g. Other comments: It would be helpful to also show a version of Table C-6 restricted to the 47 urban areas. It would be helpful to include a table that documents the number of 12 km grid cells per CBSA since this will affect the estimates of spatial variability within CBSA.
2. Regarding Section C.2.2, I note that a major source of variation in numbers of individuals affected (the scale most of the risk estimates are reported on) across 12 km modeling regions is the size of the at-risk population in that region. This could come across a bit more clearly.
3. The robustness and validity of the risk estimates may be most sensitive to the use of the downscaler rather than one of the other national models presented in Chapter 2. This was not addressed at all in the risk assessment. (See also my Chapter 2 comments below.) I encourage EPA to evaluate this input in sensitivity analyses.
4. I would like EPA to carefully address whether they are able to include the entire US in their risk assessment or need to continue to rely on a subset of urban areas as they have done here.

### **Comments on the PA's preliminary conclusions regarding the PM<sub>2.5</sub> standards**

I agree with the draft PA preliminary conclusion that the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards should be called into question.

I agree with the "...focus on the annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short-and long-term PM<sub>2.5</sub> exposures..." (p 3-13, line 7-9) Add strong statement on short-term standard

It is appropriate for the PA to discuss support for and potential implications of putting more or less weight on various aspects of the evidence. (p 3-15)

In calling into question the current standards (in favor of lower standards), EPA puts appropriate weight on the longstanding body of health evidence for serious short- and long-term effects, noting that newer evidence supports and strengthens the previous 2009 conclusions. They also note epidemiologic evidence for effects at low PM levels and that no evidence of a threshold has been identified. They highlight that the risk assessment results suggest large numbers of deaths could be avoided with a lower standard.

In considering the alternative argument that the current standard should be retained, EPA notes that substantial weight must be placed on a number of uncertainties, including the biologic pathways, public health impacts of air quality improvements, and the risk assessment results. As was solidified during our meeting discussion and developed fully in our consensus comments, these arguments for retaining the current standard are not scientifically justified.

In discussing potential alternative standards, the arguments for the indicator, averaging time, and form are straightforward and indicate no change is needed. In discussing the level, I agree with EPA's appropriate focus on "the annual PM<sub>2.5</sub> standard as the principle means of providing increased public health protection." Their consideration is

informed by existing concentrations and their relationship with design and pseudo-design values, as well as effects in controlled human exposure studies and their risk estimates. Together these justify a lower alternative standard. Furthermore, as became apparent during our meeting discussion, the standards will be less protective if only the annual standard is lowered without lowering the 24-hour standard. Thus I do not concur with EPA's recommendation to retain the 24-hour standard. There are locations where the 24-hour standard is the controlling standard and in order to protect public health in those locations the 24-hour standard also should be lowered.

#### **EPA-6: Future research areas**

A few topics to add to the research agenda:

- Better understanding of exposure models and their features, comparing and contrasting their utility for epidemiology vs. risk assessment. (Builds on comment on p 2-48, lines 24-27)
- There is ongoing need for characterization of the performance of modeled ambient concentration fields estimated using hybrid modeling methods. We need to better understand the different implications of the hybrid models.
- Methods for mixtures and effect modification.
- Causal inference methods. See for instance the commentary by Carone, Dominici, and Sheppard, *Epidemiology*, in press.

#### **Chapter 2 comments**

- P 3-37 l 3-4: Please add the size bins
- P 2-38 figure 2-22: The legend of this figure is confusing since the x axis is year for both y-axis measures. Also I suggest a time series plot is clearer if plotted using connected lines rather than points as shown. The best fit lines can still be included. Also, please clarify whether there are data missing in the plot, or whether some values are overplotted. (A line plot would be less confusing w.r.t. this point.)
- It seems to me that the key goal of Section 2.3.3 Predicted Ambient PM<sub>2.5</sub> Based on Hybrid Modeling Approaches is to present results from several models and provide context for the one selected for use in the risk assessment.
  - I suggest reframing this section and eliminating extra detail. As part of this reframing I suggest presenting the link of the models discussed to the health studies to the models used in the risk assessment. (However, priorities in choosing exposure models useful for risk assessment are distinct from those for epidemiologic inference. For risk assessment (and in contrast to epidemiology), it is essential to capture the full variation of the population exposure distribution. For epidemiology it is the quality of the predicted mean and the spatial alignment of the data used in the model with the target health study population that are important for inference.)
  - P 2-39: I find EPA's use of "hybrid" terminology confusing. Its first use on line 3 seems clear enough to me and consistent with my understanding of the common usage for air pollution prediction models. This brief mention acknowledges the reference to "hybrid" is to capture the explicit combination of data from multiple sources. It does not refer to weighting of all the same kind of data (e.g. as in inverse distance weighting), or using some air quality

- data as predictors in a regression model. Section 2.3.3.1.1, Overview of hybrid methods, goes on to include interpolation and machine learning methods, which I would not consider “hybrid”. Regarding interpolation methods, perhaps it is the reference to including weighting by a CTM (line 23), that makes this description “hybrid”? I encourage EPA to revisit this section. One solution may be to simplify the presentation and eliminate some of the detail.
- Section 2.3.3.1.2 seems to be too much detail for the PA. Also there are many details in how R<sup>2</sup>'s are calculated that may mean the estimates reported on p. 2-41 are not comparable. One hint that this may be the case is that the Di and Hu study estimates have opposite ordering for their R<sup>2</sup> and RMSEs. Also generally the RMSEs are more interpretable scientifically and the RMSE value for the downscaler results should be reported. Finally, cross-validated results ought to appropriately capture overfitting so this should be reflected in any cross-validated model performance statistics.
  - Since maps inherently smooth over large spatial scales, it is hard to interpret the effect of showing the predictions “at their native resolution”. I suggest one set of zoomed in maps in a region with sufficient PM<sub>2.5</sub> variation with a total of 16 or 64 12-km grid cells to allow better understanding of the impact of the native resolution.
  - P 2-46 Figure 2-26: It is probably worth spelling out coefficient of variation here since the CV abbreviation is easily confused with cross-validation. Also provide an explicit definition of its use here, which I believe is the standard deviation of the estimates across the 4 models divided by the mean across these four.
  - Figure 2-28 is particularly informative and suggests to me that there are some structural features in the data and its use in the downcaler model that provide such strong bands of similar concentrations in the middle section of the US. This feature suggests to me that it would be worth considering additional exposure models in the risk assessment as the risk assessment results may be particularly sensitive to the choice of exposure model.
  - Given human contributions and causes, is it fair to classify all wildland fires as “background”? See references included in the consensus comments that suggest this is an incorrect classification.
  - We should clarify the difference in emphasis in model- vs monitor-based methods for urban areas and the relevance of this focus for the PA. p 2-48 10-13

### **Comments on CASAC and my reaction to consultant comments**

- I agree completely with Duncan Thomas' comments, a CASAC consultant. Of particular note he provides important overall perspective about the state of causal inference in epidemiology. This is a perspective CASAC hasn't heard under Dr. Cox's leadership and it is an important one. Dr. Thomas' perspective is completely consistent with my recently published commentary, available online (Carone, Dominici, Sheppard, *Epidemiology*, in press). (I also submitted this paper to the CASAC docket.)

- EPA should leverage Dr. Thomas' replies, particularly to Dr. Cox's questions, in its revisions to the PA. Particularly given the outsize attention CASAC is paying to causal inference, I think it is important for the PA to address these considerations directly.
- I want to express concern about the CASAC Chair providing references to his own research to CASAC. Given his leadership position and federal rules about conflict of interest, this self-promotion is of concern.
- I want to express concern about the apparent outsized role of the CASAC Chair in the upcoming Panel deliberations. As per the October 24-25 draft agenda, Tony Cox has been assigned to respond to charge questions for four of the five chapters and is the lead discussant for two of these. I do not think it is appropriate for CASAC perspective to be dominated by one person's views and the optics suggest CASAC's opinion will be dominated by Tony Cox. Furthermore, a more appropriate role of the Chair is to navigate consensus among all Panelists, rather than to dominate the discussion. Based on previous CASAC meetings, I am concerned that he will attempt to push the Committee to an extreme perspective without attention to consensus.

### **Causal inference, epidemiologic studies, and evidence**

Regarding application of causal inference methods, under appropriate conditions, i.e., reasonable causal assumptions, causal inference tools allow us to draw causal conclusions from epidemiologic studies, much as we would if we could experimentally manipulate the exposures in the populations under study. Causal inference relies on framing a causal question of interest in terms of counterfactual or potential outcomes, and then ensuring that the causal question can be estimated from the observed data by mapping onto these observed data the unobservable causal contrast obtained from the potential outcomes. Essential to this mapping is the validity of the required causal assumptions. These assumptions are challenging to meet in their entirety in many studies, and particularly observational studies, although as CASAC consultant Duncan Thomas notes, "these may be reasonable depending on the context." Furthermore, even when the causal assumptions cannot be met completely, a causal framework can still be useful for informing policy-relevant decision-making. I also wish to note that there are many challenges to conducting valid causal inference analyses of observational data, from the most basic framing of causal questions and ensuring the validity of the causal conditions, to actually estimating causal effects. Specific to air pollution epidemiology, some more difficult aspects of these challenges include defining a causal effect due to the complex time-varying nature of air pollution exposures, including their multi-pollutant nature; the inherent limitations of relying on observational data, particularly with regard to estimating the relatively small effects typical in air pollution studies; the challenge of accurately quantifying the exposure used in the inference; and the current emerging state of methodological research in the field of causal inference.

Given the important policy implications of the PM<sub>2.5</sub> health effect evidence from observational studies, I emphasize that the epidemiologic study evidence is credible for advancing air pollution policy. While it would be ideal if the PA could rely on recently developed causal inference methods for these policy inferences, most of the current

body of evidence was developed under conventional inferential analyses that aren't explicitly framed in a causal inference framework. Nonetheless, these existing studies give us important insights, and when taken together, combine to give a weight of evidence that is substantially stronger than any single study can provide alone. As noted by CASAC consultant Duncan Thomas, "it would be inappropriate to dismiss them [i.e., epidemiologic studies] as not addressing causation, given their concordance and the general conformity with the criteria used by epidemiologists for decades to qualitatively evaluate causation." Carone et al (in press) state that, "causal inference methods should not be used as another opportunity to weaponize science against itself." The Clean Air Act requires EPA to act to protect public health with an adequate margin of safety, even in the presence of uncertainty. Just because most air pollution epidemiology studies do not explicitly apply causal inference methods, this is not an appropriate justification for discounting or discrediting the evidence they provide.

Carone M, Dominici F, Sheppard L. In pursuit of evidence in air pollution epidemiology: The role of causally driven data science. *Epidemiology*, 2019, in press. NIHMSID 1535952

### **A few insights based on the Panel (IPMRP) discussion**

- The risk assessment in the context of acceptable risk: The risk assessment implies that the risk at the current standard is greater than 1 in 10,000 (using ~50,000 excess deaths and a US population of 330 million). This is MUCH higher than what would be considered acceptable for increased risk in the general population for cancer risk assessments. While these are not directly comparable, this is helpful perspective.
- I concur with arguments about the importance of a sub-daily standard, particularly to capture traffic-related PM in the morning and wood smoke exposures in the evening.
- There is a need for a Federal Reference method for UFP.

### **Specific details to consider in revising the PA**

- C-30 lines 16-17: Revise the wording of this section to clarify that the measured concentrations are the basis of the projection vs. the current wording, which implies that the measured concentrations are the result of the projection.
- Figures C-26 and C-28: Please use a different color scheme from the maps and define the color scale. The current presentation invites confusion.
- Add RRF – relative response factors – to the list of abbreviations.
- P 3-19 line 13 "cohorts"
- P 3-23 l 28: I agree with the judgment that the heterogeneity is multifactorial.
- A few places with discussion of the width of CIs relative to the mean PM: As I understand the text, the feature being described is a property of CIs for regression model estimates ( $\hat{Y}$ ). (e.g., p 3-51, p 3-10)
- Figure 3-11 p 3-83: It would be helpful to add some clarification in the text or as a footnote regarding the values reported on the graph that correspond to the various standards.
- P 3-85 table 3-5: Add a footnote to define the ranges reported in the table.
- P 3-87 observations about potential alternative standards should note that the study used to develop the risk estimates had more impact



- P 3-90: Fix the Figure 3-12 title to be more stand-alone, adding that it is IHD mortality and that the risk estimates come from Jerrett.
- P 3-90 footnote 68: I think the explanation should be reworded to say the risk estimates were truncated. Or perhaps the intended meaning is that what is reported are the risk estimates for a range of concentrations depicted at the integer concentration level. Clarify.
- P 3-91: It appears that the graphic is a table, not a figure. Also refine the caption.
- Figures 3-3 to 3-6, it would be helpful to add a column for the pseudo-design values and to order the studies (perhaps within country) by PM means.

## Dr. Barbara J. Turpin

**SCQ 2.1** *Regarding whether the draft PA accurately reflects and communicates the air quality related information most relevant to its subsequent evidence-based assessment of the health and welfare effects of studies, including uncertainties, as well as the development of the risk assessment for current and alternative standards?*

*In particular, do the following sections accurately reflect and communicate current scientific understanding, including uncertainties for:*

- a) relationships between annual and daily distributions of PM;*
- b) the review of hybrid modelling approaches used to estimate exposure in some studies and the risk assessment; and*
- c) information on background levels of various PM indicators?*

- a) Annual and daily:** The document notes that, in the Northwestern US, daily and sub-daily (2-hr) concentrations (and the relationship between annual and daily) are heavily influenced by wildfire emissions in the summer/fall and stagnation in the winter. ***Not reflected adequately here are the impacts of controllable emissions, including seasonal or episodic emissions on these features nor do they reflect impacts from controlled burns, which are*** a major risk reduction approach for forestry. The text implies that these high concentrations are beyond our control. It does not acknowledge that stagnation events concentrate *anthropogenic emissions* near the surface in the winter, sometimes leading to high ground-level concentrations. Local heat emissions in urban settings (the Urban Heat Dome) can contribute to local stagnation as well. The episodic but substantial contribution of residential wood combustion for home heating is one of these anthropogenic sources. It does not acknowledge that anthropogenic activities impact climate, which contributes to drought and fire in the west. Currently, the inaccurate impression that is created regarding 24 h and sub-daily concentrations is used to discount and exclude measurements in the Northwest and California from the risk assessment and the consideration of whether the annual standard can adequately control for health effects associated with short term exposures (Chapter 3).

The text in question is here:

Page 2-26 “Northwest U.S. has very high daily design values relative to the annual design values. This is due to episodically high PM<sub>2.5</sub> concentrations that affect the region, both from wintertime stagnation events and summer/fall wildfire smoke events”

2-30 Wildfires are having an important and substantial impact on Apr-Sept exposure in the western US. Only says “Most of the sites measuring these very high concentrations are in the northwestern U.S. and California, where wildfires have been relatively common in recent years”

- b) Hybrid modelling:** Performance of Methods (2.3.3.1.2) -- The most important points that should be made in this section do not come through clearly. Impressively, some of the more sophisticated methods have n-fold cross validation R<sup>2</sup> better than 80% and root-mean-square error (RMSE) of 2-3 µg/m<sup>3</sup> for daily PM<sub>2.5</sub> predictions. These methods clearly lead to improved exposure estimates in locations without samplers. The second paragraph tells where performance is worse but not where it is better. Approaches including land-use features, rather than straight Bayesian downscaling, are better at

capturing concentration gradients close to sources. The consistency of the regional concentration estimates across methods is remarkably good (Table 2-3).

Rather than focus on variability among the methods, this text should be explaining why some methods work better than others. The Bayesian downscaler does not incorporate information about locations of primary  $PM_{2.5}$  sources (i.e., surrogates such as land use variables), whereas several other methods, including the neural network, do. All these methods are designed to predict broad spatial  $PM_{2.5}$  features, but the neural network and other methods including land use variables do a better job of capturing spatial gradients near sources. Ideally, the concentrations predicted across the US from the *best* performing methods should be used to conduct risk assessment for the entire country, rather than conducting the risk assessment for only a modest number of sites. The Bayesian downscaler is the worst of these methods (especially for the Northwest and California), and yet it was the one selected for further analysis. The selection of the Bayesian downscaler likely leads to an underestimation of exposure and risk in the Northwest and California, assuming higher populations are spatially collocated with sources.

Importantly, the text is wrong as to the reason that there is worse agreement between these methods in the west. The reason is not because concentrations are low in the west, it is because spatial concentration gradients are substantially greater in the west than in the east, where  $PM_{2.5}$  is more influenced by secondary formation and more therefore regionally homogeneous.

In some cases, variations between methods are discussed with no explanation given as to why they make sense. For example:

“Predictions span a wider range of concentrations for the western regions centered on California and Arizona (Figure 2-25, panels a and c) than the eastern region centered on New Jersey (Figure 2-25, panel b).”

This makes sense – in the eastern US, a larger fraction of  $PM_{2.5}$  is secondary, formed regionally, and thus concentrations can be expected to be more spatially homogeneous. This is not explained.

“Despite general agreement among predictions for the California and the eastern U.S. areas, the spatial texture of the concentration fields differs among methods. For instance, the 12-km Bayesian downscaler produces the smoothest  $PM_{2.5}$  concentration field, and the 1-km neural network (DI2016) produces the field with the greatest variance.”

This also makes sense, since the Bayesian downscaler does not incorporate information pertaining to the locations of primary  $PM_{2.5}$  sources, whereas the neural network does. Thus, both are designed to predict broad spatial  $PM_{2.5}$  features, but the neural network will do a better job of capturing spatial gradients near sources. This is not explained, and may leave the reader without this important context.

“In Figure 2-26, the coefficient of variation (CV; i.e., the standard deviation divided by the mean) among methods is shown in percentage units based on predictions that were averaged to a common 12-km grid. The largest values occur in the western U.S. (Figure 2-26, panel a), where terrain is complex, wildfire is prevalent, monitoring is relatively

sparse, and PM<sub>2.5</sub> concentrations tend to be low. The distance from the grid-cell center to the nearest monitor is greater than 100 km for broad areas of the west (Figure 2-27)."

Yes, distance to monitors is large in many parts of the West, but the reason the simpler method (Bayesian downscaler) does not perform as well in the west is because of the larger concentration gradients, not the low concentrations. The methods that make use of land use variables (e.g. neural network) have an advantage in situation. The spatial gradients are more extreme in the west, whereas in the east regional secondary formation leads to more spatially uniform concentrations. The differences between methods make sense.

- c) **Background:** As an upperbound, background was estimated by assuming all biogenic SOA is natural. For the record, I would like to remind the authors that even though it is made from biogenic hydrocarbons, **biogenic SOA is not necessarily natural**.

There is substantial evidence that anthropogenic emissions impact the formation of SOA from biogenic VOCs. This was raised in my comments on the first draft of the Integrated Science Assessment. One important example is isoprene. Oxidation of isoprene leads to several gas phase products. A major SOA precursor is isoprene epoxydiol (IEPOX), which forms SOA when it reacts with wet acidic sulfate (anthropogenic). Thus, IEPOX SOA is formed as a result of reactions with anthropogenic emissions, and thus are controllable. Field studies measuring tracers of IEPOX SOA suggest that it is a major source of aerosol (roughly one-third of organic PM<sub>2.5</sub>) in the southeastern US in both rural and urban locations (see reference below and in the ISA).

Budisulistiorini, S., Li, X., Bairai, S.T., Renfro, J., Liu, Y., Liu, Y.J., McKinney, K.A., Martin, S.T., McNeill, V.F., Pye, H.O.T. and Nenes, A., 2015. Examining the effects of anthropogenic emissions on isoprene-derived secondary organic aerosol formation during the 2013 Southern Oxidant and Aerosol Study (SOAS) at the Look Rock, Tennessee ground site. *Atmospheric Chemistry and Physics*, 15(15), pp.8871-8888.

Budisulistiorini, S.H., Canagaratna, M.R., Croteau, P.L., Marth, W.J., Baumann, K., Edgerton, E.S., Shaw, S.L., Knipping, E.M., Worsnop, D.R., Jayne, J.T. and Gold, A., 2013. Real-time continuous characterization of secondary organic aerosol derived from isoprene epoxydiols in downtown Atlanta, Georgia, using the Aerodyne Aerosol Chemical Speciation Monitor. *Environmental science & technology*, 47(11), pp.5686-5694.

As another example, model predictions by Carlton et al, suggest that more than 50% of biogenic SOA in the Eastern U.S. could be controlled by reducing anthropogenic NO<sub>x</sub> emissions.

Carlton, A.G., Pinder, R.W., Bhawe, P.V. and Pouliot, G.A., 2010. To what extent can biogenic SOA be controlled?. *Environmental Science & Technology*, 44(9), pp.3376-3380.

The following text does not recognize that SOA from biogenic VOCs is, in part, controllable:

Page 2-3 "Natural sources of PM include...oxidation of biogenic hydrocarbons such as isoprene and terpenes to produce secondary organic aerosol (SOA),"

Page 2-50: “sources that contribute to natural background PM.... oxidation of biogenic hydrocarbons such as isoprene and terpenes to produce SOA”

Page 2-55: “As a region, the Southeast has the highest levels of biogenic aerosol production in the country, so the organic matter contribution at these three sites likely represents an upper bound for the country of what natural biogenic organic aerosol production could be under present atmospheric conditions.”

**Additionally:** Please note that water-soluble gases also contribute via multiphase reactions in clouds and aerosols. Not reflected in the following text:

Page 2-9 “In addition, atmospheric oxidation of VOCs, both anthropogenic and biogenic, is an important source of organic aerosols, particularly in summer. The semi-volatile and non-volatile products of VOC oxidation reactions can condense onto existing particles or can form new particles (U.S. EPA, 2009, section 3.3.2; U.S. EPA, 2018, section 2.3.2).”

**SCQ 3.3** *Regarding approaches described in Chapter 3 of the PA considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? Regarding rationales supporting the preliminary conclusion on the current and potential alternative primary PM<sub>2.5</sub> standards? Regarding the evidence-based approach, including:*

- e) *The preference for continuing the use of an annual PM<sub>2.5</sub> standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM<sub>2.5</sub> exposures?*

The PA presents a substantial case, using multiple lines of evidence, that the current PM<sub>2.5</sub> standards are not adequate to protect public health with a requisite margin of safety. ***It will be necessary to reduce both the annual and 24 h standard.*** The annual standard may be used as the principle means to provide public health protection against health effects associated with short- and long-term exposures, but cannot be used as the only means of protecting public health. It is clear from Figure 2-11 that lowering the annual standard vs the 24 h standard will protect different people. Lowering the annual standard alone will result in reduced short- and long-term exposures for people predominantly in the east and industrial Midwest, but will not provide protection from short term and peak exposures for people in the Northwest and California. The health of people in the Northwest and California must still be protected whether or not wildfires and stagnation events occur during summer, fall and winter seasons.

**SCQ 3.4** *Regarding the quantitative risk assessment for PM<sub>2.5</sub>, including:*

- c) *The hybrid modeling approach used...*

See response to SCQ 2.1 (b) above.

- d) *The characterization of variability and uncertainty in the risk assessment?*

Page 3-70: As stated above (comments on Chapter 2) the performance of the hybrid models (most particularly the Bayesian downscaling) is not hampered by low

concentrations. It is hampered by strong spatial concentration gradients. Hybrid methods that include land use factors related to primary sources are better able to address this. Regional secondary formation in the east means that spatial gradients are much smaller and the models perform better. It makes sense that the neural network hybrid model would perform better than the Bayesian downscaling in the west for this reason. Thus, I disagree with the following statement:

“factors likely contributing to poorer model performance often coincide with relatively low ambient PM<sub>2.5</sub> concentrations, potentially accounting for the observations that model performance for hybrid models weaken by some metrics with decreasing PM<sub>2.5</sub> concentration and that the normalized variability between predictions based on different hybrid modeling approaches increases with decreasing concentrations. Thus, uncertainty in hybrid model predictions becomes an increasingly important consideration as lower predicted concentrations are considered.”

Uncertainty is larger for Bayesian downscaling models specifically, in locations with large concentration gradients. In the west, more weight should be placed on the other hybrid models.

**SCQ 3.5** *Regarding the draft PA preliminary conclusion that, taken together, the available scientific evidence can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards.*

I agree that the evidence is strong. Specifically, ***I agree with the following statement, which is well documented in the evidence base and supported by the risk assessment.***

**(page 3-98)**

“When taken together, we reach the preliminary conclusion that the available scientific evidence, air quality analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the combination of the current annual and 24-hour primary PM<sub>2.5</sub> standards.”

Regarding the subsequent paragraph:

“In contrast to this preliminary conclusion, a conclusion that the current primary PM<sub>2.5</sub> standards do provide adequate public health protection would place little weight on the broad body of epidemiologic evidence reporting generally positive and statistically significant health effect associations, particularly for PM<sub>2.5</sub> air quality distributions likely to have been allowed by the current primary standards, or on the PM<sub>2.5</sub> risk assessment. Rather, such a conclusion would place greater weight on uncertainties and limitations in the evidence and analyses”

A conclusion that the current primary PM<sub>2.5</sub> standards do provide adequate public health protection cannot be justified based on the weight of the evidence from multiple kinds of data and analyses clearly documented in the ISA and PA. No scientific rationale is offered for affording any uncertainties and limitations greater weight than that given to the scientific results.

## **Dr. Ronald Wyzga**

### **Chapter 1 – Introduction: To what extent does the CASAC find that the information in Chapter 1 is clearly presented and that it provides useful context for the review?**

The Chapter is clearly written, but it omits key factors that set the context for this review. First of all, it does not indicate the differences in the overall review process for PM in this review as opposed to previous reviews. Secondly, there was limited review of the ISA with only one draft reviewed despite the comments made on the first draft. Thirdly, there is no formal risk and exposure assessment as has been included in previous reviews. Finally, the content of this chapter is dependent upon the science and conclusions of the ISA. Only a draft version is available; the final version is planned for release in December 2019. Given the uncertainty about the content of this document, it makes it difficult to make this document at best provisional and subject to change given changes in the ISA. This Chapter needs to recognize these factors and indicate how the overall process will accommodate them.

### **Chapter 3 – Review of the Primary PM<sub>2.5</sub> Standards: What are the CASAC views on the approaches described in Chapter 3 to considering the PM<sub>2.5</sub> health effects evidence and the risk assessment in order to inform preliminary conclusions on the primary PM<sub>2.5</sub> standards? What are the CASAC views regarding the rationales supporting the preliminary conclusions on the current and potential PM<sub>2.5</sub> standards?**

#### **SCQ-3.1 Does the panel find that the questions posed in this chapter appropriately reflect the important policy-relevant issues for the PM<sub>2.5</sub> review? Are there additional policy-relevant questions that should be addressed?**

The content is based upon a draft ISA; it is unclear whether a final ISA would influence the discussions and conclusions of this chapter. By and large the questions addressed are reasonable. I would have like to have seen more discussion of PM components other than ultrafine particles. Although virtually all PM components have been shown to have some adverse health impacts, there are some differences among major components for both respiratory and cardiovascular endpoints. Although these differences would not change the PM indicator, they are noteworthy and could help inform risk managers about the need to consider all major PM components in achieving compliance, I base my conclusions on two relatively recent reviews in which I was involved. A comprehensive review of the literature for both short-term and long-term studies found that different components were associated with respiratory and cardiovascular endpoints; moreover, although no major components of PM were exonerated, there appeared to be greater and more associations with organic particles than with other components. See A. C. Rohr and R. Wyzga, *Attributing health effects to individual particulate matter constituents. Atmospheric Environment* 62 130-152 2012 and R.E. Wyzga and A. C. Rohr. *Long-term Particulate Matter Exposure: Attributing Health Effects to Individual PM Components. J of the Air and Waste Manage. Assoc.* 2014.

#### **SCQ 3.2 What are the Panel's views on the relative weight that the draft Policy Assessment gives to the evidence-based (i.e., draft PA Section 3.2) and risk-based (i.e. draft PA, section 3.3) approaches in reaching conclusions and recommendations regarding current and alternative PM 2.5 standards?**

I like the fact that two approaches were considered; the conclusions for each were similar which adds strength to an overall conclusion. Both approaches clearly indicate

that the current standard is not protective. These sections do not consider all studies covered in the ISA. Greater justification of the studies considered need be incorporated into the PA.

**SCQ 3.3 What are the Panel's views on the evidence-based approach, including:**

- a) The emphasis on health outcomes for which the draft ISA causality determinations are “causal” or “likely causal”?**

I have no problem with considering the adverse health effects two categories. It should be noted that consideration of the “causal” and “likely causal” categories will most likely result in standards that are protective of other categories. To the extent that this may not be true, some indication could be useful.

- b) The identification of potential at-risk populations?**

The draft PA rightly indicates that very large subpopulations are at-risk. Greater specificity is not necessary.

- c) Reliance on key multicity epidemiology studies conducted in the US and Canada for assessing the PM 2.5 levels associated with health effects?**

There should be greater discussion about how the results might change if a broader set of studies considered in the ISA were included here.

- d) Characterizing air quality in these key studies using two approaches: the overall mean and 25 th /75 th percentiles of the distribution and the “pseudo design value” reflecting a monitor with the highest levels in an area?**

The approach is reasonable although there should be some discussion about the nature of the overall statistical distribution; this may be covered in Chapter2, which I have not yet reviewed.

- e) The preference for continuing the use of an annual PM 2.5 standard as the principle means of providing public health protection against the bulk of the distribution of short- and long-term PM 2.5 exposures?**

If the analysis were the other way around, would it be as useful? My concern is that some extreme events could possibly alter some of the assumptions between long-term and short-term air quality measures.

- f) The draft PA conclusions on the extent to which the current scientific information strengthens or alters conclusions reached on the last review on the health effects of PM<sub>2.5</sub>?**

I agree with the conclusions.

- g) Whether the discussions of these and other issues in Chapter 3 accurately reflect and clearly communicate the currently available health effects evidence, including important uncertainties as characterized in the ISA?**



Without seeing the final ISA, it is difficult to evaluate this question. This chapter considers a subset of studies covered in the current ISA; it would be helpful to explain further how the subset was chosen and what would be the impact of considering a wider set of studies.

**SCQ 3.4 What are the Panel's views on the quantitative risk assessment for PM<sub>2.5</sub> including:**

- a. The choice of health outcomes and studies selected for developing concentration-response functions for long and short-term effects?

I would like to see greater explanation of how the selected studies were chosen, and what the likely impact would be if additional studies were chosen as well. I was struck by the fact that the studies that used modeling as opposed to monitoring to estimate PM exposures appeared to give slightly different results. I would like to see some discussion of this. Is it because different geographic regions were considered or some other reason?

- b. The selection criteria for the 47 urban areas and PM<sub>2.5</sub> air quality scenarios analyzed?

No problems here.

- c. The hybrid modeling approach used for quantifying exposure surrogates across an area and adjusting air quality for alternative standard levels, as supplemented by interpolation/extrapolation?

It seems reasonable

- d. The characterization of variability and uncertainty in the risk assessment?

Again if additional studies were considered, would the results and their variability change much?

- e. The robustness and validity of the risk estimates?

I would like to see more discussion of the differences seen in those studies that considered modeling as opposed to monitoring to estimate PM levels.

**SCQ-3.5 What are the Panel's views on the draft PA preliminary conclusion that, taken together, the available scientific evidence, air quality analyses, and the risk assessment can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current primary PM<sub>2.5</sub> standards?**

I agree.

**SCQ-3.6 What are the Panel's views on the conclusions in the draft PA regarding developing potential PM<sub>2.5</sub> alternative standards with respect to:**

- a. The preliminary conclusion that the available information continues to support the PM<sub>2.5</sub> mass-based indicator, remains too limited to support a distinct standard for any specific PM<sub>2.5</sub> component or group of components, and remains too limited to support a distinct standard for the ultrafine fraction?

The issue here remains tied to the ISA. I agree that no major constituent of PM is exonerated, but the draft ISA, in my opinion, does not fully discuss the relative roles of major constituent categories. See my comments with regard to charge question 3.1.

- b. The preliminary conclusion to retain the annual and 24-hour averaging times?

Reasonable, but it should be pointed out that most studies make use of commonly reported air quality measures. Further research to indicate whether other averaging times would be preferred is lacking.

- c. The preliminary conclusion that it is appropriate to consider retaining the forms of the current annual and 24-hour PM<sub>2.5</sub> standards, in conjunction with revised levels?

- d. The preliminary conclusion that the range for alternative levels for the annual PM<sub>2.5</sub> standard should begin below 12 µg/m<sup>3</sup> and extend as low as 8 µg/m<sup>3</sup>?  
Reasonable

- e. The possible rationales for alternative annual PM<sub>2.5</sub> levels of 12, 10, and 8 µg/m<sup>3</sup>?

Reasonable, but I would like to see further discussion of why the Canadian studies and those studies which used modeled air quality data appear to give different results.

- f. The preliminary conclusion that, in conjunction with a lower annual standard intended to protect against both short- and long-term exposures, the evidence does not support the need for a revised level for the PM<sub>2.5</sub> 24-hour standard?

I worry about this. The arguments supporting this position are not crystal clear to me and all of the assumptions therein need be clearly articulated.

- g. The discussion of an alternative approach to lower the level of the 24 hour standard to 30 µg/m<sup>3</sup> to provide increased protection for both short- and long term exposures?

I liked this.

**Chapters 3 to 5: What are the CASAC views regarding the areas for additional research identified in Chapters 3, 4 and 5? Are there additional areas that should be highlighted?**

The current review must be based upon existing information; however, there are several areas that could inform future reviews of the standard and help reduce some of the uncertainties associated with this process.

I believe that future research should include the following:

- More detailed measurement of PM components; in particular, more detailed measurements of organic components. Several studies have suggested that some organic components may be of greater health concern than others. EC and OC are catchall categories defined by a measurement technique. Availability of such measurements would facilitate their use in future epidemiological studies.
- Research should also continue to define in more detail the physiological bases for adverse health responses to PM and its components. It may be that different components are associated with different components. If so, consideration of

components may provide a more precise understanding of the biological basis for observed responses in epidemiological studies.

- Alternative exposure metrics need to be explored. How important are peak exposures as opposed to average exposures in explaining observed health responses? What is the appropriate time average for peak exposures? Do current average measures adequately limit exposures to peak levels? Is the relative change in exposure important; research needs to consider the issue of delta exposure. How important are past exposures in explaining responses to current levels; indeed the correct question to ask is what are the impacts of current exposures given past exposures? This is particularly important when health outcomes, e.g., cancer, develop over an extended period of time and when cross-sectional designs are considered. These designs compare exposures and health responses across geographic entities. Although there are changes in air quality over time, the relative ordering of air quality across geographic entities changes minimally. What is the latency of response? Tied to this is the issue of cumulative exposure, which should be examined.
- Consideration of the NAAQS for the coarse fraction of PM is limited because measurement of the coarse fraction per se is limited. There are studies, especially considering asthmatic response, that report significant associations with PM<sub>10</sub> but not PM<sub>2.5</sub>. Statistical and other phenomena could explain these results, but they could also suggest that coarse PM, independent of fine PM, may be of health concern. More research on the relationship between asthmatic and other respiratory responses and coarse PM is needed.
- Health research tends to be focused on one pollutant at a time even when several pollutants are measured, but they are most often considered independently. How important is joint exposure to more than one pollutant in influencing health response? Is sequencing of exposures important?

People spend more of their time in indoor environments. Indoor PM levels can be high in these environments? How important are these? If they are not as important, why? What is the health impact of joint indoor and outdoor exposures? Are health responses to outdoor PM levels greater when indoor levels are high?

## Attachment D

### History, Membership Criteria, and Administrative Procedures of the Independent Particulate Matter Review Panel

#### A.1 History of the Independent Particulate Matter Review Panel

The core statutory obligation of the EPA Clean Air Scientific Advisory Committee (CASAC) is incorporated into CASAC's charter with Congress.<sup>58</sup> Under that charter, CASAC may be augmented with experts. Specifically, the charter states:

"EPA, or CASAC with the Agency's approval, may form subcommittees or workgroups for any purpose consistent with this charter. Such subcommittees or workgroups may not work independently of the chartered committee and must report their recommendations and advice to the chartered CASAC for full deliberation and discussion. Subcommittees or workgroups have no authority to make decisions on behalf of the chartered committee, nor can they report directly to the EPA."

Augmentation of CASAC with additional experts for the review of criteria and standards has been a routine practice for four decades. Additional experts have been appointed to review panels that interact with members of the chartered CASAC for all reviews since the late 1970s.<sup>59</sup> Over time, the chartered CASAC has typically been augmented with 12 or more additional experts in a given review cycle for a given criteria pollutant. The average number of experts among 20 such panels for which membership data is available is 14, and the average size of the review panels is 20 members, inclusive of participating CASAC members.

The previous four particulate matter review panels have been comprised of members of the chartered CASAC augmented with additional experts. CASAC was augmented with additional experts for the joint review of the criteria and standards for particulate matter and sulfur oxides in the early 1980s.<sup>60</sup> The CASAC Subcommittee on Health Effects of Particulate Matter and Sulfur Oxides included six experts in addition to members of the chartered CASAC. The CASAC Subcommittee on Welfare Effects of Particulate Matter and Sulfur Oxides included five additional experts in addition to members of the chartered CASAC. In total, there were 11 additional experts who augmented the chartered CASAC for this review cycle. For the 1994 to 1996 PM review, there were 6 members of the chartered CASAC and 15 additional experts on

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<sup>58</sup> United States Environmental Protection Agency Charter, Clean Air Scientific Advisory Committee, Filed with Congress, June 5, 2019, [https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/\\$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf)

<sup>59</sup> Frey, H.C., A.V. Diez Roux, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, "CASAC Review of EPA's Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018)," 34 page letter and 100 pages of attachments submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA-HQ-ORD-2014-0859, December 10, 2018. Pages E-37 to E-39, [https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/\\$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf)

<sup>60</sup> EPA, Air Quality Criteria for Particulate Matter and Sulfur Oxides, Volume 1, EPA-600/8-82-029a, U.S. Environmental Protection Agency, Research Triangle Park, NC, December 1982. [http://ofmpub.epa.gov/eims/eimscomm.getfile?p\\_download\\_id=459608](http://ofmpub.epa.gov/eims/eimscomm.getfile?p_download_id=459608)

the review panel.<sup>61</sup> For the 2001 to 2006 scientific review, and for the 2008 to 2010 scientific review, there were 7 members of the chartered CASAC and 15 additional experts.<sup>62,63</sup> From 2015 to 2018, the CASAC Particulate Review Panel had 6 members of the chartered CASAC and 20 additional experts.<sup>64</sup> Thus, the use of augmented review panels specifically for particulate matter dates back 37 years.

The 7-member chartered CASAC does not have the breadth, depth, and diversity of expertise required for a review of the particulate matter criteria and standards that meets the requirements of the Clean Air Act for a “thorough review” that “shall accurately reflect the latest scientific knowledge” of the “extent and kind of ... effects.”<sup>65</sup> The only credible way to provide a “thorough review” that “shall accurately reflect the latest scientific knowledge” is to engage scientists who are active at the leading edge of scientific work in disciplines and areas related to the subject matter of a review, as described in the February 4, 2015 Federal Register request for nominations, and as illustrated by the history of CASAC Review Panels.

On February 4, 2015, the EPA Science Advisory Board (SAB) office issued a “Request for Nominations of Experts for the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter Review Panel.”<sup>66</sup> In this notice, EPA stated that it will “form a CASAC ad hoc panel to provide advice through the chartered CASAC on the scientific and technical aspects of air quality criteria and the National Ambient Air Quality Standards (NAAQS) for particulate matter (PM).” The notice further stated:

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<sup>61</sup> Wolff, G.T., “Closure by the Clean Air Scientific Advisory Committee (CASAC) on the Staff Paper for Particulate Matter,” Letter to Carol M. Browner, EPA-SAB-CASAC-LTR-96-008, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, June 13, 1996.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/C146C65BA26865A2852571AA00530007/\\$File/casl9608.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/C146C65BA26865A2852571AA00530007/$File/casl9608.pdf)

<sup>62</sup> Henderson, R. “Clean Air Scientific Advisory Committee Recommendations Concerning the Proposed National Ambient Air Quality Standards for Particulate Matter,” EPA-CASAC-LTR-06-002, Letter to Stephen L. Johnson, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, March 21, 2006, [https://yosemite.epa.gov/sab/sabproduct.nsf/CD706C976DAC62B3852571390081CC21/\\$File/casac-ltr-06-002.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/CD706C976DAC62B3852571390081CC21/$File/casac-ltr-06-002.pdf)

<sup>63</sup> Samet, J.M., “CASAC Review of Policy Assessment for the Review of the PM NAAQS – Second External Review Draft (June 2010),” EPA-CASAC-10-015, Letter to Lisa P. Jackson, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, September 10, 2010, [https://yosemite.epa.gov/sab/sabproduct.nsf/CCF9F4C0500C500F8525779D0073C593/\\$File/EPA-CASAC-10-015-unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/CCF9F4C0500C500F8525779D0073C593/$File/EPA-CASAC-10-015-unsigned.pdf)

<sup>64</sup> Diez Roux, A., “CASAC Review of the EPA’s Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – April 2016),” EPA-CASAC-16-003, Letter to Gina McCarthy, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, August 31, 2016.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/\\$File/EPA-CASAC+2016-003+unsigned.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/$File/EPA-CASAC+2016-003+unsigned.pdf)

<sup>65</sup> Frey, H.C., A.V. Diez Roux, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “CASAC Review of EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 34 page letter and 100 pages of attachments submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA–HQ–ORD–2014-0859, December 10, 2018. Page E-39.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/\\$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/$File/PMRP+Letter+to+CA+SAC+181210+Final+181210.pdf)

<sup>66</sup> EPA, “Request for Nominations of Experts for the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter Review Panel,” *Federal Register*, 80(23):6086-6089 (February 4, 2015).  
<https://www.govinfo.gov/content/pkg/FR-2015-02-04/pdf/2015-02265.pdf>

“The SAB Staff Office is seeking nominations of nationally and internationally recognized scientists with demonstrated expertise and research in the field of air pollution related to PM. Experts are sought in: air quality and climate responses, atmospheric science and chemistry, dosimetry, toxicology, controlled clinical exposure, epidemiology, biostatistics, human exposure modeling, risk assessment/modeling, characterization of PM concentrations and light extinction, and visibility impairment and related welfare effects.”

The notice also stated:

“Selection criteria to be used for panel membership include: (a) Scientific and/or technical expertise, knowledge, and experience (primary factors); (b) availability and willingness to serve; (c) absence of financial conflicts of interest; (d) absence of an appearance of a lack of impartiality; (e) skills working in committees, subcommittees and advisory panels; and, (f) for the panel as a whole, diversity of expertise and viewpoints.”

On November 17, 2015, a memorandum from Aaron Yeow to Chris Zarba in the EPA Science Advisory Board office established the CASAC PM Review Panel.<sup>67</sup> The panel was formed for the following purpose:

“An ad hoc expert panel of the CASAC will provide independent advice through the chartered CASAC on EPA’s technical and policy assessments that support the Agency’s review of the National Ambient Air Quality Standard (NAAQS) for PM, including drafts of the Integrated Review Plan, Integrated Science Assessment, Risk/Exposure Assessment, and Policy Assessment.”

In the case of particulate matter, for which there are health effects data from multiple scientific disciplines, including epidemiology, toxicology, and controlled human studies, it has been common practice to have multiple experts in each of these disciplines to assure breadth and depth of expertise. The CASAC PM Review Panel was comprised of leading scientists recognized nationally and internationally for their expertise in multiple scientific disciplines, including air quality, exposure assessment, dosimetry, toxicology, epidemiology, medicine, risk assessment methodology, uncertainty analysis, and related fields.

The CASAC Particulate Matter Panel held teleconference meetings on May 23, 2016, and August 9, 2016, to peer review the EPA’s Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – April 2016).<sup>68</sup>

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<sup>67</sup> Yeow, A., Formation of the Clean Air Scientific Advisory Committee (CASAC) Particulate Matter (PM) Review Panel, Memorandum to C. Zarba, Science Advisory Board Staff Office, U.S. Environmental Protection Agency, Washington, DC, November 17, 2015, [https://yosemite.epa.gov/sab/sabproduct.nsf/0/EB862B233FBD0CDE85257DDA004FCB8C/\\$File/Determination%20memo-CASAC%20PM.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0/EB862B233FBD0CDE85257DDA004FCB8C/$File/Determination%20memo-CASAC%20PM.pdf)

<sup>68</sup> Diez Roux, A., “CASAC Review of the EPA’s Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – April 2016),” EPA-CASAC-16-003, Letter to Gina McCarthy, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, August 31, 2016. <https://yosemite.epa.gov/sab/sabproduct.nsf/9920C7E70022CCF98525802000702022/%24File/EPA-CASAC+2016-003+unsigned.pdf>

On October 10, 2018, then acting EPA Administrator Wheeler eliminated the CASAC PM Review Panel by press release,<sup>69</sup> with a follow-up email from the SAB office on October 11, 2018. This was done without advance notice and without prior consultation with the panel or the CASAC. There is no precedent for disbanding a review panel in the middle of a review cycle.

The EPA released the external review draft of the Integrated Science Assessment (ISA) on October 15, 2018, five days after disbanding the CASAC PM Review Panel.<sup>70</sup> The Federal Register notice announcing that the draft ISA was available for public review was dated October 16, 2018 and published on October 23, 2018.<sup>71</sup>

Compared to the chartered CASAC, the PM review panel has more experts, covers more scientific disciplines, and has multiple experts who provide diversity of perspectives in many key disciplines, such as epidemiology, toxicology, and human clinical studies, among others.

Since that time, members of the disbanded CASAC PM Review Panel have formed this Independent Particulate Matter Review Panel (IPMRP). Like the disbanded CASAC PM Review Panel, the IPMRP is committed to providing “public service” “in protecting public health and safeguarding our nation’s air,” as described in the Nov 20, 2015 appointment letters from the EPA SAB office to panelists. The panel does not require affiliation with EPA to carry on its mission. Although no longer affiliated with the U.S. EPA, the IPMRP continues as a group of independent science advisors recognized for their national leadership in policy-relevant science pertaining to the particulate matter NAAQS.

The mission of this Panel is three-fold: (1) to provide independent advice regarding technical and policy assessments pertaining to the EPA’s review of the National Ambient Air Quality Standard (NAAQS); (2) objectively observe and assess modifications to the NAAQS Review Process and their implications; and (3) educate the public about the public health and public welfare objectives of the NAAQS, the NAAQS review process, and scientific issues pertaining to the NAAQS. Given the process under which this group was originally formed as the CASAC PM Review Panel, we are recognized for our expertise and our independence.

On December 10, 2018, the IPMRP submitted public comments to the CASAC pertaining to the EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018).<sup>72</sup> The IPMRP subsequently submitted comments to the CASAC on March 27,

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<sup>69</sup> EPA, “Acting Administrator Wheeler Announces Science Advisors for Key Clean Air Act Committee Tasks Chartered Panel to Lead Review of Ozone & Particulate Matter Standards Under Reformed Process,” News Release, U.S. Environmental Protection Agency, Washington, DC, October 10, 2018, <https://www.epa.gov/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee>

<sup>70</sup> EPA, “Integrated Science Assessment for Particulate Matter (External Review Draft),” EPA/600/R-18/179, U.S. Environmental Protection Agency, Research Triangle Park, NC, October 2018. [https://yosemite.epa.gov/sab/sabproduct.nsf/0/932D1DF8C2A9043F852581000048170D/\\$File/PM-1STERD-OCT2018.PDF](https://yosemite.epa.gov/sab/sabproduct.nsf/0/932D1DF8C2A9043F852581000048170D/$File/PM-1STERD-OCT2018.PDF)

<sup>71</sup> EPA, “Integrated Science Assessment for Particulate Matter (External Review Draft),” *Federal Register*, 83(205):53471-53472 (October 23, 2019). <https://www.govinfo.gov/content/pkg/FR-2018-10-23/pdf/2018-23125.pdf>

<sup>72</sup> Frey, H.C., A.V. Diez Roux, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “CASAC Review of EPA’s Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 34 page letter and 100 pages of attachments submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency and to Docket EPA–HQ–ORD–2014-0859, December 10, 2018.

2019 with additional comments on the draft ISA.<sup>73</sup> These letters contain detail on the statutory requirements for the review of the NAAQS, history of the CASAC PM Review Panel and the IPMRP, and specific findings and recommendations related to the CASAC, NAAQS review process, and draft ISA.

In early September of 2019, EPA released an external review draft of the Policy Assessment (PA) for the PM NAAQS review.<sup>74</sup> A Federal Register notice published on September 11, 2019 indicated availability of the draft PA for public comment through November 12, 2019.<sup>75</sup> The chartered CASAC will hold a public teleconference on October 22, 2019 to receive public comments to consider in their peer review of the EPA's Policy Assessment for Particulate Matter on October 24-25, 2019.<sup>76</sup> The chartered CASAC will hold a public meeting at a location to be determined in North Carolina on October 24-25, 2019 for the purpose of conducting a peer review of EPA's Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft – September 2019).<sup>77</sup>

The CASAC stated in its April 11, 2019 letter to the EPA Administrator that “the breadth and diversity of evidence to be considered exceeds the expertise of the statutory CASAC members, or indeed of any seven individuals.”<sup>78</sup> Furthermore, the CASAC recommended that “the EPA reappoint the previous CASAC PM panel or appoint a panel with similar expertise.” The disbanding of the PM Review Panel on October 10, 2017 deprived CASAC of the needed expertise. The EPA Administrator responded in a letter dated July 25, 2019 that disregarded CASAC's advice to reappoint the disbanded panel or form a new panel. Specifically, the

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[https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/\\$File/PMRP+Letter+to+CASAC+181210+Final+181210.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/086D8B853E0B63AE8525835F004DC679/$File/PMRP+Letter+to+CASAC+181210+Final+181210.pdf)

<sup>73</sup> Frey, H.C., A.V. Diez Roux, P. Adams, G. Allen, J. Balmes, J.C. Chow, D.W. Dockery, J.R. Harkema, J. Kaufman, D.M. Kenski, M. Kleinman, R. McConnell, R.L. Poirot, J.A. Sarnat, E.A. Sheppard, B. Turpin, and S. Vedal, “03-07-19 Draft CASAC Review of EPA's Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft – October 2018),” 19 page letter submitted to Chair, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, March 27, 2019.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/A491FD482BB83BEE852583CA006A2548/\\$File/Written+Comments+from+17+Members+of+the+CASAC+PM+Review+Panel+that+was+Disbanded+on+October+11+2018+rev.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/A491FD482BB83BEE852583CA006A2548/$File/Written+Comments+from+17+Members+of+the+CASAC+PM+Review+Panel+that+was+Disbanded+on+October+11+2018+rev.pdf)

<sup>74</sup> EPA (2019), Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft, EPA-452/P-19-001, U.S. Environmental Protection Agency, Research Triangle Park, NC, September 2019. [https://www.epa.gov/sites/production/files/2019-09/documents/draft\\_policy\\_assessment\\_for\\_pm\\_naaqs\\_09-05-2019.pdf](https://www.epa.gov/sites/production/files/2019-09/documents/draft_policy_assessment_for_pm_naaqs_09-05-2019.pdf)

<sup>75</sup> EPA, “Release of a Draft Document Related to the Review of the National Ambient Air Quality Standards for Particulate Matter,” *Federal Register*, 84(176):47944-47945 (September 11, 2019).  
<https://www.govinfo.gov/content/pkg/FR-2019-09-11/pdf/2019-19627.pdf>

<sup>76</sup> Public Teleconference of the Chartered Clean Air Scientific Advisory Committee (CASAC) on Particulate Matter, 10/22/2019, 12:00 PM - 04:00 PM.  
<https://yosemite.epa.gov/sab/sabproduct.nsf/MeetingCalCASAC/A2DF51609E3DFC9C85258473006CF120?OpenDocument>

<sup>77</sup> Public Meeting of the Chartered Clean Air Scientific Advisory Committee (CASAC) on Particulate Matter, 10/24/2019 to 10/25/2019.  
<https://yosemite.epa.gov/sab/sabproduct.nsf/MeetingCalCASAC/49FAF8892AD2D38285258473006D1F4A?OpenDocument>

<sup>78</sup> Cox, L.A. (2019), “CASAC Review of the EPA's Integrated Science Assessment for Particulate Matter (External Review Draft – October 2018),” EPA-CASAC-19-002, Letter to A. Wheeler, Clean Air Scientific Advisory Committee, U.S. Environmental Protection Agency, Washington, DC, April 2019.  
[https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002+.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002+.pdf)



Administrator stated that he would instead “create a pool of subject matter experts.”<sup>79</sup> In addition, he rejected the CASAC request for the augmented committee to review a revised draft of the ISA. On August 7, 2019, EPA issued a Federal Register notice to request nominations for consultants to support CASAC reviews of particulate matter and ozone.<sup>80</sup>

The use of a “pool of subject matter experts” rather than a review panel to augment the chartered CASAC is unprecedented. Review Panels augment and report through the chartered CASAC, working in parallel and in collaboration with the members of the chartered CASAC. Members of review panels are nominated by the public and the nominations are subject to public comment. The SAB staff office reviews, vets, and appoints members of review panels. Members of review panels participate in meetings with members of the chartered CASAC, and deliberate interactively with members of the chartered CASAC on complex subject matter. The chartered CASAC is ultimately responsible for the content of advice sent to the Administrator, but the formulation of that advice is informed based on deliberations with panelists who provide the breadth, depth, and diversity of needed scientific expertise.

In contrast, there has been no opportunity for public comment on the nominees for the pool of subject matter experts, who were named in an EPA press release on September 13, 2019.<sup>81</sup> The decision regarding appointments of ad hoc consultants to serve as subject matter experts was made by the Administrator, not by the SAB Staff Office. All interactions between CASAC and the subject matter experts will be done solely through the Designated Federal Official (DFO) for CASAC and the CASAC chair, in writing. Subject matter experts will not be allowed to participate in deliberative meetings with CASAC. For example, subject matter experts are not allowed to, unless invited in writing by the chair, respond to all charge questions that might be of interest to the consultant. Subject matter experts will not be allowed to deliberate or interact with the CASAC other than in writing. The appointment of subject matter experts by the Administrator is not correcting the deficiencies in CASAC’s ability to conduct a thorough review that have resulted from disbanding the PM Review Panel.

Therefore, the IPMRP will continue to provide its expert advice, based on the breadth, depth, and diversity of its expertise, and based on interactive deliberation among its members. The IPMRP will submit its review and advice as a public comment to the CASAC and as a public comment to docket EPA-HQ-OAR-2015-0072 for the PM NAAQS review.

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<sup>79</sup> Wheeler, A.R. (2019), Letter to L.A. Cox, EPA Clean Air Scientific Advisory Committee, from Administrator, U.S. Environmental Protection Agency, Washington, DC, July 25, 2019, [https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002\\_Response.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002_Response.pdf)

<sup>80</sup> EPA, “Request for Nominations of Consultants To Support the Clean Air Scientific Advisory Committee (CASAC) for the Particulate Matter and Ozone Reviews,” *Federal Register*, 84(152):38625 (August 7, 2019). <https://www.govinfo.gov/content/pkg/FR-2019-08-07/pdf/2019-16913.pdf>

<sup>81</sup> EPA, “Administrator Wheeler Announces New CASAC Member, Pool of NAAQS Subject Matter Experts,” News Release, U.S. Environmental Protection Agency, Washington, DC, September 13, 2019. <https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>.

## A.2 Membership Criteria for the Independent Particulate Matter Review Panel

The criteria for membership on the IPMRP are that any member of the CASAC PM Review Panel from any time during the CASAC PM Review Panel existence from 2015 until being disbanded on October 10, 2018, and any member of the chartered CASAC from any time during the CASAC PM Review Panel's existence, is eligible, with the exception of any such persons currently serving as members of the chartered CASAC. All of the members of the IPMRP were originally appointed by EPA as Special Government Employees (SGEs) and were subject to disclosure requirements and ethics review. Members of the IPMRP have submitted updates of these disclosures for review by a former EPA Deputy Ethics official in a good faith effort to meet or exceed peer review process and ethics requirements.

On October 31, 2017, EPA Administrator Scott Pruitt signed a memorandum that changed membership criteria for EPA advisory committees.<sup>82</sup> The memorandum states that “no member of an EPA federal advisory committee currently receive EPA grants,” but that this “principle should not apply to state, tribal, or local government agency recipients of EPA grants.” This is inconsistent with the Federal Advisory Committee Act and inappropriate for four reasons. One is the obvious inconsistency of implying that receiving a grant creates a conflict of interest for one but not another class of persons. The second is the longstanding recognition that receipt of a peer-reviewed scientific research grant, for which the Agency does not manage the work nor control the output, is not a conflict of interest. Per the Office of Management and Budget (OMB): “When an agency awards grants through a competitive process that includes peer review, the agency’s potential to influence the scientist’s research is limited. As such, when a scientist is awarded a government research grant through an investigator-initiated, peer-reviewed competition, there generally should be no question as to that scientist’s ability to offer independent scientific advice to the agency on other projects.”<sup>83</sup> A 2013 report by the EPA Office of Inspector General reaffirmed that receipt of an EPA research grant is not a conflict of interest.<sup>84</sup> However, there can be situations in which a member of an advisory committee should recuse themselves from discussions that might pertain to their own work. Thus, third, the CASAC has had recusal policies in place for dealing with this issue and situations in which a member’s work may come up for deliberation. Fourth, the memorandum does not acknowledge that persons with financial or professional ties to regulated industries have, at the very least, the appearance of conflict of interest. With respect to members who currently hold or have recently held EPA STAR research grants, we reject Administrator Pruitt’s restrictions.

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<sup>82</sup> Pruitt, E.S., “Strengthening and Improving Membership on EPA Federal Advisory Committees,” Memorandum, U.S. Environmental Protection Agency, October 31, 2017. <https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>.

<sup>83</sup> Office of Management and Budget, “Final Information Quality Bulletin for Peer Review,” *Federal Register*, 70(10):2664-2677 (January 14, 2005), <https://www.govinfo.gov/content/pkg/FR-2005-01-14/pdf/05-769.pdf>

<sup>84</sup> EPA, “EPA Can Better Document Resolution of Ethics and Partiality Concerns in Managing Clean Air Federal Advisory Committees,” Report No. 13-P-0387, Office of Inspector General, U.S. Environmental Protection Agency, Washington, DC, September 11, 2013. <https://www.epa.gov/sites/production/files/2015-09/documents/20130911-13-p-0387.pdf>

### **A.3 Administrative Procedures for the October 10-11, 2019 and October 18, 2019 Meetings of the Integrated Particulate Matter Review Panel**

The meeting was opened with remarks from a person filling the role of a designated official who described the ethics review procedure and the status of the members with respect to ethics compliance. We had a period for public comments. Following that, the panel deliberated on charge questions or groups of charge questions in a sequential order. A former EPA lawyer and a former EPA air science/policy expert were available as a resource for IPMRP questions.

The goal of the deliberations was to develop “consensus” panel responses to charge questions relating to the review of the draft Policy Assessment and elicit the Panel’s recommendation on the criteria and standards, as well as to consider other statements that the panel may wish to make. “Consensus” does not mean that all members of the panel must share or agree to the same viewpoints. “Consensus” means that all members of the panel agree that the written responses to charge questions and other written statements from the panel accurately reflect the views of the panel. If there are topics for which there are a diversity of viewpoints among members of the panel, the “consensus” response should accurately reflect such diversity of viewpoints. If a consensus response could not be achieved then it is acceptable for one or several panel members to express a dissenting opinion on all or part of the final report. The dissenting opinions, if any, should be captured in writing and included in the final report or the appendices.

The role of the chair is to facilitate the work of the panel. Examples of responsibilities of the chair are to monitor and guide progress on the agenda, enable panelists to have an opportunity to provide input and deliberate, assist the panel in identifying areas of consensus, and assist the panel in focusing on issues that require deliberation. The chair can also address issues regarding the scope of the Panel’s work and recommend approaches to formulating and communicating advice.

The following are the most common procedural considerations for this type of meeting:

- The deliverable from the panel meeting is a written report. The written report includes the following key elements: (1) a summary letter; (2) consensus responses to charge questions; and (3) individual member comments. The letter may additionally include consensus responses on other issues identified by the panel. The purpose of the letter is to concisely communicate the high level key findings and advice of the panel. The purpose of the consensus responses to charge questions is to provide more detail regarding the Panel’s findings and advice.
- All panelists were invited and encouraged to prepare written pre-meeting comments that address charge questions relevant to each panelist’s expertise, as well as any other issues that the panelist may want to address that generally relate to the scope of issues for review of the draft Policy Assessment and of the PM NAAQS.
- The panel is in deliberation if more than half of its members are interacting in formulating a written or oral statement on an issue. Panel deliberations must occur in public. Small groups of panelists, representing up to less than one-half of the panel members, may interact offline to refine draft materials.
- For each charge question or related group of charge questions, discussants and lead discussants were assigned. Discussants prepare draft responses to the charge questions. During deliberations at the public meeting, the lead discussant, with assistance from the

other discussants, formulated draft consensus written responses to the charge questions. Drafts of consensus responses were circulated among discussants for editing and revision, as long as the discussant group had fewer than 50% of panel members.

- During the course of the meeting, the lead discussant for each charge question identified the top “bullet points” that might be included in the Panel’s letter. This enabled the full panel to deliberate on key points for inclusion in the Panel’s letter.
- All key points for the main letter from the panel to the Administrator and the docket, and for the consensus responses to charge questions, were deliberated in a public meeting. No information not deliberated in a public meeting was included in the letter or consensus responses to charge questions.
- Comments from individual members that were reported only as individual comments did not have to be deliberated in the public meeting. However, any individual comments that might inform the formulation of panel consensus on an issue were deliberated with the panel.
- Individual panelists did not engage in deliberations on studies that they authored or co-authored, or research for which they are or were a principal investigator or co-principal investigator, other than to respond to clarifying questions.
- After the October 10-11, 2019 meeting and prior to the follow-up teleconference on October 18, 2019, a draft letter was prepared by the chair and drafts of consensus responses to charge questions was prepared by the charge question discussant groups. The panel deliberated during the follow-up teleconference to revise, as needed, the draft letter and consensus responses to charge questions and approve the final letter and consensus responses to charge questions.
- Individual members of the panel submitted a final version of their individual comments for attachment to the final letter.

## **Biosketches**

### **Independent Particulate Matter Review Panel**

#### **H. Christopher Frey (Panel Chair)**



Dr. H. Christopher Frey is the Glenn E. Futrell Distinguished University Professor of Environmental Engineering in the Department of Civil, Construction, and Environmental Engineering at North Carolina State University. Dr. Frey's research includes quantification of uncertainty in engineering process technologies and emission factors, probabilistic methods for exposure assessment, measurement and modeling of human exposure to air pollution, and measurement and modeling of vehicle emissions. He has been the principal investigator or co-principal investigator for 75 externally sponsored research projects, and has published 133 journal papers, 216 conference papers, 75 technical reports, 8 book chapters, and one book. He teaches courses on air pollution control, environmental exposure and risk assessment, and sustainable infrastructure. Dr. Frey is an adjunct professor in the Division of the Environment and Sustainability at the Hong Kong University of Science and Technology.

Dr. Frey served as a member (2008-2012) and chair (2012-2015) of the U.S. Environmental Protection Agency's Clean Air Scientific Advisory Committee (CASAC), has chaired CASAC Review Panels on Lead, Nitrogen Dioxide, and Ozone, and has served on CASAC Review Panels for all criteria pollutants including Lead, Nitrogen Dioxide, Ozone, Carbon Monoxide, Particulate Matter, and Sulfur Oxides. He served on the U.S. EPA Science Advisory Board from 2012 to 2018. For the National Greenhouse Gas Inventory Program of the Intergovernmental Panel on Climate Change (IPCC), he served as an expert and Lead Author for the chapter on uncertainties for the 2006 IPCC Guidelines on National Greenhouse Gas Emission Inventories, and in 2016 was an invited expert regarding updates to the 2006 Guidelines. Additionally, he was a technical contributor to the U.S. Department of Transportation's 2010 Report to Congress regarding Transportation's Role in Reducing U.S. Greenhouse Gas Emissions. He served on a World Health Organization working group that developed guidance on uncertainty in exposure assessment (2006). He served on two National Research Council (NRC) committees and was a member (2009-2012) of the NRC Board of Environmental Studies and Toxicology. He currently serves on the MOVES Model Review Work Group of the Mobile Sources Technical Review Subcommittee of the EPA Clean Air Act Advisory Committee (CAAAC).

In the last two years, Dr. Frey has been the principal investigator of research grants and contracts at North Carolina State University sponsored by the North Carolina Department of Transportation, the U.S. Environmental Protection Agency via the Health Effects Institute and Eastern Research Group, and the Urban Air Initiative. Dr. Frey's research work at HKUST is funded by the HSBC 150th Anniversary Charity Programme. Dr. Frey has also conducted work for the Hong Kong Environmental Protection Department. Dr. Frey's current affiliations include serving as a member of the Transportation and Air Quality (ADC20) Committee of the Transportation Research Board, and as a member of the Publications Committee and the Critical Review Committee of the Air & Waste Management Association (AWMA).

Dr. Frey is a Fellow of the Air & Waste Management Association (AWMA) and of the Society for Risk Analysis (SRA), served on the AWMA Board of Directors (2015-2018), and was President of SRA in 2006. He received the Chauncey Starr Award from SRA in 1999, the Lyman A. Ripperton Award from AWMA in 2012, and the Frank A. Chambers Award from AWMA in 2019. He has a B.S. in mechanical engineering from the University of Virginia, a master of engineering in mechanical engineering from Carnegie Mellon University, and Ph.D. in engineering and public policy from Carnegie Mellon.

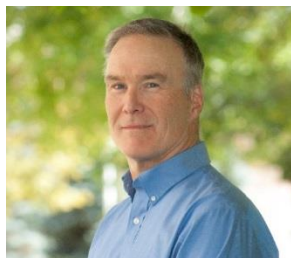
## Peter Adams



Dr. Peter Adams is a Professor in the Civil and Environmental Engineering Department and the Engineering and Public Policy Department at Carnegie Mellon University. Dr. Adams' research largely focuses on development of chemical transport models and their application to decision-making, especially related to PM<sub>2.5</sub>. Dr. Adams also has extensive expertise in the simulation of aerosol microphysical processes, ultrafine particles and the formation of cloud condensation nuclei in global climate models. Areas of research have also included the effects of climate change on air quality, short-lived climate forcers, atmospheric ammonia and particulate matter formation from livestock operations, and the simulation organic particulate matter. Dr. Adams was selected for a Fulbright grant to collaborate with researchers at the Institute of Atmospheric Sciences and Climate in Bologna, has been a Visiting

Senior Research Scientist at the National Aeronautics and Space Administration's Goddard Space Flight Center, and received the Sheldon K. Friedlander Award for outstanding doctoral thesis from the American Association for Aerosol Research. He has previously served on the Commonwealth of Pennsylvania's Air Quality Technical Advisory Committee and the Allegheny County Health Department's Air Toxics New Guidelines Proposal Committee as well as service to the American Association for Aerosol Research. His research is supported primarily by the Environmental Protection Agency, the National Science Foundation, the National Aeronautics and Space Administration, the Department of Energy, and the Department of Defense. Dr. Adams received his B.S. degree in Chemical Engineering, summa cum laude, from Cornell University. He was awarded a Hertz Foundation Applied Science Fellowship for graduate study and received M.S. and Ph.D. degrees in Chemical Engineering from the California Institute of Technology. He also holds an associated faculty position in the Chemical Engineering department at Carnegie Mellon.

## John L. Adgate



John L. Adgate, Ph.D is Professor and Chair of the Department of Environmental and Occupational Health at the Colorado School of Public Health, University of Colorado. His exposure science research focuses on improving public health and epidemiological studies by documenting the magnitude and variability of human exposure to air pollutants, pesticides, metals, and allergens. His research projects have included evaluation of methods to reduce lead and allergen exposure exploration of longitudinal exposure to indoor and outdoor air pollutants, and, most recently, assessing the environmental and human health impacts of unconventional oil and gas development and the impact of climate change on indoor environments.

Dr. Adgate has served on multiple U.S.EPA Science Advisory Panels exploring technical and policy issues related to residential exposure to pesticides, metals, and implementation of the Food Quality Protection Act of 1996. He was also a member of US Institute of Medicine's Committee on Research Ethics in Housing Related Health Hazard Research in Children and the National Research Council's 2011 Committee on Indoor Air and Climate Change. Most recently he has advised the States of New York, Maryland, and Michigan on the potential public health impacts of high volume hydraulic fracturing, and leading studies exploring the public health impacts of hydraulic fracturing funded by the National Science Foundation, the National Institutes of Environmental Health Sciences, and the Department of Energy. His current research is focused on characterizing the exposures and impacts of the wide range of chemical and non-chemical stressors found in and around oil and gas development sites and indoor air and climate change funded by USEPA.

Dr. Adgate received a B.Sc. in biology from Calvin College, an M.S.P.H. from the University of North Carolina at Chapel Hill, and a Ph.D. in Environmental Health Sciences jointly awarded by Rutgers University and the University of Medicine and Dentistry of New Jersey. He has held faculty positions at the University of Minnesota and has current appointments at the University of Colorado Denver and Colorado State University. In 2006-7 he was a Fulbright Visiting Scholar at the Pontificia Universidad Católica de Chile in Santiago, where he taught risk analysis and worked on air quality research. He has served as an elected Councilor of the International Society of Exposure Science (ISES), was a recipient of its Joan M. Daisey Outstanding Young Scientist Award, and co-chaired ISES's 2009 meeting. He has taught graduate level courses on Risk Analysis and Communication, Advanced Methods in Exposure Science, Introduction to Environmental and Occupational Health, and Occupational Health and Safety.

## George Allen



Mr. George Allen is the Chief Scientist at NESCAUM (Northeast States for Coordinated Air Use Management), an interagency association of the eight Northeastern States. He holds a B.S. in Electrical Engineering from Tufts University (1974). At NESCAUM, Mr. Allen is responsible for monitoring and exposure assessment activities across a range of wide range of air topics, including regional haze, air toxics, on and off-road diesel, wood smoke, and continuous aerosol measurement technologies. He served on the chartered EPA CASAC from 2010 to 2016, has been a member of several CASAC review panels since 2004, and is the author or co-author of more than 30 peer-reviewed journal papers on development and evaluation of measurement methods, exposure assessment, and air pollution health effects. Before joining NESCAUM in 2002, Mr. Allen was on the professional staff at the Harvard School of Public Health (HSPH) in Boston for more than 20 years, working on a wide range of U.S. Environmental Protection Agency (EPA) and National Institutes of Health- funded air pollution studies. While at HSPH, he developed several new techniques for real-time aerosol measurements. Currently, Mr. Allen is serving as the lead for the NESCAUM Monitoring and Assessment Committee. He also represents states interests to EPA in the National Association of Clean Air Agencies (NACAA) Monitoring Steering Committee, and is a member of the EPA AIRNow Steering Committee. Mr. Allen's current and pending research support pertains to scientific, technical, analytical, and policy support for NESCAUM states' air quality and climate programs, with a focus on air pollution exposure assessment and measurement methods development. These funders include New York State Energy Research and Development Authority (NYSERDA) (characterization of biomass air pollution), Massachusetts Department of Environmental Protection (spatial and temporal trends of black carbon), NESCAUM member states and Federal Land Managers (CAMNET visibility network), NESCAUM member states and US EPA (support member states' air quality programs).



## John Balmes



Dr. John Balmes is Professor of Medicine at the University of California, San Francisco (UCSF) and Professor of Environmental Health Sciences in the School of Public Health at UC Berkeley. He is a member of the faculty of the UCSF Division of Occupational and Environmental Medicine and the UCSF Division of Pulmonary and Critical Care Medicine at Zuckerberg San Francisco General Hospital. He is the Director of the UC Berkeley-UCSF Joint Medical Program and the Northern California Center for Occupational and Environmental Health. Dr. Balmes received a BA in Psychology from the University of Illinois in Urbana and his MD from Mount Sinai School of Medicine. He completed a residency in Internal Medicine at Mount Sinai and a post-doctoral fellowship in Pulmonary Medicine at Yale. For over 9 years, Dr. Balmes has been studying the effects of exposures to occupational and environmental agents on respiratory and cardiovascular health. In the UCSF Human Exposure Laboratory, he has conducted controlled human exposure studies with sampling of respiratory tract lining fluid to characterize acute exposure-response relationships for oxidant pollutant-induced airway inflammation in subjects with and without asthma, and more recently, investigation of acute cardiovascular responses. Recently, his group has been funded by the Health Effects Institute to participate in a multi-center study designed to determine whether experimental exposure to ozone induces cardiovascular toxicity (decreased heart rate variability, epithelial dysfunction, and a pro-thrombotic state) and whether any of these effects are associated with airway inflammation, systemic oxidative stress, and systemic inflammation. At UC Berkeley, Dr. Balmes has collaborated on a number of studies of the chronic effects of air pollutants on respiratory health. He has investigated the effects of exposures to air pollutants on respiratory symptoms, growth of lung function, and immune dysfunction in children with and without asthma in Fresno, CA. He contributed to the first randomized controlled trial (RCT) of a chimney stove to prevent pneumonia among infants in Guatemala and led follow-up studies on the effects of exposure to biomass smoke on lung function in both the children and their mothers. He has also participated in a second RCT of a cleaner-burning biomass stove to prevent childhood pneumonia in Malawi. He contributed to an investigation of whether chronic environmental exposure to hydrogen sulfide is associated with adverse effects on respiratory health in Rotarura, New Zealand. Dr. Balmes is one of the multiple of the Children's Health and Air Pollution Study (CHAPS) grant that is studying the adverse effects of air pollution on children living in the San Joaquin Valley. The Center project that he leads is investigating the potential effects of exposure to polycyclic aromatic hydrocarbons on risk of obesity and glucose dysregulation. Dr. Balmes has received multiple awards for his research from various organizations, including the American Thoracic Society (ATS), the American College of Occupational and Environmental Medicine (ACOEM), the Western Occupational and Environmental Medicine Association, the American Lung Association of California, and the South Coast Air Quality Management District. He is a member of the ATS, the American College of Chest Physicians, and the ACOEM. He has served on several US EPA advisory committees, including CASAC panels on ozone, NO<sub>2</sub>, SO<sub>2</sub> and PM<sub>2.5</sub>, as well as on various National Academy of Sciences/Institute of Medicine committees. In addition to his experience in air pollution health effects research, Dr. Balmes also has policy experience in the regulation of air quality and climate change mitigation in his role as Physician Member of the California Air Resources Board (since January 2008).

## Kevin Boyle



Dr. Kevin Boyle is an environmental economist who specializes in the valuation of resources that are not traded in markets. Dr. Boyle is Professor of Agricultural and Applied Economics and Willis Blackwood Director, Program in Real Estate at Virginia Tech. Prior to this position he was Head of the Department of Agricultural and Applied Economics at Virginia Tech. He was formerly Distinguished Maine Professor and Libra Professor of Environmental Economics at the University of Maine. Dr. Boyle holds a Ph.D. from the University of Wisconsin. Dr. Boyle has held editorial positions with the *Journal of Environmental Economics and*

*Management and Marine Resource Economics*. Dr. Boyle was recognized by the Carnegie Foundation for the Advancement of Teaching as “U.S. Professor of the Year, Maine” in 2004. He is a member of Virginia Tech’s Academy of Faculty Leadership, a Fellow of the Agricultural and Applied Economics Association and a Fellow of the Association of Environmental and Resource Economists. Dr. Boyle’s research investigates the validity of nonmarket valuation methods, including stated preferences (contingent valuation and attribute-based choices), revealed preferences (travel-cost models, averting behavior and hedonic, property-value models), and benefit transfers. Dr. Boyle served as the co-PI for a National Park Service grant to develop procedures to estimate the value of changes in the annual distribution of visibility in Class 1 areas in accordance with U.S. EPA’s Regional Haze Program. He is also participating in research, through Industrial Economics, to model the effects on property values from air-pollution induced changes in visibility for the U.S. EPA and the South Coast Air Quality Management District.

## Judith Chow



Dr. Judith Chow holds the Nazir and Mary Ansari Chair in Science and Entrepreneurialism and is a Research Professor in the Division of Atmospheric Sciences of the Desert Research Institute (DRI) of the Nevada System of Higher Education in Reno, Nevada. She was the founder and has been the director of DRI's Environmental Analysis Facility (EAF) since 1985. EAF specializes in method development and chemical analysis of airborne particles for multielements, ions, and carbon. Dr. Chow earned a B.S. degree in Biology from Fu-Jen Catholic University (Taiwan), and a S.M. degree in Environmental Health Science and a Sc.D. degree in Environmental Science and Physiology from Harvard University. For more than 40 years, she has conducted studies and performed data

analysis to improve understanding of air quality effects on human health, visibility, historical treasures, ecosystems, and climate. Dr. Chow has been principal investigator or a major collaborator in more than 50 air quality studies across the United States and in other countries. Her current research includes tracking changes in air quality with control measures at the ports of Los Angeles and Long Beach, investigating the chemical nature and composition of atmospheric brown carbon aerosol, and evaluating nitrogen partitioning and evolution of particulate organic nitrogen in fresh and aged peat fire emissions. She was a member of the National Research Council's (NRC) committees on Research Priorities for Airborne Particulate Matter (1998–2003) and Energy and Air Pollution Futures in the U.S. and China (2004–2008); she also served on the NRC Board on Environmental Studies and Toxicology (2002–2005). Dr. Chow prepared and revised sections of the U.S. EPA's PM Criteria Document pertaining to chemical analysis and source emissions and contributed to EPA guidance documents on network design, continuous particulate monitoring, and particulate matter chemical speciation. She served as a chartered member (2015–2018) of EPA's Clean Air Scientific Advisory Committee (CASAC) and a member (2004–2018) of CASAC's Air Monitoring and Methods Subcommittee (AMMS, formerly the Ambient Air Monitoring and Methods Subcommittee). Dr. Chow was chair of the Publications Committee and Editorial Review Board for the Journal of the Air & Waste Management Association, serves as a Thematic Editor for Particuology, and is on Editorial Boards for Air Quality, Atmosphere, & Health; Aerosol and Air Quality Research; Atmospheric Pollution Research; and Aerosol Science and Engineering. She is the principal author or co-author of more than 350 peer-reviewed articles and more than 90 peer-reviewed book chapters. She been recognized by ISI HighlyCited.com in ecology and environment with more than 20,000 citations of her work with a h-index of 78. Dr. Chow has received the Air & Waste Management Association (A&WMA)'s 2016 Arthur C. Stern Award for Distinguished Paper; California Air Resources Board's 2011 Haagen-Smit Clean Air Award for her contributions to air quality science and technology; A&WMA's 2002 Frank A. Chambers Excellence in Air Pollution Control Award; and the Nevada System of Higher Education's 2001 Regents' Researcher Award.

## **Douglas W. Dockery**



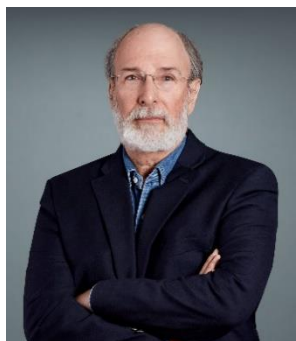
Dr. Douglas W. Dockery is the John L. Loeb and Frances Lehman Research Professor of Environmental Epidemiology in the Departments of Environmental Health and of Epidemiology at the Harvard TH Chan School of Public Health. He was Chair of the Department of Environmental Health (2005-2016) and Director of the Harvard-National Institute of Environmental Health Sciences (NIEHS) Center for Environmental Health Sciences (2008-2019). He received a B.S. in physics from the University of Maryland, an M.S. in meteorology from the Massachusetts Institute of Technology, and a ScD in environmental health from the Harvard School of Public Health. Dr. Dockery has been studying air pollution exposures and their health effects for more than four decades. He served as Principal Investigator of the Harvard Six Cities Study of the Respiratory Health Effects of Respirable Particles and Sulfur Oxides. His recent work includes assessment of the health benefits of air pollution controls. Dr. Dockery has published over two hundred peer-reviewed articles. His 1993 New England Journal of Medicine paper on air pollution and mortality in the Harvard Six Cities study is the single most cited air pollution paper. In 1998, the International Society of Environmental Epidemiology honored with the first John Goldsmith Award from for Outstanding Contributions to the field.

## Henry (Dirk) Felton



Mr. Henry (Dirk) Felton is currently employed by the New York State Department of Environmental Conservation (NYSDEC) as a Research Scientist III. He has a Bachelor of Arts undergraduate degree in Physics from Kenyon College, Gambier Ohio (1987), and a Master of Science in Environmental Engineering from Stevens Institute of Technology in Hoboken, New Jersey (1993). He is also a Civil Engineer licensed in the State of New York. Mr. Felton's professional work has been entirely focused on ambient air monitoring. His first independent work involved setting up a monitoring network for criteria, toxic and tracer compounds around the Freshkills Landfill on Staten Island. Since then he has worked to optimize monitoring technology to operate a rural upwind PAMS site for NARSTO-NE, conducted several experiments to evaluate new automated mass measurement technologies, initiated speciated Mercury and ultrafine monitoring programs and has designed the PM-2.5 FRM and PM speciation monitoring program in New York. Mr. Felton also was the lead for his Agency's participation in the New York PMTACS EPA SuperSite program, participated on the Board of Science Counselors review of EPA ORD's Clean Air Research program and was a two-term member of the CASAC Ambient Air Monitoring and Methods subcommittee (AAMMs). Mr. Felton currently participates on the NESCAUM Monitoring Assessment Committee (MAC), the NACAA Monitoring Steering Committee (MSC) and recently was elected to his local school board.

## Terry Gordon



Dr. Terry Gordon holds the rank of Professor of Environmental Medicine at the New York University (NYU) School of Medicine. He holds a B.S. in Physiology (1974) and an M.S. in Toxicology (1976) from the University of Michigan, and a Ph.D. in Toxicology from Massachusetts Institute of Technology (1981), and was appointed to the faculty of the Department of Environmental Medicine in 1989. He has served as an ad hoc member of grant review panels and/or site visit teams for the National Institute of Environmental Health Services (NIEHS), National Institute of Allergy and Infectious Diseases (NIAID), National Coalition for Cancer Research (NCCR), U.S. Department of Defense (DOD), Bureau of Mines, Health Canada, and the U.S. Environmental Protection Agency (EPA).

Dr. Gordon currently serves as Chair of the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value committee, a volunteer organization that publishes occupational exposure levels that are used as workplace safety guidelines throughout the world. Dr. Gordon's broad research interest is in inhalation toxicology. The major focus of his research lab is the identification and understanding of the role of genetic host factors in the pathogenesis of the adverse pulmonary effects produced by inhaled environmental and occupational agents. Because inter-individual responses to inhaled particles and gases vary so greatly in both human subjects and test animals, Dr. Gordon has hypothesized that genetic susceptibility factors play a major role in environmental and occupational lung disease. In collaboration with a number of investigators in the department, his laboratory uses classic murine genetics models, computational genomics, and DNA microarrays to identify genes involved in the acute response as well as in the development of tolerance to repeated exposure to inhaled toxicants. Dr. Gordon also plays a major role in the particulate matter (PM) research program at NYU, and was among the first researchers to use concentrator technology to study the adverse cardiopulmonary effects of ambient PM. He also led a large collaborative effort amongst EPA's five original PM research centers to evaluate the in vitro and in vivo toxicity of size-segregated PM collected in the U.S. and Europe. Dr. Gordon's research has been supported by grants from both government agencies and private companies, with core grant research support primarily being from the federal government (U.S. Environmental Protection Agency, Centers for Disease Control, National Institute of Environmental Health Sciences), with additional grant support from state and local governments, and industry. Dr. Gordon is an active member of the Society of Toxicology (SOT), and has served on the Program Committee (2002-2005), the Placement Service (1998-2001), Membership Committee (2009-2012), and as President of its Inhalation Specialty Section during 2002-2003. He has served as a consultant/author to the EPA on issues of pulmonary toxicology related to the development of various documents, and he served on EPA's Clean Air Scientific Advisory Committee (CASAC) Oxides of Nitrogen (NO<sub>x</sub>) and Sulfur Oxides (SO<sub>x</sub>) Primary National Ambient Air Quality Standards (NAAQS) Review Panels.

## Jack R. Harkema



Dr. Jack R. Harkema, DVM, Ph.D., DACVP, is a University Distinguished Professor at Michigan State University in East Lansing, MI. Dr. Harkema received a DVM (veterinary medicine) from Michigan State University (MSU) and a Ph.D. (comparative pathology) from the University of California, Davis (UCD). After completing a National Institutes of Health (NIH)-sponsored research/residency training program in comparative pathology and toxicology at the UCD, Dr. Harkema joined the scientific staff of the Lovelace Inhalation Toxicology Research Institute in Albuquerque, NM in 1985 as an experimental and toxicologic pathologist. He later became the institute's project manager for pathogenesis research. In 1994, Dr. Harkema joined the faculty of the Department of Pathobiology and Diagnostic Investigation in the College of Veterinary Medicine at MSU. His primary research is designed to understand the pathobiology and toxicology underlying the health effects of outdoor and indoor air pollutants. In 2011, he became the director of the Great Lakes Air Center for Integrated Environmental Research, one of four U.S. Environmental Protection Agency (EPA)-funded Clean Air Research Centers in the nation. Dr. Harkema has authored or co-authored over 220 peer-reviewed scientific publications and has served on several scientific advisory committees, including those for the National Institute of Environmental Health Sciences (NIEHS), the National Toxicology Program, the EPA, and the National Academy of Sciences. Besides training graduate students, residents, and postdoctoral fellows in biomedical research, Dr. Harkema also moderates didactic courses in advanced general pathology, integrative toxicology, and pulmonary pathobiology. Dr. Harkema is a diplomate of the American College of Veterinary Pathologists (by examination) and a member of the Society of Toxicologic Pathologists, the Society of Toxicology, and the American Thoracic Society. He currently receives research funding through grants or contracts from a variety of sources including the EPA to explore and elucidate the health effects of multipollutant atmospheres in the Great Lakes region, the American Chemistry Council to study the nasal pathology and toxicology of inhaled olefin compounds, and the NIEHS/National Institutes of Health to identify the molecular mechanisms underlying toxicity of dioxin-like compounds.



## Joel Kaufman



Dr. Kaufman is a physician-epidemiologist, board-certified in internal medicine and occupational medicine. A graduate of the University of Michigan (B.A., M.D.) and the University of Washington (MPH), he has been a full-time faculty member at the University of Washington (UW) since 1997. He is currently Professor in the departments of Environmental & Occupational Health Sciences, Medicine, and Epidemiology. His current research activities are primarily focused on environmental factors in cardiovascular and respiratory disease. He is the principal investigator of a major epidemiological prospective cohort study of air pollution and cardiovascular disease (The Multi-Ethnic Study of Atherosclerosis and Air Pollution, or “MESA Air”). He directs the UW Northlake Controlled Exposure Facility, a facility customized for experimental inhalation toxicology studies on health effects of combustion-derived pollutants. He has also served as principal investigator of a National Institutes of Health-funded Specialized Center for Research at the University of Washington on Cardiovascular Disease and Traffic-Related Air Pollution. From 2016 to 2018, he was the interim dean of the University of Washington School of Public Health. He is a Fellow of the American College of Physicians, the American College of Occupational and Environmental Medicine, and the American Heart Association. Dr. Kaufman's research integrates the disciplines of epidemiology, exposure sciences, toxicology, and clinical medicine.



## Patrick Kinney



Dr. Kinney, at the Boston University School of Public Health, has a broad background in environmental health sciences, with specific training and expertise in exposure assessment, respiratory health and climate change. He completed his doctoral studies in Environmental Science and Physiology at the Harvard School of Public Health in 1986. As a junior faculty member at New York University, he developed and led epidemiologic research on lung function and inflammatory biomarker changes in relation to chronic exposures to ozone and other air pollutants. Moving to Columbia in 1994, he expanded his research to include community-based studies of traffic pollutant exposures and health outcomes in underprivileged neighborhoods in New York City, leading and contributing to several large-scale studies over the following 20 years. He has contributed to the periodic reviews of the National Ambient Air Quality Standards for ozone and particulate matter, and served on the EPA Clean Air Scientific Advisory Committee for reviews of the Nitrogen Dioxide and Sulfur Dioxide standards. He developed and directed the Climate and Health Program at Columbia. He also has directed research on indoor and outdoor air quality and health in Africa, including a randomized stove trial in Ghana funded by NIEHS. Other recent funding sources include the National Oceanic and Atmospheric Administration, and the National Aeronautics and Space Administration. In 2017, he was appointed the inaugural Beverly A Brown Professor of Urban Health at Boston University. His work at BU focuses on assessing health impacts of air pollution and climate extremes in cities, and the health and climate co-benefits that can be achieved through carefully-planned mitigation and adaptation strategies.

## Michael T. Kleinman



Dr. Michael T. Kleinman is an Adjunct Professor of Toxicology in the Department of Medicine's Occupational and Environmental Medicine Division at the University of California, Irvine (UCI), with a joint appointment in the Program in Public Health. He was previously employed by the U.S. Atomic Energy Commission (AEC) as an environmental scientist and he directed the Aerosol Exposure and Analytical Laboratory at Rancho Los Amigos Hospital in Downey, CA. He has more than 40 years of experience researching the health effects of environmental contaminants. He holds a M.S. in Chemistry (Biochemistry) from the Polytechnic Institute of Brooklyn and a Ph.D. in Environmental Health Sciences from New York University. He is the Co-Director of the Air Pollution Health Effects Laboratory at UCI. He has published more than 115 peer-reviewed journal articles on effects of

environmental contaminants on cardiopulmonary and immunological systems and on global and regional distribution of environmental contaminants including heavy metals and radioactive contaminants from nuclear weapons testing. He has directed more than 50 controlled exposure studies of human volunteers and laboratory animals to ozone and other photochemical oxidants, carbon monoxide, ambient particulate matter (PM) and laboratory-generated aerosols containing chemically or biologically reactive metals such as lead, cadmium, iron and manganese. He has served on two National Academy committees to examine issues in protecting deployed U.S. Forces from the effects of chemical and biological weapons. Dr. Kleinman's current research focuses on neurological and cardiopulmonary effects of inhaled particles, including nanomaterials and ultrafine, fine and coarse ambient particles in humans and laboratory animals. His recent health effects studies have the role of inhaled combustion-generated particles on the promotion of airway allergies and acceleration of development of cardiovascular disease and how these effects are mediated by organic and elemental carbon components of PM. Dr. Kleinman's current research grants and contracts include a grant to examine the effects of inhaled particles on brain stem cells related to tumor development from the California Brain and Lung Tumor Foundation, a contract from the California Environmental Protection Agency to study the role of semi-volatile components of fine and ultrafine PM on cardiac function and atherosclerosis, and a contract to examine the effects of long term inhalation exposure to concentrated fine particles on brain inflammation. Dr. Kleinman has previously served on the U.S. EPA Clean Air Scientific Advisory Committee (CASAC) Ozone, PM and NO<sub>2</sub> panels and was appointed to Chair the Scientific Review Panel for Toxic Substances for the state of California. Dr. Kleinman's current research focuses on neurological and cardiopulmonary effects of inhaled particles, including nanomaterials and ultrafine, fine and coarse ambient particles in humans and laboratory animals. His recent health effects studies have the role of inhaled combustion-generated particles on the promotion of airway allergies and acceleration of development of cardiovascular disease and how these effects are mediated by organic and elemental carbon components of PM. Dr. Kleinman is a co-Investigator on grants from NIH and NSF as well as contracts from the California Brain and Lung Tumor Foundation and from the California Environmental Protection Agency to study the role of semi-volatile components of fine and ultrafine PM on cardiac function, atherosclerosis, and effects of subchronic and chronic inhalation exposures to concentrated fine particles on brain inflammation.

## Rob McConnell



Rob McConnell is a physician and epidemiologist, a Professor of Preventive Medicine, and the director of Southern California Children's Environmental Health Center at the University of Southern California, where he has studied the effects of air pollution on children's health. He has been the principal investigator or project director on several large National Institutes of Health-funded R01s or Centers supporting the Southern California Children's Health Study, a large, ongoing longitudinal cohort study that has made important contributions to understanding the role of air pollution in childhood origins of respiratory and cardiometabolic health and obesity. His research interests include, in addition, novel methods for assessment of environmental exposure and understanding susceptibility to the effects of air pollution related to psychosocial stress and social factors, exercise, co-exposures associated with housing conditions, as well as genetics. Other interests include the development of methods for estimating the burden of disease associated with near-roadway air pollution and for assessing exposure in environmental epidemiology. He directs the Career Development Program of the NIEHS-supported Southern California Environmental Health Sciences Center. Before coming to USC, Dr. McConnell directed a World Health Organization (WHO) regional center for environmental health in Latin America and the Caribbean, where he was a member of advisory committees to the Ministries of Health in the Americas and of the senior management team to the WHO Regional Director for the Americas. He is a fellow of the American Association for the Advancement of Science.

## Richard Poirot



Mr. Richard Poirot is an independent consultant who recently retired as the Air Quality Planning Chief with the Vermont Department of Environmental Conservation, where he's worked since 1978. During his 37 years in VT state government, Rich's responsibilities included developing and implementing State Implementation Plans to ensure attainment and maintenance of federal and state air quality standards for ozone, particulate matter, and regional haze. He developed interests and expertise in drawing inference on the nature of pollution sources from analysis of ambient air quality and meteorological measurement data. Rich has been an active participant on the Acid Deposition Committee and the Ambient Monitoring and Assessment Committee for the Northeast States for Coordinated Air Use Management (NESAUM); the U.S. Environmental Protection Agency (EPA) Acid Rain Advisory Committee; the Data Analysis Workgroup for the Ozone Transport Assessment Group (OTAG); the Science and Technical Support Workgroup for the Federal Advisory Committee on Ozone, Particulate Matter and Regional Haze (OPRHA); the Monitoring and Data Analysis Workgroup for the Mid Atlantic/Northeast Visibility Union (MANE-VU), the Steering Committees for the Interagency Monitoring of Protected Visual Environments (IMPROVE); the Subcommittee on Scientific Cooperation for the U.S./Canada Air Quality Agreement; the EPA Clean Air Scientific Advisory Committee (CASAC), the CASAC Ambient Air Monitoring and Methods Subcommittee, the CASAC Panels for Particulate Matter, Ozone, Lead, and Secondary SO<sub>x</sub> and NO<sub>x</sub> National Ambient Air Quality Standards Review; the NARSTO External Review Panel; the U.S. EPA Advisory Council on Clean Air Compliance Analysis and the Council Subcommittee on Ambient Air Modeling; and the Board on Environmental Studies and Toxicology (BEST) for the National Research Council. He is not currently a recipient of research grants from the Environmental Protection Agency, other federal agencies, or the private sector.

## Jeremy A. Sarnat



Dr. Jeremy Sarnat is currently an Associate Professor of Environmental Health at the Rollins School of Public Health of Emory University. He holds an Sc.D. in Environmental Health from the Harvard School of Public Health. Dr. Sarnat's research focuses primarily on characterizing exposures to urban air pollution in various populations, in particular panels of sensitive cohorts such as children, older adults and individuals with cardiorespiratory disease. Much of his work examines how exposure science informs environmental epidemiology; the impact of exposure misclassification and confounding on air pollution epidemiologic findings; and the application of these findings towards the development of novel spatiotemporal models of personal air pollution exposures. Currently, Dr. Sarnat is the Principal Investigator of several panel studies investigating exposures to primary traffic pollution in cohorts of healthy and asthmatic subjects and corresponding acute cardiorespiratory response. He is also the co-Director of the Southeastern Center for Air Pollution and Epidemiology (SCAPE), based jointly at Emory University and the Georgia Institute of Technology.

### Elizabeth A. (Lianne) Sheppard



Dr. Elizabeth A. (Lianne) Sheppard, PhD is Professor in the Departments of Environmental and Occupational Health Sciences and Biostatistics at the University of Washington School of Public Health. She holds a B.A. in psychology and a Sc.M. in biostatistics from Johns Hopkins University, and a Ph.D. in biostatistics from University of Washington. Her research interests focus on modeling and understanding the health effects of environmental and occupational exposures with particular emphasis on statistical methods for environmental and occupational epidemiology. She is principal investigator of the ACT-AP study, which aims to determine whether air pollutants are associated with cognitive decline and dementia incidence, as well as markers of Alzheimer's disease, in the Puget Sound, an area with relatively low levels of air pollution. She actively collaborates on a variety of research projects in the environmental and occupational health sciences and has been the lead statistician for the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) study, a 10-year study funded by EPA to determine the effect of long-term air pollution exposure on subclinical progression of cardiovascular disease. Dr. Sheppard directs a NIEHS-funded program for quantitative training in the environmental health sciences. She is a fellow of the American Statistical Association, recently served on the Health Effects Institute's Review Committee, and has served on the Clean Air Scientific Advisory Committee and its special panels, as well as other EPA review panels.

## Barbara J. Turpin



Dr. Barbara J. Turpin is a Professor in Environmental Sciences and Engineering, University of North Carolina at Chapel Hill. She specializes in atmospheric organic chemistry that transforms gaseous emissions into particulate matter (PM) affecting health, visibility and climate. She was the first to publish time-resolved measurements that provided atmospheric evidence for the formation of secondary organic aerosol (Turpin et al., 1991). Her group was the first to recognize that secondary organic aerosol forms through gas followed by aqueous chemistry in clouds, fogs and wet aerosols (Blando et al., AE 2000).

Her 2000 organic aerosol review paper has been called a “primer” on organic aerosol measurement and was awarded Atmospheric Environment’s Haagen Smit Prize. Professor Turpin’s research continues to provide novel insights into the sources, properties and behavior of atmospheric organic aerosol. She also makes substantive contributions to the understanding of PM exposure and has conducted collaborative PM and health research.

Dr. Turpin obtained a BS at the California Institute of Technology, a PhD from OGI - Oregon Health Sciences University and did postdoctoral research at the University of Minnesota Particle Technology Laboratory. She joined the faculty of Rutgers University in 1994 and moved to the University of North Carolina at Chapel Hill in 2015. Professor Turpin is a Fellow of the American Association for the Advancement of Science (AAAS), the American Geophysical Union (AGU), and the American Association for Aerosol Research (AAAR). She is a recipient of AAAR’s Sinclair Award for “sustained excellence in aerosol research and technology by an established scientist still active in his/her career.” She is an Associate Editor of the prestigious journal, *Environmental Science and Technology*. Professor Turpin is a Past President of the American Association for Aerosol Research and just completed her term as a member of the International Commission for Atmospheric Chemistry and Global Pollution (iCACGP). She has published over 100 peer-reviewed papers (avg citations/paper = 71; h-index = 44) and received over \$10M of research funding from sources such as the Environmental Protection Agency (STAR), National Science Foundation, National Oceanic and Atmospheric Administration, Sloan Foundation, Health Effects Institute, New Jersey Department of Environmental Protection and Electric Power Research Institute.

## Ronald Wyzga



Dr. Ronald Wyzga is Technical Executive in the Air Quality Health Effects program area of the Environment Sector at the Electric Power Research Institute. He received an AB degree in mathematics from Harvard College and an M.S. degree in statistics from Florida State University. He also received a Sc.D. degree in biostatistics from Harvard University. Dr. Wyzga has authored an extensive list of publications on his research. His current research activities focus on understanding the relationship between health effects and air pollution, an area in which he has worked for over 30 years.

Dr. Wyzga is particularly interested in the design, conduct, and interpretation of epidemiological studies that examine this relationship. He is also interested in health risk assessment methods. Dr. Wyzga has studied the relationship between health effects and air pollution since he joined EPRI in 1975. In addition, he has worked on methods to attach economic values to air pollution damage and effects. Dr. Wyzga has served on, and has chaired, several committees for the EPA Science Advisory Board and National Academy of Sciences. He has also served on advisory oversight committees for several research programs on the health effects of air pollution. In 1990, Dr. Wyzga was elected a Fellow of the American Statistical Association by his peers. Prior to joining EPRI, he worked at the Organization for Economic Cooperation and Development (OECD) in Paris, where he co-authored a book on economic evaluation of environmental damage.



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Harold P. Wimmer

November 12, 2019

Administrator Andrew Wheeler  
U.S. Environmental Protection Agency  
1200 Pennsylvania Avenue NW  
Washington D.C. 20460

Sent via Regulations.gov.

Re: Comments on Docket ID No. EPA-HQ-OAR-2015-0072

Dear Administrator Wheeler:

The American Lung Association appreciates the opportunity to provide comments on the Policy Assessment for Review of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft, September 2019 (Draft PA).

**[EPA's revised review process undermines the protection of public health](#)**

The Lung Association continues to express our objections to the changes to the process that EPA has adopted in this review. EPA's changes restrict the full discussion and review of the information, undermining the core purpose of this process: to set standards that "protect health with an adequate margin of safety." While the Lung Association has long supported and, indeed, taken legal action to ensure the completion of the reviews in a timely manner, the Lung Association opposes the current process because it undermines the ability of CASAC and EPA to arrive at appropriate and adequate decisions on these standards. The revised process threatens that the decisions you make as Administrator would not be based on a thorough review of the evidence.

The review process adopted in 2006 followed an open, deliberative discussion led by CASAC of the changes needed to improve the process. Based on the desire to provide an informed and robust assessment of the information, EPA established a protocol that included separate reviews of two separate drafts of critical documents, including a separate Risk and Exposure Assessment (REA). That process ensured that EPA would have reached conclusions on the scientific evidence about health and welfare impacts before beginning work on the policy implications. CASAC had agreed to such a plan for the review of the PM NAAQS in 2016, at the

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beginning of this process. EPA upended that agreement and reversed the long-established process with no public or CASAC review of the proposed changes.

EPA has set up an unprecedented, flawed process to truncate the review of the particulate matter and the ozone NAAQS. With these changes, critical information that forms the basis of the decisions is absent or unresolved. The Draft PA for particulate matter should have been fully informed by the Integrated Science Assessment (ISA). EPA should not have released the Draft PA until EPA finalized the ISA. Until the ISA is final, no final, reliable determination of the air quality criteria exists; that is, there is no full conclusion on the information which “accurately reflect[s] the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from presence to such pollutant in the ambient air.” 42 U.S.C 7408(a)(2).

Without first finalizing the ISA, EPA impairs the determination of the relevant policy decisions in accessing the Draft PA. EPA’s process unacceptably handicaps the review.

Further, EPA dismissed the established independent advisory panel for particulate matter, a step that cost the CASAC and EPA essential expertise in the complex avenues that the documents explore. Such panels had served multiple CASAC reviews for decades. EPA offered flawed arguments for eliminating the in-place, working panel: that under the Clean Air Act, only CASAC can advise EPA, and that such elimination would expedite the review. Both arguments are specious. The independent panels have always provided expert assistance only to CASAC, which CASAC then used to advise EPA. The panels have worked closely with CASAC to assist in an accurate and thorough scientific review following the adopted schedule.

Not until CASAC itself acknowledged its limitations and requested assistance in a letter to you did EPA take limited steps to provide additional assistance.<sup>1</sup> EPA appointed a new pool of advisors who lacked experience in the NAAQS review process and PM, and then mangled the process again by limiting advisory actions to one single panelist’s opinion, by letter, in response to written questions. This restricted process eliminated the traditional approach that provided a more complete and open discussion with multiple, experienced panelists who contributed independent perspectives and deliberated their consensus recommendations on topics throughout the reviews of each document.

The shining light in this damaged process is the diligence and thoroughness of the EPA staff in preparing this assessment. In general, there is much to support in their assessment. They have attempted to provide a full, extensive review, albeit based on the draft ISA, which should provide added evidence to EPA that more protective standards are needed for particulate matter.

The Lung Association strongly urges EPA to issue a second draft PA, reappoint the independent CASAC advisory panel and restore the process that the Agency had previously followed to complete this review to protect public health. The Lung Association also urges that EPA publish the final ISA before releasing a second draft PA.

#### Millions of Americans face increased risk from particulate matter

The Lung Association agrees with the finding in the draft PA, that “a substantial portion of the U.S. population” face increased risk from breathing particulate matter. The Lung Association supports recognition of these groups as at risk, as mentioned in the draft PA: children and teenagers; older adults; people with chronic lung diseases or cardiovascular diseases; people who are overweight

or obese; people with specific genetic variants; Hispanics; non-Hispanic blacks; and people who have low incomes. Evidence also indicates that healthy adults who work and exercise outdoors also face higher risk. The Lung Association particularly calls attention to the new evidence showing that African Americans face a three-time higher risk from PM than the entire population.<sup>2</sup> This adds to the evidence that African Americans, Hispanics and low-income communities face higher risk because social and environmental disparities.

The Lung Association is acutely concerned about the impacts to millions of people with lung diseases. In 2017, estimates show that 15.3 million adults had chronic obstructive pulmonary disease (COPD) and 25.2 million Americans had asthma, including 6.2 million children.<sup>3</sup> In 2019, more than 228,000 Americans will be diagnosed with lung cancer.<sup>4</sup>

All Americans deserve to have their health protected by stronger PM standards.

#### The current fine particulate standards fail to protect public health

One of the key findings in this Draft PA is overwhelming evidence that the current annual fine particulate (PM<sub>2.5</sub>) standard fails to protect public health. The Lung Association strongly supports that conclusion, as discussed below. However, strong evidence exists that the 24-hour PM<sub>2.5</sub> standard also fails to provide that required protection to public health. The Lung Association urges EPA to strengthen both the annual and the 24-hour PM<sub>2.5</sub> standards.

Today, more robust evidence than ever before supports the increased risk of premature death from levels below the current standards. No evidence exists of a threshold to that risk, as EPA has acknowledged. Recent U.S. studies that restrict the analysis to long-term exposures below 10 µg/m<sup>3</sup><sup>5</sup> and Canadian studies that find evidence down to and below 8 µg/m<sup>3</sup><sup>6</sup> all found premature deaths at those lower levels. A large study looking at short-term exposures also added evidence of harm below the current annual standard.<sup>7</sup> These studies offer new information that answered questions in previous reviews. The Canadian studies, in particular, offer data on low levels of exposures over long periods.

The Draft PA estimates that the current standards allow “a substantial number” of deaths from PM<sub>2.5</sub> exposure in the U.S. today. The analysis in Table 3-5 estimates that, using 2015 air quality data adjusted to just meet the 2015 annual standard, the median estimated annual mortality ranges from 13,500 to 52,100 premature deaths.<sup>8</sup> The evidence extends to the 24-hour standard, where even with the current standard, the median estimated mortality from short-term exposures ranges from 1,200 to 3,870 premature deaths annually.<sup>9</sup> These studies show consistent evidence that the current standards allow significant and unacceptable increased risk to health, unquestionably failing to provide the legally required protection. In addition, EPA’s estimates do not explore the evidence of harm from multiple other health effects of PM exposure, including COPD and asthma exacerbations. The Draft PA therefore does not consider the full impact of these inadequate standards.

The vast evidence that PM<sub>2.5</sub> shortens lives remains consistent as it has from the landmark studies in the 1990s. The follow-up studies that further tracked those cohorts mirror the findings in newer studies looking at lower levels of pollution<sup>10</sup> including some with one of the largest data sets in history, the 61 million people in the Medicare cohort.<sup>11</sup> The research included studies that examined the evidence using different ways of accessing exposure,<sup>12</sup> diverse regions of the country and diverse populations,<sup>13</sup> and different statistical models.<sup>14</sup>

We strongly disagree with the specious arguments provided in the Draft PA to create some justification for retaining the current standard. The powerful evidence from these epidemiological studies alone undercut these rationalizations of uncertainty. The evidence from the toxicological studies and clinical trials support these conclusions. The lack of studies examining exposures down to zero create no valid uncertainty over whether reducing PM levels further would add to the health benefits. We urge EPA to remove those arguments and that option from consideration.

We also disagree with EPA's evaluation that the 24-hour PM<sub>2.5</sub> standard provides sufficient protection for public health. EPA argues that its primary use is to supplement the annual standard. Even with that role, that combination fails to provide protection for many communities across the nation where the annual level is quite low. For communities in Alaska, parts of the Northwest and parts of New England, shorter-term exposures pose the primary risk because of the emissions from woodstoves and other sources that create elevated levels of PM<sub>2.5</sub>. Nearly all these areas have year-round concentrations that are well under the annual standard. The risk assessment in the Draft PA did not include areas in these parts of the nation, limiting the assessment of exposures to these sources.

Further, while wildfires do pose a significant source of the 24-hour exposures at high levels, they should not be dismissed as not caused by human action. Droughts exacerbated by climate change and fires from flawed electrical infrastructure as seen in California this year provide two examples of the human actions that contribute to the expanding burden of wildfires in the United States. Research supports this. In a study examining wildfires nationwide, researchers estimated that human activities caused 84 percent of wildfires between 2009 and 2012.<sup>15</sup>

The Lung Association continues to support changes to the form of the short-term standard, recommending at 99<sup>th</sup> percentile rather than at the 98<sup>th</sup>. The Draft PA continues to argue that the 98<sup>th</sup> percentile offers more stability to the standard than would at 99<sup>th</sup> percentile standard. While that is true, stability fails public health protection when the 98<sup>th</sup> percentile allows as many as 21 days to be exempted before meeting the standard.

Further, the Lung Association urges the consideration of a rolling 24-hour standard, rather than one that covers the 24 hours of a single calendar day. As one of the main sources of 24-hour PM spikes, woodstove smoke often peaks during shorter, overnight periods that may not be appropriately captured in the split that occurs at midnight. The Lung Association also urges EPA to require PM<sub>2.5</sub> monitoring to be a continuous monitoring network. The continued use of monitoring limited to data capture only every three or six days adds to the gaps in protection that Americans deserve from this deadly pollutant.

#### [The coarse particulate standard should be strengthened](#)

The PM<sub>10</sub> standard has stood in place, unaltered, since its adoption in 1987. That comes despite the long recognition that, while intended to provide protection against the coarse particles (PM<sub>10-2.5</sub>), inadequate monitoring and research limit the standard's ability to protect health from these particles. In 2010, CASAC advised EPA to consider a stronger PM<sub>10</sub> standard with a different form (98<sup>th</sup> percentile) and a more protective level, down to 65 µg/m<sup>3</sup>.<sup>16</sup> This Draft PA did not examine that recommendation.

EPA should reconsider the CASAC 2010 recommendation on PM<sub>10</sub> in a second Draft PA. The Lung Association supports strengthening the standard, based on a more complete review of the 2010 CASAC recommendations. In addition, EPA should expand the monitoring data for the coarse

fraction (PM<sub>10-2.5</sub>), to provide sufficient information to assess exposure and health risks that may be different from those solely of the fine particle fraction.

#### Recommendations for proposed PM<sub>2.5</sub> standards

Real world studies demonstrate that the current standards fail to protect health. EPA must adopt stronger standards based on the best available evidence that would protect health with an adequate margin of safety

Based on the information in the Draft ISA and Draft PA, the Lung Association urges EPA to strengthen the annual PM<sub>2.5</sub> standard to 8 µg/m<sup>3</sup> and the 24-hour standard to 25 µg/m<sup>3</sup>. The Draft PA considers annual standard levels down to 8 µg/m<sup>3</sup> based on the current evidence. These studies—including the Medicare cohort study<sup>17</sup> that found mortality associated with levels as low as 7 µg/m<sup>3</sup>—provide sufficient, robust evidence that the standard should be no higher than 8 µg/m<sup>3</sup> to protect public health with an adequate margin of safety.

The Lung Association recommends adopting a stronger standard of 25 µg/m<sup>3</sup> for the 24-hour standard, as well as changing the form of the standard to the 99<sup>th</sup> percentile.

#### EPA should support additional research

Research forms the basis of our understanding of the complex sources, composition, size, transmission, and health risks from particulate matter. The Lung Association urges EPA to increase research on particulate matter health impacts, including improved monitoring and health effect research on ultrafines, PM<sub>10-2.5</sub> and speciation, especially on respiratory health.

#### EPA should return to the previous review process with an experienced CASAC and independent advisory panel

The flawed process imposed on this review by EPA poses serious limits to the Agency's ability to fulfill its requirements under the Clean Air Act to protect human health. The inadequate review is now accompanied by an even more egregiously reduced review of the ozone NAAQS in an even shorter time. These reviews create an overwhelmingly impossible task for CASAC and EPA to complete in any reasonably appropriate way.


The Lung Association strongly urges EPA to restore the review process previously adopted and to restore the prior CASAC members and the prior independent panel. Only by returning to the full process that EPA abandoned will EPA be able to fulfill its duties required under the Clean Air Act.

Sincerely,



Deborah Brown  
Chief Mission Officer



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- <sup>10</sup> U.S. EPA. Draft Integrated Science Assessment for Particulate Matter. 2018 sections 11.2.2.2, 11.2.5
- <sup>11</sup> Di Q et al., 2017.
- <sup>12</sup> EPA 2018, section 11.2.5.1
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# The Legal Consequences of EPA's Disruption of the NAAQS Process

by Joe Goffman, Laura Bloomer

## INTRODUCTION

Under the Clean Air Act, EPA has a statutory mandate to set health-based air quality standards for six pervasive pollutants: carbon monoxide, lead, ground-level ozone, nitrogen dioxide, particulate matter, and sulfur dioxide. These standards, called the National Ambient Air Quality Standards (NAAQS), are a cornerstone of EPA's mission to achieve and maintain clean air. EPA must set the NAAQS at levels that protect the public health and welfare with an adequate margin of safety, and must review the NAAQS every five years. In practice, this means that as public health science advances and the health impacts of air pollutants are better understood, the agency's five-year reviews of the NAAQS often result in more protective standards.

Reviewing the NAAQS is a major effort that requires diverse scientific expertise. Understanding the importance of this responsibility, Congress charged an independent group of experts, now called the Clean Air Scientific Advisory Committee (CASAC), with assisting EPA in reviewing and revising the NAAQS. For over four decades, EPA –



with the help of CASAC – has developed and carried out a scientifically sound and widely lauded approach to determining the NAAQS.

EPA conducts comprehensive assessments of the relevant science, the risks to human health and the environment associated with exposure to the regulated pollutants, and the implications of alternative policy options. As EPA's website (<https://www.epa.gov/criteria-air-pollutants/process-reviewing-national-ambient-air-quality-standards>) states, "Scientific review during the development of these documents is thorough and extensive. Drafts of all documents are reviewed by CASAC and the public has an opportunity to comment on them." EPA and the independent review panels use the "weight of the evidence" framework (<https://science.sciencemag.org/content/early/2019/03/20/science.aaw9460?versioned=true>) to analyze the impact of air pollutants on public health by considering evidence from many scientific disciplines. This process ensures that the agency meets its statutory obligation to set the NAAQS at levels that protect the public's health with an adequate margin of safety. CASAC's involvement insulates the NAAQS from politics and gives credibility to NAAQS rulemaking.[1] Like the EPA itself, the courts rely on CASAC's scientific recommendations and EPA's response to CASAC's recommendations when considering legal challenges to the NAAQS.

## THE TRUMP ERA

Now, the Trump Administration's attacks on EPA's independent scientific review committees (<https://eelp.law.harvard.edu/2018/02/removing-scientists-from-advisory-panels/>) are crippling the NAAQS review efforts and threatening EPA's ability to meet its statutory obligation. They are obstructing CASAC as it seeks to advise EPA on the NAAQS for ozone and particulate matter (PM) through a series of disruptions to the review process. A recent decision by the D.C. Circuit Court of Appeals, Murray v. EPA (<https://law.justia.com/cases/federal/appellate-courts/cadc/15-1385/15-1385-2019-08-23.html>), No. 15-1385 (D.C. Cir. Aug. 23, 2019) highlighted the legal importance of maintaining the integrity of CASAC. The court emphasized EPA's



statutory obligation to follow CASAC's scientific advice or otherwise provide "substantial evidence" supporting an alternative determination. The court's essential presumption regarding the validity of CASAC's scientific advice reinforces that CASAC's determinations must reflect the best understanding of the current science and assist the agency in meeting its statutory duty to protect the public health.

Even so, the Trump administration is reshaping CASAC in a way that threatens the validity of CASAC's advice. The court may soon find itself at a fork in the road. In one direction, it could follow precedent and continue to make CASAC recommendations an essential component of its review. In that case, the court may unwittingly sanction an inferior scientific process to the detriment of the quality of the NAAQS and ultimately to public health. If it goes the other way, if CASAC loses credibility with the court, the court will need to reformulate its approach to reviewing the NAAQS.

In this post, we recount the steps that brought us here and discuss the consequences of these changes for setting air quality standards at the statutorily mandated level and for defending those standards in court.

## **EPA'S DIRECTIVES POLITICIZING & WEAKENING ADVISORY PANELS**

While we provide a summary here of the recent actions by the Trump administration to undermine the scientific expertise within and available to CASAC, for a more thorough explanation of these actions, please see posts by Gretchen Goldman at the Union of Concerned Scientists (<https://blog.ucsusa.org/author/gretchen-goldman#.Xim2XGhKiU>).

### **Restricting the science EPA can consider**

In early 2018, EPA released a proposed rule (<https://www.federalregister.gov/documents/2018/04/30/2018-09078/strengthening-transparency-in-regulatory-science>) that would severely limit the scientific studies that EPA and its

advisory committees could consider when reviewing the NAAQS and undertaking other actions. Under the proposed rule, EPA would only be able to consider studies if the underlying data could be made publicly available, which would exclude many essential epidemiological studies that are foundational to understanding the harmful impacts of pollutants on human health. After a contentious public comment period during which public health and environmental organizations united in opposition to the rule, EPA recently announced it will not finalize the original proposal and instead expects to publish a supplemental proposal that will be available for public comment in early 2020. Regardless, the 2018 proposed rule signals EPA's preference to avoid considering epidemiological studies in its NAAQS reviews.

### **Accelerating the NAAQS process**

On May 9, 2018, Administrator Scott Pruitt signed a Memorandum, Back-to-Basics Process for Reviewing National Ambient Air Quality Standards (<https://www.epa.gov/sites/production/files/2018-05/documents/image2018-05-09-173219.pdf>), which accelerated the process for reviewing and setting the NAAQS. The Administrator directed the agency to complete the current reviews of the ozone NAAQS by October 2020 and the PM NAAQS by December 2020. Scientists contend (<https://science.house.gov/imo/media/doc/Samet%20Testimony.pdf>) that these proposed timelines (which appear to have been pushed back slightly ([https://www.epa.gov/sites/production/files/2019-08/documents/o3-irp-aug27-2019\\_final.pdf](https://www.epa.gov/sites/production/files/2019-08/documents/o3-irp-aug27-2019_final.pdf)) since the original memo) may prevent CASAC and EPA from effectively reviewing the NAAQS.

### **Altering the NAAQS process**

The back-to-basics memo also proposed modifications to the NAAQS process that threaten its public health safeguards and scientific integrity. Prior to the May 2018 memo, EPA would first establish the air quality standard by considering only public health science. Once the standard was established, EPA could incorporate economic and

technological considerations when determining how the standards would be met. These two determinations occurred separately to ensure that the scientific determination of the appropriate air quality standard was insulated from other considerations. As Janet McCabe explained in our CleanLaw podcast (<https://eelp.law.harvard.edu/2018/06/subverting-the-process-of-setting-health-based-air-quality-standards-eelp-interviews-janet-mccabe/>), the back-to-basics memo compresses the review steps and could inject implementation considerations into the standard-setting process. This removes the public health safeguard of requiring EPA to establish health-based standards and implementation guidance separately. Nearly two decades ago, the Supreme Court in *Whitman v. American Trucking Associations, Inc.*, ruled that EPA is not allowed to consider the costs of implementation when setting the NAAQS. Yet the memo suggests that EPA will consider economic effects during the NAAQS review process, given that those considerations are relevant to the implementation guidance.

### **Disqualifying many academic experts**

In late 2017, EPA issued a directive ([https://www.epa.gov/sites/production/files/2017-10/documents/final\\_draft\\_fac\\_directive-10.31.2017.pdf](https://www.epa.gov/sites/production/files/2017-10/documents/final_draft_fac_directive-10.31.2017.pdf)) that removed distinguished scientists from CASAC by prohibiting any person who had received a grant from EPA from serving as a member of independent scientific advisory panels. There is no parallel prohibition on experts who are compensated by or affiliated with industries regulated by EPA. This policy tips the balance against academic experts and towards industry-affiliated scientists, as many academic experts routinely receive government funding for research. What's more – this approach to an alleged conflict-of-interest has long been discredited by the federal courts, see e.g. *Cargill, Inc., v. United States*, 173 F. 3d 323, 339 (1999).

### **Appointing anti-regulatory, industry-affiliated experts to CASAC**

By the end of 2018, EPA replaced the entire seven member CASAC panel (<https://www.epa.gov/newsreleases/acting-administrator->

wheeler-announces-science-advisors-key-clean-air-act-committee).

The current CASAC does not include an epidemiologist. On the other hand, several industry-affiliated members have called into question the regulation of harmful particulates. As of September 2019, only two university-affiliated experts serve on the panel.

### **Eliminating essential panels of experts**

EPA failed to convene an ozone review panel and disbanded the additional panel of 26 multidisciplinary experts it had formed to assist in reviewing the PM NAAQS. Previous NAAQS reviews have involved forming an auxiliary panel of experts to help the chartered CASAC in areas where it lacks expertise. Instead, Administrator Wheeler appointed twelve consultants (<https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>) who the CASAC chair may engage with on specific topics.

### **Changing appointment processes**

A recent GAO report found that EPA did not follow the agency's established process for vetting members of CASAC. As the report explains (<https://www.gao.gov/assets/710/700171.pdf>), during the vetting process, EPA creates an appointment packet for each candidate that includes rationales from career EPA staff "recommending the candidates EPA's staff deem best qualified and most appropriate for achieving balanced committee membership." The GAO report found that CASAC's 2018 appointment packets did not include this documentation (<https://eelp.law.harvard.edu/2019/08/gao-report-on-epas-science-advisory-panels/>).

## **IMPACTS TO THE DEVELOPMENT OF AIR QUALITY STANDARDS**

These recent actions call into question whether the agency will be able to meet its statutory mandate to set the ozone and PM NAAQS at a level requisite to protect public health. Unlike EPA Administrator Wheeler, the members of CASAC understand what's at stake. On April

11, 2019, CASAC wrote a letter to Administrator Wheeler ([https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/\\$File/EPA-CASAC-19-002+.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002+.pdf)) recommending that EPA reappoint the disbanded PM panel and add expertise in diverse scientific fields to ensure that “meaningful independent scientific review” could occur.

CASAC’s letter followed multiple letters (<https://blog.ucsusa.org/gretchen-goldman/uncharted-territory-the-epas-science-advisors-just-called-out-administrator-wheeler>) from former CASAC chairs, review panel members, and independent experts urging EPA to reinstate the panel. As a December 2018 letter from past members of EPA’s independent review panels stated, “the Charter CASAC, simply based on its number, cannot span the scope of science considered by the EPA as it guides the Administrator in assuring that the NAAQS will protect human health with an adequate margin of safety, as mandated by the Clean Air Act.” The letter went on to say that the rushed schedule for ozone and particulate matter “will reduce transparency, opportunity for public input, and the quality of review.”

Since Administrator Wheeler denied CASAC’s request to reconvene the panel and instead appointed consultants, CASAC members can only engage with the independent experts in writing and through Chair Tony Cox. Replacing the panel with consultants reduces transparency and quality of review, which ultimately threatens the outcomes of the NAAQS review process. Previously the panels held public meetings with CASAC, CASAC members were not required to filter questions through the Chair, and career EPA staff had significant input regarding the selection of the expert panel members. EPA also announced (<https://www.epa.gov/newsreleases/administrator-wheeler-announces-new-casac-member-pool-naaqs-subject-matter-experts>) the appointment of an additional scientist to CASAC to replace a member who retired. Rather than appointing an epidemiologist or public health expert, as many experts have requested, EPA appointed a wildlife toxicologist.

Furthermore, many scientists and CASAC members have drawn attention to the substance of the scientific review. For example, experts have raised concerns regarding Tony Cox's outlier approach to scientific assessment. As we explain in a [previous post](https://eelp.law.harvard.edu/2019/08/gao-report-on-epas-science-advisory-panels/), (<https://eelp.law.harvard.edu/2019/08/gao-report-on-epas-science-advisory-panels/>) rather than using the weight of the evidence framework, his proposal would require that EPA only consider studies that show a direct causation between an air quality standard and a health benefit. This approach would severely narrow the science that EPA could use to set the NAAQS. Scientists write that it would “[fundamentally change](https://science.sciencemag.org/content/363/6434/1398) (<https://science.sciencemag.org/content/363/6434/1398>) the EPA's process for scientific assessment” and would weaken air pollution standards.

CASAC members also requested a second review of the integrated science assessment for PM, stating that the first draft “does not provide a sufficiently comprehensive, systematic assessment of the available science....” Though this call for a second review is not exceptional, it underscores the importance of elevating quality of review over efficiency of review.

As many voices have made clear, the dramatic cutting of independent scientific expertise from CASAC and the NAAQS process is directly impacting EPA's ability to set air quality standards for ozone and PM that adequately protect the public health and welfare. In fact, the situation presents such a large threat to public health and to EPA's statutory mandate that 21 members of the disbanded PM Review Panel have re-organized as the **Independent Particulate Matter Review Panel (IPMRP)** (<https://news.ncsu.edu/2019/09/frey-clean-air-q-and-a-2019/>). Though the IPMRP is now unaffiliated with CASAC and EPA, the panel submitted formal comments on the draft Integrated Science Assessment and will meet again in October 2019 to review the draft Policy Assessment. As the chair of IPMRP, Chris Frey, stated, “We will do what EPA tried to prevent us from doing: advise CASAC, EPA staff, the EPA administrator, and the public regarding our scientific advice pertaining to key science and policy

issues.”

## POTENTIAL LONG-TERM LEGAL CONSEQUENCES

The legal impacts of the Trump’s administration’s attacks on CASAC may outlive the administration. As the D.C. Circuit made clear in Murray v. EPA (<https://law.justia.com/cases/federal/appellate-courts/cadc/15-1385/15-1385-2019-08-23.html>), CASAC’s determinations – and EPA’s decision to follow or diverge from CASAC’s advice – are important components of the court’s review of the NAAQS. Under Section 307 of the Clean Air Act, EPA must offer an explanation of any important differences between a NAAQS rulemaking and the findings, recommendations, and comments by scientific review committees. Should EPA fail to adequately meet the requirements of Section 307, the court can remand the issue to the agency to either modify its determination or provide a more thorough explanation. As the D.C. Circuit acknowledged in a 2013 case, “Congress intended that CASAC’s expert scientific analysis aid not only EPA in promulgating NAAQS but also the courts in reviewing EPA’s decisions.” *Mississippi v. EPA*, 744 F.3d 1334, 1355 (D.C. Cir. 2013).

The *Murray* opinion raises an important question regarding the legal implications for EPA’s current actions: what happens when CASAC is no longer comprised of independent, reputable scientists?

In one potential future, the court would continue to defer to CASAC’s advice and would rely on its own precedent to compel EPA to heed the advice of CASAC, regardless of its scientific credibility. In another future, the court could question CASAC’s legitimacy and decide to abandon its effective presumption towards CASAC’s scientific advice. This outcome would give the EPA Administrator, a political appointee, more authority to determine health standards and could shift the NAAQS process away from hard science. Such a shift in the balance of power away from science, already under way via EPA’s science-exclusion proposal and its disruption of CASAC’s make-up and deliberative processes, seems to be exactly what EPA is seeking.

There is also a potential future in which the court defers to CASAC only to the extent CASAC represents independent, scientific expertise. But that reality would require significant legal advocacy on behalf of EPA and other litigants to prove the level of deference merited by each iteration of CASAC. The court should be able to rely on the assurance that committees mandated by the Clean Air Act to provide expert, independent scientific analysis will provide precisely that.

Ultimately, Trump's EPA is destabilizing an essential and basic premise of air quality standards and public health protections: that EPA's scientific advisory committees will provide necessary, independent scientific advice based on the best available science. These actions could have significant legal repercussions in the future – both within the agency as it seeks to fulfil its statutory duty and within the courts as it seeks to defend its decisions.

[1] For an in-depth discussion of NAAQS and the role of independent science, see William Boyd, *The Clean Air Act's National Ambient Air Quality Standards* in LESSONS FROM THE CLEAN AIR ACT 15-55 (Ann Carlson & Dallas Burtraw eds., 2019).

**Download this post as a PDF (<http://eelp.law.harvard.edu/wp-content/uploads/Legal-Consequences-of-NAAQS-Changes.pdf>)**

*For information on events that occurred after this post, please see:*

- *Listen to our follow up [CleanLaw Podcast with Gretchen Goldman](https://eelp.law.harvard.edu/2019/12/cleanlaw-laura-bloomer-speaks-to-gretchen-goldman-about-epa-science-advisory-panels/) (<https://eelp.law.harvard.edu/2019/12/cleanlaw-laura-bloomer-speaks-to-gretchen-goldman-about-epa-science-advisory-panels/>)*
- *View our post, [Ongoing Changes to the Air Quality Standards Review Process](https://eelp.law.harvard.edu/2019/12/ongoing-changes-to-the-air-quality-standards-review-process/) (<https://eelp.law.harvard.edu/2019/12/ongoing-changes-to-the-air-quality-standards-review-process/>)*





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# Canadian Ambient Air Quality Standards

Alberta air zone reports and regional action plans.

## On this page:

- [Overview](#)
- [Management plans](#)

## Overview

The Canadian Ambient Air Quality Standards (CAAQS) are part of a collaborative national Air Quality Management System (AQMS), to better protect human health and the environment.

In October, 2012, the Canadian Council of Ministers of the Environment (CCME) agreed to the new CAAQS for fine particulate matter (PM<sub>2.5</sub>) and ozone. New CAAQS for sulphur dioxide (SO<sub>2</sub>) and nitrogen dioxide (NO<sub>2</sub>) were developed and will be effective in 2020.

The CCME have developed a [State of the Air website](#) to provide information on the AQMS and CAAQS across Canada.

## Management levels and standards

As part of the AQMS, Alberta was divided into six air zones. The ambient monitoring data for each air zone is assessed annually to make sure they meet the national air quality standards and management levels.

The management levels are represented by the colours green, yellow, orange, and red. Each management level has an air quality objective:

Management	Air quality objective
------------	-----------------------

level	
Green	To maintain good air quality through proactive air management measures to keep clean areas clean.
Yellow	To improve air quality using early and ongoing actions for continuous improvement.
Orange	To improve air quality through active air management and prevent exceedance of the CAAQS.
Red	To reduce pollutant levels below the CAAQS through advanced air management actions.

When an air zone report indicates that an air zone is in the orange or red management level, a management plan must be developed for that air zone. The Management plans section below provides information on the management plans in place for Alberta.

## Air zone reports

The annual air zone reports along with supplementary information on the CAAQS and the document outlining Alberta's implementation of the Air Zone Management Framework are available below.

- [Alberta Air Zones Reports](#)
- [Alberta Implementation of the Air Zone Management Framework for Fine Particulate Matter and Ozone](#)

## CAAQS tables

The following tables outline the CAAQS effective dates and management levels for each substance.

Unit	Definition
ppb	Parts per billion
$\mu\text{g m}^{-3}$	The weight, in micrograms, of the substance in one cubic metre of air

**Table 1: O<sub>3</sub>**

Management Level	O <sub>3</sub> (ppb)
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	<b>2015</b>	<b>2020</b>	<b>2025</b>
Red (CAAQS)	> 63	> 62	> 60
Orange	57 to 63	57 to 62	57 to 60
Yellow	51 to 56	51 to 56	51 to 56
Green	≤ 50	≤ 50	≤ 50

**Table 2: PM<sub>2.5</sub>**

<b>Management Level</b>	<b>PM<sub>2.5</sub> 24-hour (µg m<sup>-3</sup>)</b>		<b>PM<sub>2.5</sub> annual (µg m<sup>-3</sup>)</b>	
	<b>2015</b>	<b>2020</b>	<b>2015</b>	<b>2020</b>
Red (CAAQS)	> 28	> 27	> 10.0	> 8.8
Orange	20 to 28	20 to 27	6.5 to 10.0	6.5 to 8.8
Yellow	11 to 19	11 to 19	4.1 to 6.4	4.1 to 6.4
Green	≤ 10	≤ 10	≤ 4.0	≤ 4.0

**Table 3: SO<sub>2</sub>**

<b>Management Level</b>	<b>SO<sub>2</sub> 1-hour (ppb)</b>		<b>SO<sub>2</sub> annual (ppb)</b>	
	<b>2020</b>	<b>2025</b>	<b>2020</b>	<b>2025</b>
Red (CAAQS)	> 70	> 65	> 5.0	> 4.0
Orange	51 to 70	51 to 65	3.1 to 5.0	3.1 to 4.0
Yellow	31 to 50	31 to 50	2.1 to 3.0	2.1 to 3.0
Green	≤ 30	≤ 30	≤ 2.0	≤ 2.0

**Table 4: NO<sub>2</sub>**

<b>Management Level</b>	<b>NO<sub>2</sub> 1-hour (ppb)</b>	<b>NO<sub>2</sub> annual (ppb)</b>
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	<b>2020</b>	<b>2025</b>	<b>2020</b>	<b>2025</b>
Red (CAAQS)	> 60	> 42	> 17.0	> 12.0
Orange	32 to 60	32 to 42	7.1 to 17.0	7.1 to 12.0
Yellow	21 to 31	21 to 31	2.1 to 7.0	2.1 to 7.0
Green	≤ 20	≤ 20	≤ 2.0	≤ 2.0

## Alberta's air zones

The following map shows Alberta's six air zones, which are based on the Land-use Framework Regions.



- [Alberta's Land-use Framework](#)

The CAAQS replace the Canada-wide Standards for Fine Particulate Matter and Ozone, which were established in June 2000 by the Canadian Council of Ministers of the Environment (CCME). The Clean Air Strategic Alliance (CASA) developed a Particulate Matter and Ozone Management Framework and the Government of Alberta adopted this framework as Alberta's commitment to achieve Canada-wide Standard levels by the 2010 target date.

For more information on the CASA Particulate Matter and Ozone Management Framework and results of historical annual assessments, please see:

- [Particulate Matter and Ozone Management History](#)

## Management plans

The Canadian Ambient Air Quality Standards (CAAQS) assessment titled Alberta: Air Zones Report 2011 - 2013 for fine particulate matter and ozone was published in September 2015 for 2011 to 2013

monitoring data. In response to this report, Alberta committed to developing regional action plans for all six air zones.

The following reports provide information on the management level for each air zone and a summary of management actions committed to by the Government of Alberta in response to the assigned management level in each region.

## **Lower Athabasca Air Zone**

- [Lower Athabasca Region Air Zone CAAQS Response – Action Plan](#)

## **North Saskatchewan Air Zone**

- [North Saskatchewan Region Air Zone CAAQS Response – Action Plan](#)

## **Peace Air Zone**

- [Peace Region Air Zone CAAQS Response – Action Plan](#)

## **Red Deer Air Zone**

### **2017**

- [Red Deer Region Air Zone CAAQS Response – Action Plan](#)

### **2016**

- [Red Deer Air Zone Fine Particulate Matter Response – Action Plan](#)
- [Red Deer Fine Particulate Matter Response](#)
- [Red Deer Fine Particulate Matter Response Science Report](#)

## **South Saskatchewan Air Zone**

- [South Saskatchewan Region Air Zone CAAQS Response – Action Plan](#)

## **Upper Athabasca Air Zone**

- [Upper Athabasca Region Air Zone CAAQS Response – Action Plan](#)

# Ambient (outdoor) air pollution

2 May 2018

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## Key facts

- Air pollution is a major environmental risk to health. By reducing air pollution levels, countries can reduce the burden of disease from stroke, heart disease, lung cancer, and both chronic and acute respiratory diseases, including asthma.
- The lower the levels of air pollution, the better the cardiovascular and respiratory health of the population will be, both long- and short-term.
- The WHO Air Quality Guidelines: Global Update 2005 provide an assessment of health effects of air pollution and thresholds for health-harmful pollution levels.
- In 2016, 91% of the world population was living in places where the WHO air quality guidelines levels were not met.
- Ambient (outdoor air pollution) in both cities and rural areas was estimated to cause 4.2 million premature deaths worldwide in 2016.
- Some 91% of those premature deaths occurred in low- and middle-income countries, and the greatest number in the WHO South-East Asia and Western Pacific regions.
- Policies and investments supporting cleaner transport, energy-efficient homes, power generation, industry and better municipal waste management would reduce key sources of outdoor air pollution.
- In addition to outdoor air pollution, indoor smoke is a serious health risk for some 3 billion people who cook and heat their homes with biomass, kerosene fuels and coal.

## Background

Outdoor air pollution is a major environmental health problem affecting everyone in low-, middle-, and high-income countries.

Ambient (outdoor) air pollution in both cities and rural areas was estimated to cause 4.2 million premature deaths worldwide per year in 2016; this mortality is due to exposure to small particulate matter of 2.5 microns or less in diameter (PM<sub>2.5</sub>), which cause cardiovascular and respiratory disease, and cancers.

People living in low- and middle-income countries disproportionately experience the burden of outdoor air pollution with 91% (of the 4.2 million premature deaths) occurring in low- and middle-income countries, and the greatest burden in the WHO South-East Asia and Western Pacific regions. The latest burden estimates reflect the very significant role air pollution plays in cardiovascular illness and death. More and more, evidence demonstrating the linkages between ambient air pollution and the cardiovascular disease risk is becoming available, including studies from highly polluted areas.

WHO estimates that in 2016, some 58% of outdoor air pollution-related premature deaths were due to ischaemic heart disease and strokes, while 18% of deaths were due to chronic obstructive pulmonary disease and acute lower respiratory infections respectively, and 6% of deaths were due to lung cancer.

Some deaths may be attributed to more than one risk factor at the same time. For example, both smoking and ambient air pollution affect lung cancer. Some lung cancer deaths could have been averted by improving ambient air quality, or by reducing tobacco smoking.

A 2013 assessment by WHO's International Agency for Research on Cancer (IARC) concluded that outdoor air pollution is carcinogenic to humans, with the particulate matter component of air pollution most closely associated with increased cancer incidence, especially lung cancer. An association also has been observed between outdoor air pollution and increase in cancer of the urinary tract/bladder.

Addressing all risk factors for noncommunicable diseases – including air pollution – is key to protecting public health.

Most sources of outdoor air pollution are well beyond the control of individuals and demands concerted action by local, national and regional level policy-makers working in sectors like transport, energy, waste management, urban planning, and agriculture.

There are many examples of successful policies in transport, urban planning, power generation and industry that reduce air pollution:

- for industry: clean technologies that reduce industrial smokestack emissions; improved management of urban and agricultural waste, including capture of methane gas emitted from waste sites as an alternative to incineration (for use as biogas);
- for energy: ensuring access to affordable clean household energy solutions for cooking, heating and lighting;
- for transport: shifting to clean modes of power generation; prioritizing rapid urban transit, walking and cycling networks in cities as well as rail interurban freight and passenger travel; shifting to cleaner heavy-duty diesel vehicles and low-emissions vehicles and fuels, including fuels with reduced sulfur content;
- for urban planning: improving the energy efficiency of buildings and making cities more green and compact, and thus energy efficient;
- for power generation: increased use of low-emissions fuels and renewable combustion-free power sources (like solar, wind or hydropower); co-generation of heat and power; and distributed energy generation (e.g. mini-grids and rooftop solar power generation);
- for municipal and agricultural waste management: strategies for waste reduction, waste separation, recycling and reuse or waste reprocessing; as well as improved methods of biological waste management such as anaerobic waste digestion to produce biogas, are feasible, low cost alternatives to the open incineration of solid waste. Where incineration is unavoidable, then combustion technologies with strict emission controls are critical.

In addition to outdoor air pollution, indoor smoke from household air pollution is a serious health risk for some 3 billion people who cook and heat their homes with biomass fuels and coal. Some 3.8 million premature deaths were attributable to household air pollution in 2016. Almost all of the burden was in low-middle-income countries. Household air pollution is also a major source of outdoor air pollution in both urban and rural areas.

The 2005 *WHO Air quality guidelines* offer global guidance on thresholds and limits for key air pollutants that pose health risks. The Guidelines indicate that by reducing particulate matter (PM<sub>10</sub>) pollution from 70 to 20 micrograms per cubic metre (µg/m), we can cut air pollution-related deaths by around 15%.

The Guidelines apply worldwide and are based on expert evaluation of current scientific evidence for:

- particulate matter (PM)
- ozone (O<sub>3</sub>)
- nitrogen dioxide (NO<sub>2</sub>)
- sulfur dioxide (SO<sub>2</sub>).

Please note that the *WHO Air quality guidelines* are currently under revision with an expected publication date in 2020.



# Particulate matter (PM)

## Definition and principal sources

PM is a common proxy indicator for air pollution. It affects more people than any other pollutant. The major components of PM are sulfate, nitrates, ammonia, sodium chloride, black carbon, mineral dust and water. It consists of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air. While particles with a diameter of 10 microns or less, ( $\leq \text{PM}_{10}$ ) can penetrate and lodge deep inside the lungs, the even more health-damaging particles are those with a diameter of 2.5 microns or less, ( $\leq \text{PM}_{2.5}$ ).  $\text{PM}_{2.5}$  can penetrate the lung barrier and enter the blood system. Chronic exposure to particles contributes to the risk of developing cardiovascular and respiratory diseases, as well as of lung cancer.

Air quality measurements are typically reported in terms of daily or annual mean concentrations of  $\text{PM}_{10}$  particles per cubic meter of air volume ( $\text{m}^3$ ). Routine air quality measurements typically describe such PM concentrations in terms of micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ). When sufficiently sensitive measurement tools are available, concentrations of fine particles ( $\text{PM}_{2.5}$  or smaller), are also reported.

## Health effects

There is a close, quantitative relationship between exposure to high concentrations of small particulates ( $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ ) and increased mortality or morbidity, both daily and over time. Conversely, when concentrations of small and fine particulates are reduced, related mortality will also go down – presuming other factors remain the same. This allows policy-makers to project the population health improvements that could be expected if particulate air pollution is reduced.

Small particulate pollution has health impacts even at very low concentrations – indeed no threshold has been identified below which no damage to health is observed. Therefore, the WHO 2005 guideline limits aimed to achieve the lowest concentrations of PM possible.

## WHO Air quality guideline values

### Particulate matter (PM)

## Guideline values

### Fine particulate matter (PM<sub>2.5</sub>)

10 µg/m<sup>3</sup> annual mean

25 µg/m<sup>3</sup> 24-hour mean

### Coarse particulate matter (PM<sub>10</sub>)

20 µg/m<sup>3</sup> annual mean

50 µg/m<sup>3</sup> 24-hour mean

In addition to guideline values, the *WHO air quality guidelines* provide interim targets for concentrations of PM<sub>10</sub> and PM<sub>2.5</sub> aimed at promoting a gradual shift from high to lower concentrations.

If these interim targets were to be achieved, significant reductions in risks for acute and chronic health effects from air pollution can be expected. Achieving the guideline values, however, should be the ultimate objective.

The effects of PM on health occur at levels of exposure currently being experienced by many people both in urban and rural areas and in developed and developing countries – although exposures in many fast-developing cities today are often far higher than in developed cities of comparable size.

"*WHO air quality guidelines*" estimate that reducing annual average fine particulate matter (PM<sub>2.5</sub>) concentrations from levels of 35 µg/m<sup>3</sup>, common in many developing cities, to the WHO guideline level of 10 µg/m<sup>3</sup>, could reduce air pollution-related deaths by around 15%. However, even in the European Union, where PM concentrations in many cities do comply with guideline levels, it is estimated that average life expectancy is 8.6 months lower than it would otherwise be, due to PM exposures from human sources.

In low- and middle- income countries, exposure to pollutants in and around homes from the household combustion of polluting fuels on open fires or traditional stoves for cooking, heating and lighting further increases the risk for air pollution-related diseases, including acute lower respiratory infections, cardiovascular disease, chronic obstructive pulmonary disease and lung cancer.

There are serious risks to health not only from exposure to PM, but also from exposure to ozone (O<sub>3</sub>), nitrogen dioxide (NO<sub>2</sub>) and sulfur dioxide (SO<sub>2</sub>). As with PM, concentrations are often highest

largely in the urban areas of low- and middle-income countries. Ozone is a major factor in asthma morbidity and mortality, while nitrogen dioxide and sulfur dioxide also can play a role in asthma, bronchial symptoms, lung inflammation and reduced lung function.

## **Ozone (O<sub>3</sub>)**

### **Guideline values**

O<sub>3</sub>

100 µg/m<sup>3</sup> 8-hour mean

The recommended limit in the 2005 Air Quality Guidelines was reduced from the previous level of 120 µg/m<sup>3</sup> in previous editions of the "WHO Air Quality Guidelines" based on recent conclusive associations between daily mortality and lower ozone concentrations.

### **Definition and principal sources**

Ozone at ground level – not to be confused with the ozone layer in the upper atmosphere – is one of the major constituents of photochemical smog. It is formed by the reaction with sunlight (photochemical reaction) of pollutants such as nitrogen oxides (NO<sub>x</sub>) from vehicle and industry emissions and volatile organic compounds (VOCs) emitted by vehicles, solvents and industry. As a result, the highest levels of ozone pollution occur during periods of sunny weather.

### **Health effects**

Excessive ozone in the air can have a marked effect on human health. It can cause breathing problems, trigger asthma, reduce lung function and cause lung diseases.

## **Nitrogen dioxide (NO<sub>2</sub>)**

### **Guideline values**

NO<sub>2</sub>

40 µg/m<sup>3</sup> annual mean

200  $\mu\text{g}/\text{m}^3$  1-hour mean

The current WHO guideline value of 40  $\mu\text{g}/\text{m}^3$  (annual mean) was set to protect the public from the health effects of gaseous.

## Definition and principal sources

As an air pollutant,  $\text{NO}_2$  has several correlated activities. At short-term, concentrations exceeding 200  $\mu\text{g}/\text{m}^3$ , it is a toxic gas which causes significant inflammation of the airways.

$\text{NO}_2$  is the main source of nitrate aerosols, which form an important fraction of  $\text{PM}_{2.5}$  and, in the presence of ultraviolet light, of ozone. The major sources of anthropogenic emissions of  $\text{NO}_2$  are combustion processes (heating, power generation, and engines in vehicles and ships).

## Health effects

Epidemiological studies have shown that symptoms of bronchitis in asthmatic children increase in association with long-term exposure to  $\text{NO}_2$ . Reduced lung function growth is also linked to  $\text{NO}_2$  at concentrations currently measured (or observed) in cities of Europe and North America.

## Sulfur dioxide ( $\text{SO}_2$ )

### Guideline values

$\text{SO}_2$

20  $\mu\text{g}/\text{m}^3$  24-hour mean

500  $\mu\text{g}/\text{m}^3$  10-minute mean

A  $\text{SO}_2$  concentration of 500  $\mu\text{g}/\text{m}^3$  should not be exceeded over average periods of 10 minutes' duration. Studies indicate that a proportion of people with asthma experience changes in pulmonary function and respiratory symptoms after periods of exposure to  $\text{SO}_2$  as short as 10 minutes. Health effects are now known to be associated with much lower levels of  $\text{SO}_2$  than previously believed. A greater degree of protection is needed. Although the causality of the effects of low concentrations of  $\text{SO}_2$  is still uncertain, reducing  $\text{SO}_2$  concentrations is likely to decrease exposure to co-pollutants.

## Definition and principal sources

SO<sub>2</sub> is a colourless gas with a sharp odour. It is produced from the burning of fossil fuels (coal and oil) and the smelting of mineral ores that contain sulfur. The main anthropogenic source of SO<sub>2</sub> is the burning of sulfur-containing fossil fuels for domestic heating, power generation and motor vehicles.

## Health effects

SO<sub>2</sub> can affect the respiratory system and the functions of the lungs, and causes irritation of the eyes. Inflammation of the respiratory tract causes coughing, mucus secretion, aggravation of asthma and chronic bronchitis and makes people more prone to infections of the respiratory tract. Hospital admissions for cardiac disease and mortality increase on days with higher SO<sub>2</sub> levels. When SO<sub>2</sub> combines with water, it forms sulfuric acid; this is the main component of acid rain which is a cause of deforestation.

# WHO response

WHO Member States recently adopted a resolution (2015) and a road map (2016) for an enhanced global response to the adverse health effects of air pollution.

WHO is custodial agency for 3 air pollution-related Sustainable Development Goals indicators:

- 3.9.1 Mortality from air pollution
- 7.1.2 Access to clean fuels and technologies
- 11.6.2 Air quality in cities.

WHO develops and produces air quality guidelines recommending exposure limits to key air pollutants (indoor and outdoor).

WHO creates detailed health-related assessments of different types of air pollutants, including particulates and black carbon particles, and ozone.

WHO produces evidence regarding the linkage of air pollution to specific diseases, such as cardiovascular and respiratory diseases and cancers, as well as burden of disease estimates from existing air pollution exposures, at country, regional, and global levels.

WHO develops tools such as AirQ+ for assessing the health impacts from various pollutants, but also the Health Economic Assessment Tool (HEAT) to assess walking and cycling interventions, the

Green+ tool to raise importance of green space and health, the Sustainable Transport Health Assessment Tool (STHAT) and the Integrated Transport and Health Impact Modelling Tool (ITHIM).

WHO is developing a Clean Household Energy Solutions Toolkit (CHEST) to provide countries and programmes with the tools needed to create or evaluate policies that expand clean household energy access and use, which is particularly important as pollutants released in and around the household (household air pollution) contribute significantly to ambient pollution. CHEST tools include modules on needs assessment, guidance on standards and testing for household energy devices, monitoring and evaluation, and materials to empower the health sector to tackle household air pollution.

WHO assists Member States in sharing information on successful approaches, on methods of exposure assessment and monitoring of health impacts of pollution.

WHO is leading the Joint Task Force on the Health Aspects of Air Pollution within the Convention on Long-range Transboundary Air Pollution to assess the health effects of such pollution and to provide supporting documentation.

The WHO co-sponsored Pan European Programme on Transport Health and Environment (PEP), has built a model of regional, Member State, and multisectoral cooperation for mitigation of air pollution and other health impacts in the transport sector, as well as tools for assessing the health benefits of such mitigation measures.

# Exposure to Formaldehyde and Asthma Outcomes: A Systematic Review, Meta-Analysis, and Economic Assessment

--Manuscript Draft--

<b>Manuscript Number:</b>	PONE-D-19-21348R4
<b>Article Type:</b>	Research Article
<b>Full Title:</b>	Exposure to Formaldehyde and Asthma Outcomes: A Systematic Review, Meta-Analysis, and Economic Assessment
<b>Short Title:</b>	Exposure to Formaldehyde and Asthma Outcomes
<b>Corresponding Author:</b>	T.J Woodruff University of California San Francisco San Francisco, UNITED STATES
<b>Keywords:</b>	Formaldehyde; Systematic review; meta-analysis; asthma; Epidemiology; Evidence Integration; Navigation Guide; Benefit-Cost Analysis
<b>Abstract:</b>	<p>Background Every major federal regulation in the United States requires an economic analysis estimating its benefits and costs. Benefit-cost analyses related to regulations on formaldehyde exposure have not included asthma in part due to lack of clarity in the strength of the evidence. Objectives 1) To conduct a systematic review of evidence regarding human exposure to formaldehyde and diagnosis, signs, symptoms, exacerbations, or other measures of asthma in humans; and 2) quantify the annual economic benefit for decreases in formaldehyde exposure. Methods We developed and registered a protocol in PROSPERO (Record ID #38766). We conducted a comprehensive search of articles published up to March 15, 2018. We evaluated potential risk of bias for included studies, identified a subset of studies to combine in a meta-analysis, and rated the overall quality and strength of the evidence. We quantified economics benefit to children from a decrease in formaldehyde exposure using assumptions consistent with EPA's proposed formaldehyde rule. Results We screened 4,482 total references and identified 150 human studies that met inclusion criteria; of these, we focused on 85 studies reporting asthma status of all participants with quantified measures of formaldehyde directly relevant to our study question. Nine studies were combinable in a meta-analysis for childhood asthma diagnosis and four combinable for exacerbation of childhood asthma (wheezing and shortness of breath). Studies had low to probably-low risk of bias across most domains. A 10-<math>\mu\text{g}/\text{m}^3</math> increase in formaldehyde exposure was associated with increased childhood asthma diagnosis (OR=1.08, 95% CI: [1.02, 1.14]). We also found a positive association with exacerbation of childhood asthma (OR=1.08, 95% CI: [0.92, 1.28]). The overall quality and strength of the evidence was rated as "moderate" quality and "sufficient" for asthma diagnosis and asthma symptom exacerbation in both children and adults. We estimated that EPA's proposed rule on pressed wood products would result in 1,197 fewer asthma cases and total economic benefit of \$90 million annually. Conclusion We concluded there was "sufficient evidence of toxicity" for associations between exposure to formaldehyde and asthma diagnosis and asthma symptoms in both children and adults. Our research documented that when exposures are ubiquitous, excluding health outcomes with relatively "small" effect estimates from benefit-cost analysis can underestimate the true benefits to health from environmental regulations.</p>
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<b>Opposed Reviewers:</b>	
<b>Response to Reviewers:</b>	All of our comments can be found in our cover letter.
<b>Additional Information:</b>	
<b>Question</b>	<b>Response</b>
<p><b>Financial Disclosure</b></p> <p>Enter a financial disclosure statement that describes the sources of funding for the work included in this submission. Review the <a href="#">submission guidelines</a> for detailed requirements. View published research articles from <a href="#">PLOS ONE</a> for specific examples.</p> <p>This statement is required for submission and <b>will appear in the published article</b> if the submission is accepted. Please make sure it is accurate.</p> <p><b>Unfunded studies</b> Enter: <i>The author(s) received no specific funding for this work.</i></p> <p><b>Funded studies</b> Enter a statement with the following details:</p> <ul style="list-style-type: none"> <li>• Initials of the authors who received each award</li> <li>• Grant numbers awarded to each author</li> <li>• The full name of each funder</li> <li>• URL of each funder website</li> <li>• Did the sponsors or funders play any role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript?</li> <li>• <b>NO</b> - Include this sentence at the end of your statement: <i>The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.</i></li> <li>• <b>YES</b> - Specify the role(s) played.</li> </ul> <p>* typeset</p>	<p>Funding source: JPB Foundation (Grant #681), NIEHS P01ES022841, USEPA RD 83543301.</p> <p>The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript</p>
<b>Competing Interests</b>	The authors have declared that no competing interests exist.
Use the instructions below to enter a competing interest statement for this submission. On behalf of all authors,	



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#### Human Subject Research (involving human participants and/or tissue)

- Give the name of the institutional review board or ethics committee that approved the study
- Include the approval number and/or a statement indicating approval of this research
- Indicate the form of consent obtained (written/oral) or the reason that consent was not obtained (e.g. the data were analyzed anonymously)

#### Animal Research (involving vertebrate animals, embryos or tissues)

- Provide the name of the Institutional Animal Care and Use Committee (IACUC) or other relevant ethics board that reviewed the study protocol, and indicate whether they approved this research or granted a formal waiver of ethical approval
- Include an approval number if one was obtained
- If the study involved *non-human primates*, add *additional details* about animal welfare and steps taken to ameliorate suffering
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Include the following details if this study involves the collection of plant, animal, or other materials from a natural setting:

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Additional data availability information:	



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February 18, 2021

Dear Dr. Amegah,

Thank you for your recent message and additional edits regarding our manuscript appeal to PLOS ONE. Below we have responded to your comment.

**General Comment 1: In order to improve the readability of the article, please move tables 3a and 3b to the supplementary material. In addition, please consider dividing table 3b by study design.**

*We have moved both tables as requested to the supplement and have updated the table order in the manuscript. The new manuscript is down to ~70 pages (clean) and we thank the editorial team for making this suggestion to improve the readability of our manuscript.*

*Table 3a: We have moved this to the supplement, and it is now the new Supplemental Table 99. We have made the old Supplemental Table 99 ("Study characteristics by population/outcome") into Supplemental Table 100.*

*Table 3b: We've moved Table 3b to the supplement and split it up by study design (cohort, case-control, cross-sectional, non-randomized controlled trial, randomized controlled trial, case report) as requested. We have further stratified each table by studies considered for meta-analysis and studies not considered for meta-analysis, similar to (new) Supplemental Table 100. Table 3b is now Supplemental Table 101 ("Study characteristics by study design").*

We appreciate your time and patience throughout this process. Let us know if you have any additional questions.

Thank you,

A handwritten signature in black ink, appearing to read "Tracey Woodruff". The signature is written in a cursive, flowing style.

Dr. Tracey Woodruff

# **Exposure to Formaldehyde and Asthma Outcomes: A Systematic Review, Meta-Analysis, and Economic Assessment**

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## **Competing financial interests:**

The authors declare no competing financial interests.

## **Disclaimer:**

The views expressed in this paper are those of the authors and do not necessarily reflect the view or policies of the U.S. Environmental Protection Agency. MDC is a member of the United States Preventative Services Task Force (USPSTF). This article does not necessarily represent the views and policies of the USPSTF.

## Abstract

**Background:** Every major federal regulation in the United States requires an economic analysis estimating its benefits and costs. Benefit-cost analyses related to regulations on formaldehyde exposure have not included asthma in part due to lack of clarity in the strength of the evidence.

**Objectives:** 1) To conduct a systematic review of evidence regarding human exposure to formaldehyde and diagnosis, signs, symptoms, exacerbations, or other measures of asthma in humans; and 2) quantify the annual economic benefit for decreases in formaldehyde exposure.

**Methods:** We developed and registered a protocol in PROSPERO (Record ID #38766, CRD 42016038766). We conducted a comprehensive search of articles published up to April 1, 2020. We evaluated potential risk of bias for included studies, identified a subset of studies to combine in a meta-analysis, and rated the overall quality and strength of the evidence. We quantified economics benefit to children from a decrease in formaldehyde exposure using assumptions consistent with EPA's proposed formaldehyde rule.

**Results:** We screened 4,821 total references and identified 150 human studies that met inclusion criteria; of these, we focused on 90 studies reporting asthma status of all participants with quantified measures of formaldehyde directly relevant to our study question. Ten studies were combinable in a meta-analysis for childhood asthma diagnosis and five combinable for exacerbation of childhood asthma (wheezing and shortness of breath). Studies had low to probably-low risk of bias across most domains. A 10- $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure was associated with increased childhood asthma diagnosis (OR=1.20, 95% CI: [1.02, 1.41]). We also found a positive association with exacerbation of childhood asthma (OR=1.08, 95% CI:

[0.92, 1.28]). The overall quality and strength of the evidence was rated as “moderate” quality and “sufficient” for asthma diagnosis and asthma symptom exacerbation in both children and adults. We estimated that EPA’s proposed rule on pressed wood products would result in 2,805 fewer asthma cases and total economic benefit of \$210 million annually.

**Conclusion:** We concluded there was “sufficient evidence of toxicity” for associations between exposure to formaldehyde and asthma diagnosis and asthma symptoms in both children and adults. Our research documented that when exposures are ubiquitous, excluding health outcomes from benefit-cost analysis can underestimate the true benefits to health from environmental regulations.

## **Introduction**

Formaldehyde exposure is ubiquitous and occurs in homes, communities, and workplaces. Formaldehyde is a high-volume production chemical with numerous industrial and commercial uses as a solution, disinfectant, preservative or to produce industrial resins used to manufacture adhesives and binders in wood, paper, and other products. It is present in many household products, such as foam insulation, cleaning and personal care products, pressed wood products such as particleboard and plywood, and as a result is a common indoor air pollutant found in virtually all homes and buildings (1-9). Homes are impacted by off-gassing of formaldehyde from new housing materials, with availability and rates of ventilation having minimal impact on exposure levels (10).

In particular, formaldehyde is an environmental justice and affordable housing concern. Lower-income communities are disproportionately at risk of exposure to formaldehyde and resulting health effects from pressed wood products in homes built with less costly building materials.



Formaldehyde exposure extends beyond residential homes—for instance, formaldehyde has been measured at levels exceeding exposure limits in childcare settings in California. Workplace exposure to formaldehyde occurs in a wide variety of industries and occupations, such as in the manufacture or production of formaldehyde or formaldehyde-based products or during firefighting, embalming, carpentry, and pathology lab work.

Asthma is a complex disease caused by chronic inflammation of the airways that results in episodic airway hyper responsiveness, excessive mucous secretion, and airway obstruction.

Exposure to formaldehyde occurs primarily through inhalation and also as a respiratory contact irritant (11). The relationship between exposure to formaldehyde and asthma has been actively under evaluation by government agencies for the last few decades (12-14). A substantial amount of research exploring relationships between formaldehyde exposure and exacerbation of asthma has been conducted, but few systematic reviews (with a pre-established protocol, systematic literature search, pre-defined criteria for evaluating studies and categories to assess the strength of evidence) are available providing a comprehensive overview of the evidence.

The U.S. Environmental Protection Agency (EPA) released its review of formaldehyde health risks in its Integrated Risk Information System (IRIS) assessment in 1990, initiated a reassessment in 1998, and released a draft report in 2010, which included a review of the asthma health outcome (Fig 2). A review of the draft assessment by the National Academy of Sciences (NAS) highlighted many methodological limitations of the IRIS process, such as EPA's study selection and evaluation criteria that led to the advancement of one study (15) with potential misclassification of infection-associated wheezing in young children as asthma (14). EPA's conclusion of a causal relationship between formaldehyde exposure and asthma incidence and

subsequent derivation of a candidate Reference Concentration (RfC) was ultimately challenged by the NAS committee (14).

In 2010, Congress required EPA to issue a rule on pressed wood products and emissions of formaldehyde; ultimately EPA issued a final rule on formaldehyde in 2016 (Fig 2). EPA conducted a benefits cost analysis of this rule under an Executive Order that requires every significant regulation in the U.S. be accompanied by an economic analysis of the benefits and costs of implementation. EPA initially included asthma in the benefit-cost analysis for the proposed rule; however, asthma was removed from the analysis after interagency review. In the U.S., asthma affects approximately 23 million people, including 6 million children (16), impacting approximately 8% of both children and adults (17). The omission of asthma from the benefit-cost analysis could significantly underestimate the true value of regulating formaldehyde in pressed wood products.

To assess the evidence of formaldehyde's contribution to asthma outcomes, we conducted a systematic review of human studies to answer the question of whether exposure to formaldehyde is associated with diagnosis, signs, symptoms, exacerbation, or other measures of asthma in humans. We used results from the quantitative evaluation of the evidence to estimate the benefits of the reduction in asthma cases implied by the proposed EPA rule on pressed wood products.

## **Methods**

We applied the Navigation Guide systematic review methodology, a systematic and transparent method for synthesizing the available scientific evidence designed specifically for environmental exposures (18, 19). The method is based on Cochrane and GRADE methods (20, 21) and includes the same elements (protocol development, risk of bias evaluation, evidence evaluation,

etc.). However, one main difference is that this method accounts for differences in evidence and decision context inherent to environmental health assessments, i.e., the reliance on human observational studies in the absence of randomized controlled trials (RCTs), and the fact that population exposure to exogenous chemicals precedes evidence of their safety.

### ***Protocol***

We developed a protocol prior to initiating the review and registered it in PROSPERO in May 2016 (<http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD 42016038766).

### ***Study question***

Our systematic review objective was to answer the question: “Is exposure to formaldehyde associated with the diagnosis, signs, symptoms, exacerbation, or other measures of asthma in humans?”

The “Participants”, “Exposure,” “Comparator” and “Outcomes” (PECO) statement is briefly outlined below, with additional specifics available in the protocol.

**Participants:** Humans.

**Exposure:** Any indoor or outdoor sources of airborne inhalation exposure to formaldehyde, including but not limited to occupational, outdoor ambient, indoor household settings, and/or exposure to household products that occurred prior or concurrent to health outcome.

**Comparator:** Humans exposed to lower levels of formaldehyde than the more highly exposed humans.

**Outcomes:** Any of the following asthma-related outcomes: diagnosis of asthma, asthma signs or symptoms, asthma exacerbation, or indirect measures of asthma.

## ***Data Sources***

We searched the databases PubMed, ISI Web of Science, Biosis Previews, Embase, Google Scholar, and Toxline from the inception of each database up to April 1, 2020 using the search terms in S1-5 Tables. We did not limit our search by language or initial publication date. We used the Medical Subject Headings (MeSH) database to compile synonyms for formaldehyde and asthma-related outcomes. Our search terms and search strategy were developed by two librarians trained in systematic review methodology (LS, EW). We also supplemented these results by searching toxicological and grey literature databases (S6-7 Tables), consulting with subject matter experts, and hand-searching references by reviewing reference lists of included studies and review papers on the topic as well as searching for references that cited included studies (“snowball searching”).

## ***Study Selection***

We included studies that contained original data from human studies that measured or reported formaldehyde exposure prior to evaluating the health outcome. We screened references for inclusion using structured forms in DistillerSR (Evidence Partners; available at: <http://www.systematic-review.net>). Two of four possible reviewers (EK, ND, AP, HV) independently reviewed titles and abstracts of each reference to determine eligibility in a non-random assignment (to ensure that the same two authors did not always screen the same references). In the event that an abstract was missing or there were discrepancies between the two reviewers, the default was to move the reference forward for full text review. Two of the same four reviewers (EK, ND, AP, HV) then independently performed a full-text review to evaluate inclusion criteria of each reference not excluded by title/abstract screening. An

additional reviewer (JL) screened five percent of the titles/abstracts and full-texts for quality assurance.

We excluded studies if any one of the following criteria was met: 1) the report did not contain original data; 2) the article did not involve human subjects; 3) there was no report of formaldehyde exposure; 4) there was no report of diagnosis of asthma, asthma signs or symptoms, asthma exacerbation, or indirect measures of asthma (such as daily use of inhaler); or 5) there was no comparator—control group or exposure range comparison (S1 Methods). We translated the title and abstracts of studies using freely available online software (i.e., Google Translate) that were not published in English to evaluate its relevance.

### ***Data Extraction***

We extracted data from studies in duplicate in a Health Assessment Workplace Collaborative database (HAWC; available at: <https://hawcproject.org/about/>). Two of three possible extractors (SE, EM, DB) independently extracted data relating to study characteristics and outcome measures (S2 Methods) from each included article. A third extractor (PH, BV) performed QA/QC on all the studies to resolve any discrepancies between the two independent extractors; subsequently, two authors (JL, EK) reviewed all studies to further ensure the accuracy of extracted data. When information was missing from a published article, we contacted corresponding study authors to request additional information.

*Rate the quality and strength of the evidence*

**Statistical analyses:** Prior to study selection, we developed a list of study characteristics (contained in our protocol: <http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD 42016038766) to identify studies suitable for meta-analysis. After evaluating the characteristics of all the studies, we grouped studies into four study population and health outcome combinations: 1) child asthma diagnosis; 2) child asthma exacerbation and symptoms; 3) adult asthma diagnosis; and 4) adult asthma exacerbation and symptoms.

To differentiate child from adult studies, we initially planned to use the age of 18 years as a cutoff for children, but a number of the studies used a cutoff age of 15 years to distinguish between children and adults. Given that the onset of asthma commonly occurs during preschool years and recent increases in asthma incidence over the past few decades has been observed to increasingly affect children and adolescents aged 1 to 14 years, we decided to use age 15 years as the cutoff to group child vs. adult studies. We did not include studies in the meta-analysis that reported effect estimates with only mixed children and adult populations in the meta-analysis due to concerns that differences in adult-onset versus childhood-onset of asthma would be masked. We also did not consider these data in our overall rating of study quality and strength, but we did include these data in visual scatterplots of data for comparison with child and adult data.

For the adult studies, we considered the body of evidence to include all adult population studies, regardless of whether exposure occurred in the general population or at work, as biologically, the relationship between exposure and health outcome is independent of where the exposure occurred. We distinguished the adult general population study results from the adult occupational study results on the visual scatterplots for comparison.

For cohorts with multiple publications (for instance, if a cohort was followed over time), we utilized results from the latest time point where our relevant outcome of interest was measured, but also considered information provided collectively across the publications for evaluating study quality. Where available, we used adjusted odds ratios to conduct the meta-analysis but if adjusted results were not reported, we included unadjusted ORs in the analyses. We converted effect estimates to an OR and 95% confidence interval (CI) for the association between asthma per 10- $\mu\text{g}/\text{m}^3$  unit increase in formaldehyde exposure to standardize across studies, transforming units of exposure when necessary. Where a meta-analysis was not possible, we created visual scatterplots of data across studies reporting on similar outcomes and subpopulations to consider all available data in assessing the evidence. We also applied a mixed models approach for repeated data to evaluate outcomes at various doses, using exchangeable correlation structures for repeated measurements within the same study.

We evaluated statistical heterogeneity across study estimates in the meta-analysis using  $I^2$  with  $p \leq 0.05$  as our cut off for statistical significance, as previously described. If statistical heterogeneity was present, we used leave-one-out analysis to identify the study or studies contributing, evaluated potential study characteristics (e.g., study location, study population, study design, adjusted confounders, timing of exposure, etc.) to determine if we could explain the source, and incorporated hierarchical cluster structures in the data analysis to statistically account for heterogeneity. We also investigated the relative contribution of each study to the overall meta-analysis association and conducted sensitivity analysis to investigate the impacts of removing highly influential studies from the analysis. Data management was performed with Microsoft Excel. Statistical analyses were performed using STATA 13.1 software (StataCorp, 2011). We pooled estimates using inverse variance-weighted models, fixed-effects models and

the DerSimonian and Laird random-effects models. We used the *metan*, *metareg*, *metainf*, *metafunnel*, *metabias* and *metatrim* packages in STATA version 13.1.

To investigate the effect of publication bias on our meta-analysis, we created funnel plots and used Egger's test. We also quantitatively evaluated each meta-analysis for the potential effect that a new study might have on changing the interpretation of our overall results. Specifically, the association estimate of a new or unpublished study necessary to alter the results of the meta-analysis was calculated under two scenarios: 1) the 95% confidence interval of the meta-analysis overlapped zero, and 2) the meta-analysis central association estimate was greater than zero (moved to the opposite direction—i.e., such that increases in formaldehyde exposures would be associated with decreases in asthma outcomes). In making this calculation, we assumed that the new hypothetical study would have a standard error equal to the smallest in our group of studies.

**Assessing the risk of bias for each included study:** We evaluated risk of bias separately for each of the four study population/outcome group combinations using The Navigation Guide Risk of Bias Tool, a modified instrument based on the Cochrane Collaboration and Agency for Healthcare Research and Quality (AHRQ) domains , with customized instructions for each domain based on the type of evidence anticipated beforehand (S3 Methods).

We evaluated nine risk of bias domains (Source Population, Blinding, Outcome Assessment, Confounding, Incomplete Outcome, Exposure Assessment, Selective Reporting, Financial Conflict of Interest, and Other). We assigned each domain as “low,” “probably low,” “probably high,” or “high” risk of bias, or “not applicable” (domain not applicable to study) according to specific criteria as described in our risk of bias instruments (S3 Methods). Two of three possible reviewers (SE, EM, RB) independently recorded risk of bias determinations for each included



study. We held an in-person meeting for all review authors (JL, EK, PS, AMP, MDC, HV, ND, EW, TJW) to review risk of bias ratings and rationales for each study, come to consensus to ensure consistency, and record our final rationale. One review author (EK) independently reviewed all final risk of bias ratings for QA/QC.

**Rating the quality of evidence across all included studies:** We separately rated the quality of the overall body of evidence as “high,” “moderate,” or “low” for each of the four study population/outcome group combinations. We assigned an initial rating of “moderate” quality for each group of human observational studies prior to evaluating the included studies, based on previously described rationale—briefly, observational human studies are recognized as a reliable source of evidence and generally the most appropriate for answering environmental health-related questions. From the initial “moderate” quality rating, we then considered potential adjustments (“downgrades” or “upgrades”) to the quality rating based on 8 categories of considerations: risk of bias, indirectness, inconsistency, imprecision, potential for publication bias, large magnitude of effect, dose response, and whether residual confounding would minimize the overall effect estimate; the specific factors and criteria considered are outlined in S4 Methods. Possible ratings were 0 (no change from initial quality rating), -1 (1 level downgrade) or -2 (2 level downgrade), +1 (1 level upgrade) or +2 (2 level upgrade). Review authors independently evaluated the quality of the evidence and then we compared ratings as a group and recorded the consensus and rationale for each decision.

**Rating the strength of the evidence across all included studies:** We assigned an overall strength of evidence rating separately for the four study population/outcome group combinations based on four considerations: (1) Quality of body of evidence (i.e., the rating from the previous step); (2) Direction of effect; (3) Confidence in effect (likelihood that a new study would change

265 our conclusion); and (4) Other compelling attributes of the data that may influence certainty.  
266 Possible ratings were “sufficient evidence of toxicity,” “limited evidence of toxicity,”  
267 “inadequate evidence of toxicity,” or “evidence of lack of toxicity” (Table 1), based on  
268 categories used by the International Agency for Research on Cancer (IARC), the U.S. Preventive  
269 Services Task Force, and U.S. EPA (22-25). Review authors independently evaluated the quality  
270 of the evidence following directions as outlined in S4 Methods and then compared ratings as a  
271 group and recorded the consensus and rationale.

**Table 1.** Strength of evidence definitions for human evidence

Strength Rating    Definition

Sufficient evidence of toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies <sup>1</sup> .
Limited Evidence of Toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding cannot be ruled out with reasonable confidence. Confidence in the relationship is constrained by such factors as: the number, size, or quality of individual studies, or inconsistency of findings across individual studies <sup>2</sup> . As more information becomes available, the observed effect could change, and this change may be large enough to alter the conclusion.
Inadequate Evidence of Toxicity	The available evidence is insufficient to assess effects of the exposure. Evidence is insufficient because of: the limited number or size of studies, low quality of individual studies, or inconsistency of findings across individual studies. More information may allow an assessment of effects.
Evidence of Lack of Toxicity	No relationship is observed between exposure and outcome, and chance, bias and confounding can be ruled out with reasonable confidence. The available evidence includes consistent results from more than one well-designed, well-conducted study at the full range of exposure levels that humans are known to encounter, and the conclusion is unlikely to be strongly affected by the results of future studies <sup>3</sup> . The conclusion is limited to the age at exposure and/or other conditions and levels of exposure studied.

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<sup>1</sup> The Navigation Guide rates the quality and strength of evidence of human and non-human evidence streams separately as “sufficient”, “limited”, “inadequate” or “evidence of lack of toxicity” and then these two ratings are combined to produce one of five possible statements about the overall strength of the evidence of a chemical’s reproductive/developmental toxicity. The methodology is adapted from the criteria used by the International Agency for Research on Cancer (IARC) to categorize the carcinogenicity of substances except as noted.

<sup>2</sup>Language for the definitions of the rating categories were adapted from descriptions of levels of certainty provided by the U.S. Preventive Services Task Force Levels of Certainty Regarding Net Benefit. <http://www.uspreventiveservicestaskforce.org/uspstf07/methods/benefit.htm>

<sup>3</sup> Language for the definitions of the rating categories were adapted from descriptions of levels of certainty provided by the U.S. Preventive Services Task Force Levels of Certainty Regarding Net Benefit.

**Economic analysis:** We combined quantitative assessment of exposure-response from our systematic review with incidence rates of asthma and annual values of asthma control to estimate the monetized benefits of avoiding asthma in EPA’s proposed rule on pressed wood products. We used the standard EPA approach of “willingness to pay” to calculate benefits, which measures the maximum amount of money that an individual is willing to pay to reduce the probability of an adverse health outcome assumed to be related to an environmental exposure [54].

To estimate the reduction in risk for asthma diagnosis, we used standardized risk estimates from our meta-analyses to estimate the reduction in risk per 1 ppb decrease in formaldehyde exposure. We assumed a Cox proportional hazard model so the number of reduced cases of asthma from a reduction in formaldehyde exposure is the exposed population times the baseline asthma risk times  $(1 - \exp(\ln(OR) * (\text{change in exposure})))$ . Using the tables for annual asthma benefits from EPA’s economic analysis for the proposed rule, we derived the exposure reduction for structures built new or renovated in the past eleven years. We used the change in indoor formaldehyde exposure for new and renovated homes at various ages (ranging from 0.124 to 3.390 ppb), the assumed baseline annual risk of asthma of 0.83%, used in EPA’s economic analysis and the estimated number of children aged 4-17 in 2017 in each housing type from the U.S. Census Bureau, with the proportional hazard model to estimate the reduced number of asthma cases associated with the proposed rule (26). We estimated the annual benefits for lowering formaldehyde emissions once the impacts of the reduction have reached steady-state (26).

To quantify the economic benefits of the reduction in asthma risk, we used estimates reported in the literature for the annual willingness to pay for full asthma control (inflated to 2018 dollars) from three studies. Full asthma control is equivalent to avoiding a case of asthma. Blomquist et

al. (27) used a two-stage contingent valuation survey of parents of asthmatic children aged 4–17 years and of adults to elicit the willingness to pay for a hypothetical drug that would control asthma symptoms [55]. The mean annual willingness to pay for children was \$3,434 and the mean annual value for adults was \$2,368. Blumenschein and Johannesson (28) used a contingent valuation bidding game to estimate asthma patients’ willingness to buy a new treatment that cured their asthma, finding a mean value of \$3,621. O’Conor and Blomquist (29) used a two-stage contingent valuation survey of adults with asthma to elicit the tradeoff between hypothetical medication of varying degrees of safety and efficacy and estimated a mean annual willingness to pay for full asthma control of \$2,413 using the value of statistical life. The average annual value of asthma control for adults across all three studies is \$2,801 and the annual value for children is \$3,434 from Blomquist et al. (27). The total value to an individual to not develop asthma at a given age is the present discounted value (3% discount rate) of the annual values over the life expectancy of that individual.

## Results

### *Included studies*

We retrieved a total of 4,821 unique records (4,482 from the initial search on March 15, 2016, an additional 254 from an updated search on March 15, 2018, and an additional 85 from an updated search on April 1, 2020), of which 150 ultimately met the inclusion criteria. Given the large number of diverse references identified, we decided to focus on studies where the asthma status of all study participants was measured (90 studies) (Fig 1). Our rationale was that these studies provided the most robust evidence for understanding the relationship between formaldehyde exposure and asthma because they all had quantitative measures of formaldehyde exposure,

participants for whom asthma status was known, and included asthmatics. Lists of all other studies are provided in the supplemental materials (S1 Results). Several included studies contained information from multiple records, such as a graduate thesis and a published manuscript following the cohort over time; the information from these records were combined into one record and listed as the main published manuscript. Four studies were identified that looked at similar outcomes from the same study population, so we combined these and focused on the publication for which the most relevant information was reported, supplementing with additional information from the related publications when necessary. We contacted corresponding study authors for 21 studies to request additional information missing from their published articles and received useable data from three.

Studies were further categorized separately into four combinations of study population and outcome (with some studies reporting on multiple populations/outcomes falling in multiple categories): 1) Child asthma diagnosis (n=24); 2) Child asthma exacerbation and symptoms (n=23); 3) Adult (general population and occupational) asthma diagnosis (n=20); Adult (general population and occupational) asthma exacerbation and symptoms (n=26). Presentation of results below include separate discussions for each of these four population/outcome categories. In particular, S99 Table presents study characteristics for included studies stratified by these group population/outcome categories.

### **Characteristics of included studies—Demographics**

The 90 included studies were published between 1969 and 2019, were conducted in 23 different countries (including 32% (n=29) within the U.S.), and included a range of 7 to 15,837 participants (Table 2, S99-S101 Table).

**Table 2.** Summary of included studies (n=90)

Study Characteristics	N (%)	Study Characteristics	N (%)
<b>Publication Year</b>		<b>Formaldehyde Exposure</b>	
<i>1969</i>	1 (1%)	<i>Measured exposure level</i>	82 (91%)
<i>1977</i>	1 (1%)	<i>Categorized exposure level</i>	8 (9%)
<i>1980-1989</i>	17 (19%)		
<i>1990-1999</i>	16 (18%)		
<i>2000-2009</i>	22 (24%)		
<i>2010-2019</i>	33 (37%)		
<b>Study Design</b>		<b>Study Participants*</b>	
<i>Case-control</i>	7 (8%)	<b>Child</b>	37 (41%)
<i>Nested case-control</i>	3 (3%)	<i>Asthma***</i>	24 (65%)
<i>Prospective cohort</i>	15 (17%)	<i>Asthma symptoms***</i>	23 (62%)
<i>Cohort</i>	2 (2%)	<i>Pulmonary function***</i>	5 (14%)
<i>Cross-sectional</i>	46 (51%)	<b>Adult (General and occupational)</b>	54 (60%)
<i>Cross-sectional and case-control</i>	2 (2%)	<i>Asthma***</i>	20 (37%)
<i>Non-randomized controlled trial</i>	6 (7%)	<i>Asthma symptoms***</i>	26 (48%)
<i>Randomized controlled trial</i>	5 (6%)	<i>Pulmonary function***</i>	35 (65%)
<i>Case report</i>	4 (4%)	<b>Mixed child and adults</b>	2 (2%)
<b>Sample Size</b>		<i>Asthma***</i>	1 (50%)
<i>0-50</i>	24 (26%)	<i>Asthma symptoms***</i>	2 (100%)
<i>51-100</i>	16 (18%)	<i>Pulmonary function***</i>	1 (50%)
<i>101-200</i>	12 (13%)	<b>Unspecified</b>	1 (1%)
<i>201-500</i>	14 (16%)	<i>Asthma symptoms***</i>	1 (100%)
<i>501-1000</i>	5 (6%)	<i>Pulmonary function***</i>	1 (100%)
<i>&gt;1000</i>	17 (19%)		
<i>Not reported</i>	2 (2%)		
<b>Country**</b>		<b>Population Source</b>	
<i>Egypt, Estonia, Indonesia, Iran, Japan, Malta, New Zealand, Poland, Russia, Thailand, United Arab Emirates</i>	1 (12%)	<b>General population (Adult and child)</b>	59 (66%)
<i>Canada, Finland, Portugal, Romania</i>	2 (9%)	<i>Asthma***</i>	33 (56%)
<i>Denmark</i>	3 (3%)	<i>Asthma symptoms***</i>	30 (51%)
<i>France</i>	4 (4%)	<i>Pulmonary function***</i>	18 (31%)
<i>Australia, China</i>	5 (11%)	<b>Occupational</b>	31 (34%)
<i>United Kingdom</i>	5 (6%)	<i>Asthma***</i>	11 (35%)
<i>South Korea</i>	7 (8%)	<i>Asthma symptoms***</i>	19 (61%)
<i>Sweden</i>	13 (14%)	<i>Pulmonary function***</i>	20 (65%)
<i>United States</i>	29 (32%)		

\*Studies that reported child versus adult data separately fell into both categories (as opposed to studies that reported collectively on children and adults mixed in the study population)—therefore total % is greater than 100%

\*\*Due to the variety of different countries represented, countries with similar counts have been grouped together for reporting. For instance, there are 5 studies located in Australia and 5 other studies located in China.

\*\*\*Many studies report multiple asthma outcomes—therefore total % is greater than 100%. Percentages are calculated out of the category sub-total; for instance, the percentage of asthma studies in children is calculated as 24/37.

Child studies were published relatively recently (1990-2016 for asthma diagnosis, 1984-2019 for asthma symptoms) whereas adult studies had a wider range of publication years including more older studies (Table 3). Almost half of child studies (11/24 for asthma diagnosis and 9/23 for asthma symptoms) had sample sizes greater than 1,000, whereas more adult studies had smaller sample sizes (13/20 for asthma diagnosis and 21/26 for asthma symptoms with sample size <500) (Table 3). Combined, child studies reported on a total of over 34,000 participants for asthma diagnosis and 32,000 participants for asthma symptoms. Adult studies reported on a total of over 8,000 participants for asthma diagnosis and 12,000 for asthma symptoms (S100 and S101 Table).

A little over half (51%, n=46) of the included studies were cross-sectional in study design, and the remainder were cohort (n=17), controlled trials (n=11), case-control (n=7), case reports (n=4), or of mixed study design (e.g., cross-sectional and case-control) (n=5) (Table 2). A similar trend in study design was observed in that the majority of studies in all four population/outcome combinations were of cross-sectional study design. Children studies reporting on asthma diagnosis were mostly cross-sectional (58%) and case-control (21%) whereas those reporting on asthma symptoms were mostly cross-sectional (52%) and prospective cohort (22%) (Table 3). Adult studies reporting on asthma diagnosis were mostly cross-sectional (80%) and cohort (15%), and similarly for those reporting on asthma symptoms (58% cross-sectional, 27% cohort) (Table 3).



## **Characteristics of included studies—Exposure measures**

Most studies (91%, n=82) reported association estimates between asthma outcomes and quantitative measurements of formaldehyde exposure. In the remainder of studies (n=8), although quantitative formaldehyde exposure measures were reported (leading to the study's inclusion), these estimates were not used by study authors directly to calculate association estimates, but rather they used categorized formaldehyde levels (i.e., high, medium, and low exposures) (Table 2). Formaldehyde levels were measured in school (n=14), home (n=30), work (n=16), vehicles (n=1), and outdoor environments (n=6), as well as using personal monitors (n=13) or given as experiment doses to healthy volunteers (n=12) (S100 and S101 Table). School formaldehyde measurements were used in 10 child asthma diagnosis and 10 child asthma symptom studies (and in no adult studies). Home formaldehyde measurements were used in 9 studies each for child asthma diagnosis and symptom studies and 7 studies each for adult asthma diagnosis and symptom studies. Work formaldehyde measurements were used in 6 adult asthma diagnosis studies and 11 adult symptom studies (and in no child studies). Outdoor exposure measurements were mostly used in child studies (3 studies of child asthma diagnosis, 4 for child asthma symptoms, and 2 for adult asthma diagnosis) whereas personal monitor measurements were mostly used in adult studies (5 studies of adult asthma diagnosis, 7 for adult asthma symptoms, and 2 each for child asthma diagnosis and asthma symptoms) (S100 and S101 Table).

**Table 3.** Study Characteristics, stratified by population health outcome group.

	<b>Child asthma n (%)</b>	<b>Child asthma symptoms n (%)</b>	<b>Adult asthma n (%)</b>	<b>Adult asthma symptoms n (%)</b>
<b>Publication Year</b>				
<i>1969</i>	0	0	0	1 (4%)
<i>1977</i>	0	0	0	1 (4%)
<i>1980-1989</i>	0	1 (4%)	2 (10%)	6 (23%)
<i>1990-1999</i>	3 (13%)	1 (4%)	4 (20%)	7 (27%)
<i>2000-2009</i>	7 (29%)	6 (26%)	5 (25%)	6 (23%)
<i>2010-2019</i>	14 (58%)	15 (65%)	9 (45%)	5 (19%)
<b>Study design</b>				
<i>Case-control</i>	5 (21%)	2 (9%)	1 (5%)	0
<i>Nested case-control</i>	2 (8%)	0	0	1 (4%)
<i>Prospective cohort</i>	2 (8%)	5 (22%)	2 (10%)	7 (27%)
<i>Cohort</i>	0	0	1 (5%)	0
<i>Cross-sectional</i>	14 (58%)	12 (52%)	16 (80%)	15 (58%)
<i>Cross-sectional and case-control</i>	1 (4%)	2 (9%)	0	0
<i>Non-randomized controlled trial</i>	0	1 (4%)	0	3 (11%)
<i>Randomized controlled trial</i>	0	1 (4%)	0	0
<i>Case report</i>	0	0	0	0
<b>Sample size</b>				
<i>0-50</i>	0	2 (9%)	1 (5%)	6 (23%)
<i>51-100</i>	3 (12%)	2 (9%)	5 (25%)	6 (23%)
<i>101-200</i>	6 (25%)	2 (9%)	1 (5%)	4 (15%)
<i>201-500</i>	1 (4%)	7 (30%)	6 (30%)	5 (19%)
<i>501-1000</i>	2 (8%)	0	4 (20%)	2 (8%)
<i>&gt;1000</i>	11 (46%)	9 (39%)	2 (10%)	3 (11%)
<i>Not reported</i>	1 (4%)	1 (4%)	1 (5%)	0

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## **Characteristics of included studies—Outcome measures**

Of the 90 total included studies, 41 evaluated asthma diagnosis outcomes (21 studies in children, 17 in adults, and 3 in both children and adults) and 48 evaluated asthma-related symptoms (22 studies in children, 25 in adults, and 1 in both children and adults). Asthma diagnosis was ascertained either by questionnaire (for instance, the International Study of asthma and Allergies in Childhood (ISAAC) (30)) medical records, or a physical examination (S100 and S101 Table).

Studies reported on a wide range of asthma-related outcomes, including current/ever asthma (n=33), asthma attacks (n=3), respiratory symptoms (n=9), wheeze (n=32), shortness of breath/dyspnea/breathlessness (n=17), chest tightness and pain (n=10), pulmonary bronchial hyperresponsiveness (n=1), asthma medication use (n=6), hospitalizations (n=2), emergency room visits (n=1), and results from asthma control (n=2), pulmonary function (n=35), and bronchial provocation tests (n=5) (S100 and S101 Table).

Studies reporting on child asthma symptoms reported most commonly on wheeze (n=16) and current/ever asthma (n=14); all other asthma-related outcomes listed were reported in  $\leq 5$  studies. No child studies reported on outcomes of chest tightness and pain, pulmonary bronchial hyperresponsiveness, or bronchial provocation (S100 and S101 Table).

Studies reporting on adult asthma symptoms reported most commonly on pulmonary function (n=28), current/ever asthma (n=19), wheeze (n=15), and shortness of breath /dyspnea/breathlessness (n=13), chest tightness and pain (n=9), and respiratory symptoms (n=6); all other asthma-related outcomes listed were reported in  $< 5$  studies. No adult studies reported on hospitalizations or emergency room visits (S100 and S101 Table).

## **Risk of bias assessment**

We rated risk of bias separately by outcome (asthma diagnosis versus symptoms exacerbation), but since our ratings were ultimately identical by outcome, risk of bias results are presented by study only. A limited number (n=3) of studies (31-33) reported results for mixed children/adult populations (aged 6-63 years); we excluded these studies from rating the quality of the evidence due to concerns with combining outcomes across a wide age range, given the unique issues in diagnosing and assessing asthma in children (especially at very young ages) compared to adults (34, 35). Overall, the majority of studies were rated “low” or “probably low” risk of bias across all domains (Fig 3, S1-3 Figs). We evaluated the risk of bias separately by each of the four-study population/health outcome groups.

### **Group 1: Childhood asthma diagnosis**

Overall, the majority of childhood asthma diagnosis studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included a small number of “probably high” ratings—source population (three “probably high” ratings), outcome assessment (four), incomplete outcome data (one), and exposure assessment (three). These were not consistent across any one study—i.e., only no study was rated “probably high” across all three of these domains. Generally, studies rated “probably high” were for similar reasons—i.e., for source population, three studies (39-41) reported high non-participation rates but failed to compare characteristics from study participants to those refusing to participate to explore potential selection bias. Similarly, for outcome assessment four studies (42-45) relied on self-reported outcomes by study participants (i.e., through a survey, self-administered spirometry, or daily diaries) but lacked follow-up by study investigators to

438 evaluate the validity of reported outcomes. Furthermore, two studies were rated “high” risk of  
439 bias for the other category—Huang et al. (46) due to cases having formaldehyde levels sampled  
440 more during the summer when formaldehyde exposures were lower versus controls who were  
441 sampled more during the summer when formaldehyde exposures were higher and Madureira et  
442 al. (47) who published a similar paper in a different journal the year prior with similar reported  
443 results.

444 The most problematic domain appeared to be confounding, where six studies were rated  
445 “probably high” and four were rated as “high.” Consistent with the instructions from our  
446 protocol, studies were rated as “probably high” for the confounding domain if studies evaluated  
447 some but not all of confounders pre-determined to be important (age, smoking status or exposure  
448 to environmental tobacco smoke, and socioeconomic status or parental education) and some but  
449 not all of other confounders pre-determined to be potentially important (race/ethnicity, sex,  
450 height, weight, BMI, obesity status, parental or family history of asthma, allergies, and additional  
451 environmental exposures), and were rated “high” if the study did not account for or evaluate  
452 many of the important or potentially important confounders. Studies most commonly adjusted  
453 for age, sex, and exposure to smoking. Adjusting for socioeconomic status was often  
454 accomplished through incorporating variables of family income or parent’s academic  
455 background. Few studies adjusted for environmental co-exposures; those that did included  
456 exposures to allergens (house dust mites or pets), indoor dampness or mold, proximity to traffic,  
457 or certain contaminants such as nitrogen dioxide or particulate matter.

458 Overall, review authors felt confident that the majority of children asthma diagnosis studies were  
459 rated predominantly “low” or “probably low” risk of bias, particularly for studies that were  
460 ultimately included in the meta-analysis. In particular, of the nine studies that were ultimately

included in the meta-analysis, four received “low” or “probably low” ratings across all risk of bias domains and accounted for 44% of the weight in estimating the overall association estimate. Studies generally that were rated “probably high” or “high” were not for reasons that were consistent across this body of evidence, and did not produce compelling reasons to downgrade the overall body of evidence as a result.

## **Group 2: Childhood asthma exacerbation and symptoms**

Overall, the majority of childhood asthma exacerbation and symptoms studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included a couple “probably high” or “high” ratings—blinding (one “probably high” rating), outcome assessment (two “probably high ratings), conflict of interest (one “probably high” and one “high” rating), and other (one “high” rating). These were not consistent across any one study—i.e., only no study was rated “probably high” or “high” across all domains. One study (48) was rated “probably high” for blinding because children and parents were recruited based on existence of airway respiratory symptoms and parents were responsible for deploying and retrieving in-home environmental samples and media as well as recording outcomes in diaries, thus making it unlikely that the reporting of outcomes was competed by someone without knowledge of exposure status. Two studies (45, 49) were rated as “probably high” for outcome assessment due to lack of physician confirmation or in-person interviews by study investigators to confirm asthma symptoms. One study (48) appeared to have a financial conflict of interest, with research grants provided from several private foundations from the pharmaceutical field (i.e., AstraZeneca). Another study (15) received a

“high” rating for the other domain because of an apparent typographical error in the reporting of results that could not be confirmed by authors upon personal communication.

A few other domains included a higher number of “probably high” or “high” ratings—source population (five “probably high” ratings), confounding (five “probably high” and two “high” ratings), incomplete outcome data (two “probably high” and one “high” ratings), and exposure assessment (three “probably high” ratings). Similar to the child asthma diagnosis studies, the most problematic risk of bias domain appeared to be confounding, where several studies did not adjust for or consider several of the important or potentially important adjustment factors outlined in our protocol. Studies most commonly adjusted for age, sex, and exposure to smoking. Adjusting for socioeconomic status was often accomplished through incorporating variables of family income or parent’s academic background. Few studies adjusted for environmental co-exposures; those that did included exposures to allergens (house dust mites or pets), indoor dampness or mold, proximity to traffic, or certain contaminants such as nitrogen dioxide or particulate matter.

Overall, review authors felt confident that the majority of children asthma diagnosis studies were rated predominantly “low” or “probably low” risk of bias, particularly for studies that were ultimately included in the meta-analysis. In particular, of the five studies that were ultimately included in the meta-analysis, three received “low” or “probably low” ratings across all risk of bias domains and accounted for 90% of the weight in estimating the overall association estimate for wheeze and 100% of the weight for shortness of breath. In particular, a number of studies were rated consistently as “low” or “probably low” risk of bias across all domains, increasing the review authors’ confidence that a sufficient body of evidence was available with minimal risk of bias to rate the overall body of evidence for this study population/health outcome group. Studies

that were rated “probably high” or “high” were not for reasons that were consistent across this body of evidence, and did not produce compelling reasons to downgrade the overall body of evidence as a result.

### **Group 3: Adult population asthma diagnosis**

Overall, the majority of adult asthma diagnosis studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included a one to two “probably high” or “high” ratings—outcome assessment (one “probably high”), confounding (two “high”), and conflict of interest (one “probably high”). These studies were rated higher risk of bias for lack of validation for self-reported outcomes (50), failure to adjust for or consider several of the important or potentially important adjustment factors outlined in our protocol (50, 51), or receiving funding from a private company without including a statement of the role of this company in influencing the study (52). Unlike for included children studies, confounding did not appear as problematic for the adult studies, likely because many studies were occupational and relied on either matching participants based on baseline characteristics or were pre- and post-experimental tests that used each individual subject as their own control.

Other domains included a higher number of “probably high” or “high” ratings—blinding (five “probably high”), exposure assessment (five “probably high”) and other (five “probably high”). These were not consistent across studies—only one study (53) received “probably high” ratings across four of these domains. This study (53) received high risk of bias ratings due to lacking detail on recruitment methods, failure to address blinding and the existing potential for bias if



investigators knew exposure status of participants, exposure measurements that were assessed by self-administered, proctored questionnaires that ultimately used work assignment as a proxy for high versus low exposure groups, and the existence of potential healthy worker effect. Blinding was more generally problematic for adult studies compared to those in children since many were occupational studies where study participants were likely already aware of their exposure and/or outcome status, and blinding was not a possibility. For the other domain, all five studies that received “probably high” ratings were occupational studies where potential for healthy worker effect either likely existed or was likely.

Overall, review authors felt confident that the majority of adult asthma diagnosis studies were rated predominantly “low” or “probably low” risk of bias. In particular, one study (54) received “low” risk of bias ratings across all domains, another study (33) was rated consistently as “low” or “probably low” risk of bias across all domains, and several studies (52, 55, 56) only received a “probably high” rating in one category, increasing the review author’s confidence that a sufficient body of evidence was available with minimal risk of bias to rate the overall body of evidence for this study population/health outcome group. Studies that were rated “probably high” or “high” were not for reasons that were consistent across this body of evidence, and did not produce compelling reasons to downgrade the overall body of evidence as a result.

#### **Group 4: Adult population asthma symptoms**

Overall, the majority of adult asthma diagnosis studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included one to two “probably high” or “high” ratings—source population

(one “probably high” and one “high”), confounding (two “probably high”), incomplete outcome data (one “high”), exposure assessment (two “probably high”), and conflict of interest (one “probably high”). These studies were rated higher risk of bias for lacking details regarding recruiting and inclusion/exclusion criteria (53, 57), failure to adjust for or consider several of the important or potentially important adjustment factors outlined in our protocol (58, 59), measuring exposure only for a portion of study participants (60), relying on self-reported outcomes by study participants but lacking follow-up for validation (53), or receiving funding from a private company without including a statement of the role of this company in influencing the study (52). Unlike for included children studies, confounding did not appear as problematic for the adult studies, likely because many studies were occupational and relied on either matching participants based on baseline characteristics or were pre- and post-experimental tests that used each individual subject as their own control.

A few other domains included a higher number of “probably high” or “high” ratings—blinding (five “probably high” and one “high”) and other (four “probably high” and one “other”). Similar to adult asthma diagnosis studies, blinding was generally more problematic for included occupational studies where study participants likely were already aware of their exposure and/or outcome status and blinding was not a possibility. For the other risk of bias domain, all five studies that received high risk of bias ratings were occupational studies where potential for healthy worker effect either likely existed or was likely (for instance, de Vos et al. (61) specifically excluded individuals with “unstable asthma, current acute or chronic respiratory illness, or any other chronic or severe illnesses,” thus likely leading to selection bias that favored healthier individuals.

Overall, review authors felt confident that the majority of adult asthma diagnosis studies were rated predominantly “low” or “probably low” risk of bias. In particular, one study (54) received “low” risk of bias ratings across all domains, another study (33) was rated consistently as “low” or “probably low” risk of bias across all domains, and a number of studies (52, 59, 62) only received a “probably high” rating in one category, increasing the review author’s confidence that a sufficient body of evidence was available with minimal risk of bias to rate the overall body of evidence for this study population/health outcome group. Studies that were rated “probably high” or “high” were not for reasons that were consistent across this body of evidence, and did not produce compelling reasons to downgrade the overall body of evidence as a result.

All adult studies with pulmonary measure outcomes received “probably high” or “high” ratings for the source population domain, each for slightly different reasons but all stemming from the fact that these were randomized controlled exposure trials with small sample sizes. For instance, Witek et al. (63) received a “probably high” rating because all 14 participants were a self-selected group of individuals responding to a recruitment advertisement (S86 Table). The ‘other’ risk of bias domain was used predominantly to capture healthy worker bias for included occupational studies—the phenomenon that occupations where chemical exposures occur often tend to avoid employment of older, younger, or ill individuals, and hence select out for susceptible individuals (36-38) (Figs 3-4). Studies considered in the meta-analysis or sensitivity analysis were generally high quality, with only “probably high” or “high” ratings in the domains blinding, outcome assessment, or confounding (Fig 4).

Occupational studies received higher risk of bias ratings for the domains of exposure assessment and ‘other’ compared to general population studies (S2 Fig), resulting from reliance on job exposure matrices to classify formaldehyde exposures (based solely on job titles without

measuring formaldehyde levels) or potential healthy worker effects. In contrast, over a third of general population studies received “probably high” or “high” ratings for the confounding domain from failure to account for the important confounding variables as outlined in our protocol. In contrast, many occupational studies incorporated matching study participants in the study design—for example matching exposed and unexposed by age, ethnicity, or job functions from similar socioeconomic status—and thus resulted in lower risk of bias ratings for confounding.

## **Statistical analysis**

### **Group 1: Childhood asthma diagnosis**

Of the 37 studies reporting on child populations, 24 reported on outcomes related to asthma diagnosis (i.e., children having been diagnosed by a physician as having asthma or based on self-reported asthma diagnosis). Nine of these studies were identified as combinable in a meta-analysis (41-44, 64-68) . The remaining studies could not be combined because they either categorized formaldehyde exposures or reported outcomes that could not be converted to an odds ratio (i.e., median formaldehyde exposures for those with asthma versus those without) .

Attempts to obtain estimates that could be standardized to an odds ratio from the study authors were unsuccessful.

One study, Rumchev et al. (2002) , was excluded from the meta-analysis because it included very young children (between 6 months and 3 years old), which could potentially have resulted in misclassification of infection-associated wheezing in young children as asthma (14), leading the NAS to conclude that this study should not be included in meta-analyses of formaldehyde and asthma. The estimate from another study in the meta-analysis, Krzyzanowski et al. (1990)

(44) was investigated in a sensitivity analysis removing the estimate because it was the only unadjusted estimate included.

One study considered for the meta-analysis measured incident asthma cases—Smedje et al. (2001) followed children over time to identify new asthma diagnoses (43). The remaining studies measured prevalent cases based on self-reported or physician ever having diagnosed with asthma, but because they all incorporated some requirement of current asthma symptoms (i.e., use of asthma medication or wheezing in the past 12 months) we decided that it was acceptable to combine prevalent and incident asthma cases. All studies measured indoor formaldehyde exposures, either at home or in school classrooms.

A meta-analysis combining effect estimates from the 9 children's asthma diagnosis studies using random effects modeling found an elevated OR (1.20) with 95% CI range above 1 (95% CI: [1.02, 1.41]), predicting an 20% increased odds of being diagnosed with asthma per 10- $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure (Fig 5). Removing the estimate from Krzyzanowski et al. (44), the only study reporting unadjusted estimates, slightly elevated the odds ratio (1.20 to 1.26) with a similar 95% CI [1.04, 1.53] (Table 4). (15)

**Table 4.** Meta-Analysis and Sensitivity Analysis of Childhood Asthma Diagnosis (N=9 studies) Pooled ORs and 95% CIs for random-effects models.

		Random-effects model	
	Number of studies	OR (95% CI) per 10- $\mu\text{g}/\text{m}^3$ increase	I <sup>2</sup> (p-value)
<b>Asthma Diagnosis</b>	9	1.20 (1.02, 1.41)	27% (p=0.2)
<b>Sensitivity Analysis</b>			
(-) Krzyzanowski et al. 1990	8	1.26 (1.04, 1.53)	31% (p=0.18)
(-) Kim et al. 2011	8	1.27 (1.06, 1.54)	28% (p=0.21)

(-) indicates removing a study from the meta-analysis for sensitivity analysis

The two most statistically influential studies in the meta-analysis were Krzyzanowski et al. (44) and Kim et al. (65). We removed these study to determine how this might impact the overall effect estimate. The impact of removing Krzyzanowski et al. (44) as discussed above as part of the sensitivity analysis was minimally impactful; removing Kim et al. (65) had a similar null effect, only slightly elevating the odds ratio (1.27) and changing the 95% CI [1.06, 1.54] (Table 4). (68).

We used a funnel plot and used Egger's test for small-study effects to statistically test for publication bias in the eight studies in the meta-analyses. Our funnel plots revealed no evidence of overall publication bias ( $p$ -value=0.35) (S98 Table; S4 Fig)—however, the small number of studies (<10) might result in no indication of publication bias when in fact it might exist.

We also investigated the potential impact of a new or unpublished hypothetical study necessary to alter the results of the meta-analysis. In making this calculation, we assumed that the new hypothetical study would have a standard error equal to the smallest in our group of studies—0.14 for children asthma diagnosis (44, 66, 68). We determined that a new study would be required to have an estimate of OR=0.97, 95% CI: [0.74, 1.27] to change the 95% confidence interval of the meta-analysis overlapping one. We judged the existence of a study with such a result to be possible, given that this association estimate and confidence interval was within the range of other included studies, but not likely given that this point estimate would be in the opposite direction of all studies included in the meta-analysis.

To shift our meta-analysis to have an overall association estimate just below zero (i.e., increases in formaldehyde exposures would be associated with decreases in asthma outcomes) would require a new study reporting an OR=0.05, 95% CI: [0.04, 0.07]. We judged the existence of a

well-conducted study with such a result to be very unlikely, given that this association estimate and confidence interval was considerably outside the range of the estimates from almost every included study.

Data that could not be combined into a meta-analysis were visually depicted on scatterplots when possible. The categorical odds and risk ratios (n=14), formaldehyde levels (n=6), and asthma prevalence (n=5) were visually displayed for consideration in rating the overall body of evidence (S5-7 Figs). Several studies with estimates included in the meta-analysis also reported secondary estimates (for instance, outcomes of self-reported current asthma) that were included on these scatterplots. Overall, these data appeared generally consistent with each other (i.e., increasing exposure to formaldehyde associated with increasing odds/risk ratios, asthma prevalence, and asthma status), and with the results of the meta-analysis. The secondary estimates from studies included in the meta-analysis (42, 43, 64-68) were also within the range of studies included in the meta-analysis (S5 Fig). Additional studies further supported the meta-analysis estimate; for instance, Tavernier et al. (39) reported odds ratios for self-reported asthma confirmed by physician by tertile of formaldehyde exposure, with an estimate of 1.22 (95%CI: [0.49, 3.07]) comparing the third to first tertile (S5 Fig). Several studies reported associations with asthma and categorical exposures to formaldehyde, which allowed review authors to evaluate the potential for a dose-response relationship. Rumchev et al. (15) reported a consistent relationship between increasing exposure (across four exposure groups ranging from 10 to >50  $\mu\text{g}/\text{m}^3$ ) and increased odds for asthma diagnosis. However, other studies did not illustrate a similar relationship—for instance, Annesi Maesano (69) reported increased odds (OR=1.1, 95% CI [0.87, 1.38]) for self-reported asthma comparing the medium to low tertile for formaldehyde exposure, but decreased odds (OR=0.9, 95% CI: [0.76, 1.08]) comparing the high to low tertile (S5 Fig). Similarly, some

studies reporting asthma prevalence with increasing formaldehyde exposure supported a dose-response relationship with increasing exposure (40, 54, 70, 71) whereas others did not (44) (S6 Fig). Review authors concluded that these data supported the meta-analysis results and association between formaldehyde exposure and asthma diagnosis, but that there was limited evidence supporting a dose-response relationship.

## **Group 2: Childhood asthma exacerbation and symptoms**

Twenty-three studies reported symptoms related to asthma—asthma attack, wheeze, or breathlessness/shortness of breath (Table 3). Of these, six studies (40, 41, 64-67) were initially identified as potentially combinable in a meta-analysis for the association between indoor formaldehyde exposures and wheeze or daytime shortness of breath. One study reported a crude OR estimate for respiratory symptoms including wheeze and shortness of breath, but did not provide an estimate of variability (i.e., confidence limits or standard error) and therefore could not be included in the meta-analysis. Efforts to contact study authors to obtain this information were unsuccessful. Thus, we ultimately combined five studies in our meta-analysis (Fig 6). Several studies provided multiple effect estimates to the meta-analysis—e.g., Kim et al. reported effect estimates for wheeze symptoms and daytime breathlessness associated with indoor formaldehyde exposure. Overall, separate combined effects for wheeze and shortness of breath were similar and the combined effects were moderate (OR=1.08, 95% CI: [0.92, 1.28]) (Fig 6). Due to the small number of studies contributing estimates to the meta-analysis, we did not conduct a statistical analysis of potential publication bias.



Since the meta-analysis association estimate 95% lower bound CI was below 1, we only explored the sensitivity of shifting our meta-analysis to have an overall association estimate just below zero (i.e., such that increases in formaldehyde exposures would be associated with decreases in asthma outcomes). We assumed that the new hypothetical study would have a standard error equal to the smallest in our group of studies, 0.12 (66). We concluded this would require a new study reporting an OR=0.84, 95% CI: [0.66, 1.017]. We judged the existence of a well-conducted study with such a result to be possible, given that this association estimate and confidence interval was within the range and overlapped with most of the included studies and aligned with the estimate of one study in particular.

The categorical odds ratios (n=10), formaldehyde levels by asthma status (n=2), and symptom scores (n=1) were visually displayed on the same figure for consideration in rating the overall body of evidence (S8-9 Figs). Most studies identified elevated association estimates from exposures to formaldehyde, but lower 95% CI was below 1. Several studies (41, 64, 65, 67, 72) reported on different asthma symptoms (asthma attacks, asthma symptoms, or wheeze) per 1  $\mu\text{g}/\text{m}^3$  formaldehyde exposure and reported consistent estimates of positive odds ratios ranging from 0.96-1.2 (S7 Fig). Several studies (48, 73-75) reported on categorical formaldehyde exposures, but did not demonstrate a consistent dose-response relationship (S7 Fig). For instance, Raaschou-Nielsen (48) reported on wheezing symptom across five exposure categories (ranging from 0 to  $>25.6 \mu\text{g}/\text{m}^3$  formaldehyde) with increased odds ratios across three groups (OR=1.11, 1.21, 1.4) but a negative odds ratio for the highest exposure group (OR=0.67). Review authors concluded that these data supported the meta-analysis results and association between formaldehyde exposure and asthma symptoms, but that there was limited evidence supporting a dose-response relationship.

Four studies reported pulmonary function measures in children, but because two studies reported on peak expiratory flow rates (PEFR) and two others reported on forced expiratory volume in one second (FEV<sub>1</sub>) and forced vital capacity (FVC), a comparison between such a small number of studies was determined not to be useful.

### **Group 3: Adult population asthma diagnosis**

Seventeen total studies included outcomes of whether subjects had been previously diagnosed by a physician with having asthma (most commonly ascertained through use of a self-reported questionnaire (n=11) or through medical records or physician examination (n=6)). None of these 17 studies reported sufficient data to evaluate outcomes with respect to a continuous 10-µg/m<sup>3</sup> increase in formaldehyde. Three studies reported results for at least two measured exposure categories; the majority of studies reported exposures categorically, such as exposed versus unexposed or by job category. Due to the small number of studies and high amount of heterogeneity in key study characteristics, the studies were not amenable to meta-analysis to combine effect estimates. We identified three studies reporting similar ranges of exposure categories to assess for a dose-response relationship for asthma diagnosis and identified a positive trend (Fig 7), although review authors noted the small number of studies and limited dose groups included.

The formaldehyde levels by categorical odds ratios (n=4) and asthma prevalence (n=4) were visually displayed for consideration in rating the overall body of evidence (S10-11 Figs). Although the categorical odds ratios varied considerably in how formaldehyde exposures were categorized (i.e., high vs. low, exposed to newly painted dwelling/workplace vs. not, occupations

exposed to formaldehyde vs. not, etc.), there was a consistent increase in odds of asthma diagnosis with increased category of exposure (S10 Figure). For instance, Billionnet et al. (56) reported an increased odds (OR=1.43) for those in the high exposure group ( $\geq 28.03 \mu\text{g}/\text{m}^3$ ) compared to those in the low exposure group ( $< 28.03 \mu\text{g}/\text{m}^3$ ). However, review authors noted a limitation with Billionnet et al. (56) in that no estimates of statistical confidence (i.e., standard error, 95% confidence interval) were reported with these estimates. Although all four studies reported increased odds with increased category of exposure, only Herbert et al. (76) reported a statistically significant increase (comparing exposed versus non-exposed occupational groups). Similarly, the scatterplot of prevalence data by formaldehyde exposure categories demonstrated a similar pattern of supporting increases in asthma prevalence with increasing formaldehyde exposure (S11 Figure). For instance, Elshaer and Mahmoud (50) reported dramatic prevalence increases in exposed occupational workers for asthma (53.3%) versus non-exposed workers.

Considering the overall evidence, review authors concluded that there did appear to be evidence supporting a relationship between increasing formaldehyde exposure and asthma diagnosis, although the number of studies was low and the variety of exposure categories made it challenging to easily compare across different studies.

#### **Group 4: Adult population asthma symptoms**

Twenty studies reported on asthma-related symptoms—i.e., asthma attack, wheeze, or breathlessness/shortness of breath (Table 3). All studies reported categorical formaldehyde exposures and therefore could not be combined in a meta-analysis. The categorical odds ratios

(n=5), asthma prevalence (n=4), and symptom score (n=1) were visually displayed for consideration in rating the overall body of evidence (S9-11 Figs). The symptom score study and most studies reporting odds ratios documented increased risk of symptoms with exposure to formaldehyde, with several reporting statistically significant findings (S9-10 Figs). For instance, Herbert et al. (76) reported a statistically significant increase in asthma symptoms (attacks of wheeze) comparing exposed versus non-exposed occupational groups. Asthma prevalence estimates were generally greater with increased exposure to formaldehyde, but these studies lacked confidence intervals around the point estimates (S11 Fig). However, there were few studies reporting on prevalence outcomes and results were not consistent across studies. For instance, Kilburn, Seidman, and Warshaw (53) reported consistent increases in asthma symptom prevalence in an occupational setting with increases in the hours of exposure to formaldehyde but Thetkathuek et al. (58) reported an inconsistent relationship with wheeze symptoms across low, moderate, and high formaldehyde exposure groups (lower prevalence in the moderate exposure group compared to low exposure group).

There were also 32 total studies that reported on pulmonary lung measures in adults. We decided to focus on studies reporting associations between formaldehyde exposure and Forced Expiratory Volume in 1 second (FEV1) outcomes, following recommendations from National Institute of Health (NIH) to use FEV1 as a supplemental outcome related to asthma exacerbation. Most studies reported FEV1 outcomes (n=27), but not all reported associations with formaldehyde exposures. Several studies reported FEV1 percentage changes comparing to baseline values (either to a comparator group or standardized values, for instance standardized predicted values based on age, height and gender published by the American Thoracic Society (77)—we decided not to plot these on the same figure due to lack of comparability across studies using different

comparisons or standardized values. Of the 27 studies, 7 reported associations between FEV1 measured values with formaldehyde exposures. These were visually displayed for consideration in rating the overall body of evidence (S12 Fig). Four of the studies reported confidence intervals for association estimates that overlapped between exposed and comparator groups but did not find consistent changes in FEV1 with formaldehyde exposures (i.e., comparing formaldehyde-exposed participants to controls, two studies reported decreases in FEV1 while the other two reported increases .

**Considering the overall evidence, review authors concluded that there did appear to be evidence supporting a relationship between increasing formaldehyde exposure and asthma symptoms, although the number of studies was low and the variety of exposure categories made it challenging to easily compare across different studies. Rating quality and strength of the body of evidence**

Based on the comparison of the body of evidence to pre-specified criteria in our protocol (S4 Methods), the review authors concluded that there was “moderate” quality for the body of evidence for each of the four-study population/health outcome groups (Table 5). Review authors did not apply any upgrades (for large magnitude of effect, dose-response relationship, or confounding that minimizes effect) or downgrades (for risk of bias, indirectness, inconsistency, imprecision, or publication bias) to criteria across the body of evidence, which led to the final rating of “moderate”.

812 **Table 5.** Summary of rating quality and strength of the human evidence, by population/  
outcome group

813 **A. Children asthma diagnosis**

Category	Downgrades	Rationale
<b>Initial Rating of human evidence = Moderate</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. The confounding domain appeared to be most frequently problematic due to failure to adjust for the important confounders outlined in the protocol; however, a number of included studies were rated as “low” or “probably low” risk of bias, including several studies ultimately included in the meta-analysis. Review authors concluded that this did not appear to warrant downgrading for risk of bias across all studies.
Indirectness	0	The population, exposure, and outcome were all directly related to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.
Inconsistency	0	Studies included in the meta-analysis have similar point estimates with overlap among the confidence intervals. Effect estimates across studies were mostly positive (showing increased risk). Estimates from the meta-analysis indicate that statistical heterogeneity was moderate, but not statistically significant ( $I^2=46.5\%$ , $p\text{-value}=0.06$ ).
Imprecision	0	No concern regarding the imprecision in effect estimates across studies.

Publication bias	0	Could not rule out publication bias, but there is no affirmative evidence of its existence—in particular, funnel plots revealed no evidence of overall publication bias (p-value = 0.35).
	Upgrades	
Large magnitude of effect	0	The overall effect size from the meta-analysis is small but precise. Authors concluded there was not enough evidence to warrant upgrading for this domain.
Dose-response	0	Results from the meta-analysis between formaldehyde exposure and child asthma diagnosis, which assumes a linear dose-response relationship, appeared to support the existence of an association of increasing response with increased dose. However, there was limited data to statistically evaluate whether there was a dose-response relationship, primarily due to the small number of studies and the heterogeneity in reporting of effect estimates. Review authors did not believe that results from the meta-analysis were sufficient to warrant upgrading the body of evidence for evidence of a dose-response relationship.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more

		well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.
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815 **B. Children asthma exacerbation and symptoms**

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. The confounding domain appeared to be most consistently problematic due to failure to adjust for the important confounders outlined in the protocol; however, a number of included studies were rated as “low” or “probably low” risk of bias, including several studies ultimately included in the meta-analysis. Review authors concluded that this did not warrant downgrading for risk of bias across all studies.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.



Inconsistency	0	Effect estimates across studies were consistent across the body of evidence, in particular as seen by the categorical odds ratios and the prevalence data visual scatterplots.
Imprecision	0	No concern regarding the imprecision in effect estimates across studies.
Publication bias	0	Number of studies included were too small (i.e., <10) for a statistical evaluation of potential publication bias. Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Studies that found positive relationship between exposure and outcome were interpreted as a minimal magnitude of effect; insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Results from the meta-analysis between formaldehyde exposure and children asthma exacerbation and symptoms, which assumes a linear dose-response relationship, appeared to support the existence of an association of increasing response with increased dose.

		However, there was not enough evidence to statistically evaluate existence of a dose-response relationship, primarily due to the small number of studies and the heterogeneity in reporting of effect estimates. Review authors did not believe that results from the meta-analysis were sufficient to warrant upgrading the body of evidence for evidence of a dose-response relationship.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.

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823 **C. Adult asthma diagnosis**

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. Most studies were rated “low” risk of bias across most domains with only one or two “probably high” ratings, with the exception of only a few studies. Occupational studies received “probably high” ratings for blinding, exposure assessment and “other” domains, but review authors did not feel this warranted a downgrade to the overall body of evidence.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.
Inconsistency	0	Effect estimates across studies were generally consistent across the body of evidence; heterogeneity likely explained by the differing study designs, and data demonstrate a tendency towards increased asthma diagnosis and therefore would not warrant a downgrade for this domain.

Imprecision	0	Confidence intervals appeared to be somewhat wide, but review authors did not feel there was enough evidence to warrant downgrading for this domain.
Publication bias	0	Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Studies that found positive relationship between exposure and outcome were interpreted as a minimal magnitude of effect; insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Data supported the existence of a dose-response relationship, but review authors did not feel it was strong enough to warrant an upgrade for this domain.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.

<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.
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#### 826 **D. Adult asthma exacerbation and symptoms**

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear problematic across all studies. Occupational studies appeared to have probably high ratings for blinding, exposure assessment and other domains, but review authors did not feel this warranted a downgrade to the overall body of evidence.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.

Inconsistency	0	Effect estimates across studies were generally consistent across the body of evidence; heterogeneity likely explained by other factors, and data demonstrate a tendency towards increased asthma exacerbation and symptoms and therefore would not warrant a downgrade for this domain.
Imprecision	0	Confidence intervals appeared to be somewhat wide, but review authors did not feel there was enough evidence to warrant downgrading for this domain.
Publication bias	0	Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Some studies illustrate large impact, but this is not consistent across the studies and so review authors concluded there was insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Some data supported the existence of a dose-response relationship, but review authors did not feel it was strong enough to warrant an upgrade for this domain.

Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.

828

829 Review authors noted that risk of bias limitations did exist across each of the study  
830 population/health outcome groups. Concerns were generally limited to the domains of blinding,  
831 confounding, exposure assessment, and “other” (the latter being predominantly limited to  
832 occupational studies that were rated for potential healthy worker bias) domains. For instance,  
833 several child asthma diagnosis studies were rated “high” (n=4) or “probably high” (n=6) for  
834 confounding due to the failure to adjust for the important confounders outlined in our pre-  
835 published protocol. A number of other studies were rated as “probably high” for various other  
836 domains (source population, outcome assessment, incomplete outcome, and exposure  
837 assessment). However, review authors felt that overall a sufficient number of studies were rated  
838 “low” or “probably low” risk of bias across all domains, in particular several studies ultimately  
839 included in the meta-analysis (i.e., (65-68)) and review authors concluded that these limitations

840 did not rise to the level of a downgrade, in accordance with the instructions outlined in the  
841 protocol (<http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD 42016038766).  
842 Review authors came to similar conclusions in evaluating the risk of bias for each of the other  
843 three study population/health outcome groups. In particular, review authors noted that many of  
844 the “high” or “probably high” risk of bias ratings were assigned to a select subgroup of studies  
845 (i.e., those with issues stemming from small sample sizes or occupational studies due to healthy  
846 worker bias concerns) but the remaining included studies did not suffer from such limitations and  
847 had minimal risk of bias concerns. Review authors did not apply downgrades to the evidence for  
848 the other domains for any of the study population/health outcome groups because there lacked  
849 sufficient evidence supporting existence of indirectness, inconsistency, imprecision, or  
850 publication bias.

851 Review authors also did not apply any upgrade factors for any of the study population/health  
852 outcome groups. For child asthma diagnosis and child asthma symptoms evidence, although we  
853 were able to conduct a meta-analysis that supported an association between increasing response  
854 with increased dose (based on an assumption of model linearity), there were too few studies to  
855 support the formal analysis of a dose-response relationship. Furthermore, as discussed above  
856 visual inspections of scatterplots of data not able to be combined in a meta-analysis provided  
857 mixed evidence supporting the existence of a consistent dose-response relationship. Review  
858 authors concluded that overall this evidence was not sufficient enough to warrant upgrading the  
859 evidence for dose-response relationship,

860 Ultimately, review authors rated the overall strength of evidence as “sufficient” for each of the  
861 four outcome groups (Table 5), based on: a) “moderate” quality of the body of evidence; b)  
862 direction of the association (i.e., consistent evidence of a positive association between



formaldehyde exposure and outcomes of either asthma diagnosis or exacerbation in symptoms, in both adults and children; c) confidence in the association with multiple well-conducted studies (i.e., several studies were prospective cohort studies that were of “low” or “probably low” risk of bias overall; and positive and/or statistically significant overall estimates of association from the combination of similar studies in a meta-analysis (Figs 5-6).

### **Economic analysis**

We valued the outcome of avoiding a case of asthma in children, as it had the strongest support from well-conducted combinable studies with minimal risk of bias concerns. We used the OR estimate of 1.20 per 10  $\mu\text{g}/\text{m}^3$  (95% CI: [1.02, 1.41]) (Fig 5) based on the random effects meta-analysis model for asthma diagnosis in children from indoor formaldehyde exposure.

We rescaled this OR to estimate the reduction in risk per 1 ppb decrease in formaldehyde exposure (OR of 1.02265 per 1 ppb change in formaldehyde). We estimated that EPA’s proposed rule on pressed wood products would have resulted in 2,805 fewer asthma cases annually once the impacts of the reduction has reached steady-state.

We estimated a willingness to pay for a treatment that would eliminate asthma of \$75,024, which translates into total economic benefits for asthma reduction from EPA’s rule of approximately \$210 million annually across all children in the U.S. over a 30-year period. (Table 6).

**Table 6.** Cases reduced and willingness to pay for a reduction in Formaldehyde exposure implied by the proposed EPA rule on pressed wood products (once the impacts of the rule have reached steady-state)

	<b>Exposure reduction (ppb)</b>	<b>Individuals Affected</b>	<b>Cases avoided</b>	<b>Benefits with WTP = \$75,024</b>
Structure age 0-1	-3.390085	599,822	364.0	\$27,311,030
Structure age 1-2	-2.178523	599,822	237.1	\$17,787,752
Structure age 2-3	-1.408503	599,822	154.6	\$11,599,437
Structure age 3-4	-0.926590	599,822	102.3	\$7,671,854
Structure age 4-5	-0.624871	599,822	69.2	\$5,191,181
Structure age 5-6	-0.431493	599,822	47.9	\$3,592,426
Structure age 6-7	-0.306329	599,822	34.0	\$2,553,938
Structure age 7-8	-0.229512	599,822	25.5	\$1,915,142
Structure age 8-9	-0.181581	599,822	20.2	\$1,516,000
Structure age 9-10	-0.152852	599,822	17.0	\$1,276,554
Structure age 10-11	-0.133711	599,822	14.9	\$1,116,939
0-1 years post-ren.	-2.363858	1,306,316	559.1	\$41,948,116
1-2 years post-ren.	-1.525697	1,306,316	364.3	\$27,327,908
2-3 years post-ren.	-1.002335	1,306,316	240.7	\$18,058,604
3-4 years post-ren.	-0.668362	1,306,316	161.1	\$12,086,556
4-5 years post-ren.	-0.458218	1,306,316	110.7	\$8,305,820
5-6 years post-ren.	-0.323412	1,306,316	78.3	\$5,871,128
6-7 years post-ren.	-0.239982	1,306,316	58.1	\$4,360,639
7-8 years post-ren.	-0.189089	1,306,316	45.8	\$3,437,825
8-9 years post-ren.	-0.156738	1,306,316	38.0	\$2,850,684
9-10 years post-ren.	-0.133647	1,306,316	32.4	\$2,431,347
10-11 years post-ren.	-0.124415	1,306,316	30.2	\$2,263,624
<b>Total</b>		<b>20,967,514</b>	<b>2,805</b>	<b>\$210,474,503</b>

## Discussion

We found “sufficient” evidence of an association between exposure to formaldehyde and asthma diagnosis and asthma symptoms in children and adults. The definition of “sufficient” was predefined in our protocol (Table 1). Our review had several strengths, including that we used

892 the Navigation Guide systematic review methodology, which specifically accounted for the  
893 weaknesses identified by the NAS in the IRIS formaldehyde assessment, i.e., explicit and  
894 transparent study selection and evaluation criteria, including exclusion of a study in which  
895 asthma may have been misclassified . Moreover, our review was based only on studies where the  
896 asthma status of participants was known and which reported quantitative measures of  
897 formaldehyde exposure, and our methods accounted for several considerations of causality as  
898 part of the evaluation, specifically, our PECO statement limited included evidence based on  
899 temporality criteria and the evaluation of the strength and quality of evidence incorporated  
900 considerations of strength, consistency, and biological gradient.

901 We retrieved six self-identified “systematic reviews” of formaldehyde and asthma conducted  
902 between 2011 and 2015 in the literature search for our review (78-83),. Of the three reviews with  
903 findings consistent with our review, two conducted a meta-analysis of the data (78, 83) and the  
904 third cited the McGwin et al. meta-analysis (82). The three reviews which did not find  
905 compelling evidence for an association between asthma and formaldehyde exposure did not  
906 conduct a meta-analysis, and there was a wide disparity in the number and type of papers  
907 included in these reviews. Specifically, our review included 22, 17, 17, and 20 studies on child  
908 asthma diagnosis, child asthma symptoms, adult asthma diagnosis and adult asthma symptoms,  
909 respectively.

910 In contrast, Patelarou et al. (81) included 2 studies on formaldehyde and asthma and wheezing in  
911 children up to 5 years old; Baur et al. (80) included 8 studies on formaldehyde and asthma in  
912 occupational settings; and Nurmatov et al. (79) included 17 studies on formaldehyde and asthma  
913 etiology, 1 study on formaldehyde and asthma exacerbation, and 14 studies on asthma etiology  
914 and exacerbation (among which the authors found a positive association between formaldehyde

and wheezing in young children on the basis of a “well-conducted, low-risk of bias” randomized controlled trial, which was consistent with our findings). While none of these six self-described systematic reviews fully met all of the criteria for a systematic review as specified in the Literature Review Appraisal Toolkit (<http://policyfromscience.com/lrat/about-the-lra-toolkit/>), the transparency of their methods allowed for better understanding the discrepant results.

In 2016, EPA published its final rule to regulating formaldehyde in pressed wood products as well as household and other finished goods. The regulations set by this final rule did not consider the benefits of preventing asthma; estimated annualized benefits (from avoided incidence of eye irritation and nasopharyngeal cancer outcomes only) ranged from \$64-186 million per year. Our results show that using assumptions consistent with EPA’s proposed rule [24], the final rule excluded approximately \$210 million annually in total economic benefits associated with 2,805 fewer asthma cases. Furthermore, these benefits were calculated based on the willingness to pay for asthma control, and could potentially represent an underestimate of the true valuation of one’s willingness to pay for avoiding an asthma diagnosis in the first place.

Formaldehyde is a high-production volume chemical ubiquitous in homes, communities, and workplaces and asthma is a prevalent and costly chronic health outcome. While our results show that the association between exposure to formaldehyde and asthma is robust, the effect estimate is relatively small, i.e., an 8% increase in children’s asthma diagnosis per 10-fold increase in exposure. These findings underscore that preventing relatively “low” risks brings “high” health benefits when exposures are ubiquitous. Our results demonstrate that benefits analyses that inform regulatory action need to account for all relevant health outcomes as to not do so could underestimate benefits.

Formaldehyde is a well-defined respiratory irritant and has been identified as a known respiratory carcinogen in humans. There are several proposed mechanisms supporting the role of formaldehyde exposure in asthma development. Formaldehyde is a small molecule with the ability to conjugate with large serum protein molecules such as albumin. This can provoke the formation of IgE antibodies, leading to degranulation of mast cells with allergic asthma response (84). As a small molecule, formaldehyde may bind to the amino group in proteins acquiring antigenic capacities, causing immune response with the formation of specific antibodies and triggering a local mast cell response (85). Formaldehyde is also readily absorbed into respiratory tract tissue, where it may increase T-helper cell type 2 (Th2) mediated inflammatory response and lead to cytokine mediators (3g., IL4, IL5, and IL13) release, epithelial mucous cell metaplasia, and airway recruitment of eosinophils (84). Lastly, formaldehyde may also react with the thiol group and interfere *S*-nitrosogluthathione function, triggering an airway response(86). Our systematic review had several limitations. First, we focused on evaluating only studies where asthma status of all study participants was measured and excluded other studies, namely studies relevant to our PECO statement but where the asthma status of participants was unknown or there were no asthmatics included, reported no quantitative measured of formaldehyde, or non-English studies. This likely would not influence our findings as studies with missing assessments for exposure and outcome are of poorer quality. We also did not independently evaluate temporality of exposure and note that included cross-sectional studies where exposures were measured concurrent to asthma outcomes may not accurately represent exposures occurring prior to asthma outcomes. Second, while our review documented an association between formaldehyde exposure and increased childhood asthma diagnosis, symptoms and exacerbation, it did not address whether

960 formaldehyde directly causes childhood asthma, or rather, is a trigger for childhood asthma.

961 Asthma is a complex chronic disease that can be challenging to diagnose accurately and for  
962 which symptoms are apparent only when there is a trigger. The trigger does not necessarily cause  
963 ‘asthma’, but will cause an ‘asthma flare up’, which helps lead to the diagnosis. Thus, it is  
964 possible that formaldehyde is a ‘trigger’ for a child who is yet to be diagnosed with asthma or it  
965 can be that formaldehyde exposure leads to the development of asthma. It is impossible to  
966 determine this unless without a human interventional study.

967 Third, key estimates utilized in the economic analysis (i.e., baseline asthma risk and willingness  
968 to pay for asthma reduction) were U.S.-based estimates. Thus, the economic evaluation and  
969 monetized value of benefits from formaldehyde exposure reduction may not be directly  
970 applicable in other global settings. However, inclusion of studies in the systematic review was  
971 not limited by geographic location and we ultimately included studies from a variety of countries  
972 (Sweden, France, Australia, China, South Korea, Denmark, Finland, Poland, Portugal, United  
973 Kingdom, New Zealand, Romania, Russia, Japan, Indonesia, Thailand, Iran, the United Arab  
974 Emirates), with the first five countries in addition to the U.S. contributing to the meta-analysis  
975 estimates. Thus, results and conclusions from the systematic review are likely relevant to  
976 international settings and results from the economic analyses may be modified with geographic-  
977 specific estimates to gauge potential economic benefits in international settings.

978 Our results underscore that the inability to combine studies in a meta-analysis due to lack of  
979 reporting in published studies is a major challenge for systematic reviews in environmental  
980 health specifically, and for environmental health decision-making more broadly. The association  
981 between asthma and formaldehyde exposure is well-studied, as demonstrated by the large  
982 number of epidemiology studies. However, even with a large number of included studies, there

were multiple limitations to the studies that restricted our ability to combine estimates into a meta-analysis—for instance, if studies only reported categorical formaldehyde exposures or if they did not report odds ratio or relative risk estimates. Visual scatterplots of data assisted review authors' evaluation of the consistency and interpretation of data results, but many studies did not provide data amenable to extraction for scatterplots. For example, of the 26 adult (occupational and general population) asthma diagnosis studies, only 17 studies included outcome data on a physician diagnosis; none of these 17 studies reported sufficient data to evaluate outcomes with respect to a continuous increase in formaldehyde; and few studies reported results for at least two measured exposure categories. Hence, quantitative data from 9 papers were not reported in a manner that they could be objectively incorporated (i.e., not using the author's conclusions but rather just by extracting the data) into this review. Checklists such as Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational human studies to guide the reporting of elements necessary to describe studies comprehensively and transparently may assist with these efforts and have already been incorporated into the publication process of several high-impact journals. Furthermore, journal reviews and editors may contribute to addressing this issue by requesting increased reporting or open-access of quantitative data in a format conducive to future data analyses. Conducting a systematic review prior to the development and initiation of a new study could help design efficient studies that are intended to build on existing data and address research gaps intentionally to support future systematic reviews, risk assessment, and timely decision-making on environmental chemicals.

## Conclusion

The review authors concluded that there was “sufficient” evidence supporting an association between childhood and adult exposures to formaldehyde with asthma diagnosis and symptoms. Although studies supported modest associations (our meta-analysis for childhood exposure to formaldehyde with asthma symptoms resulted in a combined OR=1.08), ubiquitous exposure to formaldehyde can result in potentially large impacts to population health. Our economic analysis identified annual benefits of 2,805 fewer asthma cases in the U.S.; the total economic benefit for asthma reduction from U.S. EPA’s rule would be approximately \$210 million annually. Thus, excluding asthma health outcomes when conducting regulatory benefit-cost analysis can underestimate the true population benefits and lead to decisions that are not fully protective of the public. Although these economic estimates are specific to the U.S., the inclusion of studies from broad geographic range indicate that results and conclusions from the systematic review are likely relevant to international settings. Our findings document that preventing formaldehyde exposure in adults and children could reduce the occurrence and impacts of a serious, chronic disease and provide significant health and economic benefits.

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1041

## 1042 **Figure Legends**

### 1043 **Figure 1.**

1044 PRISMA flowchart showing the literature search and screening process for studies relevant to  
1045 formaldehyde exposure and asthma outcomes. Our search was not limited by language or  
1046 publication date (search was conducted up until April 1, 2020). The search terms used for each  
1047 database are provided in S1-7 Tables.

1048

### 1049 **Figure 2.**

1050 Timeline of U.S. Environmental Protection Agency (EPA) action on formaldehyde from  
1051 September 1990-December 2016, highlighting Integration Risk Information System (IRIS) final  
1052 assessments releases, reassessments, internal and external reviews, and final rules issued.

1053

### 1054 **Figure 3.**

1055 Cumulative risk of bias ratings (low, probably low, probably high, or high) across all human  
1056 studies included in our systematic review of formaldehyde exposure and asthma outcomes. Risk  
1057 of bias designations for individual studies are assigned by review authors according to criteria  
1058 provided in S3 Methods (Risk of Bias instructions) and the justifications for each study are  
1059 provided in S8-95 Tables.

1060

### 1061 **Figure 4.**

1062 Risk of bias ratings (low, probably low, probably high, or high) for all human studies included in  
1063 our systematic review of formaldehyde exposure and asthma outcomes, organized by study  
1064 population (children or adult) and outcome (asthma diagnosis, asthma symptoms, or pulmonary  
1065 measures). Risk of bias designations for individual studies are assigned by review authors

according to criteria provided in S3 Methods (Risk of Bias instructions) and the justifications for each study are provided in S8-95 Tables.

**Figure 5.**

Meta-analysis of human studies (n=9 studies, including a total of 9,049 children) for formaldehyde exposure for asthma diagnosis assessed in children up to 15 years of age: reported effect estimates and 95% confidence interval (CI) from individual studies (inverse-variance weighted, represented by size of rectangle) and overall pooled estimate from random effects (RE) model per 10  $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure. Heterogeneity statistics:  $I^2 = 27.2\%$ ,  $p=0.202$ .

**Figure 6.**

Meta-analysis of human studies (n=5 studies, including a total of 7,662 children) for formaldehyde exposure for asthma symptoms (wheeze and shortness of breath) assessed in children up to 15 years of age: reported effect estimates and 95% confidence interval (CI) from individual studies (inverse-variance weighted, represented by size of rectangle) and overall pooled estimate from random effects (RE) model per 10  $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure. Heterogeneity statistics:  $I^2 = 0\%$ ,  $p=0.899$ .

**Figure 7.**

Dose-response relationship (n=3 studies, including a total of 3,600 adult participants) between formaldehyde exposure ( $\mu\text{g}/\text{m}^3$ ) and relative risk of asthma diagnosis in adults. Dose-response data from Yeatts et al. 2012 (63), Billonnet et al. 2011 (92), Matsunaga et al. 2008 (93). Data were modeled with random-effects log linear models with restricted cubic splines mixed effects methods with exchangeable covariance structure of multivariable-adjusted relative risks. Lines with long dashes represent the 95% confidence interval (CI) bounds for the fitted nonlinear trend (solid line). Symbols (triangles, circles, and squares) represent point estimates.

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## Supplemental Information

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 1418 **S89 Table. Characteristics of Yoon and Lin 2014**  
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- 1453    **S1 Results. List of included studies not considered**
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Figure 1. PRISMA Diagram of included/excluded studies

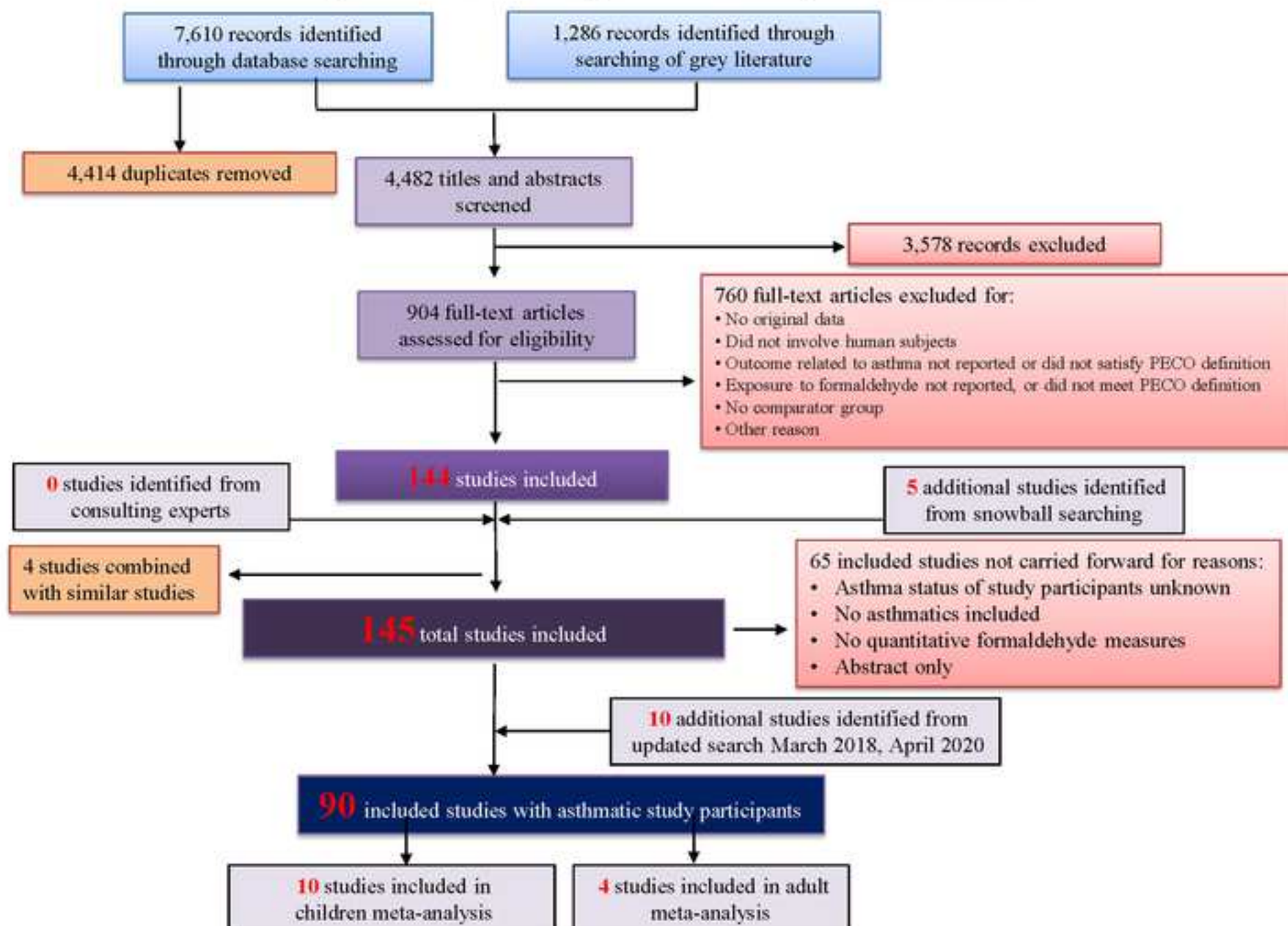


Figure 2. Timeline of EPA formaldehyde assessment

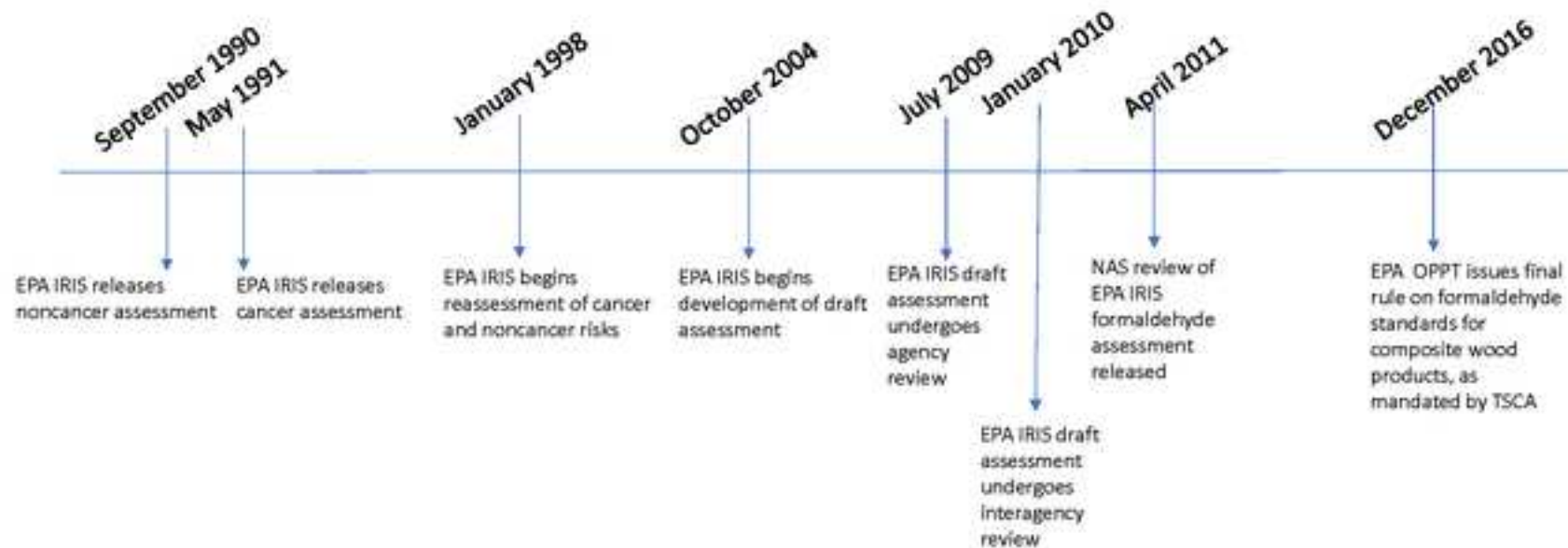
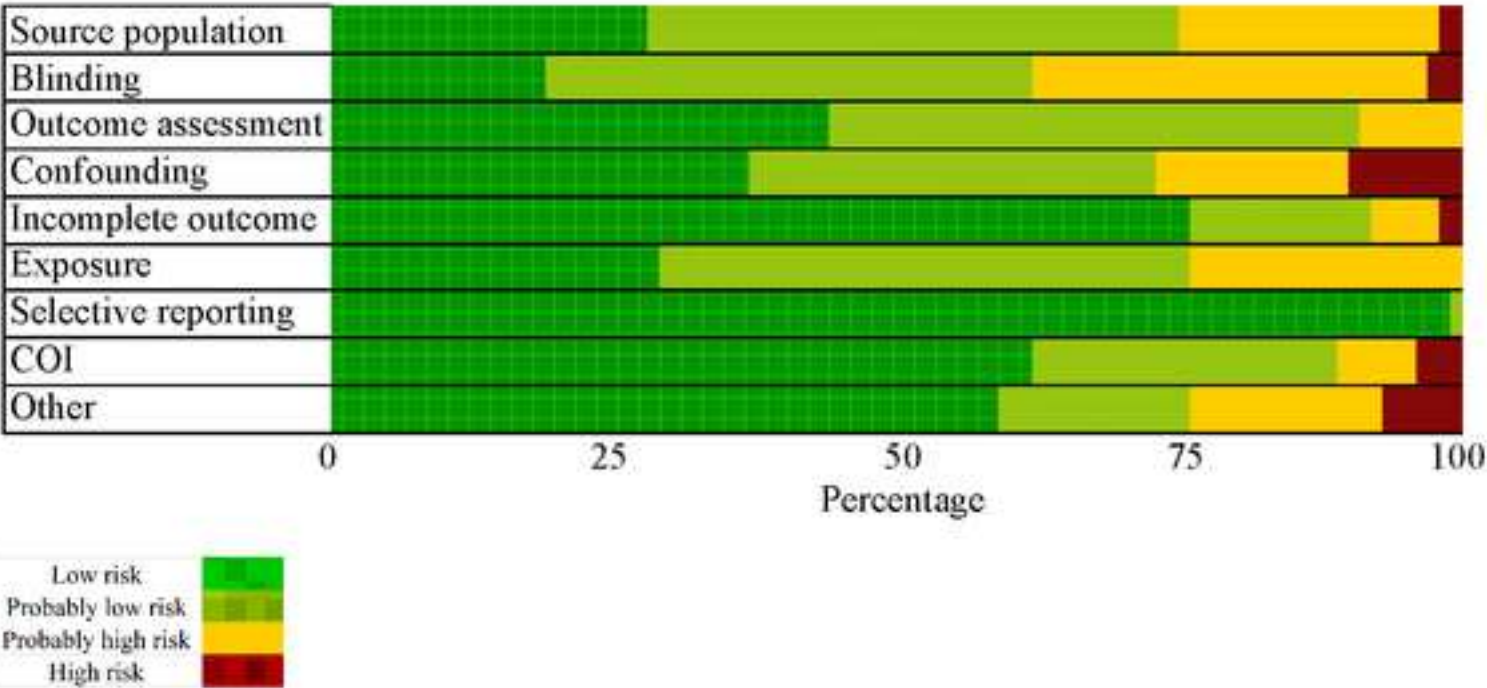


Figure 3. Risk of bias ratings across all included studies



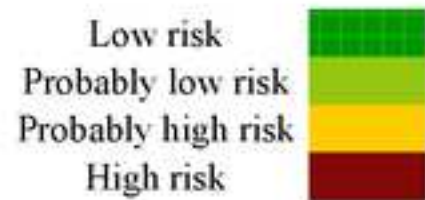


Figure 4. Risk of bias ratings for each included study, by study population and outcome

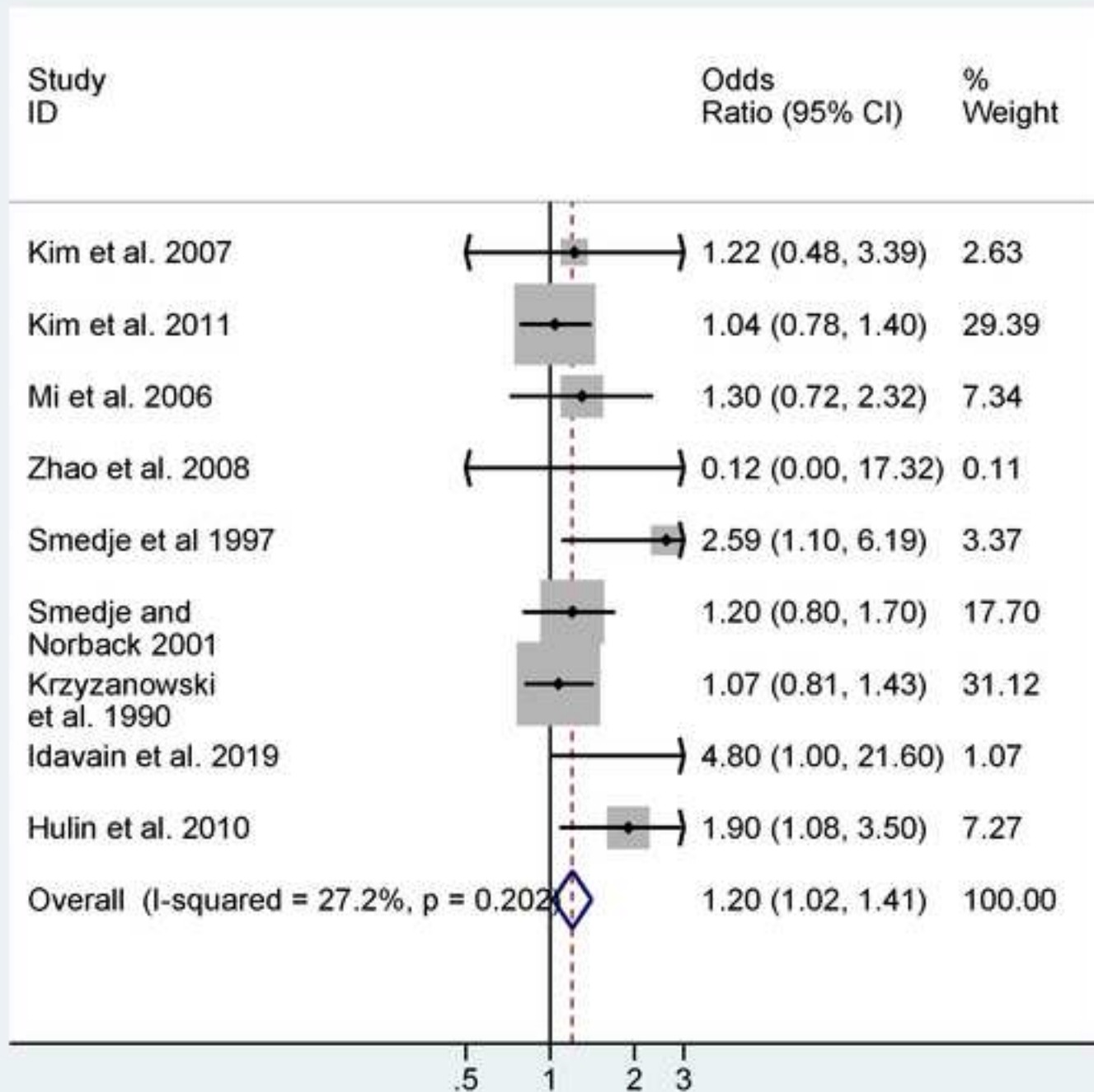
Population/Outcome	Study	Source population	Blinding	Outcome assessment	Confounding	Incomplete outcome	Exposure assessment	Selective reporting	COE	Other
Children/yellow flagstone	Zhou et al. 2013									
	McIntyre et al. 2010									
	Chen et al. 2008*									
	Adams-McCormick et al. 2012									
	Ford et al. 2012									
	Form and Lee 2014									
	Sheng et al. 2013									
	Charalabous et al. 2014									
	Wells et al. 2010*									
	Kim et al. 2011*									
	Kryzhanovskiy et al. 1990*									
	Frisk et al. 1996									
	Vanman et al. 2006									
	Seung et al. 2011									
	Shenoy and Nathani 2000									
	Chen et al. 1999									
	Pollock 1998									
	Shenoy and Nathani 2001*									
	Marston et al. 2010									
	Se et al. 2008*									
	Kim et al. 2007*									
Children/yellow flagstone	Shen et al. 2012									
	Sheng et al. 2014									
	McIntyre et al. 2010									
	Shen et al. 2009									
	Shen et al. 2011*									
	Wells et al. 2010*									
	McIntyre et al. 2010									
	Shen et al. 2009									
	Shen et al. 2011*									
	Shen et al. 2011									
Children/yellow flagstone	Wells et al. 2010*									
	McIntyre et al. 2010									
	Ford et al. 2012									
	McIntyre et al. 2010									
	Carson et al. 2013									
	Chen et al. 2008*									
	Se et al. 2008*									
	Shen et al. 2009									
	Kim et al. 2011*									
	Wells et al. 2010									
	Form et al. 2009									
	Seung et al. 2011									
	Commenet et al. 2011									
	Chen et al. 1999									
	Shenoy and Nathani 2000									
Children/yellow flagstone	Form et al. 2013									
	Marston et al. 2010*									
	Wells et al. 2010									
	Kim et al. 2007*									
	Shen et al. 2009									
	Shen et al. 2011									
	Shen et al. 2011									
	Shen et al. 2011									
	Shen et al. 2011									
	Shen et al. 2011									
Children/yellow flagstone	Carson et al. 2013									
	McIntyre et al. 2010									
	Kryzhanovskiy et al. 1990									
	Wells et al. 2010									
	Shen et al. 2011*									
Children/yellow flagstone	Carson et al. 2013									
	McIntyre et al. 2010									
	Kryzhanovskiy et al. 1990									
	Wells et al. 2010									
	Shen et al. 2011*									



	Teates et al. 2012									
	Bilconnet et al. 2011									
	Wendlandt et al. 1999									
	Matsunaga et al. 2007									
	Kilburn, Erdman, and Warsaw 1985									
	Stehbert et al. 1994									
	Jacobson et al. 2009									
	Kilburn et al. 1985									
	Linaver et al. 2017									
	Pourmahabadian et al. 2006									
Adult/infants symptoms	Chai et al. 2013									
	Teates et al. 2012									
	Uba et al. 1989									
	Wendlandt et al. 1999									
	Prisk et al. 2006									
	Kilburn, Erdman, and Warsaw 1985									
	Stehbert et al. 1994									
	Jacobson et al. 2009									
	Kilburn et al. 1985									
	De Vos et al. 2009									
	Thothathoor et al. 2016									
	Gauder et al. 1987									
Adult/pulmonary measures	Quirk-Smith et al. 1989									
	Wolke, Jr et al. 1986									
	Kilburn et al. 1985									
	Shoppard et al. 1984									
	Wolke, Jr et al. 1987									
	Gauder et al. 1987									

\*Studies included in meta-analysis

Low risk	
Probably low risk	
Probably high risk	
High risk	



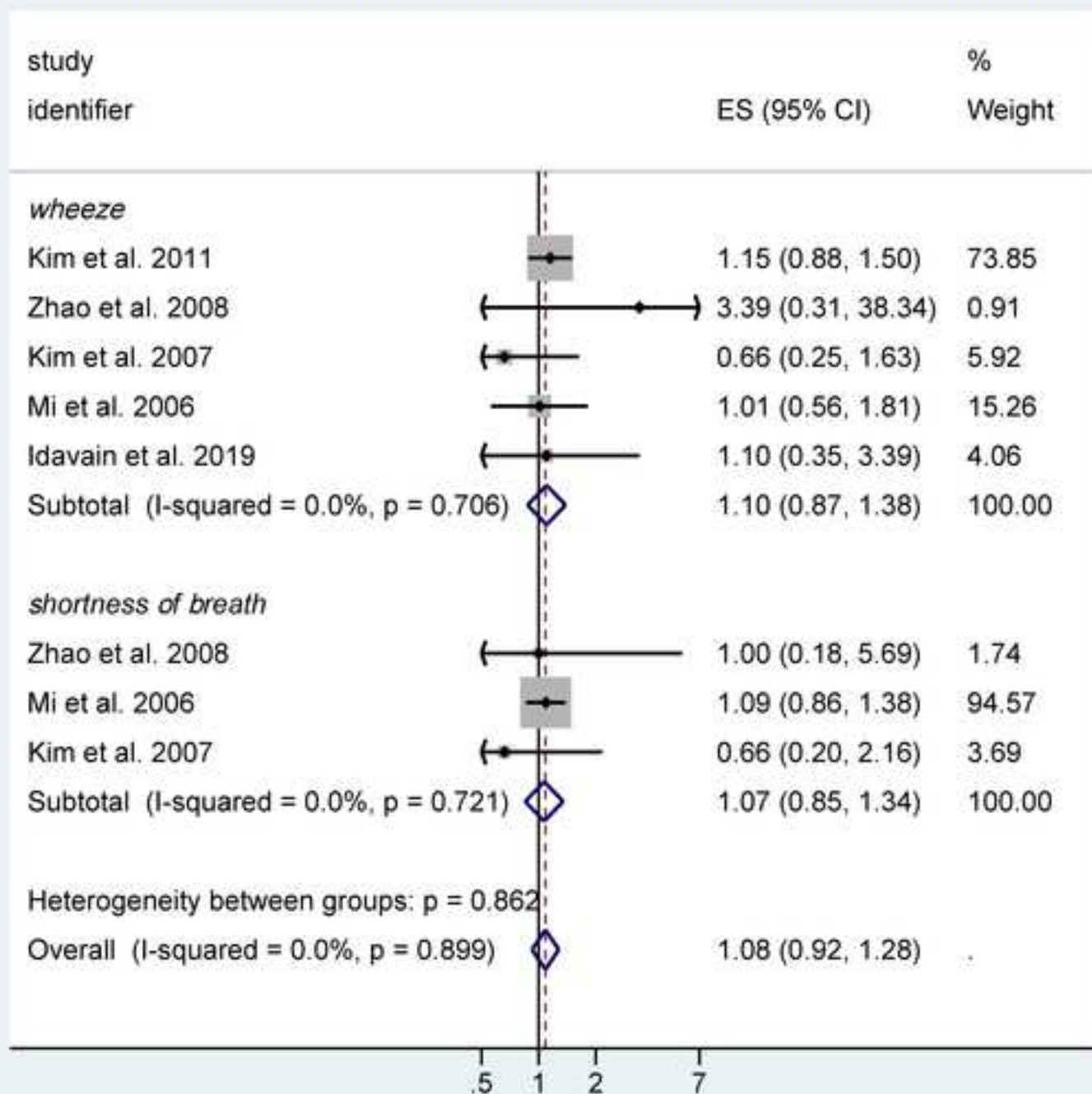
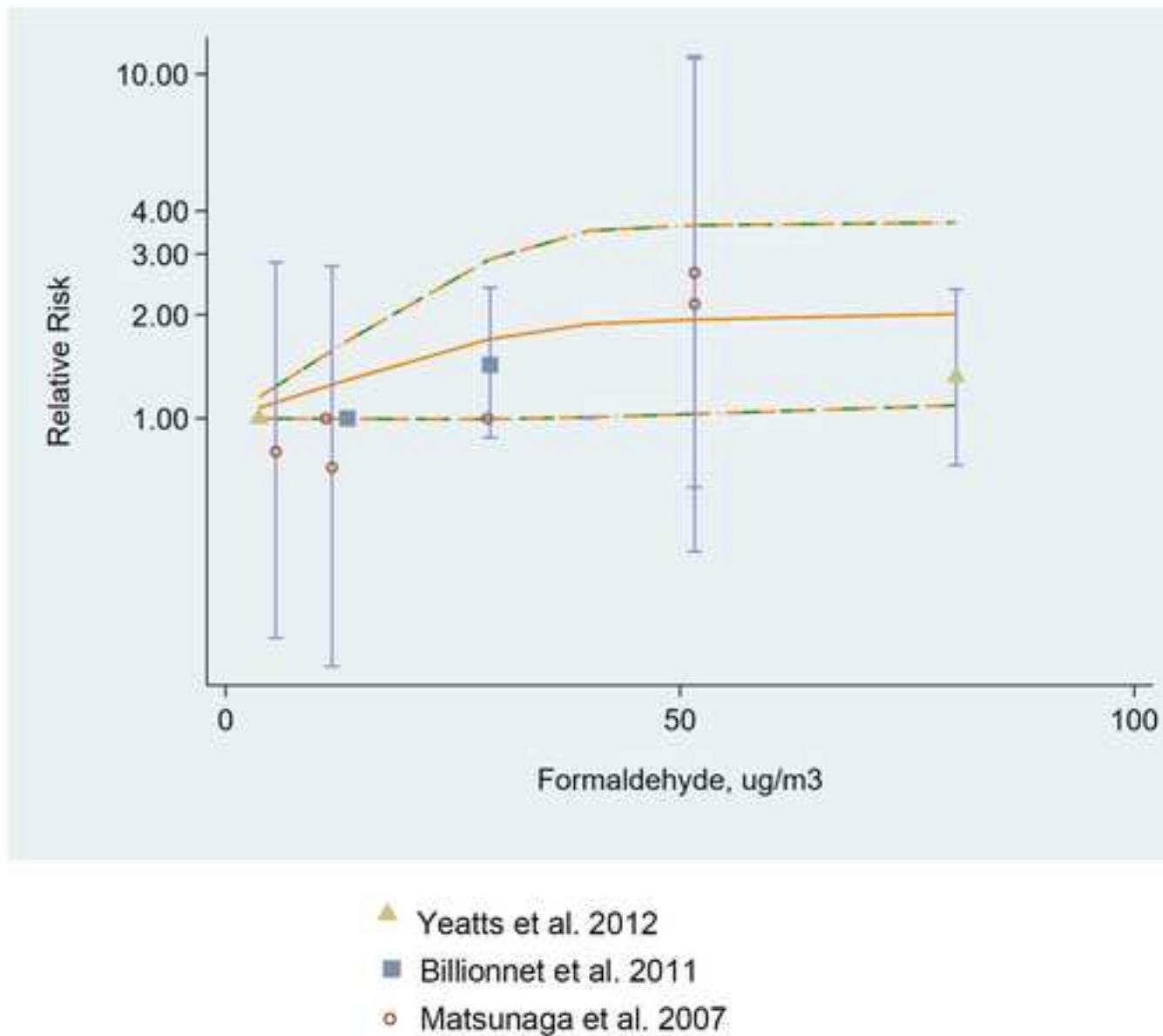


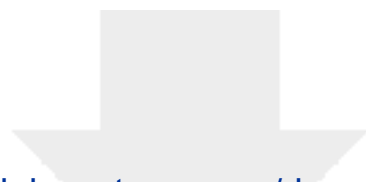
Figure 7. Dose-response relationship between formaldehyde exposure and relative risk of asthma in adults





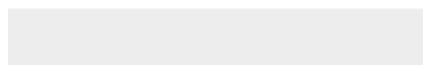
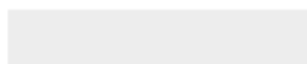
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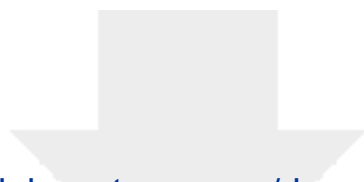




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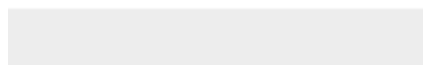
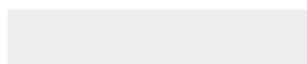
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**Supporting Information**  
**Supplemental Materials\_tracked.docx**



**Exposure to Formaldehyde and Asthma Outcomes: A Systematic Review, Meta-Analysis, and Economic Assessment**

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**Competing financial interests:**

The authors declare no competing financial interests.

**Disclaimer:**

The views expressed in this paper are those of the authors and do not necessarily reflect the view or policies of the U.S. Environmental Protection Agency. MDC is a member of the United States Preventative Services Task Force (USPSTF). This article does not necessarily represent the views and policies of the USPSTF.



## Abstract

**Background:** Every major federal regulation in the United States requires an economic analysis estimating its benefits and costs. Benefit-cost analyses related to regulations on formaldehyde exposure have not included asthma in part due to lack of clarity in the strength of the evidence.

**Objectives:** 1) To conduct a systematic review of evidence regarding human exposure to formaldehyde and diagnosis, signs, symptoms, exacerbations, or other measures of asthma in humans; and 2) quantify the annual economic benefit for decreases in formaldehyde exposure.

**Methods:** We developed and registered a protocol in PROSPERO (Record ID #38766, CRD 42016038766). We conducted a comprehensive search of articles published up to April 1, 2020. We evaluated potential risk of bias for included studies, identified a subset of studies to combine in a meta-analysis, and rated the overall quality and strength of the evidence. We quantified economics benefit to children from a decrease in formaldehyde exposure using assumptions consistent with EPA's proposed formaldehyde rule.

**Results:** We screened 4,821 total references and identified 150 human studies that met inclusion criteria; of these, we focused on 90 studies reporting asthma status of all participants with quantified measures of formaldehyde directly relevant to our study question. Ten studies were combinable in a meta-analysis for childhood asthma diagnosis and five combinable for exacerbation of childhood asthma (wheezing and shortness of breath). Studies had low to probably-low risk of bias across most domains. A 10- $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure was associated with increased childhood asthma diagnosis (OR=1.20, 95% CI: [1.02, 1.41]). We also found a positive association with exacerbation of childhood asthma (OR=1.08, 95% CI:

[0.92, 1.28]). The overall quality and strength of the evidence was rated as “moderate” quality and “sufficient” for asthma diagnosis and asthma symptom exacerbation in both children and adults. We estimated that EPA’s proposed rule on pressed wood products would result in 2,805 fewer asthma cases and total economic benefit of \$210 million annually.

**Conclusion:** We concluded there was “sufficient evidence of toxicity” for associations between exposure to formaldehyde and asthma diagnosis and asthma symptoms in both children and adults. Our research documented that when exposures are ubiquitous, excluding health outcomes from benefit-cost analysis can underestimate the true benefits to health from environmental regulations.

## Introduction

Formaldehyde exposure is ubiquitous and occurs in homes, communities, and workplaces. Formaldehyde is a high-volume production chemical with numerous industrial and commercial uses as a solution, disinfectant, preservative or to produce industrial resins used to manufacture adhesives and binders in wood, paper, and other products. It is present in many household products, such as foam insulation, cleaning and personal care products, pressed wood products such as particleboard and plywood, and as a result is a common indoor air pollutant found in virtually all homes and buildings (1-9). Homes are impacted by off-gassing of formaldehyde from new housing materials, with availability and rates of ventilation having minimal impact on exposure levels (10).

In particular, formaldehyde is an environmental justice and affordable housing concern. Lower-income communities are disproportionately at risk of exposure to formaldehyde and resulting health effects from pressed wood products in homes built with less costly building materials.

69 Formaldehyde exposure extends beyond residential homes—for instance, formaldehyde has been  
70 measured at levels exceeding exposure limits in childcare settings in California. Workplace  
71 exposure to formaldehyde occurs in a wide variety of industries and occupations, such as in the  
72 manufacture or production of formaldehyde or formaldehyde-based products or during  
73 firefighting, embalming, carpentry, and pathology lab work.

74 Asthma is a complex disease caused by chronic inflammation of the airways that results in  
75 episodic airway hyper responsiveness, excessive mucous secretion, and airway obstruction.  
76 Exposure to formaldehyde occurs primarily through inhalation and also as a respiratory contact  
77 irritant (11). The relationship between exposure to formaldehyde and asthma has been actively  
78 under evaluation by government agencies for the last few decades (12-14). A substantial amount  
79 of research exploring relationships between formaldehyde exposure and exacerbation of asthma  
80 has been conducted, but few systematic reviews (with a pre-established protocol, systematic  
81 literature search, pre-defined criteria for evaluating studies and categories to assess the strength  
82 of evidence) are available providing a comprehensive overview of the evidence.

83 The U.S. Environmental Protection Agency (EPA) released its review of formaldehyde health  
84 risks in its Integrated Risk Information System (IRIS) assessment in 1990, initiated a  
85 reassessment in 1998, and released a draft report in 2010, which included a review of the asthma  
86 health outcome (Fig 2). A review of the draft assessment by the National Academy of Sciences  
87 (NAS) highlighted many methodological limitations of the IRIS process, such as EPA's study  
88 selection and evaluation criteria that led to the advancement of one study (15) with potential  
89 misclassification of infection-associated wheezing in young children as asthma (14). EPA's  
90 conclusion of a causal relationship between formaldehyde exposure and asthma incidence and

91 subsequent derivation of a candidate Reference Concentration (RfC) was ultimately challenged  
92 by the NAS committee (14).

93 In 2010, Congress required EPA to issue a rule on pressed wood products and emissions of  
94 formaldehyde; ultimately EPA issued a final rule on formaldehyde in 2016 (Fig 2). EPA  
95 conducted a benefits cost analysis of this rule under an Executive Order that requires every  
96 significant regulation in the U.S. be accompanied by an economic analysis of the benefits and  
97 costs of implementation. EPA initially included asthma in the benefit-cost analysis for the  
98 proposed rule; however, asthma was removed from the analysis after interagency review. In the  
99 U.S., asthma affects approximately 23 million people, including 6 million children (16),  
100 impacting approximately 8% of both children and adults (17). The omission of asthma from the  
101 benefit-cost analysis could significantly underestimate the true value of regulating formaldehyde  
102 in pressed wood products.

103 To assess the evidence of formaldehyde's contribution to asthma outcomes, we conducted a  
104 systematic review of human studies to answer the question of whether exposure to formaldehyde  
105 is associated with diagnosis, signs, symptoms, exacerbation, or other measures of asthma in  
106 humans. We used results from the quantitative evaluation of the evidence to estimate the benefits  
107 of the reduction in asthma cases implied by the proposed EPA rule on pressed wood products.

## 108 **Methods**

109 We applied the Navigation Guide systematic review methodology, a systematic and transparent  
110 method for synthesizing the available scientific evidence designed specifically for environmental  
111 exposures (18, 19). The method is based on Cochrane and GRADE methods (20, 21) and  
112 includes the same elements (protocol development, risk of bias evaluation, evidence evaluation,

113 etc.). However, one main difference is that this method accounts for differences in evidence and  
114 decision context inherent to environmental health assessments, i.e., the reliance on human  
115 observational studies in the absence of randomized controlled trials (RCTs), and the fact that  
116 population exposure to exogenous chemicals precedes evidence of their safety.

### 117 ***Protocol***

118 We developed a protocol prior to initiating the review and registered it in PROSPERO in May  
119 2016 (<http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD 42016038766).

### 120 ***Study question***

121 Our systematic review objective was to answer the question: “Is exposure to formaldehyde  
122 associated with the diagnosis, signs, symptoms, exacerbation, or other measures of asthma in  
123 humans?”

124 The “Participants,” “Exposure,” “Comparator” and “Outcomes” (PECO) statement is briefly  
125 outlined below, with additional specifics available in the protocol.

126 **Participants:** Humans.

127 **Exposure:** Any indoor or outdoor sources of airborne inhalation exposure to formaldehyde,  
128 including but not limited to occupational, outdoor ambient, indoor household settings, and/or  
129 exposure to household products that occurred prior or concurrent to health outcome.

130 **Comparator:** Humans exposed to lower levels of formaldehyde than the more highly exposed  
131 humans.

132 **Outcomes:** Any of the following asthma-related outcomes: diagnosis of asthma, asthma signs or  
133 symptoms, asthma exacerbation, or indirect measures of asthma.

134 ***Data Sources***

135 We searched the databases PubMed, ISI Web of Science, Biosis Previews, Embase, Google  
136 Scholar, and Toxline from the inception of each database up to April 1, 2020 using the search  
137 terms in S1-5 Tables. We did not limit our search by language or initial publication date. We  
138 used the Medical Subject Headings (MeSH) database to compile synonyms for formaldehyde  
139 and asthma-related outcomes. Our search terms and search strategy were developed by two  
140 librarians trained in systematic review methodology (LS, EW). We also supplemented these  
141 results by searching toxicological and grey literature databases (S6-7 Tables), consulting with  
142 subject matter experts, and hand-searching references by reviewing reference lists of included  
143 studies and review papers on the topic as well as searching for references that cited included  
144 studies (“snowball searching”).

145 ***Study Selection***

146 We included studies that contained original data from human studies that measured or reported  
147 formaldehyde exposure prior to evaluating the health outcome. We screened references for  
148 inclusion using structured forms in DistillerSR (Evidence Partners; available at:  
149 <http://www.systematic-review.net>). Two of four possible reviewers (EK, ND, AP, HV)  
150 independently reviewed titles and abstracts of each reference to determine eligibility in a non-  
151 random assignment (to ensure that the same two authors did not always screen the same  
152 references). In the event that an abstract was missing or there were discrepancies between the  
153 two reviewers, the default was to move the reference forward for full text review. Two of the  
154 same four reviewers (EK, ND, AP, HV) then independently performed a full-text review to  
155 evaluate inclusion criteria of each reference not excluded by title/abstract screening. An

156 additional reviewer (JL) screened five percent of the titles/abstracts and full-texts for quality  
157 assurance.

158 We excluded studies if any one of the following criteria was met: 1) the report did not contain  
159 original data; 2) the article did not involve human subjects; 3) there was no report of  
160 formaldehyde exposure; 4) there was no report of diagnosis of asthma, asthma signs or  
161 symptoms, asthma exacerbation, or indirect measures of asthma (such as daily use of inhaler); or  
162 5) there was no comparator—control group or exposure range comparison (S1 Methods). We  
163 translated the title and abstracts of studies using freely available online software (i.e., Google  
164 Translate) that were not published in English to evaluate its relevance.

165

166 ***Data Extraction***

167 We extracted data from studies in duplicate in a Health Assessment Workplace Collaborative  
168 database (HAWC; available at: <https://hawcproject.org/about/>). Two of three possible extractors  
169 (SE, EM, DB) independently extracted data relating to study characteristics and outcome  
170 measures (S2 Methods) from each included article. A third extractor (PH, BV) performed  
171 QA/QC on all the studies to resolve any discrepancies between the two independent extractors;  
172 subsequently, two authors (JL, EK) reviewed all studies to further ensure the accuracy of  
173 extracted data. When information was missing from a published article, we contacted  
174 corresponding study authors to request additional information.

175 ***Rate the quality and strength of the evidence***

176 **Statistical analyses:** Prior to study selection, we developed a list of study characteristics  
177 (contained in our protocol: <http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD  
178 42016038766) to identify studies suitable for meta-analysis. After evaluating the characteristics  
179 of all the studies, we grouped studies into four study population and health outcome  
180 combinations: 1) child asthma diagnosis; 2) child asthma exacerbation and symptoms; 3) adult  
181 asthma diagnosis; and 4) adult asthma exacerbation and symptoms.

182 To differentiate child from adult studies, we initially planned to use the age of 18 years as a  
183 cutoff for children, but a number of the studies used a cutoff age of 15 years to distinguish  
184 between children and adults. Given that the onset of asthma commonly occurs during preschool  
185 years and recent increases in asthma incidence over the past few decades has been observed to  
186 increasingly affect children and adolescents aged 1 to 14 years, we decided to use age 15 years as  
187 the cutoff to group child vs. adult studies. We did not include studies in the meta-analysis that  
188 reported effect estimates with only mixed children and adult populations in the meta-analysis due  
189 to concerns that differences in adult-onset versus childhood-onset of asthma would be masked.

190 We also did not consider these data in our overall rating of study quality and strength, but we did  
191 include these data in visual scatterplots of data for comparison with child and adult data.

192 For the adult studies, we considered the body of evidence to include all adult population studies,  
193 regardless of whether exposure occurred in the general population or at work, as biologically, the  
194 relationship between exposure and health outcome is independent of where the exposure  
195 occurred. We distinguished the adult general population study results from the adult occupational  
196 study results on the visual scatterplots for comparison.



197 For cohorts with multiple publications (for instance, if a cohort was followed over time), we  
198 utilized results from the latest time point where our relevant outcome of interest was measured,  
199 but also considered information provided collectively across the publications for evaluating  
200 study quality. Where available, we used adjusted odds ratios to conduct the meta-analysis but if  
201 adjusted results were not reported, we included unadjusted ORs in the analyses. We converted  
202 effect estimates to an OR and 95% confidence interval (CI) for the association between asthma  
203 per 10- $\mu\text{g}/\text{m}^3$  unit increase in formaldehyde exposure to standardize across studies, transforming  
204 units of exposure when necessary. Where a meta-analysis was not possible, we created visual  
205 scatterplots of data across studies reporting on similar outcomes and subpopulations to consider  
206 all available data in assessing the evidence. We also applied a mixed models approach for  
207 repeated data to evaluate outcomes at various doses, using exchangeable correlation structures  
208 for repeated measurements within the same study.

209 We evaluated statistical heterogeneity across study estimates in the meta-analysis using  $I^2$  with  
210  $p \leq 0.05$  as our cut off for statistical significance , as previously described. If statistical  
211 heterogeneity was present, we used leave-one-out analysis to identify the study or studies  
212 contributing, evaluated potential study characteristics (e.g., study location, study population,  
213 study design, adjusted confounders, timing of exposure, etc.) to determine if we could explain  
214 the source, and incorporated hierarchical cluster structures in the data analysis to statistically  
215 account for heterogeneity. We also investigated the relative contribution of each study to the  
216 overall meta-analysis association and conducted sensitivity analysis to investigate the impacts of  
217 removing highly influential studies from the analysis. Data management was performed with  
218 Microsoft Excel. Statistical analyses were performed using STATA 13.1 software (StataCorp,  
219 2011). We pooled estimates using inverse variance-weighted models, fixed-effects models and

220 the DerSimonian and Laird random-effects models. We used the *metan*, *metareg*, *metainf*,  
221 *metafunnel*, *metabias* and *metatrim* packages in STATA version 13.1.

222 To investigate the effect of publication bias on our meta-analysis, we created funnel plots and  
223 used Egger’s test. We also quantitatively evaluated each meta-analysis for the potential effect  
224 that a new study might have on changing the interpretation of our overall results. Specifically,  
225 the association estimate of a new or unpublished study necessary to alter the results of the meta-  
226 analysis was calculated under two scenarios: 1) the 95% confidence interval of the meta-analysis  
227 overlapped zero, and 2) the meta-analysis central association estimate was greater than zero  
228 (moved to the opposite direction—i.e., such that increases in formaldehyde exposures would be  
229 associated with decreases in asthma outcomes). In making this calculation, we assumed that the  
230 new hypothetical study would have a standard error equal to the smallest in our group of studies.

231 **Assessing the risk of bias for each included study:** We evaluated risk of bias separately for  
232 each of the four study population/outcome group combinations using The Navigation Guide Risk  
233 of Bias Tool, a modified instrument based on the Cochrane Collaboration and Agency for  
234 Healthcare Research and Quality (AHRQ) domains , with customized instructions for each  
235 domain based on the type of evidence anticipated beforehand (S3 Methods).

236 We evaluated nine risk of bias domains (Source Population, Blinding, Outcome Assessment,  
237 Confounding, Incomplete Outcome, Exposure Assessment, Selective Reporting, Financial  
238 Conflict of Interest, and Other). We assigned each domain as “low,” “probably low,” “probably  
239 high,” or “high” risk of bias, or “not applicable” (domain not applicable to study) according to  
240 specific criteria as described in our risk of bias instruments (S3 Methods). Two of three possible  
241 reviewers (SE, EM, RB) independently recorded risk of bias determinations for each included

242 study. We held an in-person meeting for all review authors (JL, EK, PS, AMP, MDC, HV, ND,  
243 EW, TJW) to review risk of bias ratings and rationales for each study, come to consensus to  
244 ensure consistency, and record our final rationale. One review author (EK) independently  
245 reviewed all final risk of bias ratings for QA/QC.

246 **Rating the quality of evidence across all included studies:** We separately rated the quality of  
247 the overall body of evidence as “high,” “moderate,” or “low” for each of the four study  
248 population/outcome group combinations. We assigned an initial rating of “moderate” quality for  
249 each group of human observational studies prior to evaluating the included studies, based on  
250 previously described rationale—briefly, observational human studies are recognized as a reliable  
251 source of evidence and generally the most appropriate for answering environmental health-  
252 related questions. From the initial “moderate” quality rating, we then considered potential  
253 adjustments (“downgrades” or “upgrades”) to the quality rating based on 8 categories of  
254 considerations: risk of bias, indirectness, inconsistency, imprecision, potential for publication  
255 bias, large magnitude of effect, dose response, and whether residual confounding would  
256 minimize the overall effect estimate; the specific factors and criteria considered are outlined in  
257 S4 Methods. Possible ratings were 0 (no change from initial quality rating), -1 (1 level  
258 downgrade) or -2 (2 level downgrade), +1 (1 level upgrade) or +2 (2 level upgrade). Review  
259 authors independently evaluated the quality of the evidence and then we compared ratings as a  
260 group and recorded the consensus and rationale for each decision.

261 **Rating the strength of the evidence across all included studies:** We assigned an overall  
262 strength of evidence rating separately for the four study population/outcome group combinations  
263 based on four considerations: (1) Quality of body of evidence (i.e., the rating from the previous  
264 step); (2) Direction of effect; (3) Confidence in effect (likelihood that a new study would change

265 our conclusion); and (4) Other compelling attributes of the data that may influence certainty.  
266 Possible ratings were “sufficient evidence of toxicity,” “limited evidence of toxicity,”  
267 “inadequate evidence of toxicity,” or “evidence of lack of toxicity” (Table 1), based on  
268 categories used by the International Agency for Research on Cancer (IARC), the U.S. Preventive  
269 Services Task Force, and U.S. EPA (22-25). Review authors independently evaluated the quality  
270 of the evidence following directions as outlined in S4 Methods and then compared ratings as a  
271 group and recorded the consensus and rationale.

**Table 1.** Strength of evidence definitions for human evidence

Strength Rating    Definition

Sufficient evidence of toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies <sup>1</sup> .
Limited Evidence of Toxicity	A positive relationship is observed between exposure and outcome where chance, bias, and confounding cannot be ruled out with reasonable confidence. Confidence in the relationship is constrained by such factors as: the number, size, or quality of individual studies, or inconsistency of findings across individual studies <sup>2</sup> . As more information becomes available, the observed effect could change, and this change may be large enough to alter the conclusion.
Inadequate Evidence of Toxicity	The available evidence is insufficient to assess effects of the exposure. Evidence is insufficient because of: the limited number or size of studies, low quality of individual studies, or inconsistency of findings across individual studies. More information may allow an assessment of effects.
Evidence of Lack of Toxicity	No relationship is observed between exposure and outcome, and chance, bias and confounding can be ruled out with reasonable confidence. The available evidence includes consistent results from more than one well-designed, well-conducted study at the full range of exposure levels that humans are known to encounter, and the conclusion is unlikely to be strongly affected by the results of future studies <sup>3</sup> . The conclusion is limited to the age at exposure and/or other conditions and levels of exposure studied.

272

<sup>1</sup> The Navigation Guide rates the quality and strength of evidence of human and non-human evidence streams separately as “sufficient”, “limited”, “inadequate” or “evidence of lack of toxicity” and then these two ratings are combined to produce one of five possible statements about the overall strength of the evidence of a chemical’s reproductive/developmental toxicity. The methodology is adapted from the criteria used by the International Agency for Research on Cancer (IARC) to categorize the carcinogenicity of substances except as noted.

<sup>2</sup> Language for the definitions of the rating categories were adapted from descriptions of levels of certainty provided by the U.S. Preventive Services Task Force Levels of Certainty Regarding Net Benefit. <http://www.uspreventiveservicestaskforce.org/uspstf07/methods/benefit.htm>

<sup>3</sup> Language for the definitions of the rating categories were adapted from descriptions of levels of certainty provided by the U.S. Preventive Services Task Force Levels of Certainty Regarding Net Benefit.

**Economic analysis:** We combined quantitative assessment of exposure-response from our systematic review with incidence rates of asthma and annual values of asthma control to estimate the monetized benefits of avoiding asthma in EPA’s proposed rule on pressed wood products. We used the standard EPA approach of “willingness to pay” to calculate benefits, which measures the maximum amount of money that an individual is willing to pay to reduce the probability of an adverse health outcome assumed to be related to an environmental exposure [54].

To estimate the reduction in risk for asthma diagnosis, we used standardized risk estimates from our meta-analyses to estimate the reduction in risk per 1 ppb decrease in formaldehyde exposure. We assumed a Cox proportional hazard model so the number of reduced cases of asthma from a reduction in formaldehyde exposure is the exposed population times the baseline asthma risk times  $(1 - \exp(\ln(\text{OR}) * (\text{change in exposure})))$ . Using the tables for annual asthma benefits from EPA’s economic analysis for the proposed rule, we derived the exposure reduction for structures built new or renovated in the past eleven years. We used the change in indoor formaldehyde exposure for new and renovated homes at various ages (ranging from 0.124 to 3.390 ppb), the assumed baseline annual risk of asthma of 0.83%, used in EPA’s economic analysis and the estimated number of children aged 4-17 in 2017 in each housing type from the U.S. Census Bureau, with the proportional hazard model to estimate the reduced number of asthma cases associated with the proposed rule (26). We estimated the annual benefits for lowering formaldehyde emissions once the impacts of the reduction have reached steady-state (26).

To quantify the economic benefits of the reduction in asthma risk, we used estimates reported in the literature for the annual willingness to pay for full asthma control (inflated to 2018 dollars) from three studies. Full asthma control is equivalent to avoiding a case of asthma. Blomquist et

al. (27) used a two-stage contingent valuation survey of parents of asthmatic children aged 4–17 years and of adults to elicit the willingness to pay for a hypothetical drug that would control asthma symptoms [55]. The mean annual willingness to pay for children was \$3,434 and the mean annual value for adults was \$2,368. Blumenschein and Johannesson (28) used a contingent valuation bidding game to estimate asthma patients' willingness to buy a new treatment that cured their asthma, finding a mean value of \$3,621. O'Connor and Blomquist (29) used a two-stage contingent valuation survey of adults with asthma to elicit the tradeoff between hypothetical medication of varying degrees of safety and efficacy and estimated a mean annual willingness to pay for full asthma control of \$2,413 using the value of statistical life. The average annual value of asthma control for adults across all three studies is \$2,801 and the annual value for children is \$3,434 from Blomquist et al. (27). The total value to an individual to not develop asthma at a given age is the present discounted value (3% discount rate) of the annual values over the life expectancy of that individual.

## Results

### *Included studies*

We retrieved a total of 4,821 unique records (4,482 from the initial search on March 15, 2016, an additional 254 from an updated search on March 15, 2018, and an additional 85 from an updated search on April 1, 2020), of which 150 ultimately met the inclusion criteria. Given the large number of diverse references identified, we decided to focus on studies where the asthma status of all study participants was measured (90 studies) (Fig 1). Our rationale was that these studies provided the most robust evidence for understanding the relationship between formaldehyde exposure and asthma because they all had quantitative measures of formaldehyde exposure,

318 participants for whom asthma status was known, and included asthmatics. Lists of all other  
319 studies are provided in the supplemental materials (S1 Results). Several included studies  
320 contained information from multiple records, such as a graduate thesis and a published  
321 manuscript following the cohort over time; the information from these records were combined  
322 into one record and listed as the main published manuscript. Four studies were identified that  
323 looked at similar outcomes from the same study population, so we combined these and focused  
324 on the publication for which the most relevant information was reported, supplementing with  
325 additional information from the related publications when necessary. We contacted  
326 corresponding study authors for 21 studies to request additional information missing from their  
327 published articles and received useable data from three.

328 Studies were further categorized separately into four combinations of study population and  
329 outcome (with some studies reporting on multiple populations/outcomes falling in multiple  
330 categories): 1) Child asthma diagnosis (n=24); 2) Child asthma exacerbation and symptoms  
331 (n=23); 3) Adult (general population and occupational) asthma diagnosis (n=20); Adult (general  
332 population and occupational) asthma exacerbation and symptoms (n=26). Presentation of results  
333 below include separate discussions for each of these four population/outcome categories. In  
334 particular, S99 Table presents study characteristics for included studies stratified by these group  
335 population/outcome categories.

336 **Characteristics of included studies—Demographics**

337 The 90 included studies were published between 1969 and 2019, were conducted in 23 different  
338 countries (including 32% (n=29) within the U.S.), and included a range of 7 to 15,837  
339 participants (Table 2, [S99-S101 Table-3b](#)).



Table 2. Summary of included studies (n=90)

Study Characteristics	N (%)	Study Characteristics	N (%)
<b>Publication Year</b>		<b>Formaldehyde Exposure</b>	
1969	1 (1%)	Measured exposure level	82 (91%)
1977	1 (1%)	Categorized exposure level	8 (9%)
1980-1989	17 (19%)		
1990-1999	16 (18%)		
2000-2009	22 (24%)		
2010-2019	33 (37%)		
<b>Study Design</b>		<b>Study Participants*</b>	
Case-control	7 (8%)	Child	37 (41%)
Nested case-control	3 (3%)	Asthma***	24 (65%)
Prospective cohort	15 (17%)	Asthma symptoms***	23 (62%)
Cohort	2 (2%)	Pulmonary function***	5 (14%)
Cross-sectional	46 (51%)	Adult (General and occupational)	54 (60%)
Cross-sectional and case-control	2 (2%)	Asthma***	20 (37%)
Non-randomized controlled trial	6 (7%)	Asthma symptoms***	26 (48%)
Randomized controlled trial	5 (6%)	Pulmonary function***	35 (65%)
Case report	4 (4%)	Mixed child and adults	2 (2%)
<b>Sample Size</b>		Asthma***	1 (50%)
0-50	24 (26%)	Asthma symptoms***	2 (100%)
51-100	16 (18%)	Pulmonary function***	1 (50%)
101-200	12 (13%)	Unspecified	1 (1%)
201-500	14 (16%)	Asthma symptoms***	1 (100%)
501-1000	5 (6%)	Pulmonary function***	1 (100%)
>1000	17 (19%)		
Not reported	2 (2%)		
<b>Country**</b>		<b>Population Source</b>	
Egypt, Estonia, Indonesia, Iran, Japan, Malta, New Zealand, Poland, Russia, Thailand, United Arab Emirates	1 (12%)	General population (Adult and child)	59 (66%)
Canada, Finland, Portugal, Romania	2 (9%)	Asthma***	33 (56%)
Denmark	3 (3%)	Asthma symptoms***	30 (51%)
France	4 (4%)	Pulmonary function***	18 (31%)
Australia, China	5 (11%)	Occupational	31 (34%)
United Kingdom	5 (6%)	Asthma***	11 (35%)
South Korea	7 (8%)	Asthma symptoms***	19 (61%)
Sweden	13 (14%)	Pulmonary function***	20 (65%)
United States	29 (32%)		

\*Studies that reported child versus adult data separately fell into both categories (as opposed to studies that reported collectively on children and adults mixed in the study population)—therefore total % is greater than 100%  
\*\*Due to the variety of different countries represented, countries with similar counts have been grouped together for reporting. For instance, there are 5 studies located in Australia and 5 other studies located in China.  
\*\*\*Many studies report multiple asthma outcomes—therefore total % is greater than 100%. Percentages are calculated out of the category sub-total; for instance, the percentage of asthma studies in children is calculated as 24/37.

**Table 3a.** Study categorization by study population and outcome.

Study	Children	Adults	Asthma Diagnosis	Asthma Symptoms	Pulmonary Measures	Meta Analysis
Akbar-Khanzadeh et al. 1994 (Cross-sectional)	-	-	-	-	-	-
Akbar-Khanzadeh et al. 1997 (Cross-sectional)	-	-	-	-	-	-
Annesi-Maesano et al. 2012 (Cross-sectional)	-	-	-	-	-	-
Billionnet et al. 2011 (Cross-sectional)	-	-	-	-	-	-
Burge et al. 1984 (Case reports)	-	-	-	-	-	-
Chatzidiakou et al. 2014 (Cross-sectional)	-	-	-	-	-	-
Choi et al. 2009 (Case-control)	-	-	-	-	-	-
Dannemiller et al. 2013 (Cross-sectional)	-	-	-	-	-	-
De Vos et al. 2009	-	-	-	-	-	-
Delfino et al. 2003 (Cross-sectional)	-	-	-	-	-	-
Dumas et al. 2017 (Case-control study nested within prospective cohort)	-	-	-	-	-	-
Elschaer et al. 2017 (Cross-sectional)	-	-	-	-	-	-
Ezratty et al. 2007 (Non-randomized controlled trial)	-	-	-	-	-	-
Fornander et al. 2014 (Cross-sectional)	-	-	-	-	-	-
Fransman et al. 2003 (Cross-sectional)	-	-	-	-	-	-
Frey et al. 2014 (Cross-sectional)	-	-	-	-	-	-
Frigas et al. 1984 (Case reports)	-	-	-	-	-	-
Frisk et al. 2002 (Cohort (Prospective))	-	-	-	-	-	-

Frisk et al. 2006 (Case-control)	-	-	-	-	-	-
Frisk et al. 2009 (Cross-sectional)	-	-	-	-	-	-
Fsadni et al. 2018 (Cohort (Prospective))	-	-	-	-	-	-
Gannon et al. 1995 (Case reports)	-	-	-	-	-	-
Garrett et al. 1999 (note Garrett et al. 1998 used same cohort so combined to one record) (Cross-sectional)	-	-	-	-	-	-
Gorski et al. 1991 (Cohort)	-	-	-	-	-	-
Green et al. 1987 (Randomized-controlled trial)	-	-	-	-	-	-
Hanson et al. 1993 (Cross-sectional)	-	-	-	-	-	-
Harving et al. 1990 (Randomized-controlled trial)	-	-	-	-	-	-
Hendrick et al. 1977 (Cohort (Prospective))	-	-	-	-	-	-
Herbert et al. 1994 (Cross-sectional)	-	-	-	-	-	-
Horvath et al. 1988 (Cross-sectional)	-	-	-	-	-	-
Hsu et al. 2012 (Case-control)	-	-	-	-	-	-
Huang et al. 2016 (Nested-case-control)	-	-	-	-	-	-
Hulin et al. 2010 (Nested-case-control)	-	-	-	-	-	-
Hwang et al. 2011 (Case-control)	-	-	-	-	-	-
Idavain et al. 2019 (Cross-sectional)	-	-	-	-	-	-
Jacobsen et al. 2009 (Cohort (Prospective))	-	-	-	-	-	-

Jeong et al. 2011 (Cross-sectional)	-	-	-	-	-	-
Kilburn et al. 1985 (Cohort (Prospective))	-	-	-	-	-	-
Kilburn, Seidman, and Warshaw 1985 (Cross-sectional)	-	-	-	-	-	-
Kim et al. 2007 (Cross-sectional)	-	-	-	-	-	-
Kim et al. 2011 (Cross-sectional)	-	-	-	-	-	-
Kim et al. 2014 (Non-randomized controlled trial)	-	-	-	-	-	-
Kriebel et al. 1993 (Cross-sectional)	-	-	-	-	-	-
Kriebel et al. 2001 (Cohort (Prospective))	-	-	-	-	-	-
Krzyzanowski et al. 1990 (Cross-sectional)	-	-	-	-	-	-
Lajoie et al. 2015 (Randomized controlled trial)	-	-	-	-	-	-
Liu et al. 1991 (Cross-sectional)	-	-	-	-	-	-
Lofstedt et al. 2009 (Cohort (Prospective))	-	-	-	-	-	-
Lofstedt et al. 2011 (Cohort (Prospective))	-	-	-	-	-	-
Low et al. 1985 (Cross-sectional)	-	-	-	-	-	-
Madureira et al. 2015 (note same cohort of children as Madureira et al. 2015b, but more comprehensive, so combined to one record) (Cross-sectional and case- control)	-	-	-	-	-	-
Madureira et al. 2016 (Cross-sectional and case- control)	-	-	-	-	-	-

Malaka et al. 1990 (Cross-sectional)	-	-	-	-	-	-
Mapou et al. 2013 (Cross-sectional)	-	-	-	-	-	-
Marks et al. 2010 (Non-randomized controlled-trial)	-	-	-	-	-	-
Matsunaga et al. 2007 (Cross-sectional)	-	-	-	-	-	-
Mi et al. 2006 (Cross-sectional)	-	-	-	-	-	-
Milton et al. 1996 (Cross-sectional)	-	-	-	-	-	-
Neamtiu et al. 2019 (Cohort (Prospective))	-	-	-	-	-	-
Norback et al. 1995 (Cross-sectional)	-	-	-	-	-	-
Norback et al. 2000 (Cross-sectional)	-	-	-	-	-	-
Nordman et al. 1985 (Case reports)	-	-	-	-	-	-
Popa et al. 1969 (Cohort (Prospective))	-	-	-	-	-	-
Pourmababadian et al. 2006 (Cross-sectional)	-	-	-	-	-	-
Quackenboss et al. 1989 (Cross-sectional)	-	-	-	-	-	-
Raaschou-Nielsen et al. 2010 (Cohort (Prospective))	-	-	-	-	-	-
Rumchev et al. 2002 (Case-control)	-	-	-	-	-	-
Sauder et al. 1987 (Non-randomized controlled-trial)	-	-	-	-	-	-
Schenker et al. 1982 (Cross-sectional)	-	-	-	-	-	-
Sheppard et al. 1984 (Non-randomized controlled-trial)	-	-	-	-	-	-

Smedje and Norback 2000 (Cohort (Prospective))	-	-	-	-	-	-
Smedje and Norback 2001 (Cohort (Prospective))	-	-	-	-	-	-
Smedje et al. 1997 (Cross-sectional)	-	-	-	-	-	-
Tavernier et al. 2006 (Cross-sectional)	-	-	-	-	-	-
Thetkathuek et al. 2016 (Cross-sectional)	-	-	-	-	-	-
Tuomainen et al. 2003 (Cohort)	-	-	-	-	-	-
Tuthill 1984 (Cross-sectional)	-	-	-	-	-	-
Uba et al. 1989 (Cohort (Prospective))	-	-	-	-	-	-
Venn et al. 2003 (Case-control)	-	-	-	-	-	-
Veremchuk et al. 2016 (Cross-sectional)	-	-	-	-	-	-
Wieslander et al. 1997 (Cross-sectional)	-	-	-	-	-	-
Willis et al. 2018 (Repeated cross-sectional)	-	-	-	-	-	-
Witek, Jr et al. 1986 (Randomized-controlled trial)	-	-	-	-	-	-
Witek, Jr et al. 1987 (Randomized-controlled trial)	-	-	-	-	-	-
Yeatts et al. 2012 (Cross-sectional)	-	-	-	-	-	-
Yon et al. 2019 (Cohort (Prospective))	-	-	-	-	-	-
Yoon and Lin 2014 (Case-control)	-	-	-	-	-	-
Zammit-Tabona et al. 1983 (Cross-sectional)	-	-	-	-	-	-
Zhai et al. 2013 (Cross-sectional)	-	-	-	-	-	-
Zhao et al. 2008 (Cross-sectional)	-	-	-	-	-	-

**Table 3b.** Study Characteristics

Study (Study Design)	Study population & location	Sample size	Exposure assessment	Exposure ranges	Outcome assessment (not including pulmonary function tests)	Outcomes Reported	Confounders	Results
<b>Studies considered for meta-analysis</b>								
Smedje and Norback 2001 (Prospective Cohort)	Children in the general population attending 39 public schools in Uppsala county in Sweden (follow-up study to Smedje and Norback 2000)	1347 students in 1st, 4th or 7th grade (mean age 10.3 years in 1993 and 14.3 years in 1997)	Formaldehyde was measured for 4 hours in 2-5 secondary school classrooms and for each primary school classroom for each school in 1993 (prior to installation of new ventilation system) and in 1995 (after installation of new ventilation system)	Arithmetic mean: 8 ug/m <sup>3</sup> (range <5-72); geometric mean: 4 ug/m <sup>3</sup> (SD 2.3)	Questionnaire completed by subjects in 1993 and 1997 included question on whether student had ever had asthma and if diagnosis made by physician; additional questions on lower respiratory symptoms based on questionnaire from European Community Respiratory Health Survey (ECRHS)	Asthma diagnosis (incidence)	Age, atopy, smoking, sex	OR=1.2, 95% CI [0.8-1.7] for asthma diagnosis per 10 ug increase in formaldehyde in classroom air

Rumechev et al. 2002 (Case-control)	Children in the general population with asthma identified by the Accident and Emergency Dept at the Princess Margaret Hospital for Children and nonasthmatic controls identified through the Health Dept of Western Australia, Perth, Western Australia	88 asthmatic children (mean age 25 months) and 104 nonasthmatic controls (mean age 20 months) (ages 6 months-3 years)	Formaldehyde measured in the living room and child's bedroom for 8 hours during the day in July-September 1998 and December 1998-March 1999	Mean bedroom: 30.2 ug/m <sup>3</sup> ; mean living room: 27.5 ug/m <sup>3</sup>	Asthma cases were children discharged with medical diagnosis of asthma from emergency department; questionnaire from American Thoracic Society completed by parents for respiratory symptoms (including wheeze) and home characteristics	Asthma diagnosis; wheeze	Age, air conditioning, allergen levels of house dust mite, atopy, child allergies, family history of asthma, humidifier and gas appliances, indoor air pollutants, indoor temperature, presence of pets, relative humidity, sex, smoking inside, socioeconomic status	OR=1.003, 95% CI [1.002-1.004] for asthma diagnosis per 10-unit (ug/m <sup>3</sup> ) increase in formaldehyde exposure. Children who reported wheeze were also exposed to high average indoor levels of formaldehyde (40.5 ug/m <sup>3</sup> ) compared to those without such symptoms (26.7 ug/m <sup>3</sup> ) and the difference was significant (p<0.01)
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Smedje et al. 1997 (Cross-sectional)	Children in the general population attending 11 public schools in Uppsala county in Sweden	627 students in 7th grade (ages 13-14 years)	Formaldehyde was measured for 4 hours in 2-3 classrooms for each school (total 28 classrooms) in 1993	Arithmetic mean: $<5$ $\mu\text{g}/\text{m}^3$ (range $<5$ -10)	Questionnaire on asthmatic symptoms amended from one used in the ECRHS completed by subjects; current asthma defined as physician diagnosis and symptoms within the last year	Current asthma (diagnosed)	Controlled for "personal factors," but no explicit discussion of what these included. May include atopy, food allergy, and whether attended day care center for several years	OR=1.1, 95% CI [1.01-1.2] for current asthma per 1-unit change ( $\text{mg}/\text{m}^3$ ) in formaldehyde concentration
Kim et al. 2011 (Cross-sectional)	Children in the general population attending 4th grade in twelve randomly-selected schools in three cities (Guri, Namyangju, and Chuncheon) in Korea	1915 total school children (mean age 10 years)	Formaldehyde measured continuously for 7 days in classrooms (n=34) and outside classroom windows (n=12) in November-December 2004	Mean (classroom) $\pm 18.2$ $\mu\text{g}/\text{m}^3$ (SD 17.3; range 2.7-52.8); mean (outdoor): 16.5 $\mu\text{g}/\text{m}^3$ (SD 12.5; range 3.3-45.3)	Questionnaire completed by subjects; current asthma defined as either having current medication or asthma attack in last 12 months	Doctor-diagnosed asthma; current asthma; wheeze	Age, sex, self-reported furry pet or pollen allergy, and home environment (remodeling, changing floor, age of home building, environmental tobacco smoke and indoor dampness)	OR=1.2, 95% CI [0.44, 3.24] self-reported wheezing during last 12 months per 10 $\mu\text{g}/\text{m}^3$ increase in outdoor formaldehyde exposure. OR=1.15, 95% CI [0.88, 1.5] self-reported wheezing during last 12 months per 10 $\mu\text{g}/\text{m}^3$ increase in indoor classroom

								formaldehyd e-exposure. OR=2.1, 95% CI [0.71, 6.23] current asthma (either having current asthma medication or having an asthma attack during the last 12 months) per 10 ug/m <sup>3</sup> increase in outdoor formaldehyd e-exposure. OR=1.04, 95% CI [0.78, 1.4] current asthma per 10 ug/m <sup>3</sup> increase in indoor classroom formaldehyd e-exposure. OR=0.8, 95% CI [0.22, 2.85] asthma diagnosis per
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								10 ug/m3 increase in outdoor formaldehyd e exposure. OR=0.92, 95% CI {0.67, 1.26} asthma diagnosis per 10 ug/m3 increase in indoor classroom formaldehyd e exposure.
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Mi et al. 2006 (Cross-sectional)	Children in the general population attending junior high schools in central Shanghai and western Shanghai near Huang Pu river in China	1414 total school children from 5 schools in each district participated by questionnaire in November-December 2000 (aged 12-14 years)	Formaldehyde measured in 30 classrooms for 4 hours using stationary monitor in November-December 2000	Mean: 9.4 ug/m <sup>3</sup> (SD 6.9; range 3-20)	Questionnaire completed by subjects and included questions about asthma (doctor diagnosed; asthma medications; asthma attacks); airway symptoms during last year without using phrase "asthma" (wheezing; breathlessness)	Current asthma (diagnosed); asthma attack; medication use; current wheeze; nocturnal breathlessness; daytime breathlessness	Age, gender, indoor molds, smoking, water leakage	OR=1.01, 95% CI [0.56, 1.81] for current wheeze per 10 ug/m <sup>3</sup> increase in formaldehyde exposure. OR=1.09, 95% CI [0.86, 1.38] for daytime breathlessness per 10 ug/m <sup>3</sup> increase in formaldehyde exposure. OR=1.26, 95% CI [0.63, 2.53] for nocturnal breathlessness per 10 ug/m <sup>3</sup> increase in formaldehyde exposure. OR=1.24, 95% CI [0.63, 2.45] for asthma attack per 10 ug/m <sup>3</sup> increase in
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								formaldehyde exposure. OR=1.26, 95% CI [0.65, 2.46] for asthma medication per 10 ug/m <sup>3</sup> increase in formaldehyde exposure. OR=1.3, 95% CI [0.72, 2.32] for current asthma per 10 ug/m <sup>3</sup> increase in formaldehyde exposure.
Kim et al. 2007 (Cross-sectional)	Children in the general population attending all eight primary schools in Knivsta Municipality in rural outskirts of Uppsala City, Sweden	1014 school children (ages 5–15 years; mean age 9 years)	Formaldehyde measured in three classrooms in each school for 6 hours in May–June 2000	Mean for 23 classrooms: 7.13 ug/m <sup>3</sup> (range 3–16)	Questionnaire completed by parents with cooperation of child in April–May 2000 and included questions about asthma (doctor-diagnosed, asthma medications, asthma attacks), airway symptoms during last year without using phrase "asthma"	Current asthma (diagnosed); current asthma medication; wheezing; nocturnal breathlessness, and daytime breathlessness	Age, gender	OR=1.03, 95% CI [0.86, 1.24] for nocturnal breathlessness per 1 ug/m <sup>3</sup> increase in formaldehyde exposure in classroom. OR=0.96, 95% CI [0.87, 1.05] for wheeze per 1 ug/m <sup>3</sup> increase in

					(wheezing, breathlessness)			formaldehyd e exposure in classroom: OR=0.96, 95% CI {0.85, 1.08} for daytime breathlessnes s per 1 ug/m3 increase in formaldehyd e exposure in classroom: OR=1.02, 95% CI {0.93, 1.13} for doctor- diagnosed asthma per 1 ug/m3 increase in formaldehyd e exposure in classroom:
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Zhao et al. 2008 (Cross-sectional)	Children in the general population attending first year classes in 10 junior high schools within urban areas of Taiyuan, China	1993 school children (mean age 12.8 years)	Formaldehyde measured continuously for 7 days in one representative location in each school	Mean (classroom): 2.3 ug/m <sup>3</sup> (SD 1.1; range 1.0–5.0); mean (outdoor): 5.8 ug/m <sup>3</sup> (SD 0.6; range 5.0–7.0)	Questionnaire completed by subjects including questions on asthma (cumulative; doctor-diagnosed; and current) and on respiratory health (wheeze; breathlessness) based on International Study of Asthma and Allergy in Childhood (ISAAC)	Cumulative asthma (diagnosed); wheeze or whistling in the chest; nocturnal and daytime attacks of breathlessness	Personal and home environmental factors; age; environmental tobacco smoke at home, indoor and outdoor pollutants; new floor and new furniture in preceding 12 months; parental asthma or allergy; recent home painting; sex	OR=1.11, 95% CI [0.55, 2.23] for cumulative asthma per 1 ug/m <sup>3</sup> increase in formaldehyde exposure indoor. OR=4.61, 95% CI [1.09, 19.5] for cumulative asthma per 1 ug/m <sup>3</sup> increase in formaldehyde exposure outdoor. OR=0.93, 95% CI [0.78, 1.1] for daytime breathlessness per 1 ug/m <sup>3</sup> increase in formaldehyde exposure indoor. OR=1.29, 95% CI [0.99, 1.68] for
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								<p>cumulative asthma per 1 ug/m3 increase in formaldehyde exposure outdoor. OR=1.11, 95% CI [0.87, 1.41] for wheeze or whistling in the chest per 1 ug/m3 increase in formaldehyde exposure indoor. OR=1.32, 95% CI [0.86, 2.04] for wheeze or whistling in the chest per 1 ug/m3 increase in formaldehyde exposure outdoor. OR=1.92, 95% CI [0.87, 1.41] for nocturnal attacks of breathlessness per 1 ug/m3</p>
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								increase in formaldehyd e-exposure indoor: OR=2.03; 95%-CI {0.91, 4.54} for nocturnal attacks of breathlessnes s per 1 ug/m <sup>3</sup> increase in formaldehyd e-exposure outdoor:
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Hulin et al. 2010 (Nested case-control)	Children in the general population attending school and living within a city (Clermont-Ferrand, Auvergne, France) who participated in the Six Cities study or in surrounding rural areas (Auvergne, France)	63 urban children (32 asthmatics and 31 controls) and 51 rural children (24 asthmatics and 27 controls) (mean age 12.6 years)	Formaldehyde measured continuously for one week in the living room; assessed during summer and winter in urban area in 2003-2004 and in summer in rural area 2006-2007	Median: 19.2 ug/m <sup>3</sup> ; maximum: 75.1 ug/m <sup>3</sup>	Questionnaire completed by parents; cases identified on basis of "yes" response to questions about ever having asthma, wheezing in last year, and use of asthma medication	Current asthma; ever asthma; asthma	Age, allergic rhinitis, exposure to passive smoking during early childhood, family history of allergy, location, season, sex	OR=1.07, 95% CI [1.01, 1.13] for asthma cases per 10 ug/m <sup>3</sup> increase in formaldehyde exposure in urban environments OR=1.9, 95% CI [1.08, 3.5] for asthma cases per 10 ug/m <sup>3</sup> increase in formaldehyde exposure in rural environments OR=0.62, 95% CI [0.18, 2.14] for ever asthma comparing high (>19.2 ug/m <sup>3</sup> ) asthma versus low (<19.2 ug/m <sup>3</sup> ) in urban environments
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								.OR=10.72, 95% CI [1.69, 67.61] for ever asthma comparing high (>19.2 ug/m3) asthma versus low (<19.2 ug/m3) in rural environments .OR=0.24, 95% CI [0.03, 2.29] for current asthma comparing high (>19.2 ug/m3) asthma versus low (<19.2 ug/m3) in urban environments .OR=9, 95% CI [1, 82] for current asthma comparing high (>19.2 ug/m3) asthma
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								versus low (<10.2 ug/m3) in rural environments
Krzyzanowski et al. 1990 (Cross-sectional)	Municipal employee households in the general population with adults and children 5-15 years of age in Pima County Arizona	298 children (ages 6-15 years) and 613 adults	Formaldehyde measured in the kitchen, main living area and each subject's bedroom for two 1-week periods	Mean from 202 households: 26 ppb; maximum: 140 ppb	Questionnaire completed by subjects and included questions about asthma (doctor diagnosed with assessment of current status) and chronic respiratory symptoms (cough/phlegm, wheezing, and shortness of breath with wheezing)	Asthma diagnosis; pulmonary function test	Current smoker; education; environmental tobacco smoke; race/ethnicity	Prevalence rates per 100 subjects of current diagnosed asthma for children: 11.7 (<=40 ppb formaldehyde), 4.2 (41-60 ppb formaldehyde), 23.8 (>60 ppb formaldehyde). Relation of PEFR (Liters/Minute) to indoor

								formaldehyd e (ppb): 1.28 (mean) +/- 0.46 (SE) for children <15 years; 0.09 (mean) +/- 0.27 (SE) for adults >15 years
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Idavain et al. 2019 (Cross-sectional)	Children in the general population from 25 schools in Ida-Viru, Lääne-Viru and Tartu Counties in Estonia	1326 children from randomly selected schools. Age of students range 8-12 years,	Annual mean concentrations of formaldehyde were modelled	Range: 2.59-4.87 ug/m <sup>3</sup>	Asthma-related outcomes were assessed through questionnaires that were distributed to students by teachers and completed by parent and child together. Questions inquired whether child ever had wheezing or whistling in the chest at any time in the past, whether they had asthma diagnosed by physician, whether child ever had attacks of asthma, and whether child had wheezing or whistling in the chest without cold in the past 12 months. Children were then invited for a clinical examination for further evaluation.	Asthma diagnosis; asthma symptoms (wheezing, whistling in chest, asthma attack)	Age, sex, BMI, parent's education and family income.	OR = 1.01, 95% CI: [0.90, 1.13] for every wheezing per 1 ug/m <sup>3</sup> formaldehyde exposure.
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**Studies not considered for meta-analysis**

Smedje and Norback 2000 (Prospective Cohort)	Children in the general population attending 39 public schools in Uppsala county in Sweden	1476 students in 1st, 4th or 7th grade (mean age 10.4 years in 1993 and 12.3 years in 1995)	Formaldehyde was measured for 4 hours in 2-5 secondary school classrooms and for each primary school classroom for each school in 1993 (prior to installation of new ventilation system) and in 1995 (after installation of new ventilation system)	Geometric mean 1993 (received new ventilation system later): 6 ug/m <sup>3</sup> ; geometric mean 1993 (no new ventilation system): 3 ug/m <sup>3</sup> ; change from 1993-1995 (new ventilation system): 4 ug/m <sup>3</sup> ; change from 1993-1995 (no new ventilation system): 4 ug/m <sup>3</sup>	Questionnaire completed by subjects in 1993 & 1995 included questions about asthma and asthma symptoms (amended from those used by ECRHS); current asthma defined as ever had asthma diagnosed by doctor and had at least one asthmatic symptom recently or using medication; asthmatic symptoms include recurrent persistent cough, persistent wheeze or shortness of breath, or during past 12 months had asthma attack, shortness of breath after exercise or nocturnal shortness of breath	Current asthma, ever asthma (doctor-diagnosed); any asthmatic symptoms; more than 1 asthmatic symptom	Age, atopy, smoking, sex	OR=0.3, 95% CI [0.1, 0.8] for any asthmatic symptoms comparing children with new ventilation to those without. OR=0.6, 95% CI [0.2, 2.8] for ever doctor's asthma diagnosis comparing children with new ventilation to those without. OR=1.2, 95% CI [0.4, 4.1] for current asthma comparing children with new ventilation to those without. OR=0.5, 95% CI [0.2, 0.97]
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								for more asthmatic symptoms in 1995 than in 1993 comparing children with new ventilation to those without.
Fsadni et al. 2018 (Prospective Cohort)	Children in the general population at five primary state school randomly selected from five geographical clusters in Malta	Sample size not reported. Age of students range 9-11 years.	Sampling took place over a 5-day period (Monday morning until Friday afternoon). Pollutant concentrations were averaged.	Indoor: mean 11.21 ug/m3 (sd: 2.95; range: 6.67-18.89). Outdoor: mean 1.81 ug/m3 (sd: 0.44; range: 1.37-2.4)	Standardized International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire focusing on wheezing symptoms reported by parents	Asthma symptoms (wheezing); pulmonary lung function tests	No confounders or adjustment factors reported	No quantitative association estimates available. Authors only report that formaldehyde is associated with more likely current wheezing, but not exercise-induced wheezing or nocturnal cough. Quantitative estimates for associations are not reported.



Yon et al. 2019 (Prospective Cohort)	Children in the general population attending eleven elementary schools in Seongnam City, Korea	427 students from 11 randomly selected classrooms, 10 with asthma. Age of students not reported, although all were in elementary school.	Formaldehyde concentrations were measured twice in each classroom (once in the first half and once in the second half of the academic year).	Average classroom concentration: 27.17 $\mu\text{g}/\text{m}^3$ ( $\pm 7.72$ , maximum 60 $\mu\text{g}/\text{m}^3$ )	Asthma defined by the presence of characteristic symptoms and/or signs during the previous 12 months, based on the International Study of Asthma and Allergies in Childhood questionnaire	Asthma symptoms	Age, sex, environmental tobacco smoke exposure, keeping a pet at home, and physician-diagnosed asthma and AD in parents	OR = 1.023, 95% CI: [0.960, 1.089] for asthma per 1 $\mu\text{g}/\text{m}^3$ increase in indoor formaldehyde exposure
Neamtiu et al. 2019 (Prospective Cohort)	Children in the general population attending five public primary schools from Alba County in Romania as part of the SINPHONE cohort study	280 students from 15 different classrooms in primary school (age not reported)	Formaldehyde exposures were measured for five days inside three classrooms and in one outside location at each school.	Indoor: Mean 34.16 $\mu\text{g}/\text{m}^3$ (sd: 15.07, range: 15.50–66.19); Outdoor: Mean 9.50 $\mu\text{g}/\text{m}^3$ (sd: 3.23, range: 6.03–12.90)	SINPHONE questionnaire completed by students inquiring about asthma-like symptoms in the past week	Asthma-like symptoms (difficulty breathing, dry cough, and wheezing in the past week)	Age, gender, NO <sub>2</sub> , CO, CO <sub>2</sub> , temperature, relative humidity, ventilation rate, and tobacco smoke exposure for the past week	OR = 2.69, 95% CI: [1.04, 6.97] for asthma-like symptoms from exposure to formaldehyde concentration (higher formaldehyde ( $\geq 35 \mu\text{g}/\text{m}^3$ ) compared to

								lower (=35 ug/m3))
Hwang et al. 2011 (Case-control)	Children in the general population attending elementary school in Seongbuk, Seoul	33 asthmatic children and 40 non-asthmatic controls (ages 8-13 years)	Formaldehyde measured using personal, indoor, and outdoor monitors for 3 days in 2008	Geometric mean (indoor): 33.3 ug/m3; geometric mean (outdoor): 5.0 ug/m3; geometric mean (personal): 27.8 ug/m3	Parents completed ISAAC questionnaire; children with self-reported asthma symptoms or physician-diagnosed included as cases	Asthma diagnosis	Age, gender, family income, parents' academic background, passive smoking	OR=1.0, 95% CI [1.0, 1.1] for childhood asthma for increasing formaldehyde exposure. Unit of increasing exposure was unclear.

Hsu et al. 2012 (Case-control)	Children in the general population attending randomly selected kindergartens and day care centers (n=335 participating) in the greater Tainan Metropolitan area of Taiwan, China	9 asthmatic children and 42 non-asthmatic controls (ages 3-9 years; mean age 7 years)	Formaldehyde measured in children's bedroom for 2 hours between August 2008 and September 2009	Median (all children): 6.2 ppb (range 25th-75th percentile 4.3-20.4); median (asthma cases): 4.3 ppb (range 25th-75th percentile 3.2-9.6); median (controls): 13.8 ppb (range 25th-75th percentile 4.3-24.6)	Medical examination by pediatrician to diagnose asthma including physical examination and standardized questionnaire	Asthma diagnosed by medical examination	The analyses with formaldehyde as the exposure of concern do not account for potential confounders. Differences were identified between study subjects and the original population, including child's gender, parental education level, parental allergic history, and parental smoking status.	Median measured 2-hour indoor formaldehyde levels in the bedrooms of physician confirmed asthma case children (4.3 ppb; 25th percentile: 3.2, 75th percentile: 9.6) were statistically significantly lower (p=0.03) than median measured formaldehyde levels in the bedrooms of non-symptomatic control children (13.8 ppb; 25th percentile: 4.3, 75th percentile: 24.6).
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Yoon and Lin 2014 (Case-control)	Children in the general population attending elementary school in Andong, Korea	162 students (mean age 11.5 years)	Formaldehyde measured using personal samplers in breathing zone for 3 working days	Geometric mean (asthma cases): 6.96 ug/m <sup>3</sup> ; geometric mean (controls): 8.31 ug/m <sup>3</sup>	Questionnaire completed by subjects and included questions from American Thoracic Society criteria; students asked if they had asthma symptoms, and if yes, what symptoms were and if they had been diagnosed with asthma by a physician	Asthma diagnosis	Age, gender, family history of asthma; family income; amount of house sunlight; distance from bus within 100m; household with smokers; outdoor chemical odors	There was no significant difference between the asthmatic group and non-asthmatic group formaldehyde exposure level; the asthmatic group had a formaldehyde exposure of 6.96 ug/m <sup>3</sup> GM, 2.26 GSD; the non-asthmatic group had an exposure of 8.31 ug/m <sup>3</sup> GM, 1.66 GSD
Tavernier et al. 2006 (Cross-sectional)	Children in the general population participating in the Indoor Pollutants, Endotoxin, Allergens, Damp and Asthma in	200 children (ages 4 to 17 years)	Formaldehyde measured during 2 visits per home 1 week apart	Not reported	Questionnaire validated against physician diagnosis of asthma	Asthma diagnosed by medical examination	Considered factors: bedroom sharing; benzene in bedroom; dust mite allergen; endotoxin; furred pet ownership; gas	OR=0.82, 95% CI [0.33, 2.05] for asthma comparing second tertile of exposure to formaldehyde to first

	Manchester (IPEADAM) study recruited as patients of 2 primary care facilities or healthy controls subjects in South Manchester, United Kingdom						cooking, nitrogen dioxide in bedroom, nitrogen dioxide in living room, number of children in household, presence of smokers, redecoration in living room, respirable suspended particles in living room, self-reported absence of dampness in home, self-reported dampness in kitchen and bathroom, single-parent family, solanesol particulate matter in bedroom, solanesol particulate matter in living room, time in	tertile. OR=1.22, 95% CI {0.49, 3.07} for asthma comparing third tertile of exposure to formaldehyde to first tertile.
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							residence. Unclear what author ultimately controlled for.	
Willis et al. 2018 (Repeated cross-sectional)	Hospitalized children in the general population living in the entire state of Pennsylvania (67 total counties)	15,837 children with pediatric asthma-related hospitalizations. Age range from 2–18 years.	Formaldehyde exposure was evaluated using the Pennsylvania Unconventional Natural Gas Emission Inventory which has annualized emissions data from Unconventional Gas Drilling (UNGD) sites. Pollutants reported in tons emitted per year and linked to	Median: 0.00021 tons/year emissions (range: 0–22.51)	Asthma hospitalizations were obtained from the Pennsylvania Healthcare Cost Containment Council hospitalization data by identifying diagnostic codes with a 493 ICD-9 code, which indicates acute asthma exacerbation.	Asthma hospitalizations and exacerbations	Sex, race, year, quarter, insurance status, zip code respiratory hazard index, county median household income quartile, county unemployment, county poverty under 18 years old, and county log population density	OR = 1.2; 95% CI = [1.06, 1.36] for asthma hospitalizations for increasing formaldehyde exposure (log-sum emissions)

			participants' zip code.					
Huang et al. 2016 (Nested case-control)	Children in the general population attending 88 randomly selected kindergartens from Shanghai's six districts	1216 children with asthma and 8651 nonsymptomatic children (ages 5-10 years)	Formaldehyde measured as in children's bedroom over seven consecutive days	Mean formaldehyde 24-hour measurements over cases and controls: 21.5 ug/m <sup>3</sup> (sd=13.0). 407 (99.3%) samples were <100 ug/m <sup>3</sup> . Children's ages ranged from 5-10 years.	Questionnaire completed by parents derived from the International Study of Asthma and Allergies in Childhood. Asked questions of whether child has been diagnosed with asthma by a doctor (yes versus no)	Asthma diagnosed by medical examination	Age, sex, located district of residence, family history of atopy, ownership of the current residence, household environmental tobacco smoke, household dampness-related exposures, inspection season	OR=1.21, 95% CI [0.60, 2.45] for childhood asthma comparing 2nd quartile of formaldehyde exposure in child's bedroom to 1st quartile; OR=0.89, 95% CI [0.64, 1.24] for 3rd quartile; OR=1.09, 95% CI

								[0.86, 1.38] for fourth quartile
Madureira et al. 2016 (Cross-sectional and case-control)	A subset of children in the general population who were involved in a cross-sectional investigation recruited from 20 schools in Porto, Portugal	38 homes of asthmatic children and 30 homes from nonsymptomatic children; average age 8.5 years	Between October 2012–April 2013 visual inspections and air sampling were performed in all homes. Samples were collected in the rear of children's bedroom 1–1.5m above the floor. Outdoor samples were collected when possible at heights of 1–	Mean (cases): 14.6 ug/m <sup>3</sup> (sd=10.4); range= 3.68–50.7. Mean (controls): 16.6 ug/m <sup>3</sup> (sd=9.49); range= 5.22–43.3	Questionnaire based on the International Study of Asthma and Allergies in Childhood completed by legal guardians of children	Self-reported asthma symptoms—wheeze (ever wheeze, wheeze in the last 30 days), asthma in school. Reported on cases diagnosed with asthma by physician, but did not show data in publication.	No statistically significant differences in conditions between case and control groups, such as the presence of pets, use of air fresheners, incense stick, humidifiers, stuffed toys and smoking habits at home	p-value=0.199 comparing formaldehyde exposure between cases (asthmatic children) and control



			2m above ground-					
Madureira et al. 2015 (note same cohort of children as Madureira et al. 2015b, but more comprehensive, so combined to one record) (Cross-sectional and case-control)	Children in the general population attending 20 public primary schools and a subset of asthmatic children in Porto, Portugal. Investigation was conducted between 2011-2013.	1099 school children for classroom exposure measurements; 38 asthmatic children and 30 nonasthmatic children for home exposure measurements (ages 8-9 years)	School measurements: formaldehyde measured over 5 day period at breathing zone in 73 classrooms; Nov. 2011-Dec. 2012 and Nov. 2012-March 2013; Home measurements: formaldehyde measured over period of 7 days in rear of child's bedroom in Nov. 2011-Dec. 2012 and Nov. 2012-March 2013	Median (school): 17.5 ug/m <sup>3</sup> ; 25th percentile (school): 13.8 ug/m <sup>3</sup> ; 75th percentile (school): 23.1 ug/m <sup>3</sup> ; median (home): 11.4 ug/m <sup>3</sup> for cases; 14.8 ug/m <sup>3</sup> for controls	Parents completed paper-based questionnaire used in the International Study of Asthma and Allergies in Childhood; asthmatic cases answered yes to at least one question on asthma (doctor-diagnosed; wheezing in last year)	For school measurements: Asthma in school, doctor-diagnosed asthma; wheeze <30 d, wheeze <12 mo, ever wheeze; pulmonary function tests; for home measurements: asthma	No confounders or adjustment factors reported	No statistical comparison between formaldehyde levels between cases (asthmatic children) and controls

Raaschou-Nielsen et al. 2010 (Prospective Cohort)	Infants in the general population born to mothers with asthma participating in Copenhagen Prospective Study on Asthma in Childhood (COPSAC) in Copenhagen, Denmark	378 infants active in cohort at 18 months of age	Formaldehyde measured in children's bedrooms three times during first 18 months of life, for 10 weeks on each occasion	Mean: 20.3 ug/m <sup>3</sup> ; median: 17.7 ug/m <sup>3</sup> ; 5th-95th percentile: 7.9-36.7 ug/m <sup>3</sup>	Questionnaire completed in daily diary by parents when children between 6-18 months of age	Wheezing symptoms	Baseline lung function, education of mother, residential area, sex	OR=1.11, 95% CI [0.47, 2.63] for wheezing symptoms comparing 2nd quintile (12.4-16.3 ug/m <sup>3</sup> ) of formaldehyde exposure to 1st quintile (<12.4 ug/m <sup>3</sup> ); OR=1.21, 95% CI [0.51, 2.92] for 3rd quintile (16.3-20.3 ug/m <sup>3</sup> ) compared to first; OR=1.4, 95% CI [0.57, 3.47] for fourth quintile (20.3-25.6 ug/m <sup>3</sup> ) compared to first; OR=0.67, 95% CI [0.29, 1.54] for 5th
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								quintile ( $>25.6$ $\mu\text{g}/\text{m}^3$ ) compared to first
Venn et al. 2003 (Case- control)	Children in the general population in primary schools participating in a study of traffic pollution exposure and childhood in 1995/1996 in Nottingham City, United Kingdom	193 children with wheeze and 223 children with no reported wheeze (ages 9- 11 years)	Formaldehy de measured in child's bedroom for 3 days between 1998-1999	Range: 0- $>32 \mu\text{g}/\text{m}^3$	Daily symptom diary over 4 weeks	Persisting wheezing illness; frequent daytime and nighttime respiratory symptoms	Age, Carstairs deprivation index, sex	OR=0.47, 95% CI [0.17, 1.25] for frequent daytime respiratory symptoms comparing 2nd quartile (16.1-22 $\mu\text{g}/\text{m}^3$ ) of formaldehyd e exposure to 1st quartile ( $<16 \mu\text{g}/\text{m}^3$ ); OR=2, 95% CI [0.71, 5.65] for 3rd quartile (22.1-32 $\mu\text{g}/\text{m}^3$ ) compared to 1st; OR=2.08;

								95% CI [0.71, 6.11] for fourth quartile (>32 ug/m3) compared to 1st, p-value for trend=0.05; OR=1.4, 95% CI [0.54, 3.62] for frequent nighttime respiratory symptoms comparing 2nd quartile of formaldehyd e exposure to 1st quartile; OR=1.61, 95% CI [0.62, 4.19] for 3rd quartile compared to 1st; OR=3.33, 95% CI [1.23, 9.01] for fourth quartile compared to 1st, p-value
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									for trend = 0.02; OR=1.14; 95% CI [0.65, 2] for persistent wheezing symptoms comparing 2nd quartile of formaldehyd e exposure to 1st quartile; OR=1.08; 95% CI [0.62, 1.86] for 3rd quartile compared to 1st; OR=1.04; 95% CI [0.59, 1.82] for fourth quartile compared to 1st, p value for trend = 0.93.
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Choi et al. 2009 (Case-control)	Child patients in the general population with atopy recruited from outpatient clinic in Seoul, South Korea	36 children with allergic asthma (mean age 16.2 years) and 28 non-atopic controls (mean age 15.4)	Formaldehyde measured inside and outside subjects' homes over period from March to June 2006	Geometric mean (indoor): 42.46 ug/m <sup>3</sup> controls; 54.15 ug/m <sup>3</sup> allergic asthma cases; geometric mean (outdoor): 5.07 ug/m <sup>3</sup> controls; 9.35 ug/m <sup>3</sup> allergic asthma cases	Medical records for diagnosis of atopy; with skin prick tests, and IgE assays	Asthma	No confounders or adjustment factors reported	Geometric mean for formaldehyde exposure = 9.35 for allergic asthma cases; 5.07 for non-atopic controls, p-value non-significant
Garrett et al. 1999 (note Garrett et al. 1998 used same cohort so combined to one record) (Cross-sectional)	Children in households in the general population recruited for study in Latrobe Valley, Victoria, Australia	148 children (ages 7–14 years; mean age 10.2 years) from 80 households	Formaldehyde measured for four days in bedrooms of children; living rooms; kitchens; and outside the home between March–April, May, and September 1994 and	Median: 15.8 ug/m <sup>3</sup> ; maximum: 139 ug/m <sup>3</sup>	Questionnaire completed by parents included questions on respiratory symptoms in previous year for cough, shortness of breath, wheeze, asthma attacks and chest tightness	Asthma; respiratory symptom score	Parental allergy and parental asthma	Mean respiratory score = 1.09; 95% CI [0.42, 1.76] for formaldehyde exposure <20 ug/m <sup>3</sup> ; 2.21, 95% CI [1.7, 2.75] for formaldehyde exposure 20–50 ug/m <sup>3</sup> ; 2.59, 95% CI

			January- February 1995					<p>[1.67, 3.48] for formaldehyd e-exposure &gt;50 ug/m3. Proportion asthmatic=16 % for formaldehyd e-exposure &lt;20 ug/m3, 39% for formaldehyd e-exposure 20-50 ug/m3, 44% for formaldehyd e-exposure &gt;50 ug/m3. Bedroom formaldehyd e-exposure groups showed no significant differences between groups, but there were significant differences between highest recorded formaldehyd e-level</p>
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								groups (chi-square=6.84, df=2, p=0.03). A higher proportion of asthmatics was seen with higher formaldehyde exposure, with a significant linear trend present (p=0.02). Adjusted odds ratio for asthma was not significantly different from 1.0 (exact OR not provided).
Chatzidiakou et al. 2014 (Cross-sectional)	Children in the general population attending two primary state schools in the greater London area	151 children (mean age 10 years)	Formaldehyde measured in three classrooms and one outdoor site for 5 consecutive days during the heating	Average (suburban school): 32.70 ug/m3 (SD 4.0); average (urban school): 12.81	Standardized questionnaire completed by subjects	Asthma	Personal (gender, age, exposure to tobacco smoke, satisfaction with the school environment, and stress levels) and psychosocial	Urban schools had almost eight times higher asthma prevalence and asthmatic symptoms (12.6%) compared



				ug/m3 (SD 3.7)			factors. There is no information on which psychosocial factors were considered.	with suburban schools (1.6%), p< 0.001
Annesi Maesano et al. 2012 (Cross- sectional)	Children in the general population attending schools recruited into the Six Cities study in France	6590 children (mean age 10.4 years)	Formaldehy de measured in schools	Median: 26.8 ug/m3; 25th percentile: 16.8 ug/m3; 75th percentile: 33.2 ug/m3	Medical examination and ISAAC questionnaire completed by parents	Asthma (allergic and non-allergic) ever-past year; exercise- induced asthma	Paternal or maternal history of asthma and allergic diseases; passive smoking; gender, age	OR=1.1, 95% CI [0.87, 1.38] for asthma in the past year for 2nd tertile of formaldehyd e exposure (19.1-28.4 ug/m3) compared to 1st tertile (≤19.1 ug/m3). OR=0.9, 95% CI [0.76, 1.08] for 3rd tertile of formaldehyd e exposure (≥28.4 ug/m3) compared to 1st, p value for trend= 0.4428. OR=0.73, 95% CI

								<p>{0.51, 1.03} for nonallergic asthma in the past year for 2nd tertile of formaldehyd e-exposure compared to 1st tertile: OR=0.82, 95% CI {0.68, 0.99} for 3rd tertile of formaldehyd e-exposure compared to 1st, p value for trend= 0.32498, OR=1.31, 95% CI {1.01, 1.71} for allergic asthma in the past year for 2nd tertile of formaldehyd e-exposure compared to 1st tertile: OR=0.96, 95% CI {0.69, 1.35} for 3rd tertile</p>
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								of formaldehyd e-exposure compared to 1st, p-value for trend = 0.9542. Correlation between formaldehyd e-exposure and exercise- induced asthma = 0.018, p- value = 0.2257.
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Jeong et al. 2011 (Cross-sectional)	Children in the general population attending second grade in 56 elementary schools (11 in Incheon, Korea and 45 in Jeju, Korea)	1226 children attending Incheon schools (mean age 9.2 years) and 1748 children attending Jeju schools (mean age 9 years)	Formaldehyde measured in 11 Incheon schools and 2 Jeju schools as a single measurement in classrooms, cafeterias, infirmaries, playgrounds, and rooftops in December 2008	Mean outdoor exposure: 28.64 $\mu\text{g}/\text{m}^3$ (SD 65.26) for Incheon and 10.00 $\mu\text{g}/\text{m}^3$ (SD 0) for Jeju; mean indoor exposure: 279.44 $\mu\text{g}/\text{m}^3$ (SD 23.83) for Incheon and 196.67 $\mu\text{g}/\text{m}^3$ (SD 87.31) for Jeju	ISAAC questionnaire completed by parents	Ever asthma; asthma treatment over last 12 months; ever wheezing or whistling; wheezing over last 12 months	Study groups were not statistically different in sex, height, and weight. Study schools where in industrial and non-industrial areas (proxies for SES), parental history of asthma, and age were also reported. Analyses were unadjusted t-tests and chi-squared tests.	N=159 (13.13%) with ever diagnosis of asthma for children living in Incheon (high levels of formaldehyde exposure) compared to N=230 (13.38%) children living in Jeju; p-value=0.47. N=50 (4.19%) with asthma treatment in last 12 months for children living in Incheon compared to N=64 (3.78%) children living in Jeju; p-value=0.57. N=304
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									(24.96%) with ever wheeze or whistling for children living in Incheon compared to N=321 (18.80%) children living in Jeju, p- value<0.01. N=116 (9.50%) with wheezing in last 12 months for children living in Incheon compared to N=115 (6.83%) children living in Jeju, p- value<0.01.
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Delfino et al. 2003 (Cross-sectional)	Children in the general population with physician-diagnosed asthma recruited through referrals from area schools in East Los Angeles County, California	24 asthmatic children (between 10-15 years of age)	Formaldehyde measured by outdoor stationary monitoring stations between November 1999-January 2000	Mean: 7.21 ppb (SD 2.41; range 4.27-14.02); interquartile range: 3-16 ppb; 90th percentile: 10.09 ppb	Questionnaire completed by subjects and included daily asthma symptoms (severity scale) and number of inhaler puffs	Asthma symptoms	Study exclusion criteria used to limit confounders such as active and passive smoking, and selected non-working Hispanic children. Confounding by weekend versus weekday, maximum temperature, and respiratory infections was also accounted for. All families in the study had low SES.	OR=1.3, 95% CI [0.33, 5.02] for bothersome or more severe asthma symptoms per 7.21 ppb change (mean) in formaldehyde exposure; lag day 0. OR=7.3, 95% CI [1.46, 36.4] for bothersome or more severe asthma symptoms per 7.21 ppb change (mean) in formaldehyde exposure; lag day 1. OR=2.27, 95% CI [0.43, 11.9] for asthma symptoms that interfered with daily
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								activities-per 7.21ppb change (mean)-in formaldehyd e-exposure; lag-day-0: OR=2.64; 95% CI [1.12, 6.21] for asthma symptoms that interfered with daily activities-per 7.21ppb change (mean)-in formaldehyd e-exposure; lag-day-1.
Dannemiller et al. 2013 (Cross- sectional)	Children in the general population participating in formaldehyde monitoring study in primarily low-income homes in Boston, Massachusetts	37 children (<18 years old)	Formaldehy de measured in homes between July 2008 and February 2010	Geometric mean: 35.1 ppb (SD 1.98; range 5-132 ppb)	Questionnaire; completed by children >12 and parent for <12	Asthma Control Test <12 (very poor control)	Age, gender, race, sources of ammonia and Nox in home. All participants were primarily low income. Data on type of housing, age of building, home ownership, and resident smoking habits	Geometric mean of formaldehyd e concentration for those with overall Asthma Control Test (ACT) <12 (very poor control) = 54.0 ppb. Geometric

							were recorded and considered.	mean formaldehyde for all other groups=34.4ppb, p-value=0.078.
Tuthill 1984 (Cross-sectional)	Children in the general population attending a school system in Western Massachusetts	399 children (youngest in household)	Formaldehyde exposure classified (Y/N)-based on interview (new construction or remodeling, new upholstered furniture, foam insulation, living in mobile home) in April 1983	Unknown	Phone interview completed by parents; questions included number of colds in school year, symptom checklist, length of episode and days of school missed, physician-diagnosed chronic bronchitis, asthma, or allergies	Respiratory episodes (greater than or equal to 2)	Authors collected information on smoking, SES, education, age, sex, number of siblings, and woodstove exposures. Unclear how these factors were used for as potential adjustment factors.	Relative Risk=2.4, 95% CI [1.7, 3.4] for more than two respiratory episodes comparing those exposed versus those not exposed to formaldehyde.



Lajoie et al. 2015 (Randomized controlled trial)	Children in the general population diagnosed with asthma at the Mother Child Centre and living in the greater Quebec area in Canada	83 asthmatic children (ages 4–13 years)	Children randomly allocated to intervention and non-intervention groups and monitored for one year in pre-intervention stage and for one year in the post-intervention stage and formaldehyde measured in the child's bedroom over 6–8 day period 2009–2011	Mean (fall/winter): 37.4 ug/m <sup>3</sup> (95% CI 32.3–43.3)	Parents were interviewed and completed ISAAC questionnaire on respiratory symptoms including daily symptoms diary	Severe wheezing, effort wheezing, episodes of wheezing, hospitalizations, emergency room visits	Age, eczema	Change in annual prevalence of severe wheezing=1.5%, 95% CI [-20, 23.1] associated with 50% reduction of formaldehyde level from baseline, p-value 0.888. Change in annual prevalence of effort wheezing=9.1%, 95% CI [-22.4, 4.1] associated with 50% reduction of formaldehyde level from baseline, p-value 0.173. Change in annual prevalence of ≥1 episode of wheezing=14.8%, 95%
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									<p>CI [-28.6, -0.9] associated with 50% reduction of formaldehyde level from baseline, p-value 0.037. Change in annual prevalence of <math>\geq 4</math> episode of wheezing = 7.2%, 95% CI [-19.6, 5.3] associated with 50% reduction of formaldehyde level from baseline, p-value 0.255. Change in annual prevalence of <math>\geq 1</math> emergency room visit = 1.6%, 95% CI [-30.5, 1.5] associated with 50% reduction of</p>
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								formaldehyd e-level from baseline, p- value 0.031. Change in annual prevalence of $\geq 1$ hospitalizatio n=7.9%, 95% CI [- 20.6, 4.6] associated with 50% reduction of formaldehyd e-level from baseline, p- value 0.218.
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Marks et al. 2010 (Non-randomized controlled trial)	Children in the general population in grades 4, 5, or 6 attending 22 schools in Blue Mountains, Southern Highlands, and Goulburn regions of NSW in Australia	400 school children (mean age 11 years)	Classrooms were alternatively heated with a low-NO <sub>x</sub> unflued-gas heater and with a flued-gas heater for 3 weeks each; formaldehyde measured in classrooms June–September 2009	Overall: 28.6 ppb; flued heater: 24.7 ppb; unflued heater: 32.6 ppb	Daily symptom and medication diary	Evening wheeze; morning wheeze; evening symptoms; multiple symptoms; morning symptoms; cough or wheeze; bronchodilator use; asthma medication; pulmonary function tests	Exposure to environmental tobacco smoke; home use of gas, open fire for heating (asthma symptoms). Clustering by subject and school, day of the week, study week (pulmonary measures).	OR=1.123, 95% CI [0.856, 1.473] for evening wheeze comparing unflued-gas heater to flued-gas heater exposure. OR=1.603, 95% CI [1.171, 2.194] for morning wheeze comparing unflued-gas heater to flued-gas heater exposure. OR=0.963, 95% CI [0.852, 1.089] for evening symptoms comparing unflued-gas heater to flued-gas heater exposure.
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								<p>OR=0.938, 95% CI [0.801, 1.098] for morning symptoms (cough or wheeze) comparing unflued gas heater to flued gas heater exposure. OR=0.89, 95% CI [0.596, 1.329] for use of bronchodilat or comparing unflued gas heater to flued gas heater exposure. Difference between unflued and flued heater exposure FEV1 in the morning mean=0.004, 95% CI [- 0.009;</p>
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								0.017]; Difference between unflued and flued heater exposure FEV1 in the evening mean=0, 95% CI [- 0.014, 0.014]; Difference between unflued and flued heater exposure PEF in the morning mean=0.719, 95% CI [- 1.239, 2.677]; Difference between unflued and flued heater exposure PEF in the evening mean=0.994, 95% CI [- 0.995, 2.983];
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Jacobsen et al. 2009 (Prospective Cohort)	Occupational cohort of adult workers exposed in woodworking factories and unexposed workers from control factories in Viborg, Denmark	1377 woodworkers and 297 control workers (male mean age 38.4 years; female mean age 37.8 years)	Formaldehyde measured using personal monitors in 24 samples from 10 factories in 2003–2005	Median: 0.05 mg/m <sup>3</sup> (range 0.03–0.2)	Questionnaire completed by subjects included questions from ECRHS; ever asthma defined as current or ever self-reported asthma; asthma symptoms defined as yes to at least one group A question (doctor-diagnosed asthma; ever had asthma; current asthma; wheeze) and 2 or more group B questions (chest tightness; wake with chest tightness; wake with wheezing; cough in morning; wheeze in cold air; wheeze with exercise; wheeze from pollen; wheeze from animals; asthma medication)	Current asthma; ever asthma; asthma symptoms; ever wheezing	Age, baseline hay fever, smoking	OR=1.5, 95% CI [0.34, 6.51] for current asthma comparing male woodworkers to male reference workers. OR=6.89, 95% CI [0.85, 55.8] for current asthma comparing female woodworkers to female reference workers. OR=1.9, 95% CI [0.44, 9.12] for ever asthma comparing male woodworkers to male reference workers. OR=3.37, 95% CI [0.91, 12.5] for ever
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								<p>asthma  comparing  female  woodworkers  to female  reference  workers.  OR=0.73,  95% CI [0.4,  1.33] for ever  wheeze  comparing  male  woodworkers  to male  reference  workers.  OR=1.58,  95% CI  [0.73, 3.42]  for ever  wheeze  comparing  female  woodworkers  to female  reference  workers.  OR=0.75,  95% CI  [0.39, 1.45]  for  respiratory  symptoms  comparing  male</p>
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								woodworkers to male reference workers: OR=1.31, 95% CI [0.6, 2.83] for respiratory symptoms comparing female woodworkers to female reference workers:
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Kilburn et al. 1985 (Prospective Cohort)	Occupational cohort of adult male fiberglass batt makers; histology technicians and hospital workers in California	20 exposed batt makers; 20 unexposed batt makers; 18 histology technicians; 26 unexposed hospital workers (ages 20-62 years)	Formaldehyde measured for histology workers; and batt makers were assumed to have higher levels; self-administered questionnaire asked about exposures to formaldehyde and competing or confounding exposures	Range (histology): 0.4-1.9 ppm; assumed higher exposure levels for batt makers	Questionnaire completed by subjects included questions on respiratory disease history and symptoms	Asthma; breathlessness; wheezing; chest tightness and pain/burning; shortness of breath at work; shortness of breath at rest; respiratory mean score; pulmonary function tests	No confounders or adjustment factors reported	Incidence asthma for hot batt makers=5% (n=20); cold batt makers=15% (n=25); histology=6% (n=18); comparison group=9% (n=26). Incidence breathlessness for hot batt makers=35%; cold batt makers=44%; histology=6%; comparison group=12%. Incidence wheezing for hot batt makers=50%; cold batt makers=36%; histology=6%; comparison group=12%. Incidence chest tightness for
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								<p>hot batt makers=50%; cold batt makers=40%; histology=1+ %; comparison group=0%. Incidence shortness of breath at rest for hot batt makers=30%; cold batt makers=24%; histology=6 %; comparison group=0%. Incidence shortness of breath at work for hot batt makers=40%; cold batt makers=40%; histology=1+ %; comparison group=4%. Mean respiratory mean score for hot batt makers=5.8;</p>
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								<p>cold-batt makers=4.9; histology=1.6; comparison group=0.8. Percentage of workers who decreased FVC by 5% or more of their before-shift values for hot-batt makers (none-cigarette smokers)=22.2%; bat makers (cigarette smokers)=8.6%; p-value&lt;0.01. Percentage of workers who decreased FEV1 by 10% or more of their before-shift values for hot-batt makers (none-cigarette smokers)=33.3%; bat makers (cigarette</p>
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								<p>smokers)=11.4%, p-value&lt;0.01. Percentage of workers who decreased FEF25-75 by 15% or more of their before-shift values for hot batt makers (nonsmokers)=33.3%, bat makers (cigarette smokers)=11.4%, p-value&lt;0.01. Percentage of workers who decreased FEF75-85 by 15% or more of their before-shift values for hot batt makers (nonsmokers)=22.2%, bat makers (cigarette smokers)=40</p>
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								%, p-value<0.01.
Frisk et al. 2006 (Case-control)	Adults in the general population selected from the Orebro, Sweden section of the 1996 FinEsS study	49 asthmatics and 48 non-asthmatic controls (ages 15-49 years)	Formaldehyde measured as mean 24-hour concentration in the bedroom between January-April 1999, October 1999-January 2000	Mean: 23 ug/m3 (range 7-98)	Questionnaire completed by subjects; cases were people who replied "yes" to all questions on asthma (doctor diagnosis; asthma medications; asthma attacks/breathlessness in last 10 years and/or 12 months; wheezing within last year)	Asthma	Matched cases and controls based on age group, gender, and type of accommodation.	Mean 24hr formaldehyde levels in bedroom=27 ug/m3 for asthma cases in single family housing; 12ug/m3 for asthma cases in multi-family housing, p-value=0.009. Mean 24hr formaldehyde levels in bedroom=26 ug/m3 for controls in single family housing; 14ug/m3 for controls in multi-family housing, p-value=0.002.

								Mean 24hr formaldehyde levels in bedroom=33 ug/m3 for asthma cases in homes built before 1975, 19ug/m3 for asthma cases in homes built after 1975, p-value=0.014.
Billionnet et al. 2011 (Cross-sectional)	Adults and children in the general population living in homes identified from population-based sample of French households	1012 individuals over 15 years of age (median age 44 years)	Formaldehyde measured for one week in the bedroom of the reference person of the household; survey took place between October 2003 and December 2005	Median: 19.4 ug/m3 (range 1.3–86.3)	Questionnaire completed by subjects; asthma in past year determined based on subjects answering "yes" to woken by shortness of breath or asthma attack in last year or current asthma medication (definition suggested by ECRHS)	Asthma	Age, highest educational level, outdoor pollution, presence of mold, presence of pets, relative humidity, sex, smoking, time of survey	OR=1.43 for asthma in the past year comparing high ( $\geq 28.03 \text{ ug/m}^3$ ) to low ( $< 28.03 \text{ ug/m}^3$ ) formaldehyde exposure

Mapou et al. 2013 (Cross-sectional)	Adults in the general population participating in Relationship of Indoor, Outdoor and Personal Air (RIOPA) Study in communities in Los Angeles County, CA, Elizabeth, NJ, and Houston, TX	90 adults	Formaldehyde measured in personal passenger vehicles from July 1999-February 2001	Median: 20.0 mg/m <sup>3</sup> (range <4.65-1095.6)	Self-reported doctor-diagnosed	Asthma	In general, authors adjusted for type of vehicle driven, season in which sampling occurred, total minutes driven, and relative humidity but authors did not state which of these factors was included in the analysis of formaldehyde and asthma association. Authors reported on the gender, education level, and household income.	OR=1.03, p-value=0.054 for self-reported doctor-diagnosed asthma per 1 ug/m <sup>3</sup> change in formaldehyde exposure in vehicle. Correlation=0.27, p-value=0.004 between formaldehyde exposure (measured inside vehicles) and self-reported doctor-diagnosed asthma
Fornander et al. 2014 (Cross-sectional)	Adult metalworkers exposed to metal working fluids in an occupational setting in Sweden	271 exposed subjects and 24 non-exposed controls	Formaldehyde measured using both stationary and personal monitors	Mean (stationary): 0.04 mg/m <sup>3</sup> ; mean (personal): 0.1 mg/m <sup>3</sup>	Questionnaire with asthma defined as "have or have had" (unclear if asthma was diagnosed or self-reported)	Asthma	No confounders or adjustment factors reported	Number and percent incidence of asthma cases by formaldehyde exposure group: 102 (11%) exposed directly, 169



								(15%) exposed indirectly, 24 (17%) not exposed
Zammit- Tabona et al., 1983 (Cross- sectional)	Adult workers with bronchial hyperreactivity and respiratory symptoms occupationally exposed at foundries in British Columbia, Canada	11 symptomatic workers (mean age 44.8 for reactors to challenge ; 41.3 for nonreactors to challenge )	Subjects exposed to 2.5ppm formaldehyde for 30 min in challenge test performed at least 1 week after the last occupational exposure on two separate days	Exposed to 2.5 ppm		Pulmonary function tests	Authors evaluated differences in study participants for characteristics age, smoking, atopic status	Mean FVC=84.3, 95% CI [71.07, 97.53] for reactors; mean FVC=96.7, 95% CI [86.2, 107.2] for nonreactors; reported no statistically significant difference. Mean MMFR=41.6 , 95% CI [26.53, 56.67] for reactors; mean MMFR=73.3 , 95% CI [50.23, 96.37] for nonreactors; p-value for

								<p>difference reported</p> <p>&lt;0.05. Mean total lung capacity=97, 95% CI [81.56, 112.44] for reactors; mean total lung capacity=99.8, 95% CI [94.07, 105.53] for nonreactors; reported no statistically significant difference.</p> <p>Mean residual volume=136.6, 95% CI [98.22, 174.98] for reactors; mean residual volume=107.7, 95% CI [88.92, 126.48] for nonreactors; reported no statistically</p>
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								<p>significant difference. Mean diffusing capacity=107.3, 95% CI [88.25, 126.35] for reactors; mean diffusing capacity=109.2, 95% CI [81.19, 137.21] for nonreactors; reported no statistically significant difference.</p>
Frey et al. 2014 (Cross-sectional)	Senior adults in the general population living in a single low-income senior housing building in	72 senior residents (56 nonsmokers)	Formaldehyde measured in each apartment unit and outdoors for one hour between	Range (indoor): 10-80 ppb; median (living room): 36.9 ppb; median (kitchen):	Questionnaire (unclear if asthma was diagnosed or self-reported)	Asthma	No confounders or adjustment factors reported	Authors report that 3 of 16 smokers and 6 of 56 nonsmokers reported asthma

	Phoenix, Arizona		June-July 2010	38.8 ppb; median (outdoor): 4.3 ppb				
Wieslander et al. 1997 (Cross-sectional)	Adults in the general population randomly selected from population register in Uppsala, Sweden	562 adults (mean age 32 years)	Formaldehyde measured in the bedroom of a random sample of dwellings of 62 participants	Mean (wall/ceiling painted): 16 ug/m3 (yes) and 21 ug/m3 (no); mean (wood painted): 32 ug/m3 (yes) and 17 ug/m3 (no); mean (kitchen painted): 18 ug/m3 (yes) and 20 ug/m3 (no); mean (bedroom painted): 24 ug/m3 (yes) and 19 ug/m3 (no); mean (bathroom painted): 19 ug/m3 (yes) and	International Union Against Tuberculosis and Lung Disease questionnaire completed by subjects; current asthma defined as combination of bronchial hyperresponsiveness and at least one symptom related to asthma in last year; symptoms included wheezing, shortness of breath, nighttime awakening from breathlessness or tightness of chest	Asthma, wheezing, at least one asthma symptom, daytime and nocturnal breathlessness, pulmonary bronchial hyperresponsiveness	Age, gender, smoking	OR=1.56, 95% CI [0.98, 2.48] for increased prevalence of newly painted dwelling for those reporting asthma v. those not. OR=1.13, 95% CI [0.63, 2.01] for increased prevalence of newly painted workplace for those reporting asthma v. those not. OR=2.33, 95% CI [1.22, 4.46] for increased prevalence of wood painted

				20 ug/m3 (no)				for those reporting asthma v. those not. OR=2.21, 95% CI [1.09, 4.51] for increased prevalence of kitchen painted for those reporting asthma v. those not. OR=1.21, 95% CI [0.83, 1.76] for increased prevalence of newly painted dwelling for those reporting wheezing v. those not. OR=1.6, 95% CI [1.02, 2.52] for increased prevalence of newly painted workplace for those
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								reporting wheezing v. those not. OR=1.6, 95% CI [0.92, 2.78] for increased prevalence of wood painted for those reporting wheezing v. those not. OR=1.7, 95% CI [0.92, 3.16] for increased prevalence of kitchen painted for those reporting wheezing v. those not. OR=1.16, 95% CI [0.75, 1.79] for increased prevalence of newly painted dwelling for those reporting daytime breathlessness
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								s v. those not. OR=1.6, 95% CI [0.96, 2.67] for increased prevalence of newly painted workplace for those reporting daytime breathlessnes s v. those not. OR=1.94, 95% CI [1.07, 3.5] for increased prevalence of wood painted for those reporting daytime breathlessnes s v. those not. OR=1.66, 95% CI [0.84, 3.3] for increased prevalence of kitchen painted for those reporting daytime breathlessnes
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								s v. those not. OR=1.57, 95% CI [1.05, 2.36] for increased prevalence of newly painted dwelling for those reporting nocturnal breathlessness s v. those not. OR=1.35, 95% CI [0.82, 2.22] for increased prevalence of newly painted workplace for those reporting nocturnal breathlessness s v. those not. OR=1.75, 95% CI [0.98, 3.14] for increased prevalence of wood painted for those reporting nocturnal
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								<p>breathlessness v. those not.  OR=2.67,  95% CI  [1.42, 5.04]  for increased  prevalence of  kitchen  painted for  those  reporting  nocturnal  breathlessness  s v. those not.  OR=1.43,  95% CI  [1.01, 2.06]  for increased  prevalence of  newly  painted  dwelling for  those  reporting at  least one  asthma  symptom v.  those not.  OR=1.63,  95% CI  [1.05, 2.54]  for increased  prevalence of  newly  painted  workplace</p>
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								<p>for those reporting at least one asthma symptom v. those not: OR=1.8, 95% CI [1.04, 3.12] for increased prevalence of wood painted for those reporting at least one asthma symptom v. those not: OR=2.24, 95% CI [1.2, 4.21] for increased prevalence of kitchen painted for those reporting at least one asthma symptom v. those not: OR=1.37, 95% CI [0.88, 2.13] for increased prevalence of</p>
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								newly painted dwelling for those reporting bronchial hyperresponsiveness v. those not: OR=1.25, 95% CI [0.73, 2.14] for increased prevalence of newly painted workplace for those reporting bronchial hyperresponsiveness v. those not: OR=2, 95% CI [1.06, 3.76] for increased prevalence of wood-painted for those reporting bronchial hyperresponsiveness v. those not: OR=2.14,
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								95% CI [1.08, 4.23] for increased prevalence of kitchen painted for those reporting bronchial hyperrespons iveness v. those not.
Matsunaga et al. 2007 (Cross- sectional)	Pregnant adult women in the general population from Osaka Maternal and Child Health Study in multiple municipalitie s in Japan	998 pregnant women	Formaldehy de measured using personal monitors November- March 2003	Median: 24 ppb; maximum: 131 ppb	Questionnaire completed by subjects and included questions on asthma; asthma considered present if received medical treatment during last year	Current asthma	Age, allergic rhinitis, atopie eczema, current passive smoking at home and work, education, family history of allergy, family income, gestation, indoor domestic pets, mite antigen in house dust, mold in the kitchen, parity, season, smoking	OR=0.8, 95% CI [0.23, 2.84] for current asthma comparing second quartile of formaldehyd e exposure (18-27 ug/m3) compared to first quartile (<18 ug/m3); OR=0.72, 95% CI [0.19, 2.77] for current asthma comparing third quartile of

								formaldehyd e-exposure (28-46 ug/m3) compared to first quartile; OR=2.15; 95% CI [0.41, 11.28] for current asthma comparing fourth quartile of formaldehyd e-exposure (≥47 ug/m3) compared to first quartile; p-value for trend=0.47; OR=2.65; 95% CI [0.63, 11.11] for current asthma comparing above 90th percentile formaldehyd e-exposure (≥47 ug/m3) to those below. Authors
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								report no statistically significant difference.
Elshaer et al. 2017 (Cross-sectional)	Adult medical students exposed to formaldehyde in and occupational setting during dissections and staff members and workers within the Anatomy department at Alexandria University (Egypt)	454 medical students in their first, second or third year and 16 exposed staff members and workers	Subjects classified as exposed to formalin or not exposed	Not measured—categorized as exposed versus not exposed	Students, staff and workers were subjected to a self-administered predesigned questionnaire to collect information on asthma-related outcomes	Work-related bronchial asthma; exacerbation of pre-existing bronchial asthma	No confounders or adjustment factors reported	Number (percentage) reporting prevalence of work-related bronchial asthma=8 (53.3%) for those exposed to formaldehyde, 0 (0%) for those not exposed to formaldehyde. Number (percentage) reporting

								exacerbation of pre- existing bronchial asthma=7 (46.7%) for those exposed to formaldehyd e, 0 (0%) for those not exposed to formaldehyd e.
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Thetkathuek et al. 2016 (Cross-sectional)	Adult employees exposed to formaldehyde in an occupational setting at a MDF furniture factory in Thailand	432 volunteers (out of 535 factory workers)	Formaldehyde measured from five work sites in factory for 24 continuous hours on two separate days in March and April 2012	Formaldehyde exposures were classified as Low (0.66–3.44 ppm), Moderate (3.45–6.89 ppm), or High (>6.89 ppm)	Questionnaire based on the American Thoracic Society Respiratory Symptoms Questionnaire, adjusted to fit working conditions in the furniture factory. Questionnaires were completed by the study subjects independently.	Atopic allergic asthma; asthma symptoms (wheeze)	Authors consider variables: education; atopic eczema; allergic asthma; allergic rhinitis history; family history; formaldehyde concentrations or MDF dust concentrations; but unclear whether these are the confounder variables in the adjusted analysis reported.	Number (percentage) reporting prevalence of having wheeze during the daytime or nighttime=56 (15%) for low formaldehyde exposure (0.66–3.44 µg/m <sup>3</sup> ), 2 (4.5%) for moderate formaldehyde exposure (3.45–6.89 µg/m <sup>3</sup> ), and 4 (18.2%) for high formaldehyde exposure (>6.89 µg/m <sup>3</sup> )
Low et al. 1985 (Cross-sectional)	Adult workers exposed to formaldehyde and unexposed controls in an occupational setting at a	46 exposed workers and 17 controls (mean ages range from	Formaldehyde exposure classified based on job	Core shop: not detected; general foundry: 2–4 ppm; shell: <LOD (1 ppm)	Modified standardized questionnaire from Medical Research Council completed by subjects	Asthma or wheeze onset before at foundry and while at foundry; wheeze at work;	Authors reported no differences in cigarette smoking between comparison groups, but noted some	Number (number attributing to specific environmental factor at work) of prevalence of asthma or



	foundry in Brisbane, Australia	25.3-39.1 years based on work area)				pulmonary function tests	differences in age	wheeze-onset while at foundry=3 (1); onset before at foundry=1 (0); wheezing while at work 1. FVC measured on Monday morning mean=91.4, 95% CI [85.89, 96.91] for aftereast (not formaldehyd e-exposed) versus FVC measured on Monday morning mean=84.1, 95% CI [77.88, 90.32] for general foundry workers (formaldehyd e-exposed); not statistically significant. Change in
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								FEV1 measured on Monday and Friday mean= 8 mL, 95% CI [- 92.8, 76.8] for aftercast versus change in FEV1 measured on Monday and Friday mean= 4 mL, 95% CI [- 62.49, 70.49] for general foundry workers, not statistically significant. Change in FEV1 over the work week mean=- 15 mL, 95% CI [- 201.55 171.55] for aftercast versus change in FEV1 over the work week mean=- 105 mL, 95%
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								CI [-220.82, 10.82] for general foundry workers, not statistically significant. FEV1/FVC% measured on Monday morning mean=80.4, 95% CI [-76.58, 84.22] for aftercast versus FEV1/FVC% measured on Monday morning=83. 1 mL, 95% CI [-78.81, 87.39] for general foundry workers, not statistically significant. FEV1 measured on Monday morning mean=89.5, 95% CI [82.93,
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								96.07] for aftercast versus FEV1 measured on Monday morning mean=84.6, 95% CI [76.66, 92.54] for general foundry workers, not statistically significant. Change in FEV1 measured at beginning and end of Monday mean= -9 mL, 95% CI [- 115, 97] for aftercast versus FEV1 measured at beginning and end of Monday mean= -57, 95% CI [- 187.83, 73.83] for general foundry
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								workers, not statistically significant.
Fransman et al. 2003 (Cross-sectional)	Adult plywood mill workers in an occupational setting in New Zealand	112 workers and 415 general population controls (mean of 4.7 years employed at mill for group of workers; mean age 34.5 yr)	Formaldehyde measured using personal monitors	Geometric mean: 0.08 ug/m <sup>3</sup> (SD 3.0); pressing section (high sample): 0.16 ug/m <sup>3</sup> (SD 2.7)	Questionnaire completed by subjects and included questions on respiratory health symptoms; asthma prevalence estimated using ECRHS definition, which is based on proportion of subjects answering "yes" to woken by shortness of breath or asthma attack in last year or current asthma medication	Asthma; wheezing; shortness of breath or wheezing or chest tightness related to work; asthma medication; asthma attack; woken by shortness of breath; shortness of breath with wheezing; wheezing without a cold	Age, gender, race/ethnicity, smoking	OR=4.3, 95% CI [0.7, 27.7] for asthma comparing high formaldehyde exposure group ( $\geq 0.08$ mg/m <sup>3</sup> ) to low exposure (<0.08 mg/m <sup>3</sup> ). OR=0.4, 95% CI [0, 5.4] for wheezing; shortness of breath or chest tightness related to work comparing high formaldehyde exposure group to low exposure. OR=1, 95% CI [0.1, 15.3] for asthma medication

								<p>use comparing high formaldehyd e-exposure group to low exposure. OR=1.6, 95% CI [0.2, 13.2] for wheezing without a cold comparing high formaldehyd e-exposure group to low exposure. OR=3.5, 95% CI [0.6, 19.1] for shortness of breath with wheezing comparing high formaldehyd e-exposure group to low exposure. OR=9.5, 95% CI [1.2, 74.7] for woken by shortness of breath comparing</p>
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								high formaldehyd e-exposure group to low exposure.
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Malaka et al. 1990 (Cross-sectional)	Male workers exposed to formaldehyde and nonexposed controls at plywood plant in East Java, Indonesia	55 exposed male workers (mean age 26.6 years) and 50 unexposed male controls (mean age 28.8 years)	Estimate of cumulative formaldehyde exposure calculated from area concentrations and length of service in current job	Mean (exposed group): 6.29 ppm-yr (SD 2.72); range (area concentrations): 0.22-3.48 ppm	Standardized respiratory questionnaire from American Thoracic Society completed by subjects	Asthma; occupational asthma; wheezing; shortness of breath; pulmonary function tests	Age, dust, smoking status, cigarettes per day, weight, height	OR=6.31 for asthma comparing those exposed to formaldehyde versus not; p-value=0. OR=2.84 for occupational asthma comparing those exposed to formaldehyde versus not; p-value=0.02. OR=1.98 for shortness of breath comparing those exposed to formaldehyde versus not; p-value=0.04. OR=1.2 for wheezing comparing those exposed to formaldehyde versus not; p-
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								<p>value=0.36.  Mean  FEV1=2.78L  ,95% CI  [2.7, 2.86]  comparing  those  exposed to  formaldehyd  e versus not,  p-  value=0.001.  Mean  FEV1/FVC=  3%  comparing  those  exposed to  formaldehyd  e versus not.  Mean  FEF25%-  75%=3.44L/s  ,95% CI  [3.28, 3.6]  for those not  exposed to  formaldehyd  e, mean  FEF25%-  75%=3.04L/s  ,95% CI  [2.88, 3.2]  for those  exposed to  formaldehyd</p>
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								<p>e, p-value=0. Multiple regression coefficient=0.043 for FEF25%-75% for continuous formaldehyde exposure; p-value&lt;0.05.</p>
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Pourmababadian et al. 2006 (Cross-sectional)	Adult workers in an occupational setting at 7 hospitals of Tehran University of Medical Sciences in Tehran, Iran	180 exposed workers from pathology labs (n=38), surgery rooms (n=65), and endoscopy (n=21) and 56 unexposed controls working in administrative affairs section	Formaldehyde measured as 8-hour continuous and spot samples in different departments of 7 hospitals in 2002-2003	Mean 8-hour sample: 0.96 ppm (pathology); 0.13 ppm (endoscopy); 0.25 ppm (surgery)	Questionnaire completed by subjects	Asthma, chest tightness, pulmonary function tests	Report on smoking, sex and age but do not adjust results for covariates	Percentage reporting asthma=7.9% (pathology); 19% (endoscopy); 1.5% (surgery) versus 5.4% (nonexposed). No statistical association reported. Percentage reporting chest tightness=31.5% (pathology); 28.6% (endoscopy); 27.7% (surgery) versus 16.1% (nonexposed). No statistical association reported. FEV1 mean=2.3L; 95% CI [2.12, 2.48] for those exposed to
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								formaldehyd e-versus mean=2.9L, 95%-CI [2.58, 3.22] for those not exposed to formaldehyd e, p- value<0.001. FVC mean=3.3L, 95%-CI [3.12, 3.48] for those exposed to formaldehyd e-versus mean=4L, 95%-CI [3.68, 4.32] for those not exposed to formaldehyd e, p- value<0.001. FEV1/FVC mean=69.7% ,95%-CI [66.02, 73.38] for those exposed to formaldehyd e-versus mean=72.5%
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Akbar Khanzadeh et al. 1994 (Cross-sectional)	Adult medical students exposed to formaldehyde in an occupational setting at an anatomy lab in Toledo, OH	34 exposed subjects (mean age 26 years) and 12 nonmedical student controls (mean age 31.5 years)	Formaldehyde in breathing zone (personal sample) and general area anatomy lab measured on 9 days of work over period of 6 weeks in fall 1992	Mean time weighted average (breathing zone): 1.24 ppm (SD 0.61; range 0.07-2.94); time weighted average (cavity): 0.49 ppm (SD 0.18); time weighted average (surface): 0.35 ppm (SD 0.13); time weighted average (area): 1.65 ppm (SD 0.92; range 1.00-2.30)	Questionnaire completed by subjects including questions from Medical Research Council standardized questionnaire; acute symptoms recorded prior to and following laboratory session	Asthma; shortness of breath; wheezing; pulmonary function tests	All subjects were nonsmokers; height and weight were similar between exposed versus non-exposed. Slight differences in age and ethnicity.	Prevalence=7% for asthma for those exposed to formaldehyde versus 0% for those not exposed. No statistical association reported. Prevalence=7% for persistent wheezing for those exposed to formaldehyde versus 0% for those not exposed. No statistical association reported. Prevalence=2% for persistent shortness of breath for those exposed to formaldehyde versus 1% for those not exposed. No statistical
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								<p>association reported.</p> <p>Percent acute change in FVC=1.4, 95% CI [-2.94, 0.14] for those exposed to formaldehyde versus -0.3, 95% CI [-3.22, 2.62] for those not exposed to formaldehyde, p-value&lt;0.1.</p> <p>Percent acute change in FEV1= 0.03, 95% CI [-1.22, 1.16] for those exposed to formaldehyde versus 1, 95% CI [-1.54, 3.54] for those not exposed to formaldehyde.</p> <p>Percent acute change in FEV3=1.2, 95% CI</p>
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									<p>[-2.67, 0.27] for those exposed to formaldehyde versus 1.3, 95% CI [-0.79, 3.39] for those not exposed to formaldehyde. Percent acute change in FEF25=75%=-2.5%, 95% CI [-0.54, 5.54] for those exposed to formaldehyde versus 2.31, 95% CI [0.59, 4.03] for those not exposed to formaldehyde. Percent acute change in FEV1/FVC=1.6%, 95% CI [0.27, 2.93] for those exposed to formaldehyde versus</p>
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								0.6%, 95% CI [-1.24, 2.44] for those not exposed to formaldehyd e, p- value<0.05.
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Uba et al. 1989 (Prospective Cohort)	Adult medical students exposed to formaldehyde in an occupational setting in an anatomy lab at the University of Southern California	103 students in-class of 1988 (81 students completed questionnaire after exposure to formaldehyde in anatomy lab and after control laboratory with no formaldehyde exposure) (ages 21-33 years; mean age 24.3 years)	Formaldehyde measured using personal samplers in breathing zones of students during anatomy laboratory in September 1984-April 1985	Time weighted average: <1 ppm; peak exposures: <5 ppm; mean (while dissecting): 1.9 ppm (range 0.1-5.0); mean (while observing dissection): 1.2 ppm (range 0.2-2.0)	Questionnaire completed by subjects including questions on acute symptoms	Acute wheezing; acute dyspnea; persistent wheezing; persistent wheezing with dyspnea; acute chest tightness; pulmonary function measures	Authors report on cigarette consumption; sex, age, height, ethnicity, and history of asthma, but analyses were not adjusted for covariates	Crude OR=0.03 for subjects reporting symptoms of persistent wheezing at end of school year compared to subjects reporting symptoms only at the beginning of the year; p-value<0.001. Crude OR for subjects reporting acute wheezing only during formaldehyde exposure compared to only during control laboratory=2/0, authors defined this OR as infinite with a 2-sided p-value determined to
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								<p>be not significant. Crude OR for subjects reporting acute chest tightened only during formaldehyde exposure compared to only during control laboratory=4/0; authors defined this OR as infinite with a 2-sided p-value=0.05. FVC mean changes on test day 1=0.012, mean changes on test day 2=0.042, p-value&lt;0.001; mean changes on test day 3=0.042, p-value&lt;0.001. FEF25-75 mean changes on</p>
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								<p>test day 1= 0.079, mean changes on test day 2= 0.089, p- value reported not significant, mean changes on test day 3=0.003, p- value reported not significant. FEV1 mean changes on test day 1= 0.031, mean changes on test day 2= 0.046, p- value=0.03, mean changes on test day 3= 0.021, p- value=0.01. FEV1/FVC mean changes on test day 1= 0.004, mean changes on test day 2= 0.003, p-</p>
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								value reported not significant; mean changes on test day 3=0.002, p- value reported not significant.
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Lofstedt et al. 2011 (Prospective Cohort)	Adult workers exposed to formaldehyde and unexposed controls in an occupational setting in three foundries producing cores with the Hot Box method in Sweden (follow up to Lofstedt et al. 2009 study)	25 exposed workers and 55 controls	Formaldehyde measured for full shift (8 hours) and considered to reflect individual exposure (mean exposure 10.2 years) in 2005	Mean (2001): 98 ug/m3 (SD 94; range 14-440); mean (2005): 45 ug/m3 (SD 43; range 10-190)	Questionnaire completed by subjects	Whistling or wheezing, attacks of breathlessness, awakenings by tightness in chest, pulmonary function tests	Report characteristics of exposed and non-exposed workers were similar by sex, smoking status, those with asthma, and childhood allergy	Number participants in 2001 reporting wheezing or whistling in chest=12/25 (exposed), 11/55 (non-exposed), p-value reported not significant. Number participants in 2005 reporting wheezing or whistling in chest=4/25 (exposed), 5/55 (non-exposed), p-value reported not significant. Number participants in 2001 reporting attacks of breathlessness=8/25 (exposed), 2/55 (non-exposed), p-
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								<p>value reported-not significant. Number participants in 2005 reporting attacks of breathlessness=4/25 (exposed), 0/55 (non-exposed), p-value reported-not significant. Number participants in 2001 reporting awakenings by tightness in chest=13/25 (exposed), 4/55 (non-exposed), p-value reported-not significant. Number participants in 2005 reporting awakenings by tightness</p>
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								<p>in chest=4/25 (exposed);2/5 5-(non- exposed), p- value reported not significant. Change comparing 2005 to 2001 in VC mean= -0.8,95%CI[- 2.53,0.93] (exposed) compared to -0.4,95%CI[- 1.47,0.67] (non- exposed). Change comparing 2005 to 2001 in FEV1 mean= -1.3,95%CI[- 3.57,0.97] (exposed) compared to 0.3,95%CI[- 1.19,1.79] (non- exposed).</p>
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Hendrick et al. 1977 (Prospective Cohort)	Adult workers in an occupational setting in a hemodialysis unit and continuously exposed to formalin at Churchill Hospital in Oxford, United Kingdom	28 staff members (mean age 45 years)	Formaldehyde exposure classified based on job; workers exposed to varying degrees of formalin in 1976	Exposed while sterilizing equipment with 34-38% solution of formalin in water w/w	Medical examination and medical history	Wheezing, pulmonary function tests	Age and smoking were described, but not accounted for in analyses	Results are descriptive only, with no analyses of data. Inhalation provocation tests with formalin were used to evaluate 4 staff members and a sister of one patient, all of whom had histories of recurrent attacks of wheezing since becoming exposed regularly to formalin. Author suggested that, while exposure to formalin did not seem to be directly responsible in all cases, it might have increased
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								<p>susceptibility to other provoking agents or induced a hyper-reactive responsiveness of the airways. All the staff were interviewed and underwent simple tests of airways function, namely peak expiratory flow (PEF) using a Wright's meter, and one second forced expiratory volume (FEV1), and forced vital capacity (FVC) using a Vitalograph dry spirometer. The results were</p>
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								<p>compared with predicted values for normal subjects from Bates et al. (1971) and from nomograms supplied by Vitalograph Limited. All staff members were asymptomatic when these tests were carried out, and all readings of FEV<sub>1</sub>, FVC, and PEF were close to predicted normal values. In all cases the ratio FEV<sub>1</sub>/FVC exceeded 70%.</p>
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Gorski et al. 1991 (Cohort)	Adult workers with respiratory symptoms and exposed to formaldehyde in an occupational setting at textile or shoe manufacturing factories (country not stated, but assume Poland)	367 workers (ages 23–52 years; mean age 46 years)	Workers occupationally exposed to formaldehyde; exposure levels not reported	Not reported	Medical examination performed according to criteria of American Thoracic Society	Bronchial asthma and chronic bronchitis and dyspnoea; pulmonary function tests	Authors report information on smoking, age, and possible co-exposures, but these were not accounted for in analyses	Results are descriptive only, with no analyses of data. 14 subjects suffered from dyspnoea with clinical signs of chronic bronchitis; acute episodes of dyspnoea; classified as bronchial asthma, were found in 2 subjects; an illness lasted 2 and 7 years, respectively. Resting ventilatory function was measured by spirometry; vital capacity (VC), forced expiratory volume (FEV1) and peak expiratory
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								<p>flow (PEF) were estimated at the beginning of the work shift and immediately afterwards; in subjects suffering from chronic cough, dyspnoea or sneezing, the test was repeated at the end of the work shift and PEF was measured during the course of a three-day clinical observation. Mean values of ventilatory parameters in the group of 367 subjects were: VC = <math>3.47 \pm 1.41</math> L; FEV1 = <math>3.1 \pm 0.19</math> L/s; in the group of bronchitis</p>
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								<p>patients the mean value of VC was <math>3.03 \pm 1.92</math> L and FEV<sub>1</sub> <math>2.13 \pm 1.82</math> L/s; in 14 bronchitic patients the changes of PEF during the three day observation did not exceed 20% of the initial value; only 2 patients reacted with a decrease of PEF at the end of a non-placebo, non-bronchodilator day, but no difference between placebo and bronchodilator days were found; the mean value of PC 20 in bronchitic patients was 5.61 mg/mL</p>
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								(± 1.79); in the 2 asthmatics a significant decrease from 3.41 to 1.97 mg/mL and from 2.70 to 2.01 mg/mL was noted after exposure.
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Dumas et al. 2017 (Case control study nested within prospective cohort)	Adult nurses exposed to formaldehyde in an occupational setting while employed and working within the United States	4,102 actively employed nurses with physician - diagnosed asthma and use of asthma medication in the past year	Formaldehyde exposure classified by nurse-specific job-task-exposure matrix designed to assign exposure level to formaldehyde as low, medium or high based on a combination of types of nursing jobs and general disinfection tasks	Not measured—categorized as high versus low exposure based on job function	Questionnaire completed by subjects with information regarding diagnosis by physician as having asthma; reported use of asthma medication in the past year; use of prescribed long-term preventative medicine; and Asthma Control Test score (range 5-25) based on five questions on activity limitations; frequency of symptoms and frequency of use of quick-relief medication in past four weeks	Categorical Asthma Control Test (ACT); asthma medication	Age, smoking status, body mass index, race and ethnicity	OR=1.33, p-value=0.02 for decrease in Asthma Control Test comparing high versus low formaldehyde exposure; based on job-task-exposure-matrix (JTEM) defining high as task performed 1-3 or 4-7 days per week versus never or <1 day per week.
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Frisk et al. 2002 (Prospective Cohort)	Adult asthmatics in the general population planning to live in their homes for the following 18 months to undergo intervention in Orebro, Sweden	21 asthmatic adults (ages 28-59)	Formaldehyde measured in homes before and after interventions, during heating season (October-April)	Mean (pre-test): 21 ug/m <sup>3</sup> (SD 9); mean (post test): 19 ug/m <sup>3</sup> (SD 5)	Questionnaire completed by subjects	Asthma medication; symptom score; pulmonary function tests	No confounders or adjustment factors reported	Mean of medicine consumption pre-test=9, 95% CI [7.18, 10.82], post test=8.7, 95% CI [6.74, 10.66], p-value reported not statistically significant. Mean of symptom score pre-test=8.6, 95% CI [6.87, 10.33], post-test=8.8, 95% CI [7.12, 10.48], p-value reported not statistically significant. Mean of VC pre-test=90, 95% CI [81.47, 98.53], post-test=88, 95% CI [78.59, 97.41], p-value reported not
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								<p>statistically significant.</p> <p>Mean of FEV1 pre-test=88,95% CI [77.7, 98.3], post-test=92,95% CI [81.4, 102.6], p-value reported not statistically significant.</p> <p>Mean of PD20 (histamine) pre test=557, 95% CI [265.16, 848.84], post-test=717, 95% CI [417.63, 1016.37], p-value reported not statistically significant.</p> <p>Mean of PEF morning value pre-test=458, 95% CI [407.77,</p>
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								<p>508.23], post- test=470, 95% CI [417.29, 522.71], p- value reported not statistically significant. Mean of PEF morning evening value pre- test=484, 95% CI [438.75, 529.25], post- test=484, 95% CI [430.79, 537.21], p- value reported not statistically significant. Mean of reversibility % of baseline pre test=7.6, 95% CI [3.71, 11.49], post test=2.3, 95% CI [- 1.42, 6.02],</p>
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								p-value=0.007.
Popa et al. 1969 (Prospective Cohort)	Adults with asthma related to occupational exposure to simple chemicals were enlisted during epidemiological survey (n=29) attended the inpatient and outpatient allergy unit in hospital (n=19) in Bucharest, Romania	48 asthmatic adults followed for six months to two years after first diagnosis	Formaldehyde used in allergologic tests	Exposed to 1:2,500 dilution of formalin for inhalation tests	Medical examination/response to tests used for diagnosis; bronchial asthma diagnosis was supported by spontaneous asthma attack and asthmatic bronchitis diagnosis supported by mild asthma attack; authors note no clear cut borderline between these diagnoses	Respiratory symptoms (bronchial asthma and asthmatic bronchitis to formalin)	No confounders or adjustment factors reported	Bronchial asthma to formalin inhalation tests were positive, but were delayed (2 to 4 to 12 hours)-chronologic delayed reaction. For skin tests all subjects had (immunologic)-delayed type reactions; delayed intradermal tests (24 to 48 hours); and positive

								patch tests. Bronchial asthma and asthmatic bronchitis to formalin inhalation tests were characteristically negative. Skin tests were positive in three subjects. PK reaction was negative. Precipitating antibodies were absent.
De Vos et al. 2009 (Non-randomized controlled trial)	Active adult fire fighters exposed in an occupational setting based in Perth Metropolitan fire stations in Australia	67 fire fighters participated in four field trials where randomly allocated to groups using respirators with different filters (P, POV,	Formaldehyde measured using personal air samples inside respirators; performed 120 min and 60 min burns	Mean (120 min): 0.245 mg/m <sup>3</sup> for P-filter; 0.021 for POV-filter; 0.017 mg/m <sup>3</sup> for POVF filter; mean (60 min): 0.44 mg/m <sup>3</sup> for P-filter; 0.027 mg/m <sup>3</sup> for POV-filter;	Subjects completed questionnaire based on Medical Research Council questionnaire on respiratory symptoms	Respiratory symptoms	FESA years, age group, pack years	OR for increase in respiratory symptoms=0.050, 95% CI [0.004, 0.597] for 0-60 minute P-filter versus POV-filter; OR=0.234, 95% CI [0.068, 0.797] for 0-60 minute P-filter versus POVF filter;

		POVF filters)		0.015 mg/m3 for POVF filter				OR=0.484, 95% CI [0.034, 6.802] for 0-60 minute POV filter versus POVF filter; OR=0.048, 95% CI [0.006, 0.358] for 0-120 minute P filter versus POV filter; OR=0.237, 95% CI [0.092, 0.613] for 0-120 minute P filter versus POVF filter; OR=1.300, 95% CI [0.149, 11.359] 0-120 minute POV filter versus POVF filter;
Kim et al. 2014 (Non-randomized controlled trial)	Adult patients in the general population diagnosed with asthma	17 asthmatics (age 30s to 60s)	Subjects assigned to two groups: households where plants were	Continuation study: decrease from 24.2 to 15.5 ug/m3;	Subjects completed Quality of Life Questionnaire for Adult Korean Asthmatics which includes questions	Respiratory symptoms; pulmonary function test	Demographic information was presented for participant gender, age, area/size of	No association found between quality of life questionnaire

	at the Medical College of Yonsei University in Seoul, Korea		introduced and continued (n=9) and households where plants were introduced and then withdrawn (n=8) in January–September 2006 and 2007	withdrawal study: decrease from 29.7 to 13.6 $\mu\text{g}/\text{m}^3$	on respiratory symptoms		residents, and year of building completion (SES proxies). In attempt to prevent any confounding effects due to occupation, most participants were housewives. These factors were not accounted for in analyses.	for adult Korean asthmatics (QLQAKA) respiratory score and formaldehyde exposure (data not shown). Mean PEFR measures first morning=405 L/min, 1st evening=416 L/min, 2nd morning=406 L/min, 2nd evening=428 L/min. First experiment PEFR continuation in morning=13.9 L/min, withdrawal in morning=24.7 L/min, p-value<0.01. PEFR continuation in evening=20.6
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								L/min, withdrawal in evening=30.2, p-value<0.01. Second experiment PEFR continuation in morning=9.69 L/min, withdrawal in morning=9.23 L/min, p-value reported not significant. PEFR continuation in evening=15.23 L/min, withdrawal in evening=15.23, p-value reported not significant.
Liu et al. 1991 (Cross-sectional)	Adults in the general population living in randomly selected mobile homes in California	1394 summer phase residents (663 mobile homes) and 1096 winter	Formaldehyde measured using monitors mailed to participants in kitchen and master bedroom for	Range: 0.01 (limit of detection)-0.46 ppm; mean (summer): 0.089 ppm; mean	Questionnaire completed by subjects; occurrence of asthma attack reported for 2 weeks prior to monitoring period	Asthma attack	Age, sex, and smoking were considered, but these were not accounted for in analyses.	Percentage reporting asthma attack for the two weeks prior to monitoring period=80%. No statistical



		phase residents (523 mobile homes)	7 days in February/March 1985 and July/August 1984	(winter): 0.088 ppm				significance reported-
Norback et al., 2000 (Cross-sectional)	Adult workers in an occupational setting at four geriatric hospitals in Ystad, Sweden		Formaldehyde measured on two different days in each building, in two 6-hour samples per building per day in January-February 1997 (1-2 weeks after medical investigations completed)	Mean (signs of dampness): 5 ug/m <sup>3</sup> (range 2-8); mean (no signs of dampness): 5 ug/m <sup>3</sup> (range 3-9)	Medical examination where subjects questioned by doctor on doctor-diagnosed asthma and respiratory symptoms over last year using questions from ECRHS (wheeze, daytime and nighttime shortness of breath); current asthma defined as reporting at least one asthma-related symptom	Asthma symptoms	Report information on smoking, SES (measured as "social status"); sex, age, atopy; and other environmental factors, but these were not accounted for in analyses	Mean formaldehyde exposure for subjects with asthma symptoms=3 ug/m <sup>3</sup> , 95% CI [1.89, 4.11]. Mean formaldehyde exposure for subjects without asthma symptoms=5 ug/m <sup>3</sup> , 95% CI [4.53, 5.47]

Hanson et al. 1993 (Cross-sectional)	Adult workers surveyed in an occupational setting at hospital in New York	88 workers exposed to operating room exhaust and 84 non-exposed workers (mean age 35.4 years)	Formaldehyde measured for 8 hours in the operating room	Peak level (operating room): 0.99 ppm; time weighted average: 0.02 ppm	Questionnaire completed by subjects including questions adapted from American Thoracic Society Respiratory Disease Questionnaire	Wheezing (over and under age 35); wheezing (RNs and non-RNs); wheezing (smokers and non-smokers); asthma (temporarily associated and work aggravated); wheezing with dyspnea; wheezing	Analyses were stratified by smoking status; age, gender, job description, and Registered Nurse status	Wheezing prevalence=40% for 4th floor exposed compared to 25% for 4th floor non-exposed, p-value reported not significant. Wheezing prevalence=43% for operating room exposed compared to 24% for operating room non-exposed, p-value<0.05. Wheezing with dyspnea prevalence=26% for 4th floor exposed compared to 25% for 4th floor non-exposed, p-value reported not significant. Wheezing
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								<p>with dyspnea prevalence=3 1% for operating room exposed compared to 21% for operating room non- exposed, p- value reported not significant. Ever asthma work aggravated prevalence=4 4% for 4th floor exposed compared to 36% for 4th floor non- exposed, p- value=1.00. Ever asthma work aggravated prevalence=5 5% for operating room exposed compared to 22% for operating</p>
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								<p>room non-exposed, p-value=0.2.</p> <p>Overall wheezing prevalence risk ratio=1.6 for 4th floor comparing exposed to non-exposed, p-value reported not significant.</p> <p>Overall wheezing prevalence risk ratio=1.8 for operating room comparing exposed to non-exposed, p-value&lt;0.05.</p> <p>Wheezing prevalence risk ratio=1.2 for the 4th floor smokers comparing exposed to non-exposed, p-value reported not significant.</p>
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								<p>Wheezing prevalence risk ratio=1.2 for the operating room non-smokers comparing exposed to non-exposed, p-value reported not significant.</p> <p>Wheezing prevalence risk ratio=1.8 for the 4th floor never smokers comparing exposed to non-exposed, p-value reported not significant.</p> <p>Wheezing prevalence risk ratio=2 for the operating room never smokers comparing exposed to non-exposed, p-</p>
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								<p>value&lt;0.05.  Wheezing prevalence  risk ratio=2.1  for the 4th  floor for RNs  comparing  exposed to  non-exposed,  p-  value&lt;0.05.  Wheezing  prevalence  risk ratio=1.1  for the  operating  room for  RNs  comparing  exposed to  non-exposed,  p-value  reported not  significant.  Wheezing  prevalence  risk ratio=1  for the 4th  floor for non-  RNs  comparing  exposed to  non-exposed,  p-value  reported not  significant.</p>
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								<p>Wheezing prevalence risk ratio=3.6 for the operating room for non-RNs comparing exposed to non-exposed; p-value&lt;0.05.</p> <p>Wheezing prevalence risk ratio=1.1 for the 4th floor for those over 35 years-old comparing exposed to non-exposed; p-value reported not significant.</p> <p>Wheezing prevalence risk ratio=1.3 for the operating room for those over 35 years-old comparing exposed to non-exposed;</p>
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								<p>p-value reported not significant. Wheezing prevalence risk ratio=2.4 for the 4th floor for those under 35 years old comparing exposed to non-exposed;</p> <p>p-value reported not significant. Wheezing prevalence risk ratio=2.2 for the operating room for those under 35 years old comparing exposed to non-exposed;</p> <p>p-value reported not significant.</p>
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Horvath et al. 1988 (Cross-sectional)	Adult workers exposed to formaldehyde in an occupational setting from particle-board or molded products at Weyerhaeuser Co in Marshfield, Wisconsin and control workers from food-processing facilities in nearby areas	109 exposed workers and 254 unexposed control workers	Formaldehyde measured using personal monitors (8 hour sample) and area monitors	Range (exposed subjects): 0.04-2.93 ppm; range (control subjects): 0.03-0.12 ppm	Subjects completed modified American Thoracic Society respiratory symptom questionnaire before and after work shift	Wheezing, shortness of breath, difficulty breathing, chest pains/aching/tightness/burning, pulmonary function tests	Evaluated impact of height, age, sex, smoking, mobile home residence, and duration of exposure, but analyses did not adjust for these variables	Wheezing prevalence number (percent)=4 (3.7%) for exposed workers compared to 7 (2.8%) for non-exposed workers, p-value reported not significant. Difficulty in breathing prevalence number (percent)=7 (6.4%) for exposed workers compared to 5 (2.0%) for non-exposed workers, p-value reported not significant. Shortness of breath prevalence number (percent)=9 (8.3%) for exposed
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								workers compared to 13 (5.1%) for non-exposed workers, p-value reported not significant. FEV1=3.62L before shift, 3.58L after shift for exposed workers, p-value<0.05. FEV1=3.59L before shift, 3.55L after shift for non-exposed workers, p-value<0.001. FVC=4.49L before shift, 4.49L after shift for exposed workers, p-value reported not significant. FVC=4.47L before shift, 4.41L after shift for non-exposed
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								workers, p-value<0.001. FEV1/FVC%=80.3 before shift, 79.4 after shift for exposed workers, p-value<0.01. FEV1/FVC%=80.5 before shift, 80.8 after shift for non-exposed workers, p-value reported not significant. PEF=8.02L/s before shift, 8.25L/s after shift for exposed workers, p-value reported not significant. PEF=8.03L/s before shift, 8.06L/s after shift for non-exposed workers, p-value reported not significant.
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								<p>FEF25%-75%=3.71L/s before shift, 3.53L/s after shift for exposed workers, p-value&lt;0.01.</p> <p>FEF25%-75%=3.68L/s before shift, 3.69L/s after shift for non-exposed workers, p-value reported not significant.</p> <p>FEF25%=6.91L/s before shift, 7.02L/s after shift for exposed workers, p-value reported not significant.</p> <p>FEF25%=6.73L/s before shift, 6.73L/s after shift for non-exposed workers, p-value reported not significant.</p>
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								<p>FEF50%=4.50L/s before shift, 4.34L/s after shift for exposed workers, p-value&lt;0.01.</p> <p>FEF50%=4.38L/s before shift, 4.43L/s after shift for non-exposed workers, p-value reported not significant.</p> <p>FEF75%=1.63L/s before shift, 1.52L/s after shift for exposed workers, p-value&lt;0.01.</p> <p>FEF75%=1.66L/s before shift, 1.66L/s after shift for non-exposed workers, p-value reported not significant.</p>
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Kilburn, Seidman, and Warshaw 1985 (Cross-sectional)	Adult women exposed to formaldehyde working in an occupational setting as histology technicians in 23 hospitals and 2 laboratories and unexposed women working as secretaries and clerks at the same institutions in Los Angeles, California	76 exposed female histology technicians and 56 unexposed female controls (mean age 40, 39.3, 39.5, and 41.5 for clerical workers 0 hr, 1-3 hr, and >4 hours, respectively)	Formaldehyde measured for 1-4 hours in 10 of 25 laboratories	Range in tissue specimen prep and sampling areas: 0.2-1.9 ppm	Questionnaire completed by subjects	Shortness of breath at work; shortness of breath at rest; chest tightness; chest pain	Matched pairs with respect to age, cigarette smoking, and ethnicity. Exposed (technicians) and unexposed (secretaries and clerks) participants had different job functions, but worked for the same organization were of similar SES status.	Prevalence=5% for chest tightness for clerical workers, 7% after 0hr exposure, 27% after 1-3hr exposure, 40% after >4hr exposure, p-value not reported. Prevalence=5% for chest pain for clerical workers, 14% after 0hr exposure, 23% after 1-3hr exposure, 40% after >4hr exposure, p-value not reported. Prevalence=0% for shortness of breath at rest for clerical workers, 0% after 0hr exposure,
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								4.5% after 1-3hr exposure, 21% after >4hr exposure, p-value not reported. Prevalence=0 % for shortness of breath at work for clerical workers, 14% after 0hr exposure, 27% after 1-3hr exposure, 38% after >4hr exposure, p-value not reported.
Herbert et al. 1994 (Cross-sectional)	Adult workers in an occupational setting at an oriented strand board plant exposed to formaldehyde and workers in oilfield and gas plant from same	99 exposed workers (mean 5.1 years of employment) and 165 non-exposed controls	Formaldehyde measured from five work sites in factory for 21 continuous hours on two separate days in March and April 2012	Range: 0.07-0.27 ppm	Subjects completed questionnaire based on International Union Against Tuberculosis and Lung Disease questionnaire	Attacks of wheeze, wheeze with chest tightness, chest tightness, attacks of chest tightness, wheeze occasionally (apart from	Smoking, age	OR=5.48, 95% CI [1.85, 16.2] for asthma comparing exposed workers versus non-exposed workers. OR=3.34, 95% CI [1.66, 6.73]

		area in United States					colds); woken by shortness of breath; shortness of breath with exercise; shortness of breath at rest; shortness of breath; asthma; pulmonary function tests		for attacks of wheeze comparing exposed workers versus non- exposed workers. OR=2.46, 95% CI [1.22, 4.94] for attacks of chest tightness comparing exposed workers versus non- exposed workers. OR=2.71, 95% CI [1.56, 4.69] for chest tightness comparing exposed workers versus non- exposed workers. OR=5.72, 95% CI [2.78, 11.8] for wheeze with chest
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								<p>tightness comparing exposed workers versus non- exposed workers: OR=2.85, 95% CI [1.63, 4.99] for wheeze occasionally (apart from colds) comparing exposed workers versus non- exposed workers: OR=6.78, 95% CI [1.4, 32.7] for woken by shortness of breath comparing exposed workers versus non- exposed workers: OR=5.44, 95% CI [2.91, 10.2] for shortness</p>
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								of breath comparing exposed workers versus non- exposed workers. OR=4.94, 95% CI [2.52, 9.68] for shortness of breath with exercise comparing exposed workers versus non- exposed workers. OR=3.16, 95% CI [1.37, 7.28] for shortness of breath at rest comparing exposed workers versus non- exposed workers. OR=1.68, 95% CI [0.54, 5.25] for FEV1/FVC<
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								<p>75% comparing exposed workers versus non- exposed workers: OR=1.08, 95% CI [0.32, 3.64] for FEV1/FVC&lt; 75% ex- smokers comparing exposed workers versus non- exposed workers: OR=2.98, 95% CI [1.1, 8.07] for FEV1/FVC&lt; 75% for current smokers comparing exposed workers versus non- exposed workers:</p>
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Norback et al. 1995 (Cross-sectional)	Adults in the general population randomly selected for screening questionnaire, identified from population register of Uppsala, Sweden	88 adults (aged 20-44 years; mean age 32 years)	Formaldehyde measured in bedrooms for two hours in October 1991-April 1992	Mean with nocturnal attacks of breathlessness (bedroom): 29 ug/m <sup>3</sup> (range in house <5-100); mean without nocturnal attacks of breathlessness (bedroom): 17 ug/m <sup>3</sup> (range in house <5-60)	Questionnaire completed by subjects	Nocturnal breathlessness, respiratory symptoms (including wheezing or whistling in chest and daytime breathlessness), pulmonary function tests	Sex, prevalence of wall-to-wall carpets; prevalence of house dust mites; current smoker; age	OR=12.5; 95% CI [2, 77.9] for nocturnal breathlessness per 10-fold increase in formaldehyde exposure. Wheezing or whistling in the chest and daytime attacks of shortness of breath reported to show no statistically significant association with formaldehyde exposure. No associations found between bronchial hyper-responsiveness, variability in PEF, FEV <sub>1</sub> %, and the indoor
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								concentration of asthma.
Kriebel et al. 2001 (Prospective Cohort)	Adult physical therapy students exposed in an occupational setting attending clinical anatomy laboratory once a week at University of Massachusetts-Lowell in Massachusetts	38 graduate students (mean age 24.9 years)	Formaldehyde measured continuously at six different locations for 2.5 hours/week for 14 weeks in the laboratory; work sampling was used to link each subject to a formaldehyde zone	Geometric mean: 0.70 ppm (SD 2.13); highest short term exposure: 10.91 ppm; average exposure: 1.1 ppm (SD 0.56); median exposure: 0.97 ppm		Pulmonary function tests	No difference in response by smoking status. Gender, age, and height were reported but not adjusted for in analysis	FVC presemester mean=4.35L, 95% CI [4.1, 4.6] compared to postsemester mean=4.34L, 95% CI [4.07, 4.61]; p-value reported not significant. FEV1 presemester mean=3.65L, 95% CI [3.43, 3.87] compared to

			measuremen t					postsemester mean=3.63, I 95% CI [3.4, 3.86], p- value reported not significant. PEF (% baseline) presemester mean= 0.75, 95% CI [- 1.38, 0.12] compared to postsemester mean=0.5, 95% CI [0.05, 0.95], p- value=0.02.
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Lofstedt et al. 2009 (Prospective Cohort)	Adult workers exposed to formaldehyde in an occupational setting and unexposed controls in four foundries producing cores with the Hot Box method in Sweden	64 exposed workers and 134 controls (mean age 44.2 years)	Formaldehyde measured for full shift (8 hours) and considered to reflect individual exposure (mean exposure 8.9 years) in 2001	Mean: 8.9 ug/m <sup>3</sup> (SD 8.8)		Pulmonary function tests	Models were adjusted for smoking and co-pollutants (methyl isocyanate and isocyanic acid). Authors explored additional characteristics, including BMI and time in present job. Time in present job did not differ between the two groups. Authors report equal proportion of females in exposed and referent groups.	Percent FEV1 change over work shift adjusting for methyl isocyanate and smoking = -1.5%, 95% CI [-4, 1], p-value reported not significant. Percent FEV1 change over work shift adjusting for isocyanic acid and smoking = -1.6%, 95% CI [-4, 0.9], p-value reported not significant. Percent FEV1 change for unexposed workers mean = 0.1%, 95% CI [-0.55, 0.75], for
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								<p>coremakers exposed mean=-2%, 95% CI [- 3.56, -0.44], p- value&lt;0.05, for die eastern exposed mean=0.3%, 95% CI [- 1.36, 1.96], p-value reported not significant, for other exposed mean=-1.1%, 95% CI [- 3.25, 1.05], p-value reported not significant, for all exposed mean=-1.4%, 95% CI [- 2.47, -0.33], p- value&lt;0.05.</p>
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Kriebel et al. 1993 (Cross-sectional)	Adult physical therapy students in an occupational setting attending clinical anatomy laboratory once a week in Massachusetts	24 graduate students (mean age 26 years)	Formaldehyde measured using personal monitors for 1-1.5 hours	Geometric mean (air): 3.6 ppm (SD 1.16; range 3.0-4.3); geometric mean (personal): 0.73 ppm (SD 1.22; range 0.49-0.93)		Pulmonary function test	Age, gender, smoking status, and asthma history were assessed.	PEF change by week adjusted beta=-2.7L/min; 95% CI [-4.98, -0.42]; p-value=0.01. PEF change by week adjusted log-transformed beta=22.6L/min; 95% CI [13.29; 31.91]; p-value<0.001. PEF change for prelaboratory (1-2 weeks) mean=538.9 L/min; 95% CI [498.23; 579.75]; PEF change for cross-laboratory (1-2 weeks) mean=-12.2L/min; 95% CI [-498.23; 579.75]; PEF change for
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								prelaboratory (9–10 weeks) mean=529.4 Lmin, 95% CI [488.03, 570.77], PEF change for cross- laboratory (9- 10 weeks) mean= 1.2L/min, 95% CI [-9.3, 6.9], PEF change for prelaboratory (24–25 weeks) mean=536.6 Lmin, 95% CI [496.26, 576.94], PEF change for cross- laboratory (24–25 weeks) mean=2.4L/ min, 95% CI [-3.92, 8.72].
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Milton et al. 1996 (Cross-sectional)	Adult male workers exposed in an occupational setting at a fiberglass wool manufacturing plant in United States	18 male maintenance workers and 19 male production workers	Formaldehyde measured for 8 hours for 5 or 6 work days using personal sampling monitor	Eight hr time weighted average: 1.2-265 ug/m3; geometric mean (low): 6.4 ug/m3; geometric mean (medium): 31.8 ug/m3; geometric mean (high): 100 ug/m3		Pulmonary function test	Cigarettes during interval; asthma medications; levels of other exposures; effect of night shift; PEF change on non-work days	OR=0.9, 95% CI [0.3, 2.2] for PEF drop $\geq 5\%$ comparing medium formaldehyde exposure (16.8-60.3 ug/m3) to low formaldehyde exposure (1.2-16.6 ug/m3) measured from start to end of work shift; adjusted for effect of night shift; non-workday PEF change. OR=2, 95% CI [0.6, 7] for PEF drop $\geq 5\%$ comparing high formaldehyde exposure (61.1-265 ug/m3) to low formaldehyde
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								e-exposure measured from start to end of work shift; adjusted for effect of night shift; non- workdays PEF change. OR=0.8, 95% CI [0.3, 2.3] for PEF drop $\geq 5\%$ comparing medium formaldehyd e-exposure to low formaldehyd e-exposure measured from start to end of work shift; adjusted for cigarettes during interval; asthma medications; levels of other exposures; effect of
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								night shift; non-workday PEF change. OR=1.1, 95% CI [0.2, 7.3] for PEF drop $\geq 5\%$ comparing high formaldehyd e-exposure to low formaldehyd e-exposure measured from start to end of work shift; adjusted for cigarettes during interval; asthma medications; levels of other exposures; effect of night shift; non-workday PEF change. OR=1.2, 95% CI [0.6, 2.2] for PEF drop $\geq 5\%$ comparing
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								medium formaldehyd e-exposure to low formaldehyd e-exposure measured from start of work shift to arising; adjusted for effect of night shift; non-workday PEF change: OR=1.4, 95% CI [0.7, 2.7] for PEF drop $\geq 5\%$ comparing high formaldehyd e-exposure to low formaldehyd e-exposure measured from start work shift to arising; adjusted for effect of night shift; non-workday PEF change:
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Akbar Khanzadeh et al. 1997 (Cross-sectional)	Adult medical students exposed to formaldehyde in an occupational setting at an anatomy lab in Toledo, OH	50 exposed subjects (female mean age 26.2 years; male mean age 24.2 years) and 36 nonmedical student controls (female mean age 24.1 years; male mean age 23.1 year)	Formaldehyde in breathing zone (personal sample) and general area of anatomy lab measured	Mean (breathing zone): 1.88 ppm; mean (middle of lab): 0.97 ppm		Pulmonary function tests	All participants were non-smoking individuals, and were similar in age, height, weight, gender ratios in the study groups, and baseline respiratory function	Mean percent increase in FEV3 (1-3hr)=0.8; 95%CI [-0.1-1.6] (exposed); 3.3; 95%CI [-1.44, 5.16] (controls). Mean percent increase in FEV1 (1-3hr)=1.2; 95%CI [-0.26-2.14] (exposed); 4.1; 95%CI [-2.41, 5.79] (controls). Mean percent increase in FVC (within 3hr)=2.5; 95%CI [-0.97-4.03] (exposed); 4.6; 95%CI [-2.43, 6.77] (controls). Mean percent increase in FVC (within 3hr)=2.5; 95%CI [-0.97-
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								<p>4.03} (exposed); 4.6, 95%CI [2.43; 6.77](control s). Mean percent increase in FEF25%- 75% (within 3hr)=2.2, 95%CI [- 0.47, 4.87] (exposed); 9.3, 95%CI [5.27; 13.33](contro ls). Mean percent increase in FEV3 (within 3hr)=2.7, 95%CI [1.39- 4.01] (exposed); 5.2, 95%CI [3, 7.4] (controls). Mean-percent increase in FEV1 (within 3hr)=245, 95%CI [0.95- 3.85] (exposed); 6.2, 95%CI</p>
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								<p>increase in FEV3 (within 1hr)=1.8, 95%CI [0.83-2.77] (exposed); 1.9, 95%CI [0.95, 2.85](controls). Mean percent increase in FEV1 (within 1hr)=1.2, 95%CI [0.01-2.39] (exposed); 2.1, 95%CI [0.61, 3.59](controls). Mean percent increase in FEF25-75%(1-3hr)=0.7, 95%CI [-1.18, 2.58] (exposed); 6.1, 95%CI [3.73, 8.47] (controls).</p>
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Frisk et al. 2009 (Cross-sectional)	Adults in the general population selected from the Orebro, Sweden section of the 1996 FinEsS study	49 asthmatics (ages 19-54 years; mean age 39 years)	Formaldehyde measured over 24 hour period between January 1999 and December 2000 during heating season	Not reported		Pulmonary function tests (including bronchial provocation n=39)	Internal moisture supply; carbon dioxide; environmental tobacco smoke; indoor domestic pets; nitrogen dioxide; prevalence of house dust mites; water content	FEV1 median=92 for 0 risk factor group; 98 for 1 risk factor group (reported no statistical significance); 95 for 2 risk factor group (reported no statistical significance); and 95.5 for >2 risk factor group (reported no statistical significance); PEF median=94 for 0 risk factor group; 99 for 1 risk factor group (reported no statistical significance); 89 for 2 risk factor group (reported no statistical significance); and 88.5 for >2 risk factor
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								group (reported no statistical significance); Bronchial hyperrespons iveness median=0.5 for 0 risk factor group; 0.86 for 1 risk factor group (reported no statistical significance); 0.64 for 2 risk factor group (reported no statistical significance); and 0.62 for >2 risk factor group (reported no statistical significance);
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Sheppard et al. 1984 (Non-randomized controlled trial)	Adults in the general population diagnosed with asthma in California	7 asthmatic adults (served as own controls)	Subjects exposed to filtered air, 1ppm, and 3ppm during moderate exercise	Exposed to 1 or 3 ppm		Pulmonary function test	All participants were non-smokers. Authors measured sex, age, height, and this was a controlled exposure study so there was no need to measure other environmental co-exposures.	Specific airway resistance (SRaw) mean=9.2, 95% CI [4.95, 13.45] before air exposure, mean=6.4, 95% CI [5.08, 16.92] after air exposure, mean=8.5, 95% CI [4.62, 12.38] before 1ppm exposure, mean=10.7, 95% CI [4.97, 16.43] after 1ppm exposure, mean=7.4, 95% CI [3.52, 11.28] before 3ppm exposure, mean=10.3, 95% CI [3.64, 16.96] after 3ppm exposure.
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Sauder et al. 1987 (Non-randomized controlled trial)	Adult volunteers in the general population with clinical history of asthma and documented hyperactive airways in United States	9 asthmatic adults (served as own controls)	Subjects exposed to clean air for 3 hours followed by 3 ppm formaldehyde one week later	Mean exposure: 2.9 ppm (SD 0.14)	Symptom questionnaire completed by subjects	Chest discomfort or tightness; pulmonary function tests	All volunteers were nonsmokers. Reported characteristics of age and sex, but analyses were not adjusted for these variables.	Mean of symptom questionnaire score=0.22 after 0min of clean air exposure; mean of symptom questionnaire score=0.22 after 0min of 3ppm formaldehyde exposure; p-value reported not significant. Mean of symptom questionnaire score=0.11 after 2min of clean air exposure; mean of symptom questionnaire score=0.33 after 2min of 3ppm formaldehyde exposure; p-value reported not significant.
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								Mean of symptom questionnaire score=0.22 after 15min of clean air exposure; mean of symptom questionnaire score=0.22 after 15min of 3ppm formaldehyde exposure; p-value reported not significant. Mean of symptom questionnaire score=0.11 after 30min of clean air exposure; mean of symptom questionnaire score=0.22 after 30min of 3ppm formaldehyde exposure; p-value reported not significant.
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								Mean of symptom questionnaire score=0.22 after 60min of clean air exposure; mean of symptom questionnaire score=0.44 after 60min of 3ppm formaldehyde exposure; p-value reported not significant. Mean of symptom questionnaire score=0.44 after 120min of clean air exposure; mean of symptom questionnaire score=0.44 after 120min of 3ppm formaldehyde exposure; p-value reported not significant.
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								Mean of symptom questionnaire score=0.44 after 180min of clean air exposure; mean of symptom questionnaire score=0.44 after 180min of 3ppm formaldehyd e-exposure; p-value reported not significant. Mean of FEV1=3.02 after 180min of clean air exposure; mean of FEV1=3.07 after 180min of 3ppm formaldehyd e-exposure; p-value reported not significant. Mean of FVC=4.11 after 180min of clean air
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								exposure; mean of FVC=4.16 after 180min of 3ppm formaldehyd e-exposure; p-value reported not significant. Mean of FEF25- 75%=2.64 after 180min of clean air exposure; mean of FEF25- 75%=2.59 after 180min of 3ppm formaldehyd e-exposure; p-value reported not significant. Mean of SGaw=0.101 after 180min of clean air exposure; mean of SGaw=0.106 after 180min of 3ppm formaldehyd
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								e-exposure, p-value reported not significant.
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Ezratty et al. 2007 (Non-randomized controlled trial)	Adult patients in the general population with intermittent asthma and allergy to pollen in France	12 adults diagnosed with intermittent asthma (between 18-44 years of age; median age 25 years)	Subjects exposed at rest to filtered air or to 0.4ppm formaldehyde for 60 min on two separate days separated by 2 weeks; crossover study where order of exposure randomized	Exposed to 500 ug/m3		Pulmonary function tests (methacholine and allergen challenge)	All subjects were nonsmokers; and the age, sex, and asthma duration of all the subjects were presented. Authors noted none of the twelve subjects were receiving anti-inflammatory therapy or other current treatments, and the study was performed outside of grass pollen season. Participants were randomized to different interventions.	Methacholine challenge (PD20) median=0.17 mg for air exposure only; median=0.23 mg for formaldehyde exposure; p-value=0.42. Allergen challenge (PD15 FEV1) median=0.25 for air exposure only; median=0.8 for formaldehyde exposure; p-value=0.06. Sputum supernatant concentrations of interleukins (IL-1, IL-4, IL-5, IL-8, IL-10); granulocyte-
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								macrophage colony-stimulating factor (GM-CSF), monocyte chemoattractive protein-1 (MCP-1), tumor necrosis factor- $\alpha$ (TNF- $\alpha$ ), interferon- $\gamma$ (IFN- $\gamma$ ), and eotaxin-1 were reported for patients after exposure to air only and to formaldehyde. None of these outcomes were significantly different for patients following exposure to formaldehyde compared to air only.
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Witek, Jr et al-1986 (Randomized controlled trial)	Adults patients in the general population with a past history of asthma in the United States	30 asthmatic adults	Subjects were exposed to 0 or 2ul/L formaldehyde for 5-40 min periods in chamber on two days at rest and on two days with exercise	Exposed to 2-ul/L		Pulmonary function test	All volunteers were nonsmokers. Reported characteristics of age and sex, but analyses were not adjusted for these variables.	Mean-percent change (standard deviation) from baseline for healthy subjects in FEV1=0.50 (4.7) after 30 minutes room air at rest; -0.37 (4.5) after 30 minutes room air with exercise; -1.15 (5.3) after 30 minutes 2.0 ug/L formaldehyde exposure at rest; 1.76 (4.9) after 30 minutes 2.0 ug/L formaldehyde exposure with exercise. Mean-percent change (standard deviation) from baseline for asthmatic
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								<p> subjects in  FEV1= 0.31  (4.1) after 30  minutes room  air at rest;  0.62 (7.5)  after 30  minutes room  air with  exercise;  0.60 (6.4)  after 30  minutes 2.0  ug/L  formaldehyd  e exposure at  rest; 1.86  (11.9) after  30 minutes  2.0 ug/L  formaldehyd  e exposure  with  exercise. </p>
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Green et al. 1987 (Randomized controlled trial)	Adults in the general population with asthma recruited by newspaper advertisement in Maryland, United States	16 asthmatics and 21 healthy normal controls (mean age 26.91 years)	Subjects exposed for one hour to both clean air and 3ppm formaldehyde and exposures separated by one week	Exposed to 3 ppm		Pulmonary function tests	All subjects were nonsmokers. Asthmatics were taken off medications 48hr prior to exposures and no subjects were allowed to take anti-histamines within 12hr of trial. Exposures were randomized.	Mean FEV1 for healthy individuals after 55 minutes air exposure=4.29, 95% CI [4.04, 4.54]; mean FEV1 for healthy individuals after 55 minutes 3.0ppm formaldehyde exposure=4.15, 95% CI [3.88, 4.42]; p-value<0.02. Mean FEV1 for asthmatic individuals after 55 minutes air exposure=3.54, 95% CI [2.94, 4.14]; mean FEV1 for asthmatic individuals after 55 minutes 3.0ppm formaldehyde
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								<p>e exposure=3.4 6, 95% CI {2.86, 4.06}; p-value reported not significant. Mean FVC for healthy individuals after 55 minutes air exposure=5.0 4, 95% CI {4.73, 5.35}; mean FVC for healthy individuals after 55 minutes 3.0ppm formaldehyd e exposure=4.9 2, 95% CI {4.61, 5.23}; p- value&lt;0.02. Mean FVC for asthmatic individuals after 55 minutes air exposure=4.6 2, 95% CI {3.81, 5.43};</p>
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								<p>mean FVC for asthmatic individuals after 55 minutes 3.0ppm formaldehyd e exposure=4.5 6, 95% CI {3.73, 5.39}; p-value reported not significant.</p> <p>Mean FEF25-75% for healthy individuals after 55 minutes air exposure=4.7 4, 95% CI {4.22, 5.26}; mean FEF25- 75% for healthy individuals after 55 minutes 3.0ppm formaldehyd e exposure=4.5 6, 95% CI {3.96, 5.16}; p-value</p>
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								<p>reported not significant.</p> <p>Mean FEF25-75% for asthmatic individuals after 55 minutes air exposure=3.14, 95% CI [2.44, 3.84]; mean FEF25-75% for asthmatic individuals after 55 minutes 3.0ppm formaldehyde exposure=3.14, 95% CI [2.34, 3.88]; p-value reported not significant.</p> <p>Mean FRC for healthy individuals after 55 minutes air exposure=3.61, 95% CI [3.26, 3.96]; mean FRC for healthy</p>
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								<p>individuals after 55 minutes 3.0ppm formaldehyd e exposure=3.6 1, 95% CI {3.23, 3.99}; p-value reported not significant. Mean FRC for asthmatic individuals after 55 minutes air exposure=3.5 8, 95% CI {3.07, 4.09}; mean FRC for asthmatic individuals after 55 minutes 3.0ppm formaldehyd e exposure=3.5 8, 95% CI {3.09, 4.07}; p-value reported not significant. Mean FEV3 for healthy</p>
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								<p>individuals after 55 minutes air exposure=4.9  3, 95% CI {4.62, 5.24};  mean FEV3 for healthy individuals after 55 minutes 3.0ppm formaldehyde exposure=4.8  , 95% CI {4.49, 5.11};  p-value&lt;0.02.  Mean FEV3 for asthmatic individuals after 55 minutes air exposure=4.3  7, 95% CI {3.62, 5.12};  mean FEV3 for asthmatic individuals after 55 minutes 3.0ppm formaldehyde exposure=3.4</p>
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								<div>6, 95% CI [3.55, 5.09]; p-value reported not significant.</div>
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Witek, Jr et al-1987 (Randomized controlled trial)	Adults in the general population with a past history of asthma recruited from the university community via bulletin board advertisement in the United States	15 asthmatic adults (ages 18-35 years; mean age 22.1 years)	Subjects were exposed to 0 or 2ppm formaldehyde for 5-40 min periods in chamber on two days at rest and on two days with exercise	Exposed to 2 ppm		Pulmonary function tests	All subjects were nonsmokers and between age 18-35; refrained from taking asthma medications and caffeinated beverages for at least 24 hours prior to session, and none reported having an upper respiratory infection during the study. Authors also report gender, height and weight. This is a controlled exposure study, hence co-exposure measurement was not necessary. Participants were randomized to interventions.	Mean percent change (standard deviation) from baseline for asthmatic subjects in FEV1= 0.31 (4.0) after 30 minutes room air at rest; 0.62 (7.5) after 30 minutes room air with exercise; 0.60 (6.4) after 30 minutes 2.0 ug/L formaldehyde exposure at rest; 1.86 (11.9) after 30 minutes 2.0 ug/L formaldehyde exposure with exercise. Mean percent change (standard deviation) from baseline for asthmatic
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								<p>subjects in FVC=0.82 (5.7) after 30 minutes room air at rest;— 0.60 (6.7) after 30 minutes room air with exercise;— 2.78 (3.1) after 30 minutes 2.0 ug/L formaldehyd e exposure at rest (p- value&lt;0.01); -2.49 (7.5) after 30 minutes 2.0 ug/L formaldehyd e exposure with exercise.</p>
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Harving et al. 1990 (Randomized controlled trial)	Adult volunteers in the general population with substantial bronchial hyperreactivity in Denmark	15 adults (ages 15-36 years; mean age 25.1 years)	Subjects assigned to 1 of 3 groups and went through same 3 experiments in randomized order with formaldehyde concentration of 0.85 mg/m <sup>3</sup> , 0.12 mg/m <sup>3</sup> , and 0 mg/m <sup>3</sup> over a 3 week period; mean exposure time 89.4 min	Exposed to 0 mg/m <sup>3</sup> (SD 0.008), 0.12 mg/m <sup>3</sup> (SD 0.07), and 0.85 mg/m <sup>3</sup> (SD 0.07)		Pulmonary function tests	Researchers controlled the use of bronchial dilators and oral medications use on the day of exposure	Mean(SD) FEV1 before 0 mg/m <sup>3</sup> formaldehyde exposure=100, after 0 mg/m <sup>3</sup> formaldehyde exposure=100.9(12.8); mean(SD) FEV1 before 0.12mg/m <sup>3</sup> formaldehyde exposure=100, after 0.12mg/m <sup>3</sup> formaldehyde exposure=99.4(8.7); mean (SD) FEV1 before 0.85mg/m <sup>3</sup> formaldehyde exposure=100, after 0.85mg/m <sup>3</sup> formaldehyde exposure=105.0(16.5);
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								Mean(SD) Raw-before 0mg/m3 formaldehyd e exposure=2.1 7(0.85),after 0mg/m3 formaldehyd e exposure=2.2 1(0.54); mean (SD) Raw-before 0.12mg/m3 formaldehyd e exposure=2.4 1(0.79),after 0.12mg/m3 formaldehyd e exposure=2.2 3(0.76); mean (SD) Raw-before 0.85mg/m3 formaldehyd e exposure=2.2 9(0.66),after 0.85mg/m3 formaldehyd e exposure=2.2 9(0.66);
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								Mean (SD) Saw before 0 mg/m3 formaldehyd e exposure=11. 21(4.26),after 0mg/m3 formaldehyd e exposure=10. 67(2.66); mean(SD) Saw before 0.12mg/m3 formaldehyd e exposure=11. 67(3.02),after 0.12mg/m3 formaldehyd e exposure=10. 63(3.10); mean(SD) Saw before 0.85mg/m3 formaldehyd e exposure=11. 61(4.47); after 0.85mg/m3 formaldehyd e
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								<del>exposure=11. 17(3.56).</del>
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Burge et al. 1984 (Case reports)	Adult workers occupationally exposed to formaldehyde who were referred for symptoms suggestive of asthma in United Kingdom	15 workers (ages 26-62)	Subjects exposed to various concentrations of formaldehyde	Exposed to 0.1%-20% solution resulting in 2.3-34 mg/m <sup>3</sup>	Medical examination	Bronchial provocation tests	Authors reported smoking, age, sex, and previous history of asthma or rhinitis but analyses were not adjusted by these factors	Bronchial provocation, reported as maximum fall in FEV <sub>1</sub> and histamine reactivity, is reported individually for the 15 cases: 3 subjects had appreciable late asthmatic reactions after formaldehyde exposure, suggesting true sensitization; 4 subjects had appreciable immediate reactions with no late reaction, and 1 subject had late reactions following formaldehyde exposure on six occasions;
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								<p>two of which were appreciable immediate reactions; breathing zone concentration s of formaldehyd e required to elicit irritant reactions was mean 4.8 mg/m3; 3 of the 4 subjects with histamine hyperreactivi ty had an immediate reaction alone when exposed to 10% formaldehyd e, 3 subjects with a normal histamine PC20 value also reacted to formaldehyd e, and 1 subject with</p>
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								histamine reactivity and 4 subjects with normal histamine PC20 values failed to respond to 1% formaldehyde.
Frigas et al. 1984 (Case reports)	Adult patients in the general population with symptoms suggestive of asthma who suspected formaldehyde exposure as	13 patients (ages 15-70 years)	Subjects exposed to room air or formaldehyde for 20 minutes at levels of 0.1, 1, and 3 ppm for	Exposed to 0.1, 1 or 3 ppm	Medical examination	Bronchial provocation tests	Authors reported on smoking, sex, age, and occupation but analyses were not adjusted by these factors	Decreases in FEV1 after placebo and formaldehyde challenges are reported individually for the 13 cases; in only one patient did the FEV1

	the cause in the United States							decline by 20% or more after exposure to formaldehyde, and in this case the placebo challenge induced almost the same decrease in FEV1 as did formaldehyde; for the rest of the patients, the FEV1 did not diminish significantly after challenge with formaldehyde gas
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Nordman et al. 1985 (Case reports)	Adults occupationally exposed to formaldehyde and suffering from asthma-like respiratory symptoms between January 1977 and May 1983 in Finland	12 adults tested positive to bronchial provocation test with formaldehyde (considered to have bronchial asthma) and cases reports presented (exposed occupationally up to 19 years)	Subjects were exposed to 1ppm and 2ppm formaldehyde for 30 minutes	First exposure: 1.2 mg/m <sup>3</sup> ; second exposure: 2.5 mg/m <sup>3</sup>	Medical examination	Bronchial provocation test to diagnose asthma	Characteristics are described in detail for 5 case patients. No discussion of potential confounders for the remaining 5 cases of formaldehyde asthma, or for the 218 subjects who did not react to the formaldehyde challenge.	Twelve patients were considered to have bronchial asthma; eight of these patients demonstrated an immediate reaction to the bronchial provocation test (i.e., within 30 minutes of beginning exposure). Six late reactions, two of which were preceded by immediate drops in PEF, were recorded. The formaldehyde concentration used in 11 cases was 2.5 mg/m <sup>3</sup> and in one case 1.2 mg/m <sup>3</sup> ;
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								three patients did not react at all on provocation to histamine, indicating the absence of bronchial hyperreactivity.
Gannon et al., 1995 (Case reports)	Adult workers occupationally exposed to glutaraldehyde and referred to specialist for testing	7 workers (age 25–53 years)	Subjects exposed to 1% formaldehyde solution on cardboard in chamber for 10 minutes	Exposed to 1% solution	Medical examination	Bronchial provocation tests	Workers' characteristics are presented (age, sex, smoking, occupation, years of exposure, agents of exposure). Case series report—not incorporation of these variables in analyses.	Percent fall in specific bronchial provocation test comparing 1.2 mg/m <sup>3</sup> formaldehyde exposure from saline control: 0 (Case 1), 0 (Case 2), 27 (Case 3), 28 (Case 4), 33 (Case 6), 0 (Case 7), 0 (Case 8). Case 5 was not challenged to formaldehyde.

								<p>e for technical reasons. Three workers also had positive late reactions on challenge to formaldehyd e.</p>
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Zhai et al. 2013 (Cross-sectional)	Households of children and adults in the general population decorated within the previous four years in main urban area of Shenyang, China	One adult per household and 82 children in 186 residential houses	Formaldehyde measured in bedrooms, living rooms, and kitchens over 1 month to 3 years	Polluted homes: 0.093 mg/m <sup>3</sup> (bedroom); 0.103 mg/m <sup>3</sup> (living room); 0.131 mg/m <sup>3</sup> (kitchen); non-polluted homes: 0.43 mg/m <sup>3</sup> (bedroom); 0.040 mg/m <sup>3</sup> (living room); 0.047 mg/m <sup>3</sup> (kitchen)	Subjects (including children with parental assistance) completed survey designed by the American Thoracic Society on respiratory health	Asthma, wheeze, respiratory symptoms for adults and children	Age, education, family history of allergy, gender, height, house facing, indoor domestic pets, occupation, smoking in the family, ventilation frequency, weight	Adult asthma prevalence= 0% for non-polluted homes; 1.68% for polluted homes, p-value reported not significant. Child asthma prevalence= 3.22% for non-polluted homes; 40% for polluted homes, p-value<0.05. Adult wheeze prevalence= 2.99% for non-polluted homes; 5.04% for polluted homes, p-value reported not significant. Child wheeze prevalence= 6.56% for non-polluted homes; 10% for polluted
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								homes, p-value reported not significant. Adult OR=2.603, 95% CI [1.77, 3.828] for respiratory symptoms. Child OR=4.250, 95% CI [2.064, 8.753] for respiratory symptoms.
Veremchuk et al. 2016 (Cross-sectional)	Adult and children residents in the general population with asthma in Vladivostok, Russia	Asthma morbidity in Vladivostok (sample size not reported)	Formaldehyde measured using air quality monitors from six stationary observation posts during 2008-2012	Not reported	Medical records	Asthma diagnosis	The study evaluated effects by age groups (children, adolescents, and adults). Climatic factors and indicators of anthropogenic air pollution in	No significant correlation reported between formaldehyde exposure and asthma outcome

							the city were also evaluated.	
Quackenbush et al. 1989 (Cross-sectional)	Households of adults and children in the general population classified as single-family homes, mobiles/trailers, and apartments/condos in the United States	151 households	Formaldehyde measured for one-week periods using samplers in the kitchen, main living area, and each subject's bedroom; homes classified as likely to have low, moderate, or higher exposures	Homes were classified as being likely to have low exposures ( $\leq 60$ ug/m <sup>3</sup> ), moderate exposures (60 to 120 ug/m <sup>3</sup> ), or higher exposures ( $> 120$ ug/m <sup>3</sup> )		Pulmonary function tests	Authors measure age, sex, smoking, previous lung disease, and day of week, but not included in analysis	Percentage of subjects with variability in PEFR exceeding "normal" limits: Formaldehyde $\leq 30$ ug/m <sup>3</sup> = 70.6% (male $\leq 15$ years old), 62.5% (males $\leq 35$ years old), 36.8% (males $> 35$ years old). Formaldehyde $> 30$ ug/m <sup>3</sup> = 90.2% (male $\leq 15$ years old), p-value = 0.06, 68.8% (males $\leq 35$ years old), p-value reported not significant,

								<p>48.8% (males&gt;35 years old), p- value reported not significant. Percentage of subjects with variability in PEFR exceeding "normal" limits: Formaldehyd e≤30 ug/m3=72.4 % (female ≤15 years old), 53.1% (females ≤35 years old), 60.4% (females&gt;35 years old). Formaldehyd e&gt;30 ug/m3=93.3 % (female ≤15 years old), p- value=0.07, 46.2% (females ≤35 years old), p-value reported not</p>
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								significant, 57.4% (females>35 years old), p- value reported not significant.
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Yeatts et al. 2012 (Cross-sectional)	Children and adults in the general population recruited based on two-stage cluster sample design in the United Arab Emirates (UAE)	1590 individuals within four age/sex categories: adult male (ages 19–50 years); adult female (ages 19–50 years); adolescent (ages 11–18 years); and child (ages 6–10 years)	Formaldehyde measured in the common living room for a 7 day period in 2009–2010	Median: <7.37 ug/m <sup>3</sup> (limit of quantification: 7.37; range: 7.37–168.2)	Subjects were interviewed and asked about ever having doctor-diagnosed asthma; respiratory symptoms assessed using ISAAC and Behavioral Risk Factor Surveillance System questions	Ever asthma; wheezing limited speech to 1 or 2 words between breaths; wheezing in last 4 months; wheezing in last 12 months; ever having wheezing and whistling in chest; shortness of breath one or more times a month; shortness of breath in last 12 months; chest tightness/difficulty breathing in last 12 months; chest tightness/difficulty breathing one or more times a month	Urban/rural area; household tobacco smoke exposure; gender, age group	OR=1.43, 95% CI [0.83, 2.46] for chest tightness/difficulty in breathing in last 12 months comparing high formaldehyde exposure group (7.37–168.2 ppm) to low formaldehyde exposure group (<7.37 ppm); OR=1.55, 95% CI [0.97, 2.48] for shortness of breath in last 12 months comparing high to low formaldehyde exposure group; OR=1.32, 95% CI [0.73, 2.37] for ever
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								<p>asthma comparing high to low formaldehyd e-exposure group: OR=1.31, 95% CI {0.71, 2.42} for ever having wheezing or whistling in chest comparing high to low formaldehyd e-exposure group: OR=0.64, 95% CI {0.21, 1.98} for wheezing in past 12 months comparing high to low formaldehyd e-exposure group: OR=3.48, 95% CI {0.81, 14.89} for wheezing in past 4 weeks</p>
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								<p>comparing high to low formaldehyd e-exposure group: OR=4.18, 95%-CI [1.23, 14.22] for wheezing limiting speech to 1 or 2 words between breaths</p> <p>comparing high to low formaldehyd e-exposure group: OR=3.68, 95%-CI [1.11, 12.27] for shortness of breath one or more times a month</p> <p>comparing high to low formaldehyd e-exposure group: OR=6.52, 95%-CI [1.91, 22.31] for chest</p>
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								tightness/diff iculty in breathing one or more times a month comparing high to low formaldehyd e-exposure group-
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Sehenker et al. 1982 (Cross-sectional)	Children and adult residents of six homes in the general population with urea formaldehyde foam insulation (UFFI) in the United States	24 residents from six homes (ages 7-63 years)	Formaldehyde measured in homes 7 to 34 months following installation of UFFI in 1979-1981	Range: 0.02-0.23 ppm	Subjects completed American Thoracic Society questionnaire and special supplementary questionnaire for subjects with formaldehyde exposure	Wheeze, pulmonary function tests	Authors report measuring smoking, age, and sex variables, but did not report these data and did not adjust analyses with these variables	Prevalence of participants reporting chronic phlegm=5/24 in UFFI houses. Prevalence of participants reporting chronic cough=11/24 in UFFI houses. Prevalence of participants reporting persistent wheeze=6/24 in UFFI houses. Change in FEV1 for participants in UFFI houses mean=-0.121. Change in FEV1/FVC for participants in UFFI houses mean=-3.74.
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Tuomainen et al. 2003 (Cohort)	Adult residents of two apartment buildings in the general population in Finland	Residents living in building built for people with respiratory diseases and building built using conventional methods as a control	Formaldehyde measured for 2-4 hours in 6 apartments from each building on 5 occasions over a 3-year period	Mean (cases at 1 year): 13 ug/m3 (SD 4; range 3-18); mean (cases at 2 years): 16 ug/m3 (SD 7; range 3-38); mean (cases at 3 years): 12 ug/m3 (SD 6; range 7-28); mean (controls at 1 year): 23 ug/m3 (SD 5; range 16-29); mean (controls at 2 years): 23 ug/m3 (SD 9; range 10-43); mean (controls at 3 years): 17 ug/m3 (SD 5; range 10-28)	Questionnaire completed by subjects (when moved into building, after 5 months, and after 1, 2, and 3 years of occupancy) included questions on asthma symptoms	Asthma symptoms	Passive smoking, stuffy air, dry air, and varying temperature	Relevant asthma symptoms were not reported, but asthmatic occupants reported that their symptoms decreased during occupancy in the case building (built for people with respiratory diseases).
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‡ Studies organized by 1) meta-analysis status (studies considered for meta-analysis and studies not considered for meta-analysis), 2) study population (children, adults, children and adults, not specified), 3) outcomes reported (asthma diagnosis, asthma diagnosis and asthma symptoms, pulmonary function measures), and the 4) study design (cohort, case-control, cross-sectional, non randomized controlled trial, randomized controlled trial, case report).

Child studies were published relatively recently (1990-2016 for asthma diagnosis, 1984-2019 for asthma symptoms) whereas adult studies had a wider range of publication years including more older studies (Table 34). Almost half of child studies (11/24 for asthma diagnosis and 9/23 for asthma symptoms) had sample sizes greater than 1,000, whereas more adult studies had smaller sample sizes (13/20 for asthma diagnosis and 21/26 for asthma symptoms with sample size <500) (Table 43). Combined, child studies reported on a total of over 34,000 participants for asthma diagnosis and 32,000 participants for asthma symptoms. Adult studies reported on a total of over 8,000 participants for asthma diagnosis and 12,000 for asthma symptoms (S100 and S101 TableTable 3b, S99-Table).

A little over half (51%, n=46) of the included studies were cross-sectional in study design, and the remainder were cohort (n=17), controlled trials (n=11), case-control (n=7), case reports (n=4), or of mixed study design (e.g., cross-sectional and case-control) (n=5) (Table 2). A similar trend in study design was observed in that the majority of studies in all four population/outcome combinations were of cross-sectional study design. Children studies reporting on asthma diagnosis were mostly cross-sectional (58%) and case-control (21%) whereas those reporting on asthma symptoms were mostly cross-sectional (52%) and prospective cohort (22%) (Table 43). Adult studies reporting on asthma diagnosis were mostly cross-sectional (80%) and cohort (15%), and similarly for those reporting on asthma symptoms (58% cross-sectional, 27% cohort) (Table 43).

### **Characteristics of included studies—Exposure measures**

Most studies (91%, n=82) reported association estimates between asthma outcomes and quantitative measurements of formaldehyde exposure. In the remainder of studies (n=8), although quantitative formaldehyde exposure measures were reported (leading to the study's inclusion), these estimates were not used by study authors directly to calculate association estimates, but rather they used categorized formaldehyde levels (i.e., high, medium, and low exposures) (Table 2). Formaldehyde levels were measured in school (n=14), home (n=30), work (n=16), vehicles (n=1), and outdoor environments (n=6), as well as using personal monitors (n=13) or given as experiment doses to healthy volunteers (n=12) (Table 3b, S100 and S101 Table). School formaldehyde measurements were used in 10 child asthma diagnosis and 10 child asthma symptom studies (and in no adult studies). Home formaldehyde measurements were used in 9 studies each for child asthma diagnosis and symptom studies and 7 studies each for adult asthma diagnosis and symptom studies. Work formaldehyde measurements were used in 6 adult asthma diagnosis studies and 11 adult symptom studies (and in no child studies). Outdoor exposure measurements were mostly used in child studies (3 studies of child asthma diagnosis, 4 for child asthma symptoms, and 2 for adult asthma diagnosis) whereas personal monitor measurements were mostly used in adult studies (5 studies of adult asthma diagnosis, 7 for adult asthma symptoms, and 2 each for child asthma diagnosis and asthma symptoms) (S100 and S101 Table Table 3b, S99 Table).



**Table 43.** Study Characteristics, stratified by population health outcome group.

	Child asthma n (%)	Child asthma symptoms n (%)	Adult asthma n (%)	Adult asthma symptoms n (%)
<b>Publication Year</b>				
<i>1969</i>	0	0	0	1 (4%)
<i>1977</i>	0	0	0	1 (4%)
<i>1980-1989</i>	0	1 (4%)	2 (10%)	6 (23%)
<i>1990-1999</i>	3 (13%)	1 (4%)	4 (20%)	7 (27%)
<i>2000-2009</i>	7 (29%)	6 (26%)	5 (25%)	6 (23%)
<i>2010-2019</i>	14 (58%)	15 (65%)	9 (45%)	5 (19%)
<b>Study design</b>				
<i>Case-control</i>	5 (21%)	2 (9%)	1 (5%)	0
<i>Nested case-control</i>	2 (8%)	0	0	1 (4%)
<i>Prospective cohort</i>	2 (8%)	5 (22%)	2 (10%)	7 (27%)
<i>Cohort</i>	0	0	1 (5%)	0
<i>Cross-sectional</i>	14 (58%)	12 (52%)	16 (80%)	15 (58%)
<i>Cross-sectional and case-control</i>	1 (4%)	2 (9%)	0	0
<i>Non-randomized controlled trial</i>	0	1 (4%)	0	3 (11%)
<i>Randomized controlled trial</i>	0	1 (4%)	0	0
<i>Case report</i>	0	0	0	0
<b>Sample size</b>				
<i>0-50</i>	0	2 (9%)	1 (5%)	6 (23%)
<i>51-100</i>	3 (12%)	2 (9%)	5 (25%)	6 (23%)
<i>101-200</i>	6 (25%)	2 (9%)	1 (5%)	4 (15%)
<i>201-500</i>	1 (4%)	7 (30%)	6 (30%)	5 (19%)
<i>501-1000</i>	2 (8%)	0	4 (20%)	2 (8%)
<i>&gt;1000</i>	11 (46%)	9 (39%)	2 (10%)	3 (11%)
<i>Not reported</i>	1 (4%)	1 (4%)	1 (5%)	0

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405 **Characteristics of included studies—Outcome measures**

406 Of the 90 total included studies, 41 evaluated asthma diagnosis outcomes (21 studies in children,  
407 17 in adults, and 3 in both children and adults) and 48 evaluated asthma-related symptoms (22  
408 studies in children, 25 in adults, and 1 in both children and adults). Asthma diagnosis was  
409 ascertained either by questionnaire (for instance, the International Study of asthma and Allergies  
410 in Childhood (ISAAC) (30)) medical records, or a physical examination ([S100 and S101](#)  
411 [TableTable 3b, S99 Table](#)).

412 Studies reported on a wide range of asthma-related outcomes, including current/ever asthma  
413 (n=33), asthma attacks (n=3), respiratory symptoms (n=9), wheeze (n=32), shortness of  
414 breath/dyspnea/breathlessness (n=17), chest tightness and pain (n=10), pulmonary bronchial  
415 hyperresponsiveness (n=1), asthma medication use (n=6), hospitalizations (n=2), emergency  
416 room visits (n=1), and results from asthma control (n=2), pulmonary function (n=35), and  
417 bronchial provocation tests (n=5) ([S100 and S101 TableTable 3b, S99 Table](#)).

418 Studies reporting on child asthma symptoms reported most commonly on wheeze (n=16) and  
419 current/ever asthma (n=14); all other asthma-related outcomes listed were reported in ≤5 studies.  
420 No child studies reported on outcomes of chest tightness and pain, pulmonary bronchial  
421 hyperresponsiveness, or bronchial provocation ([S100 and S101 TableTable 3b, S99 Table](#)).

422 Studies reporting on adult asthma symptoms reported most commonly on pulmonary function  
423 (n=28), current/ever asthma (n=19), wheeze (n=15), and shortness of breath

424 /dyspnea/breathlessness (n=13), chest tightness and pain (n=9), and respiratory symptoms (n=6);  
425 all other asthma-related outcomes listed were reported in <5 studies. No adult studies reported on  
426 hospitalizations or emergency room visits ([S100 and S101 TableTable 3b, S99 Table](#)).

#### 427 **Risk of bias assessment**

428 We rated risk of bias separately by outcome (asthma diagnosis versus symptoms exacerbation),  
429 but since our ratings were ultimately identical by outcome, risk of bias results are presented by  
430 study only. A limited number (n=3) of studies (31-33) reported results for mixed children/adult  
431 populations (aged 6-63 years); we excluded these studies from rating the quality of the evidence  
432 due to concerns with combining outcomes across a wide age range, given the unique issues in  
433 diagnosing and assessing asthma in children (especially at very young ages) compared to adults  
434 (34, 35). Overall, the majority of studies were rated “low” or “probably low” risk of bias across  
435 all domains (Fig 3, S1-3 Figs). We evaluated the risk of bias separately by each of the four-study  
436 population/health outcome groups.

#### 437 **Group 1: Childhood asthma diagnosis**

438 Overall, the majority of childhood asthma diagnosis studies were rated “low” or “probably low”  
439 risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and  
440 “probably low” but included a small number of “probably high” ratings—source population  
441 (three “probably high” ratings), outcome assessment (four), incomplete outcome data (one), and  
442 exposure assessment (three). These were not consistent across any one study—i.e., only no study  
443 was rated “probably high” across all three of these domains. Generally, studies rated “probably  
444 high” were for similar reasons—i.e., for source population, three studies (39-41) reported high  
445 non-participation rates but failed to compare characteristics from study participants to those

446 refusing to participate to explore potential selection bias. Similarly, for outcome assessment four  
447 studies (42-45) relied on self-reported outcomes by study participants (i.e., through a survey,  
448 self-administered spirometry, or daily diaries) but lacked follow-up by study investigators to  
449 evaluate the validity of reported outcomes. Furthermore, two studies were rated “high” risk of  
450 bias for the other category—Huang et al. (46) due to cases having formaldehyde levels sampled  
451 more during the summer when formaldehyde exposures were lower versus controls who were  
452 sampled more during the summer when formaldehyde exposures were higher and Madureira et  
453 al. (47) who published a similar paper in a different journal the year prior with similar reported  
454 results.

455 The most problematic domain appeared to be confounding, where six studies were rated  
456 “probably high” and four were rated as “high.” Consistent with the instructions from our  
457 protocol, studies were rated as “probably high” for the confounding domain if studies evaluated  
458 some but not all of confounders pre-determined to be important (age, smoking status or exposure  
459 to environmental tobacco smoke, and socioeconomic status or parental education) and some but  
460 not all of other confounders pre-determined to be potentially important (race/ethnicity, sex,  
461 height, weight, BMI, obesity status, parental or family history of asthma, allergies, and additional  
462 environmental exposures), and were rated “high” if the study did not account for or evaluate  
463 many of the important or potentially important confounders. Studies most commonly adjusted  
464 for age, sex, and exposure to smoking. Adjusting for socioeconomic status was often  
465 accomplished through incorporating variables of family income or parent’s academic  
466 background. Few studies adjusted for environmental co-exposures; those that did included  
467 exposures to allergens (house dust mites or pets), indoor dampness or mold, proximity to traffic,  
468 or certain contaminants such as nitrogen dioxide or particulate matter.

Overall, review authors felt confident that the majority of children asthma diagnosis studies were rated predominantly “low” or “probably low” risk of bias, particularly for studies that were ultimately included in the meta-analysis. In particular, of the nine studies that were ultimately included in the meta-analysis, four received “low” or “probably low” ratings across all risk of bias domains and accounted for 44% of the weight in estimating the overall association estimate. Studies generally that were rated “probably high” or “high” were not for reasons that were consistent across this body of evidence, and did not produce compelling reasons to downgrade the overall body of evidence as a result.

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**Group 2: Childhood asthma exacerbation and symptoms**

Overall, the majority of childhood asthma exacerbation and symptoms studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included a couple “probably high” or “high” ratings—blinding (one “probably high” rating), outcome assessment (two “probably high ratings), conflict of interest (one “probably high” and one “high” rating), and other (one “high” rating). These were not consistent across any one study—i.e., only no study was rated “probably high” or “high” across all domains. One study (48) was rated “probably high” for blinding because children and parents were recruited based on existence of airway respiratory symptoms and parents were responsible for deploying and retrieving in-home environmental samples and media as well as recording outcomes in diaries, thus making it unlikely that the reporting of outcomes was competed by someone without knowledge of exposure status. Two studies (45, 49) were rated as “probably high” for outcome assessment due to lack of physician confirmation or in-

491 person interviews by study investigators to confirm asthma symptoms. One study (48) appeared  
492 to have a financial conflict of interest, with research grants provided from several private  
493 foundations from the pharmaceutical field (i.e., AstraZeneca). Another study (15) received a  
494 “high” rating for the other domain because of an apparent typographical error in the reporting of  
495 results that could not be confirmed by authors upon personal communication.

496 A few other domains included a higher number of “probably high” or “high” ratings—source  
497 population (five “probably high” ratings), confounding (five “probably high” and two “high”  
498 ratings), incomplete outcome data (two “probably high” and one “high” ratings), and exposure  
499 assessment (three “probably high” ratings). Similar to the child asthma diagnosis studies, the  
500 most problematic risk of bias domain appeared to be confounding, where several studies did not  
501 adjust for or consider several of the important or potentially important adjustment factors  
502 outlined in our protocol. Studies most commonly adjusted for age, sex, and exposure to smoking.  
503 Adjusting for socioeconomic status was often accomplished through incorporating variables of  
504 family income or parent’s academic background. Few studies adjusted for environmental co-  
505 exposures; those that did included exposures to allergens (house dust mites or pets), indoor  
506 dampness or mold, proximity to traffic, or certain contaminants such as nitrogen dioxide or  
507 particulate matter.

508 Overall, review authors felt confident that the majority of children asthma diagnosis studies were  
509 rated predominantly “low” or “probably low” risk of bias, particularly for studies that were  
510 ultimately included in the meta-analysis. In particular, of the five studies that were ultimately  
511 included in the meta-analysis, three received “low” or “probably low” ratings across all risk of  
512 bias domains and accounted for 90% of the weight in estimating the overall association estimate  
513 for wheeze and 100% of the weight for shortness of breath. In particular, a number of studies

514 were rated consistently as “low” or “probably low” risk of bias across all domains, increasing the  
515 review authors’ confidence that a sufficient body of evidence was available with minimal risk of  
516 bias to rate the overall body of evidence for this study population/health outcome group. Studies  
517 that were rated “probably high” or “high” were not for reasons that were consistent across this  
518 body of evidence, and did not produce compelling reasons to downgrade the overall body of  
519 evidence as a result.

520

521 **Group 3: Adult population asthma diagnosis**

522 Overall, the majority of adult asthma diagnosis studies were rated “low” or “probably low” risk  
523 of bias across all domains (Fig 4). Several domains were predominantly rated “low” and  
524 “probably low” but included a one to two “probably high” or “high” ratings—outcome  
525 assessment (one “probably high”), confounding (two “high”), and conflict of interest (one  
526 “probably high”). These studies were rated higher risk of bias for lack of validation for self-  
527 reported outcomes (50), failure to adjust for or consider several of the important or potentially  
528 important adjustment factors outlined in our protocol (50, 51), or receiving funding from a  
529 private company without including a statement of the role of this company in influencing the  
530 study (52). Unlike for included children studies, confounding did not appear as problematic for  
531 the adult studies, likely because many studies were occupational and relied on either matching  
532 participants based on baseline characteristics or were pre- and post-experimental tests that used  
533 each individual subject as their own control.

534 Other domains included a higher number of “probably high” or “high” ratings—blinding (five  
535 “probably high”), exposure assessment (five “probably high”) and other (five “probably high”).

536 These were not consistent across studies—only one study (53) received “probably high” ratings  
537 across four of these domains. This study (53) received high risk of bias ratings due to lacking  
538 detail on recruitment methods, failure to address blinding and the existing potential for bias if  
539 investigators knew exposure status of participants, exposure measurements that were assessed by  
540 self-administered, proctored questionnaires that ultimately used work assignment as a proxy for  
541 high versus low exposure groups, and the existence of potential healthy worker effect. Blinding  
542 was more generally problematic for adult studies compared to those in children since many were  
543 occupational studies where study participants were likely already aware of their exposure and/or  
544 outcome status, and blinding was not a possibility. For the other domain, all five studies that  
545 received “probably high” ratings were occupational studies where potential for healthy worker  
546 effect either likely existed or was likely.

547 Overall, review authors felt confident that the majority of adult asthma diagnosis studies were  
548 rated predominantly “low” or “probably low” risk of bias. In particular, one study (54) received  
549 “low” risk of bias ratings across all domains, another study (33) was rated consistently as “low”  
550 or “probably low” risk of bias across all domains, and several studies (52, 55, 56) only received a  
551 “probably high” rating in one category, increasing the review author’s confidence that a  
552 sufficient body of evidence was available with minimal risk of bias to rate the overall body of  
553 evidence for this study population/health outcome group. Studies that were rated “probably high”  
554 or “high” were not for reasons that were consistent across this body of evidence, and did not  
555 produce compelling reasons to downgrade the overall body of evidence as a result.

556

557 **Group 4: Adult population asthma symptoms**



Overall, the majority of adult asthma diagnosis studies were rated “low” or “probably low” risk of bias across all domains (Fig 4). Several domains were predominantly rated “low” and “probably low” but included one to two “probably high” or “high” ratings—source population (one “probably high” and one “high”), confounding (two “probably high”), incomplete outcome data (one “high”), exposure assessment (two “probably high”), and conflict of interest (one “probably high”). These studies were rated higher risk of bias for lacking details regarding recruiting and inclusion/exclusion criteria (53, 57), failure to adjust for or consider several of the important or potentially important adjustment factors outlined in our protocol (58, 59), measuring exposure only for a portion of study participants (60), relying on self-reported outcomes by study participants but lacking follow-up for validation (53), or receiving funding from a private company without including a statement of the role of this company in influencing the study (52). Unlike for included children studies, confounding did not appear as problematic for the adult studies, likely because many studies were occupational and relied on either matching participants based on baseline characteristics or were pre- and post-experimental tests that used each individual subject as their own control.

A few other domains included a higher number of “probably high” or “high” ratings—blinding (five “probably high” and one “high”) and other (four “probably high” and one “other”). Similar to adult asthma diagnosis studies, blinding was generally more problematic for included occupational studies where study participants likely were already aware of their exposure and/or outcome status and blinding was not a possibility. For the other risk of bias domain, all five studies that received high risk of bias ratings were occupational studies where potential for healthy worker effect either likely existed or was likely (for instance, de Vos et al. (61) specifically excluded individuals with “unstable asthma, current acute or chronic respiratory

581 illness, or any other chronic or severe illnesses,” thus likely leading to selection bias that favored  
582 healthier individuals.

583 Overall, review authors felt confident that the majority of adult asthma diagnosis studies were  
584 rated predominantly “low” or “probably low” risk of bias. In particular, one study (54) received  
585 “low” risk of bias ratings across all domains, another study (33) was rated consistently as “low”  
586 or “probably low” risk of bias across all domains, and a number of studies (52, 59, 62) only  
587 received a “probably high” rating in one category, increasing the review author’s confidence that  
588 a sufficient body of evidence was available with minimal risk of bias to rate the overall body of  
589 evidence for this study population/health outcome group. Studies that were rated “probably high”  
590 or “high” were not for reasons that were consistent across this body of evidence, and did not  
591 produce compelling reasons to downgrade the overall body of evidence as a result.

592 All adult studies with pulmonary measure outcomes received “probably high” or “high” ratings  
593 for the source population domain, each for slightly different reasons but all stemming from the  
594 fact that these were randomized controlled exposure trials with small sample sizes. For instance,  
595 Witek et al. (63) received a “probably high” rating because all 14 participants were a self-  
596 selected group of individuals responding to a recruitment advertisement (S86 Table). The ‘other’  
597 risk of bias domain was used predominantly to capture healthy worker bias for included  
598 occupational studies—the phenomenon that occupations where chemical exposures occur often  
599 tend to avoid employment of older, younger, or ill individuals, and hence select out for  
600 susceptible individuals (36-38) (Figs 3-4). Studies considered in the meta-analysis or sensitivity  
601 analysis were generally high quality, with only “probably high” or “high” ratings in the domains  
602 blinding, outcome assessment, or confounding (Fig 4).

Occupational studies received higher risk of bias ratings for the domains of exposure assessment and ‘other’ compared to general population studies (S2 Fig), resulting from reliance on job exposure matrices to classify formaldehyde exposures (based solely on job titles without measuring formaldehyde levels) or potential healthy worker effects. In contrast, over a third of general population studies received “probably high” or “high” ratings for the confounding domain from failure to account for the important confounding variables as outlined in our protocol. In contrast, many occupational studies incorporated matching study participants in the study design—for example matching exposed and unexposed by age, ethnicity, or job functions from similar socioeconomic status—and thus resulted in lower risk of bias ratings for confounding.

**Statistical analysis**

**Group 1: Childhood asthma diagnosis**

Of the 37 studies reporting on child populations, 24 reported on outcomes related to asthma diagnosis (i.e., children having been diagnosed by a physician as having asthma or based on self-reported asthma diagnosis). Nine of these studies were identified as combinable in a meta-analysis (41-44, 64-68) . The remaining studies could not be combined because they either categorized formaldehyde exposures or reported outcomes that could not be converted to an odds ratio (i.e., median formaldehyde exposures for those with asthma versus those without) . Attempts to obtain estimates that could be standardized to an odds ratio from the study authors were unsuccessful.

One study, Rumchev et al. (2002) , was excluded from the meta-analysis because it included very young children (between 6 months and 3 years old), which could potentially have resulted

625 in misclassification of infection-associated wheezing in young children as asthma (14), leading  
626 the NAS to conclude that this study should not be included in meta-analyses of formaldehyde  
627 and asthma. The estimate from another study in the meta-analysis, Krzyzanowski et al. (1990)  
628 (44) was investigated in a sensitivity analysis removing the estimate because it was the only  
629 unadjusted estimate included.

630 One study considered for the meta-analysis measured incident asthma cases—Smedje et al.  
631 (2001) followed children over time to identify new asthma diagnoses (43). The remaining studies  
632 measured prevalent cases based on self-reported or physician ever having diagnosed with  
633 asthma, but because they all incorporated some requirement of current asthma symptoms (i.e.,  
634 use of asthma medication or wheezing in the past 12 months) we decided that it was acceptable  
635 to combine prevalent and incident asthma cases. All studies measured indoor formaldehyde  
636 exposures, either at home or in school classrooms.

637 A meta-analysis combining effect estimates from the 9 children’s asthma diagnosis studies using  
638 random effects modeling found an elevated OR (1.20) with 95% CI range above 1 (95% CI:  
639 [1.02, 1.41]), predicting an 20% increased odds of being diagnosed with asthma per 10-µg/m<sup>3</sup>  
640 increase in formaldehyde exposure (Fig 5). Removing the estimate from Krzyzanowski et al.  
641 (44), the only study reporting unadjusted estimates, slightly elevated the odds ratio (1.20 to 1.26)  
642 with a similar 95% CI [1.04, 1.53] (Table 45). (15)

643 **Table 45.** Meta-Analysis and Sensitivity Analysis of Childhood Asthma Diagnosis (N=9 studies) Pooled ORs and 95% CIs for random-effects models.

	Number of studies	Random-effects model	
		OR (95% CI) per 10-µg/m <sup>3</sup> increase	I <sup>2</sup> (p-value)
Asthma Diagnosis	9	1.20 (1.02, 1.41)	27% (p=0.2)

Sensitivity Analysis			
(-) Krzyzanowski et al. 1990	8	1.26 (1.04, 1.53)	31% (p=0.18)
(-) Kim et al. 2011	8	1.27 (1.06, 1.54)	28% (p=0.21)

(-) indicates removing a study from the meta-analysis for sensitivity analysis

The two most statistically influential studies in the meta-analysis were Krzyzanowski et al. (44) and Kim et al. (65). We removed these study to determine how this might impact the overall effect estimate. The impact of removing Krzyzanowski et al. (44) as discussed above as part of the sensitivity analysis was minimally impactful; removing Kim et al. (65) had a similar null effect, only slightly elevating the odds ratio (1.27) and changing the 95% CI [1.06, 1.54] (Table 54). (68).

We used a funnel plot and used Egger's test for small-study effects to statistically test for publication bias in the eight studies in the meta-analyses. Our funnel plots revealed no evidence of overall publication bias (p-value=0.35) (S98 Table; S4 Fig)—however, the small number of studies (<10) might result in no indication of publication bias when in fact it might exist.

We also investigated the potential impact of a new or unpublished hypothetical study necessary to alter the results of the meta-analysis. In making this calculation, we assumed that the new hypothetical study would have a standard error equal to the smallest in our group of studies—0.14 for children asthma diagnosis (44, 66, 68). We determined that a new study would be required to have an estimate of OR=0.97, 95% CI: [0.74, 1.27] to change the 95% confidence interval of the meta-analysis overlapping one. We judged the existence of a study with such a result to be possible, given that this association estimate and confidence interval was within the range of other included studies, but not likely given that this point estimate would be in the opposite direction of all studies included in the meta-analysis.

664 To shift our meta-analysis to have an overall association estimate just below zero (i.e., increases  
665 in formaldehyde exposures would be associated with decreases in asthma outcomes) would  
666 require a new study reporting an OR=0.05, 95% CI: [0.04, 0.07]. We judged the existence of a  
667 well-conducted study with such a result to be very unlikely, given that this association estimate  
668 and confidence interval was considerably outside the range of the estimates from almost every  
669 included study.

670 Data that could not be combined into a meta-analysis were visually depicted on scatterplots when  
671 possible. The categorical odds and risk ratios (n=14), formaldehyde levels (n=6), and asthma  
672 prevalence (n=5) were visually displayed for consideration in rating the overall body of evidence  
673 (S5-7 Figs). Several studies with estimates included in the meta-analysis also reported secondary  
674 estimates (for instance, outcomes of self-reported current asthma) that were included on these  
675 scatterplots. Overall, these data appeared generally consistent with each other (i.e., increasing  
676 exposure to formaldehyde associated with increasing odds/risk ratios, asthma prevalence, and  
677 asthma status), and with the results of the meta-analysis. The secondary estimates from studies  
678 included in the meta-analysis (42, 43, 64-68) were also within the range of studies included in  
679 the meta-analysis (S5 Fig). Additional studies further supported the meta-analysis estimate; for  
680 instance, Tavernier et al. (39) reported odds ratios for self-reported asthma confirmed by  
681 physician by tertile of formaldehyde exposure, with an estimate of 1.22 (95%CI: [0.49, 3.07])  
682 comparing the third to first tertile (S5 Fig). Several studies reported associations with asthma and  
683 categorical exposures to formaldehyde, which allowed review authors to evaluate the potential  
684 for a dose-response relationship. Rumchev et al. (15) reported a consistent relationship between  
685 increasing exposure (across four exposure groups ranging from 10 to >50 µg/m<sup>3</sup>) and increased  
686 odds for asthma diagnosis. However, other studies did not illustrate a similar relationship—for

instance, Annesi Maesano (69) reported increased odds (OR=1.1, 95% CI [0.87, 1.38]) for self-reported asthma comparing the medium to low tertile for formaldehyde exposure, but decreased odds (OR=0.9, 95% CI: [0.76, 1.08]) comparing the high to low tertile (S5 Fig). Similarly, some studies reporting asthma prevalence with increasing formaldehyde exposure supported a dose-response relationship with increasing exposure (40, 54, 70, 71) whereas others did not (44) (S6 Fig). Review authors concluded that these data supported the meta-analysis results and association between formaldehyde exposure and asthma diagnosis, but that there was limited evidence supporting a dose-response relationship.

**Group 2: Childhood asthma exacerbation and symptoms**

Twenty-three studies reported symptoms related to asthma—asthma attack, wheeze, or breathlessness/shortness of breath (Table 3). Of these, six studies (40, 41, 64-67) were initially identified as potentially combinable in a meta-analysis for the association between indoor formaldehyde exposures and wheeze or daytime shortness of breath. One study reported a crude OR estimate for respiratory symptoms including wheeze and shortness of breath, but did not provide an estimate of variability (i.e., confidence limits or standard error) and therefore could not be included in the meta-analysis. Efforts to contact study authors to obtain this information were unsuccessful. Thus, we ultimately combined five studies in our meta-analysis (Fig 6). Several studies provided multiple effect estimates to the meta-analysis—e.g., Kim et al. reported effect estimates for wheeze symptoms and daytime breathlessness associated with indoor formaldehyde exposure. Overall, separate combined effects for wheeze and shortness of breath were similar and the combined effects were moderate (OR=1.08, 95% CI: [0.92, 1.28]) (Fig 6).

709 Due to the small number of studies contributing estimates to the meta-analysis, we did not  
710 conduct a statistical analysis of potential publication bias.

711 Since the meta-analysis association estimate 95% lower bound CI was below 1, we only  
712 explored the sensitivity of shifting our meta-analysis to have an overall association estimate just  
713 below zero (i.e., such that increases in formaldehyde exposures would be associated with  
714 decreases in asthma outcomes). We assumed that the new hypothetical study would have a  
715 standard error equal to the smallest in our group of studies, 0.12 (66). We concluded this would  
716 require a new study reporting an OR=0.84, 95% CI: [0.66, 1.017]. We judged the existence of a  
717 well-conducted study with such a result to be possible, given that this association estimate and  
718 confidence interval was within the range and overlapped with most of the included studies and  
719 aligned with the estimate of one study in particular.

720 The categorical odds ratios (n=10), formaldehyde levels by asthma status (n=2), and symptom  
721 scores (n=1) were visually displayed on the same figure for consideration in rating the overall  
722 body of evidence (S8-9 Figs). Most studies identified elevated association estimates from  
723 exposures to formaldehyde, but lower 95% CI was below 1. Several studies (41, 64, 65, 67, 72)  
724 reported on different asthma symptoms (asthma attacks, asthma symptoms, or wheeze) per 1  
725  $\mu\text{g}/\text{m}^3$  formaldehyde exposure and reported consistent estimates of positive odds ratios ranging  
726 from 0.96-1.2 (S7 Fig). Several studies (48, 73-75) reported on categorical formaldehyde  
727 exposures, but did not demonstrate a consistent dose-response relationship (S7 Fig). For  
728 instance, Raaschou-Nielsen (48) reported on wheezing symptom across five exposure categories  
729 (ranging from 0 to  $>25.6 \mu\text{g}/\text{m}^3$  formaldehyde) with increased odds ratios across three groups  
730 (OR=1.11, 1.21, 1.4) but a negative odds ratio for the highest exposure group (OR=0.67).  
731 Review authors concluded that these data supported the meta-analysis results and association



732 between formaldehyde exposure and asthma symptoms, but that there was limited evidence  
733 supporting a dose-response relationship.

734 Four studies reported pulmonary function measures in children, but because two studies reported  
735 on peak expiratory flow rates (PEFR) and two others reported on forced expiratory volume in  
736 one second (FEV<sub>1</sub>) and forced vital capacity (FVC), a comparison between such a small number  
737 of studies was determined not to be useful.

738

739 **Group 3: Adult population asthma diagnosis**

740 Seventeen total studies included outcomes of whether subjects had been previously diagnosed by  
741 a physician with having asthma (most commonly ascertained through use of a self-reported  
742 questionnaire (n=11) or through medical records or physician examination (n=6)). None of these  
743 17 studies reported sufficient data to evaluate outcomes with respect to a continuous 10-µg/m<sup>3</sup>  
744 increase in formaldehyde. Three studies reported results for at least two measured exposure  
745 categories; the majority of studies reported exposures categorically, such as exposed versus  
746 unexposed or by job category. Due to the small number of studies and high amount of  
747 heterogeneity in key study characteristics, the studies were not amenable to meta-analysis to  
748 combine effect estimates. We identified three studies reporting similar ranges of exposure  
749 categories to assess for a dose-response relationship for asthma diagnosis and identified a  
750 positive trend (Fig 7), although review authors noted the small number of studies and limited  
751 dose groups included.

752 The formaldehyde levels by categorical odds ratios (n=4) and asthma prevalence (n=4) were  
753 visually displayed for consideration in rating the overall body of evidence (S10-11 Figs).

754 Although the categorical odds ratios varied considerably in how formaldehyde exposures were  
755 categorized (i.e., high vs. low, exposed to newly painted dwelling/workplace vs. not, occupations  
756 exposed to formaldehyde vs. not, etc.), there was a consistent increase in odds of asthma  
757 diagnosis with increased category of exposure (S10 Figure). For instance, Billionnet et al. (56)  
758 reported an increased odds (OR=1.43) for those in the high exposure group ( $\geq 28.03 \mu\text{g}/\text{m}^3$ )  
759 compared to those in the low exposure group ( $< 28.03 \mu\text{g}/\text{m}^3$ ). However, review authors noted a  
760 limitation with Billionnet et al. (56) in that no estimates of statistical confidence (i.e., standard  
761 error, 95% confidence interval) were reported with these estimates. Although all four studies  
762 reported increased odds with increased category of exposure, only Herbert et al. (76) reported a  
763 statistically significant increase (comparing exposed versus non-exposed occupational  
764 groups). Similarly, the scatterplot of prevalence data by formaldehyde exposure categories  
765 demonstrated a similar pattern of supporting increases in asthma prevalence with increasing  
766 formaldehyde exposure (S11 Figure). For instance, Elshaer and Mahmoud (50) reported dramatic  
767 prevalence increases in exposed occupational workers for asthma (53.3%) versus non-exposed  
768 workers.

769 Considering the overall evidence, review authors concluded that there did appear to be evidence  
770 supporting a relationship between increasing formaldehyde exposure and asthma diagnosis,  
771 although the number of studies was low and the variety of exposure categories made it  
772 challenging to easily compare across different studies.

773

774 **Group 4: Adult population asthma symptoms**

775 Twenty studies reported on asthma-related symptoms—i.e., asthma attack, wheeze, or  
776 breathlessness/shortness of breath (Table 3). All studies reported categorical formaldehyde  
777 exposures and therefore could not be combined in a meta-analysis. The categorical odds ratios  
778 (n=5), asthma prevalence (n=4), and symptom score (n=1) were visually displayed for  
779 consideration in rating the overall body of evidence (S9-11 Figs). The symptom score study and  
780 most studies reporting odds ratios documented increased risk of symptoms with exposure to  
781 formaldehyde, with several reporting statistically significant findings (S9-10 Figs). For instance,  
782 Herbert et al. (76) reported a statistically significant increase in asthma symptoms (attacks of  
783 wheeze) comparing exposed versus non-exposed occupational groups. Asthma prevalence  
784 estimates were generally greater with increased exposure to formaldehyde, but these studies  
785 lacked confidence intervals around the point estimates (S11 Fig). However, there were few  
786 studies reporting on prevalence outcomes and results were not consistent across studies. For  
787 instance, Kilburn, Seidman, and Warshaw (53) reported consistent increases in asthma symptom  
788 prevalence in an occupational setting with increases in the hours of exposure to formaldehyde  
789 but Thetkathuek et al. (58) reported an inconsistent relationship with wheeze symptoms across  
790 low, moderate, and high formaldehyde exposure groups (lower prevalence in the moderate  
791 exposure group compared to low exposure group).

792 There were also 32 total studies that reported on pulmonary lung measures in adults. We decided  
793 to focus on studies reporting associations between formaldehyde exposure and Forced Expiratory  
794 Volume in 1 second (FEV1) outcomes, following recommendations from National Institute of  
795 Health (NIH) to use FEV1 as a supplemental outcome related to asthma exacerbation. Most  
796 studies reported FEV1 outcomes (n=27), but not all reported associations with formaldehyde  
797 exposures. Several studies reported FEV1 percentage changes comparing to baseline values

798 (either to a comparator group or standardized values, for instance standardized predicted values  
799 based on age, height and gender published by the American Thoracic Society (77)—we decided  
800 not to plot these on the same figure due to lack of comparability across studies using different  
801 comparisons or standardized values. Of the 27 studies, 7 reported associations between FEV1  
802 measured values with formaldehyde exposures. These were visually displayed for consideration  
803 in rating the overall body of evidence (S12 Fig). Four of the studies reported confidence intervals  
804 for association estimates that overlapped between exposed and comparator groups but did not  
805 find consistent changes in FEV1 with formaldehyde exposures (i.e., comparing formaldehyde-  
806 exposed participants to controls, two studies reported decreases in FEV1 while the other two  
807 reported increases .

808 **Considering the overall evidence, review authors concluded that there did appear to be**  
809 **evidence supporting a relationship between increasing formaldehyde exposure and asthma**  
810 **symptoms, although the number of studies was low and the variety of exposure categories**  
811 **made it challenging to easily compare across different studies. Rating quality and strength**  
812 **of the body of evidence**

813 Based on the comparison of the body of evidence to pre-specified criteria in our protocol (S4  
814 Methods), the review authors concluded that there was “moderate” quality for the body of  
815 evidence for each of the four-study population/health outcome groups (Table 65). Review  
816 authors did not apply any upgrades (for large magnitude of effect, dose-response relationship, or  
817 confounding that minimizes effect) or downgrades (for risk of bias, indirectness, inconsistency,  
818 imprecision, or publication bias) to criteria across the body of evidence, which led to the final  
819 rating of “moderate”.

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823 **Table 65.** Summary of rating quality and strength of the human evidence, by population/  
outcome group

824 **A. Children asthma diagnosis**

Category	Downgrades	Rationale
<b>Initial Rating of human evidence = Moderate</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. The confounding domain appeared to be most frequently problematic due to failure to adjust for the important confounders outlined in the protocol; however, a number of included studies were rated as “low” or “probably low” risk of bias, including several studies ultimately included in the meta-analysis. Review authors concluded that this did not appear to warrant downgrading for risk of bias across all studies.
Indirectness	0	The population, exposure, and outcome were all directly related to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.
Inconsistency	0	Studies included in the meta-analysis have similar point estimates with overlap among the confidence intervals. Effect estimates across studies were mostly positive (showing increased risk). Estimates from the meta-analysis indicate that statistical heterogeneity was moderate, but not statistically significant ( $I^2=46.5\%$ , $p\text{-value}=0.06$ ).

Imprecision	0	No concern regarding the imprecision in effect estimates across studies.
Publication bias	0	Could not rule out publication bias, but there is no affirmative evidence of its existence—in particular, funnel plots revealed no evidence of overall publication bias (p-value = 0.35).
	Upgrades	
Large magnitude of effect	0	The overall effect size from the meta-analysis is small but precise. Authors concluded there was not enough evidence to warrant upgrading for this domain.
Dose-response	0	Results from the meta-analysis between formaldehyde exposure and child asthma diagnosis, which assumes a linear dose-response relationship, appeared to support the existence of an association of increasing response with increased dose. However, there was limited data to statistically evaluate whether there was a dose-response relationship, primarily due to the small number of studies and the heterogeneity in reporting of effect estimates. Review authors did not believe that results from the meta-analysis were sufficient to warrant upgrading the body of evidence for evidence of a dose-response relationship.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more

		well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.
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826 **B. Children asthma exacerbation and symptoms**

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. The confounding domain appeared to be most consistently problematic due to failure to adjust for the important confounders outlined in the protocol; however, a number of included studies were rated as “low” or “probably low” risk of bias, including several studies ultimately included in the meta-analysis. Review authors concluded that this did not warrant downgrading for risk of bias across all studies.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.

Inconsistency	0	Effect estimates across studies were consistent across the body of evidence, in particular as seen by the categorical odds ratios and the prevalence data visual scatterplots.
Imprecision	0	No concern regarding the imprecision in effect estimates across studies.
Publication bias	0	Number of studies included were too small (i.e., <10) for a statistical evaluation of potential publication bias. Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Studies that found positive relationship between exposure and outcome were interpreted as a minimal magnitude of effect; insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Results from the meta-analysis between formaldehyde exposure and children asthma exacerbation and symptoms, which assumes a linear dose-response relationship, appeared to support the existence of an association of increasing response with increased dose.



		However, there was not enough evidence to statistically evaluate existence of a dose-response relationship, primarily due to the small number of studies and the heterogeneity in reporting of effect estimates. Review authors did not believe that results from the meta-analysis were sufficient to warrant upgrading the body of evidence for evidence of a dose-response relationship.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.

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834 C. Adult asthma diagnosis

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear consistently problematic across all studies. Most studies were rated “low” risk of bias across most domains with only one or two “probably high” ratings, with the exception of only a few studies. Occupational studies received “probably high” ratings for blinding, exposure assessment and “other” domains, but review authors did not feel this warranted a downgrade to the overall body of evidence.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.
Inconsistency	0	Effect estimates across studies were generally consistent across the body of evidence; heterogeneity likely explained by the differing study designs, and data demonstrate a tendency towards increased asthma diagnosis and therefore would not warrant a downgrade for this domain.

Imprecision	0	Confidence intervals appeared to be somewhat wide, but review authors did not feel there was enough evidence to warrant downgrading for this domain.
Publication bias	0	Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Studies that found positive relationship between exposure and outcome were interpreted as a minimal magnitude of effect; insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Data supported the existence of a dose-response relationship, but review authors did not feel it was strong enough to warrant an upgrade for this domain.
Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.

<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.
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837 **D. Adult asthma exacerbation and symptoms**

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Category	Downgrades	Rationale
<b>Initial Rating of human evidence = “Moderate”</b>		
Risk of bias	0	Generally risk of bias did not appear problematic across all studies. Occupational studies appeared to have probably high ratings for blinding, exposure assessment and other domains, but review authors did not feel this warranted a downgrade to the overall body of evidence.
Indirectness	0	The population, exposure, and outcome were directly relevant to the PECO statement population, exposure, and outcome. There were no concerns regarding the indirectness of evidence in supporting the study question at hand.

Inconsistency	0	Effect estimates across studies were generally consistent across the body of evidence; heterogeneity likely explained by other factors, and data demonstrate a tendency towards increased asthma exacerbation and symptoms and therefore would not warrant a downgrade for this domain.
Imprecision	0	Confidence intervals appeared to be somewhat wide, but review authors did not feel there was enough evidence to warrant downgrading for this domain.
Publication bias	0	Publication bias cannot be ruled out, but there was no affirmative evidence of its existence. We conducted a comprehensive search to identify grey literature sources (i.e., conference abstracts and graduate theses) in an attempt to identify potential publication bias and did not find evidence of such (for instance, studies reporting null or negative findings in a conference abstract that lacked a subsequent publication in the peer-reviewed literature).
	Upgrades	
Large magnitude of effect	0	Some studies illustrate large impact, but this is not consistent across the studies and so review authors concluded there was insufficient evidence to upgrade for large magnitude of effect consideration.
Dose-response	0	Some data supported the existence of a dose-response relationship, but review authors did not feel it was strong enough to warrant an upgrade for this domain.

Confounding minimizes effect	0	There was no evidence that residual confounding influenced results.
<b>Overall Quality of Evidence</b>	<b>Moderate</b>	Review authors did not feel that the evidence was strong enough to warrant downgrading or upgrading the overall quality rating and came to a final conclusion of “moderate” evidence.
<b>Overall Strength of Evidence</b>	<b>Sufficient</b>	A positive relationship is observed between exposure and outcome where chance, bias, and confounding can be ruled out with reasonable confidence. The available evidence includes results from one or more well-designed, well-conducted studies, and the conclusion is unlikely to be strongly affected by the results of future studies.

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840 Review authors noted that risk of bias limitations did exist across each of the study  
841 population/health outcome groups. Concerns were generally limited to the domains of blinding,  
842 confounding, exposure assessment, and “other” (the latter being predominantly limited to  
843 occupational studies that were rated for potential healthy worker bias) domains. For instance,  
844 several child asthma diagnosis studies were rated “high” (n=4) or “probably high” (n=6) for  
845 confounding due to the failure to adjust for the important confounders outlined in our pre-  
846 published protocol. A number of other studies were rated as “probably high” for various other  
847 domains (source population, outcome assessment, incomplete outcome, and exposure  
848 assessment). However, review authors felt that overall a sufficient number of studies were rated  
849 “low” or “probably low” risk of bias across all domains, in particular several studies ultimately  
850 included in the meta-analysis (i.e., (65-68)) and review authors concluded that these limitations

851 did not rise to the level of a downgrade, in accordance with the instructions outlined in the  
852 protocol (<http://www.crd.york.ac.uk/PROSPERO/>; Record ID #38766, CRD 42016038766).  
853 Review authors came to similar conclusions in evaluating the risk of bias for each of the other  
854 three study population/health outcome groups. In particular, review authors noted that many of  
855 the “high” or “probably high” risk of bias ratings were assigned to a select subgroup of studies  
856 (i.e., those with issues stemming from small sample sizes or occupational studies due to healthy  
857 worker bias concerns) but the remaining included studies did not suffer from such limitations and  
858 had minimal risk of bias concerns. Review authors did not apply downgrades to the evidence for  
859 the other domains for any of the study population/health outcome groups because there lacked  
860 sufficient evidence supporting existence of indirectness, inconsistency, imprecision, or  
861 publication bias.

862 Review authors also did not apply any upgrade factors for any of the study population/health  
863 outcome groups. For child asthma diagnosis and child asthma symptoms evidence, although we  
864 were able to conduct a meta-analysis that supported an association between increasing response  
865 with increased dose (based on an assumption of model linearity), there were too few studies to  
866 support the formal analysis of a dose-response relationship. Furthermore, as discussed above  
867 visual inspections of scatterplots of data not able to be combined in a meta-analysis provided  
868 mixed evidence supporting the existence of a consistent dose-response relationship. Review  
869 authors concluded that overall this evidence was not sufficient enough to warrant upgrading the  
870 evidence for dose-response relationship,

871 Ultimately, review authors rated the overall strength of evidence as “sufficient” for each of the  
872 four outcome groups (Table 65), based on: a) “moderate” quality of the body of evidence; b)  
873 direction of the association (i.e., consistent evidence of a positive association between

874 formaldehyde exposure and outcomes of either asthma diagnosis or exacerbation in symptoms,  
875 in both adults and children; c) confidence in the association with multiple well-conducted studies  
876 (i.e., several studies were prospective cohort studies that were of “low” or “probably low” risk of  
877 bias overall; and positive and/or statistically significant overall estimates of association from the  
878 combination of similar studies in a meta-analysis (Figs 5-6).

879

#### 880 **Economic analysis**

881 We valued the outcome of avoiding a case of asthma in children, as it had the strongest support  
882 from well-conducted combinable studies with minimal risk of bias concerns. We used the OR  
883 estimate of 1.20 per 10  $\mu\text{g}/\text{m}^3$  (95% CI: [1.02, 1.41]) (Fig 5) based on the random effects meta-  
884 analysis model for asthma diagnosis in children from indoor formaldehyde exposure.

885 We rescaled this OR to estimate the reduction in risk per 1 ppb decrease in formaldehyde  
886 exposure (OR of 1.02265 per 1 ppb change in formaldehyde). We estimated that EPA’s proposed  
887 rule on pressed wood products would have resulted in 2,805 fewer asthma cases annually once  
888 the impacts of the reduction has reached steady-state.

889 We estimated a willingness to pay for a treatment that would eliminate asthma of \$75,024, which  
890 translates into total economic benefits for asthma reduction from EPA’s rule of approximately  
891 \$210 million annually across all children in the U.S. over a 30-year period. (Table 76).

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**Table 76.** Cases reduced and willingness to pay for a reduction in Formaldehyde exposure implied by the proposed EPA rule on pressed wood products (once the impacts of the rule have reached steady-state)

	<b>Exposure reduction (ppb)</b>	<b>Individuals Affected</b>	<b>Cases avoided</b>	<b>Benefits with WTP = \$75,024</b>
Structure age 0-1	-3.390085	599,822	364.0	\$27,311,030
Structure age 1-2	-2.178523	599,822	237.1	\$17,787,752
Structure age 2-3	-1.408503	599,822	154.6	\$11,599,437
Structure age 3-4	-0.926590	599,822	102.3	\$7,671,854
Structure age 4-5	-0.624871	599,822	69.2	\$5,191,181
Structure age 5-6	-0.431493	599,822	47.9	\$3,592,426
Structure age 6-7	-0.306329	599,822	34.0	\$2,553,938
Structure age 7-8	-0.229512	599,822	25.5	\$1,915,142
Structure age 8-9	-0.181581	599,822	20.2	\$1,516,000
Structure age 9-10	-0.152852	599,822	17.0	\$1,276,554
Structure age 10-11	-0.133711	599,822	14.9	\$1,116,939
0-1 years post-ren.	-2.363858	1,306,316	559.1	\$41,948,116
1-2 years post-ren.	-1.525697	1,306,316	364.3	\$27,327,908
2-3 years post-ren.	-1.002335	1,306,316	240.7	\$18,058,604
3-4 years post-ren.	-0.668362	1,306,316	161.1	\$12,086,556
4-5 years post-ren.	-0.458218	1,306,316	110.7	\$8,305,820
5-6 years post-ren.	-0.323412	1,306,316	78.3	\$5,871,128
6-7 years post-ren.	-0.239982	1,306,316	58.1	\$4,360,639
7-8 years post-ren.	-0.189089	1,306,316	45.8	\$3,437,825
8-9 years post-ren.	-0.156738	1,306,316	38.0	\$2,850,684
9-10 years post-ren.	-0.133647	1,306,316	32.4	\$2,431,347
10-11 years post-ren.	-0.124415	1,306,316	30.2	\$2,263,624
<b>Total</b>		<b>20,967,514</b>	<b>2,805</b>	<b>\$210,474,503</b>

900

901 **Discussion**

902 We found “sufficient” evidence of an association between exposure to formaldehyde and asthma  
903 diagnosis and asthma symptoms in children and adults. The definition of “sufficient” was  
904 predefined in our protocol (Table 1). Our review had several strengths, including that we used  
905 the Navigation Guide systematic review methodology, which specifically accounted for the  
906 weaknesses identified by the NAS in the IRIS formaldehyde assessment, i.e., explicit and  
907 transparent study selection and evaluation criteria, including exclusion of a study in which  
908 asthma may have been misclassified . Moreover, our review was based only on studies where the  
909 asthma status of participants was known and which reported quantitative measures of  
910 formaldehyde exposure, and our methods accounted for several considerations of causality as  
911 part of the evaluation, specifically, our PECO statement limited included evidence based on  
912 temporality criteria and the evaluation of the strength and quality of evidence incorporated  
913 considerations of strength, consistency, and biological gradient.

914 We retrieved six self-identified “systematic reviews” of formaldehyde and asthma conducted  
915 between 2011 and 2015 in the literature search for our review (78-83),. Of the three reviews with  
916 findings consistent with our review, two conducted a meta-analysis of the data (78, 83) and the  
917 third cited the McGwin et al. meta-analysis (82). The three reviews which did not find  
918 compelling evidence for an association between asthma and formaldehyde exposure did not  
919 conduct a meta-analysis, and there was a wide disparity in the number and type of papers  
920 included in these reviews. Specifically, our review included 22, 17, 17, and 20 studies on child

921 asthma diagnosis, child asthma symptoms, adult asthma diagnosis and adult asthma symptoms,  
922 respectively.

923 In contrast, Patelarou et al. (81) included 2 studies on formaldehyde and asthma and wheezing in  
924 children up to 5 years old; Baur et al. (80) included 8 studies on formaldehyde and asthma in  
925 occupational settings; and Nurmatov et al. (79) included 17 studies on formaldehyde and asthma  
926 etiology, 1 study on formaldehyde and asthma exacerbation, and 14 studies on asthma etiology  
927 and exacerbation (among which the authors found a positive association between formaldehyde  
928 and wheezing in young children on the basis of a “well-conducted, low-risk of bias” randomized  
929 controlled trial, which was consistent with our findings). While none of these six self-described  
930 systematic reviews fully met all of the criteria for a systematic review as specified in the  
931 Literature Review Appraisal Toolkit (<http://policyfromscience.com/lrat/about-the-lra-toolkit/>),  
932 the transparency of their methods allowed for better understanding the discrepant results.

933 In 2016, EPA published its final rule to regulating formaldehyde in pressed wood products as  
934 well as household and other finished goods. The regulations set by this final rule did not consider  
935 the benefits of preventing asthma; estimated annualized benefits (from avoided incidence of eye  
936 irritation and nasopharyngeal cancer outcomes only) ranged from \$64-186 million per year. Our  
937 results show that using assumptions consistent with EPA’s proposed rule [24], the final rule  
938 excluded approximately \$210 million annually in total economic benefits associated with 2,805  
939 fewer asthma cases. Furthermore, these benefits were calculated based on the willingness to pay  
940 for asthma control, and could potentially represent an underestimate of the true valuation of  
941 one’s willingness to pay for avoiding an asthma diagnosis in the first place.

942 Formaldehyde is a high-production volume chemical ubiquitous in homes, communities, and  
943 workplaces and asthma is a prevalent and costly chronic health outcome. While our results show

944 that the association between exposure to formaldehyde and asthma is robust, the effect estimate  
945 is relatively small, i.e., an 8% increase in children's asthma diagnosis per 10-fold increase in  
946 exposure. These findings underscore that preventing relatively "low" risks brings "high" health  
947 benefits when exposures are ubiquitous. Our results demonstrate that benefits analyses that  
948 inform regulatory action need to account for all relevant health outcomes as to not do so could  
949 underestimate benefits.

950 Formaldehyde is a well-defined respiratory irritant and has been identified as a known  
951 respiratory carcinogen in humans. There are several proposed mechanisms supporting the role of  
952 formaldehyde exposure in asthma development. Formaldehyde is a small molecule with the  
953 ability to conjugate with large serum protein molecules such as albumin. This can provoke the  
954 formation of IgE antibodies, leading to degranulation of mast cells with allergic asthma response  
955 (84). As a small molecule, formaldehyde may bind to the amino group in proteins acquiring  
956 antigenic capacities, causing immune response with the formation of specific antibodies and  
957 triggering a local mast cell response (85). Formaldehyde is also readily absorbed into respiratory  
958 tract tissue, where it may increase T-helper cell type 2 (Th2) mediated inflammatory response  
959 and lead to cytokine mediators (3g., IL4, IL5, and IL13) release, epithelial mucous cell  
960 metaplasia, and airway recruitment of eosinophils (84). Lastly, formaldehyde may also react with  
961 the thiol group and interfere S-nitrosoglutathione function, triggering an airway response(86).  
962 Our systematic review had several limitations. First, we focused on evaluating only studies  
963 where asthma status of all study participants was measured and excluded other studies, namely  
964 studies relevant to our PECO statement but where the asthma status of participants was unknown  
965 or there were no asthmatics included, reported no quantitative measured of formaldehyde, or  
966 non-English studies. This likely would not influence our findings as studies with missing

967 assessments for exposure and outcome are of poorer quality. We also did not independently  
968 evaluate temporality of exposure and note that included cross-sectional studies where exposures  
969 were measured concurrent to asthma outcomes may not accurately represent exposures occurring  
970 prior to asthma outcomes.

971 Second, while our review documented an association between formaldehyde exposure and  
972 increased childhood asthma diagnosis, symptoms and exacerbation, it did not address whether  
973 formaldehyde directly causes childhood asthma, or rather, is a trigger for childhood asthma.  
974 Asthma is a complex chronic disease that can be challenging to diagnose accurately and for  
975 which symptoms are apparent only when there is a trigger. The trigger does not necessarily cause  
976 ‘asthma’, but will cause an ‘asthma flare up’, which helps lead to the diagnosis. Thus, it is  
977 possible that formaldehyde is a ‘trigger’ for a child who is yet to be diagnosed with asthma or it  
978 can be that formaldehyde exposure leads to the development of asthma. It is impossible to  
979 determine this unless without a human interventional study.

980 Third, key estimates utilized in the economic analysis (i.e., baseline asthma risk and willingness  
981 to pay for asthma reduction) were U.S.-based estimates. Thus, the economic evaluation and  
982 monetized value of benefits from formaldehyde exposure reduction may not be directly  
983 applicable in other global settings. However, inclusion of studies in the systematic review was  
984 not limited by geographic location and we ultimately included studies from a variety of countries  
985 (Sweden, France, Australia, China, South Korea, Denmark, Finland, Poland, Portugal, United  
986 Kingdom, New Zealand, Romania, Russia, Japan, Indonesia, Thailand, Iran, the United Arab  
987 Emirates), with the first five countries in addition to the U.S. contributing to the meta-analysis  
988 estimates. Thus, results and conclusions from the systematic review are likely relevant to

989 international settings and results from the economic analyses may be modified with geographic-  
990 specific estimates to gauge potential economic benefits in international settings.

991 Our results underscore that the inability to combine studies in a meta-analysis due to lack of  
992 reporting in published studies is a major challenge for systematic reviews in environmental  
993 health specifically, and for environmental health decision-making more broadly. The association  
994 between asthma and formaldehyde exposure is well-studied, as demonstrated by the large  
995 number of epidemiology studies. However, even with a large number of included studies, there  
996 were multiple limitations to the studies that restricted our ability to combine estimates into a  
997 meta-analysis—for instance, if studies only reported categorical formaldehyde exposures or if  
998 they did not report odds ratio or relative risk estimates. Visual scatterplots of data assisted review  
999 authors' evaluation of the consistency and interpretation of data results, but many studies did not  
1000 provide data amenable to extraction for scatterplots. For example, of the 26 adult (occupational  
1001 and general population) asthma diagnosis studies, only 17 studies included outcome data on a  
1002 physician diagnosis; none of these 17 studies reported sufficient data to evaluate outcomes with  
1003 respect to a continuous increase in formaldehyde; and few studies reported results for at least two  
1004 measured exposure categories. Hence, quantitative data from 9 papers were not reported in a  
1005 manner that they could be objectively incorporated (i.e., not using the author's conclusions but  
1006 rather just by extracting the data) into this review. Checklists such as Strengthening the  
1007 Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational  
1008 human studies to guide the reporting of elements necessary to describe studies comprehensively  
1009 and transparently may assist with these efforts and have already been incorporated into the  
1010 publication process of several high-impact journals. Furthermore, journal reviews and editors  
1011 may contribute to addressing this issue by requesting increased reporting or open-access of

quantitative data in a format conducive to future data analyses. Conducting a systematic review prior to the development and initiation of a new study could help design efficient studies that are intended to build on existing data and address research gaps intentionally to support future systematic reviews, risk assessment, and timely decision-making on environmental chemicals.

**Conclusion**

The review authors concluded that there was “sufficient” evidence supporting an association between childhood and adult exposures to formaldehyde with asthma diagnosis and symptoms. Although studies supported modest associations (our meta-analysis for childhood exposure to formaldehyde with asthma symptoms resulted in a combined OR=1.08), ubiquitous exposure to formaldehyde can result in potentially large impacts to population health. Our economic analysis identified annual benefits of 2,805 fewer asthma cases in the U.S.; the total economic benefit for asthma reduction from U.S. EPA’s rule would be approximately \$210 million annually. Thus, excluding asthma health outcomes when conducting regulatory benefit-cost analysis can underestimate the true population benefits and lead to decisions that are not fully protective of the public. Although these economic estimates are specific to the U.S., the inclusion of studies from broad geographic range indicate that results and conclusions from the systematic review are likely relevant to international settings. Our findings document that preventing formaldehyde exposure in adults and children could reduce the occurrence and impacts of a serious, chronic disease and provide significant health and economic benefits.

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1054

1055 **Figure Legends**

1056 **Figure 1.**  
1057 PRISMA flowchart showing the literature search and screening process for studies relevant to  
1058 formaldehyde exposure and asthma outcomes. Our search was not limited by language or  
1059 publication date (search was conducted up until April 1, 2020). The search terms used for each  
1060 database are provided in S1-7 Tables.

1061

1062 **Figure 2.**  
1063 Timeline of U.S. Environmental Protection Agency (EPA) action on formaldehyde from  
1064 September 1990-December 2016, highlighting Integration Risk Information System (IRIS) final  
1065 assessments releases, reassessments, internal and external reviews, and final rules issued.

1066

1067 **Figure 3.**  
1068 Cumulative risk of bias ratings (low, probably low, probably high, or high) across all human  
1069 studies included in our systematic review of formaldehyde exposure and asthma outcomes. Risk  
1070 of bias designations for individual studies are assigned by review authors according to criteria



1071 provided in S3 Methods (Risk of Bias instructions) and the justifications for each study are  
1072 provided in S8-95 Tables.

1073  
1074 **Figure 4.**  
1075 Risk of bias ratings (low, probably low, probably high, or high) for all human studies included in  
1076 our systematic review of formaldehyde exposure and asthma outcomes, organized by study  
1077 population (children or adult) and outcome (asthma diagnosis, asthma symptoms, or pulmonary  
1078 measures). Risk of bias designations for individual studies are assigned by review authors  
1079 according to criteria provided in S3 Methods (Risk of Bias instructions) and the justifications for  
1080 each study are provided in S8-95 Tables.

1081  
1082 **Figure 5.**  
1083 Meta-analysis of human studies (n=9 studies, including a total of 9,049 children) for  
1084 formaldehyde exposure for asthma diagnosis assessed in children up to 15 years of age: reported  
1085 effect estimates and 95% confidence interval (CI) from individual studies (inverse-variance  
1086 weighted, represented by size of rectangle) and overall pooled estimate from random effects  
1087 (RE) model per 10  $\mu\text{g}/\text{m}^3$  increase in formaldehyde exposure. Heterogeneity statistics:  $I^2 =$   
1088 27.2%,  $p=0.202$ .

1089  
1090 **Figure 6.**  
1091 Meta-analysis of human studies (n=5 studies, including a total of 7,662 children) for  
1092 formaldehyde exposure for asthma symptoms (wheeze and shortness of breath) assessed in  
1093 children up to 15 years of age: reported effect estimates and 95% confidence interval (CI) from  
1094 individual studies (inverse-variance weighted, represented by size of rectangle) and overall  
1095 pooled estimate from random effects (RE) model per 10  $\mu\text{g}/\text{m}^3$  increase in formaldehyde  
1096 exposure. Heterogeneity statistics:  $I^2 = 0\%$ ,  $p=0.899$ .

1097  
1098  
1099  
1100 **Figure 7.**  
1101 Dose-response relationship (n=3 studies, including a total of 3,600 adult participants) between  
1102 formaldehyde exposure ( $\mu\text{g}/\text{m}^3$ ) and relative risk of asthma diagnosis in adults. Dose-response  
1103 data from Yeatts et al. 2012 (63), Billionnet et al. 2011 (92), Matsunaga et al. 2008 (93). Data  
1104 were modeled with random-effects log linear models with restricted cubic splines mixed effects  
1105 methods with exchangeable covariance structure of multivariable-adjusted relative risks. Lines  
1106 with long dashes represent the 95% confidence interval (CI) bounds for the fitted nonlinear trend  
1107 (solid line). Symbols (triangles, circles, and squares) represent point estimates.

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## 1340 Supplemental Information

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1462 S1 Methods. Exclusion criteria for screening references

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1467 **S1 Results. List of included studies not considered**  
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# Association of Changes in Air Quality With Incident Asthma in Children in California, 1993-2014

Erika Garcia, PhD; Kiros T. Berhane, PhD; Talat Islam, PhD; Rob McConnell, MD; Robert Urman, PhD; Zhanghua Chen, PhD; Frank D. Gilliland, MD, PhD

**IMPORTANCE** Exposure to air pollutants is a well-established cause of asthma exacerbation in children; whether air pollutants play a role in the development of childhood asthma, however, remains uncertain.

**OBJECTIVE** To examine whether decreasing regional air pollutants were associated with reduced incidence of childhood asthma.

**DESIGN, SETTING, AND PARTICIPANTS** A multilevel longitudinal cohort drawn from 3 waves of the Southern California Children's Health Study over a period of air pollution decline. Each cohort was followed up from 4th to 12th grade (8 years): 1993-2001, 1996-2004, and 2006-2014. Final follow-up for these data was June 2014. Population-based recruitment was from public elementary schools. A total of 4140 children with no history of asthma and residing in 1 of 9 Children's Health Study communities at baseline were included.

**EXPOSURES** Annual mean community-level ozone, nitrogen dioxide, and particulate matter less than 10  $\mu\text{m}$  ( $\text{PM}_{10}$ ) and less than 2.5  $\mu\text{m}$  ( $\text{PM}_{2.5}$ ) in the baseline year for each of 3 cohorts.

**MAIN OUTCOMES AND MEASURES** Prospectively identified incident asthma, collected via questionnaires during follow-up.

**RESULTS** Among the 4140 children included in this study (mean [SD] age at baseline, 9.5 [0.6] years; 52.6% female [ $n = 2179$ ]; 58.6% white [ $n = 2273$ ]; and 42.2% Hispanic [ $n = 1686$ ]), 525 incident asthma cases were identified. For nitrogen dioxide, the incidence rate ratio (IRR) for asthma was 0.80 (95% CI, 0.71-0.90) for a median reduction of 4.3 parts per billion, with an absolute incidence rate decrease of 0.83 cases per 100 person-years. For  $\text{PM}_{2.5}$ , the IRR was 0.81 (95% CI, 0.67-0.98) for a median reduction of 8.1  $\mu\text{g}/\text{m}^3$ , with an absolute incidence rate decrease of 1.53 cases per 100 person-years. For ozone, the IRR for asthma was 0.85 (95% CI, 0.71-1.02) for a median reduction of 8.9 parts per billion, with an absolute incidence rate decrease of 0.78 cases per 100 person-years. For  $\text{PM}_{10}$ , the IRR was 0.93 (95% CI, 0.82-1.07) for a median reduction of 4.0  $\mu\text{g}/\text{m}^3$ , with an absolute incidence rate decrease of 0.46 cases per 100 person-years.

**CONCLUSIONS AND RELEVANCE** Among children in Southern California, decreases in ambient nitrogen dioxide and  $\text{PM}_{2.5}$  between 1993 and 2014 were significantly associated with lower asthma incidence. There were no statistically significant associations for ozone or  $\text{PM}_{10}$ .

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Asthma is the most common pediatric chronic disease, estimated to have affected 14% of children globally in 2002-2003.<sup>1</sup> There has been much interest in the effect of outdoor air pollution on asthma risk given its ubiquity and high levels in urban areas, which, compared with rural areas, have higher rates of asthma.<sup>2</sup> Globally, outdoor air pollution has been recognized as a major public health concern and was estimated to have contributed 6.8% of the annual disability-adjusted life-years lost in 2016.<sup>3</sup> Although ambient air pollution exposure has been causally linked to asthma exacerbations in children,<sup>4</sup> evidence has been limited for a role in asthma development.<sup>2,4</sup> There has been support for a link with close proximity to busy roads,<sup>4-6</sup> but studies of regional pollutants, such as ozone, nitrogen dioxide, and particulate matter (PM), have provided less robust evidence.

This study was designed to take advantage of secular trends in air pollution to examine asthma incidence among children recruited and followed up longitudinally within the same set of communities over a period of air pollution decline. Since the early 1990s, air pollutant concentrations have decreased in Southern California.<sup>7</sup> During this time, several successive cohorts of schoolchildren were enrolled from the same set of communities as part of the Southern California Children's Health Study (CHS), a long-term study of cardiopulmonary pediatric health outcomes.<sup>8</sup> Leveraging this unique data resource, the study examined whether observed reductions in regional air pollutants, specifically ozone, nitrogen dioxide, and PM less than 10  $\mu\text{m}$  ( $\text{PM}_{10}$ ) or less than 2.5  $\mu\text{m}$  ( $\text{PM}_{2.5}$ ), were associated with asthma incidence rates within these CHS communities.

## Methods

### Study Design and Participants

All parents or guardians of participating children provided written informed consent. The study protocol was approved by the institutional review board of the University of Southern California. The study population was drawn from 3 successive cohorts of the CHS. The CHS has been described in detail elsewhere.<sup>8,9</sup> Briefly, 12 communities in Southern California were selected in 1993 based on historical air pollutant levels.<sup>8</sup> Children were recruited from participating communities through public schools and followed up prospectively until 12th grade. Fourth graders aged 9 to 10 years were recruited in 1993 ( $n = 1798$ ) and in 1996 ( $n = 2061$ ) from the 12 communities. In 2003, kindergarteners and first graders ( $n = 5736$ ) were recruited from 13 communities, resulting in a total of 16 communities contributing data to the 3 cohorts.<sup>9</sup> These 3 cohorts will hereafter be referred to as the 1993-2001, 1996-2004, and 2006-2014 cohorts. Nine communities participated in all 3 cohorts (Alpine, Lake Elsinore, Lake Gregory, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland;  $n = 6858$ ).

Baseline questionnaires regarding the children's health and exposures, as well as demographic information, were

### Key Points

**Question** What is the association between reductions in regional air pollutant concentration and incidence of childhood asthma?

**Findings** In this longitudinal study that included 4140 children, each 4.3-parts-per-billion decrease in nitrogen dioxide was associated with a reduction of 0.83 cases per 100 person-years in asthma incidence; each 8.1- $\mu\text{g}/\text{m}^3$  decrease in particulate matter less than 2.5  $\mu\text{m}$  was associated with a reduction of 1.53 cases per 100 person-years in asthma incidence. There were no statistically significant associations with change in ozone and particulate matter less than 10  $\mu\text{m}$ .

**Meaning** Declines in nitrogen dioxide and particulate matter less than 2.5  $\mu\text{m}$  may be associated with decreased childhood asthma incidence.

completed by the parents or guardians. Annual follow-up questionnaires assessing changes in the children's health, among other factors, were completed initially by the parents or guardians and later by the participating children starting at approximately age 11 years. To better facilitate the comparison of these 3 cohorts in this analysis, follow-up for the 2006-2014 cohort was realigned to begin in the fourth year (2006) of that cohort's original timeline, when most 2006-2014 cohort participants (>46%) were in the fourth grade. Final follow-up for these data was June 2014 and the data reported here are the most recent available at the time of this study from these cohorts.

### Exposure Assessment

Ambient air pollutant monitoring stations were established in each of the study communities and have been continuously measuring regional air pollution since the inception of the CHS. Data on concentrations of ozone, nitrogen dioxide,  $\text{PM}_{10}$ , and  $\text{PM}_{2.5}$  were routinely collected, as previously described.<sup>8,10</sup> Community-specific annual mean concentrations in the baseline year for each cohort (ie, 1993, 1996, and 2006 for the 1993-2001, 1996-2004, and 2006-2014 cohorts, respectively) were calculated based on 24-hour means for nitrogen dioxide,  $\text{PM}_{10}$ , and  $\text{PM}_{2.5}$ , and on the 10 AM to 6 PM mean for ozone due to its marked diurnal variation.

### Covariate Assessment

Data were obtained from the baseline questionnaires on children's date of birth, sex, race and ethnicity, history of asthma, participation in team sports, presence of a gas stove in the home, exposure to smoking in utero, exposure to secondhand smoke, parental education, parental history of asthma, and residential address. Determination of children's race/ethnicity was made by the parents or guardians who completed the baseline questionnaire based on a question with fixed categories. Race/ethnicity was included as a covariate due to its role as a potential confounder of the air pollution-asthma relationship. Exposure to secondhand smoke was classified based on a positive response to either of the following questions: "Does anyone living in this child's home currently smoke cigarettes, cigars or pipes on

a daily basis inside the home?” or “In the past, has anyone living in this child’s home ever smoked cigarettes on a daily basis inside the home while the child was living there?” Use of a Spanish-language questionnaire by the parent or guardian at baseline was also recorded. Genetic ancestry, including Native American ancestry, was estimated using 233 ancestral informative markers and the STRUCTURE program.<sup>11-13</sup>

Baseline residential address was used to estimate exposure to local near-roadway pollution, based on a line source dispersion model as previously described.<sup>6,9</sup> Inputs to this model included distance to roadways, vehicle counts, vehicle nitrogen oxide emission rates, wind speed and direction, and height of the mixing layer in each community. Mean temperature data were also collected during follow-up from measurements at monitoring stations. For each cohort baseline year, community-specific mean temperature was computed from monthly means in that year. Temperature data were not available for 1993, consequently 1994 levels were used instead for all 1993-2001 cohort participants. Additionally, 2006 temperature data were not available for 2 communities (Lake Gregory and San Dimas); therefore, data from 2001, the closest previous year with complete data, were used for 2006-2014 cohort participants in these 2 communities. Community-level annual mean temperatures did not vary greatly from year to year; the mean coefficient of variation from 1994 to 2001 and 2006, for those communities with data, was 4% (range, 2%-6%).

### Outcome Assessment

Incident asthma was defined as a newly reported physician-diagnosed case of asthma on an annual questionnaire during follow-up (ie, first time answered “yes” to the question “Has a doctor ever diagnosed this child as having asthma?” when the parent or guardian was asked or “Has a doctor ever said you have asthma?” when the child was asked). Because incident asthma cases were defined with these annual questionnaires, specific dates of diagnoses were unknown. We imputed the date of diagnosis using the midpoint of the interval between the date of the questionnaire on which asthma diagnosis was first reported and the date of the questionnaire prior to reporting asthma status. This date was used for calculating follow-up time for all statistical analyses. Children with missing questionnaires during follow-up continued to contribute person-time until they reported an asthma diagnosis or were lost to follow-up.

### Statistical Analysis

To assess association between changes in regional air quality and asthma incidence in children within community over the course of follow-up for these 3 cohorts, we fitted multilevel Poisson regression models to estimate asthma incidence rate ratios (IRRs) and 95% CIs associated with exposure to regional air pollution.<sup>10,14,15</sup> Models included an offset term for person-time (natural log-transformed) and a fixed effect for community. Additionally, to account for clustering effects of children by cohort and community, a random effect for cohort nested within community with

an unstructured covariance matrix was included in the model. Follow-up time was calculated as the number of days between joining the cohort (ie, baseline questionnaire date) and either imputed date of asthma diagnosis or date of last completed questionnaire (either 12th grade or earlier if lost to follow-up), whichever came first.

Regional air pollution exposures were defined as the community-level annual mean concentrations in the baseline year for each cohort (ie, 1993, 1996, and 2006). Data were not available for 1993 on PM<sub>10</sub> in 4 communities (Alpine, Lake Gregory, Riverside, and Upland) and PM<sub>2.5</sub> in any community, therefore, 1994 concentrations were used. Point estimates were scaled to the median change in community-level annual mean concentration among the 9 communities from 1993 to 2006. These models were designed to make inferences regarding within-community changes in regional air pollution and asthma incidence rates. Incidence rate differences were calculated to provide context on absolute change; these models used sampling weights for communities to make results interpretable for the entire sample. Additional details on modeling approach are reported in eMethods in the Supplement.

Potential confounders were identified a priori based on a directed acyclic graph.<sup>16</sup> These were baseline age (continuous), sex (female, male), ethnicity (Hispanic, non-Hispanic), race (Asian/Pacific Islander, black, Native American/other, white, mixed), presence of gas stove in home (yes, no), physical activity defined here as team sports participation (yes, no), temperature defined here as community-level mean temperature for cohort baseline year (continuous), and exposure to local near-roadway pollution (continuous). To avoid loss of sample size, missing indicators were included as needed for any categorical adjustment variable. Three sets of models were fitted for each pollutant: adjusted only for community (fixed effect), additionally adjusted for all potential confounders except local near-roadway pollution, and additionally adjusted for local near-roadway pollution. Adjustment for local pollutants was conducted separately in the third model because 198 children whose residential addresses could not be geocoded were missing these data, decreasing the sample size.

We assessed heterogeneity of the regional air pollution associations by comparing nested models using a partial likelihood ratio test with and without interaction terms for the following potential effect modifiers: sex, ethnicity, race, exposure to smoking in utero, secondhand smoke exposure, parental education, parental history of asthma, Native American ancestry (only among Hispanic children), and designation of high vs low air pollution community—based on whether a community was above or below corresponding median annual mean concentration in 1993. For evaluation of effect modification, Native American ancestry among Hispanic participants was categorized into 2 groups based on having less than or greater than 50% Native American ancestry.

Robustness of the main study findings were tested with the following sensitivity analyses: (1) excluding 1 community at a time, (2) excluding participants who reported wheeze

and/or 3 or more months of cough in the prior 12 months at baseline, (3) excluding data from the first year of follow-up, (4) excluding 2006-2014 cohort participants whose baseline asthma status was defined based on the year 3 rather than year 4 questionnaire, (5) reincluding participants with missing baseline asthma status, (6) imputing asthma diagnosis date to 6 months after completion date of prior questionnaire, (7) restricting to participants with longer follow-up (followed to year 5 or later, or to year 7 or later), (8) including additional potential covariates, (9) omitting the random effect for cohort nested within community and instead bootstrapping<sup>17</sup> at the community level to assess modeling assumptions, and (10) including a fixed effect for cohort to adjust for potential temporal confounding.

Two pollutant models were fitted whenever the correlations between covariates were found to be sufficiently low to avoid multicollinearity. In addition, a set of sensitivity analyses were conducted using Cox proportional hazards regression, using the same modeling approach as the main fully adjusted model but with no random effect. These models were used to evaluate (1) the inclusion of time-varying calendar year to adjust for potential temporal confounding and (2) the use of a time-varying air pollution exposure variable. No apparent violation of the underlying assumption of proportional hazards was detected based on inclusion of a time-dependent covariate for air pollution. Due to missing air pollution data in earlier years (as noted here) and no air pollution data after 2011 as well as missing PM<sub>2.5</sub> data for 1 community in 2005, air pollution for these years was imputed by extending the closest years' air pollution data (ie, 1994 for 1993, 2006 for 2005, and 2011 for 2012 and later years).

All hypotheses were tested assuming a .05 significance level and a 2-sided alternative hypothesis. *P* values were not adjusted for multiple comparisons because the tests were hypothesis driven. All analyses were conducted using SAS software version 9.4 (SAS Institute).

## Results

The characteristics of the 4140 children included in this study are described overall and by cohort in **Table 1**. Because the outcome was asthma incidence during follow-up, we excluded participants (in a hierarchical manner) who had no follow-up questionnaire (*n* = 1503), had physician-diagnosed asthma at baseline (*n* = 804), or were missing baseline asthma status (*n* = 143). For 2006-2014 cohort participants, data from the fourth year, according to the original 2006-2014 cohort timeline, were used to define baseline asthma status. If no questionnaire was completed that year, data from the prior year were used (*n* = 467). If data from neither the fourth-year nor third-year questionnaire were available, those participants were considered as having missing baseline asthma status and excluded from the analysis (*n* = 268). The final study population comprised 4140 children, including 1093, 1170, and 1877 from the 1993-2001, 1996-2004, and 2006-2014 cohorts, respectively.

Descriptive statistics on participants excluded from the analysis are given in eTable 1 in the **Supplement**. Mean person-years observed per child were similar across the cohorts: 5.7, 5.8, and 6.0 for the 1993-2001, 1996-2004, and 2006-2014 cohorts, respectively. The crude incidence rate for asthma was the highest for the 1996-2004 cohort (2.69 cases per 100 person-years) and lowest for the 2006-2014 cohort (1.80 cases per 100 person-years).

Regional air pollution concentrations generally decreased among the 9 communities over the course of the study period (**Figure 1**). The median changes in community-level annual mean concentration among the 9 communities from 1993 to 2006 were −8.9 parts per billion (ppb) (range, −21.4 to 4.8) for ozone, −4.3 ppb (range, −14.1 to −0.8) for nitrogen dioxide, −4.0 μg/m<sup>3</sup> (range, −10.9 to 4.3) for PM<sub>10</sub>, and −8.1 μg/m<sup>3</sup> (range, −15.2 to 0.7) for PM<sub>2.5</sub>. Reductions in air pollution were larger in communities with higher 1993 concentrations.

Plots of the unadjusted community-level data, along with community-specific regression lines, comparing asthma incidence rates with regional air pollution concentrations across the 3 cohorts are shown in **Figure 2**. Greater reductions in asthma incidence rates were observed in communities with larger decreases in either nitrogen dioxide or PM<sub>2.5</sub> concentrations. Results were less consistent for ozone and PM<sub>10</sub>.

Reductions in regional nitrogen dioxide and PM<sub>2.5</sub>, but not ozone or PM<sub>10</sub>, levels were statistically significantly associated with reductions in asthma incidence rate among children (**Table 2**). For community-level nitrogen dioxide, the IRR for asthma was 0.83 (95% CI, 0.74-0.92) when only adjusted for community (for a reduction of 4.3 ppb). When adjusted for additional potential confounders, including near-roadway pollution, the IRR was 0.81 (95% CI, 0.72-0.91). For community-level PM<sub>2.5</sub>, the IRR was 0.82 (95% CI, 0.69-0.98) (for a reduction of 8.1 μg/m<sup>3</sup>). In the adjusted model with near-roadway pollution, the IRR was 0.82 (95% CI, 0.67-0.99). Reduced risks associated with decreasing ozone and PM<sub>10</sub> did not reach statistical significance. Results for incidence rate differences showed absolute decreases of 0.83, 1.53, 0.78, and 0.46 cases per 100 person-years for nitrogen dioxide, PM<sub>2.5</sub>, ozone, and PM<sub>10</sub>, respectively.

Associations for nitrogen dioxide and PM<sub>2.5</sub> did not substantially vary by sex, ethnicity, race, exposure to smoking in utero, exposure to secondhand smoke, parental education, parental history of asthma, Native American ancestry (among Hispanic children), or high or low 1993 air pollution level (eTable 2 in the **Supplement**).

Sensitivity analyses demonstrated results for nitrogen dioxide were robust to a variety of analytical decisions as reported in eTables 3 and 4 in the **Supplement**. Results for analyses excluding participants who reported wheeze and/or 3 or more months of cough in the prior 12 months at baseline (IRR, 0.81 [95% CI, 0.71-0.92]), targeting both cough- and wheeze-variant potential asthmatic cases, as well as in analyses excluding the first year of follow-up (IRR, 0.77 [95% CI, 0.68-0.88]), targeting prevalent cases, remained similar to



Table 1. Distribution of Selected Participant Characteristics From the Children's Health Study, 1993-2014

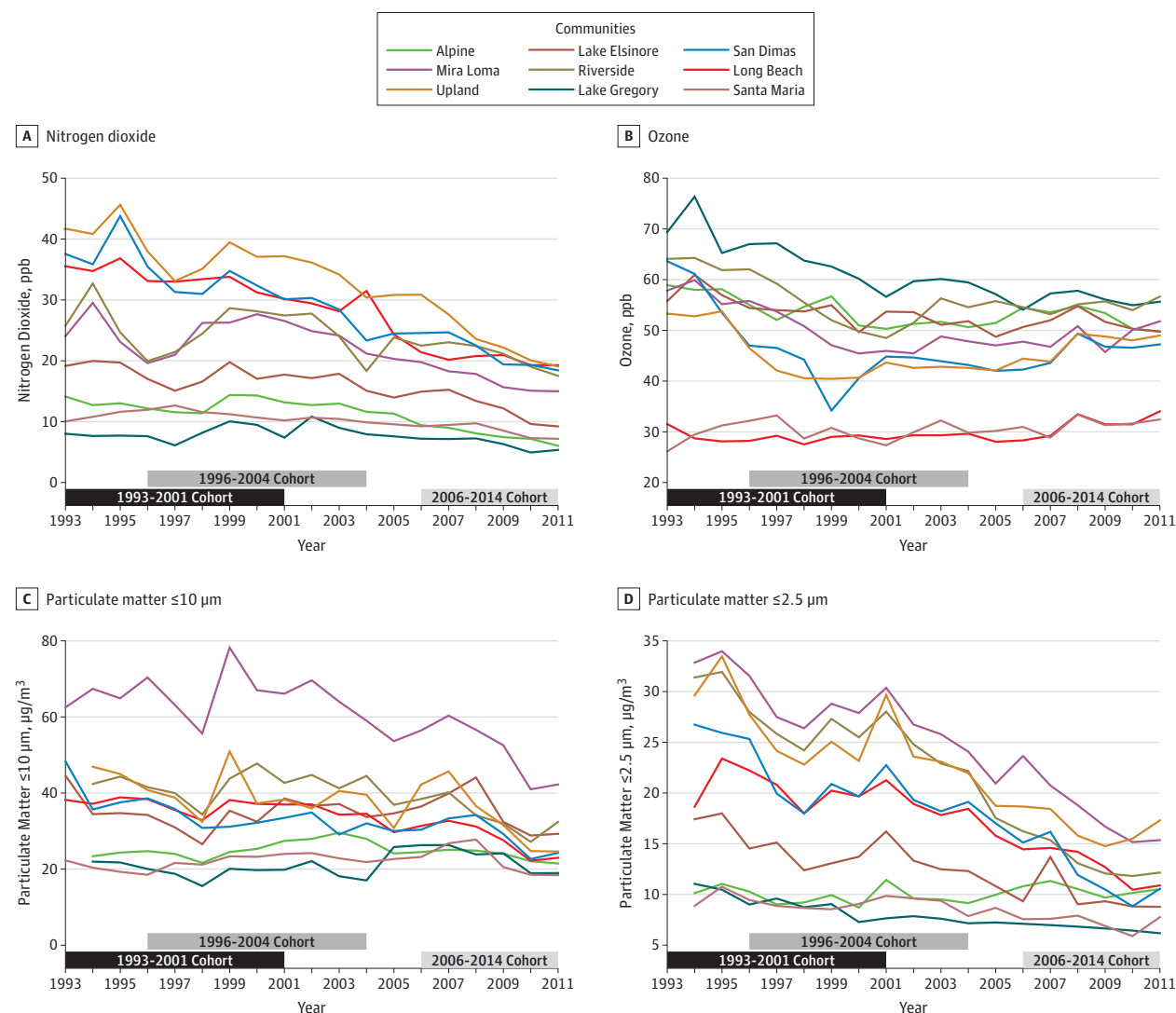
Characteristic	All Participants, No./Total No. (%)	Cohort Follow-up Period, No./Total No. (%)		
		1993-2001	1996-2004	2006-2014
Participants	4140	1093	1170	1877
Person-years of follow-up	24 254	6201	6842	11 211
Follow-up questionnaires per participant, mean (SD)	5.5 (2.2)	5.9 (2.3)	6.7 (2.5)	4.4 (1.2)
Incident asthma cases	525	139	184	202
Age at baseline, mean (SD), y	9.5 (0.6)	9.9 (0.5)	9.5 (0.4)	9.3 (0.7)
Sex				
Male	1961/4140 (47.4)	524/1093 (47.9)	564/1170 (48.2)	873/1877 (46.5)
Female	2179/4140 (52.6)	569/1093 (52.1)	606/1170 (51.8)	1004/1877 (53.5)
Ethnicity				
Hispanic	1686/3996 (42.2)	307/1083 (28.3)	413/1163 (35.5)	966/1750 (55.2)
Non-Hispanic	2310/3996 (57.8)	776/1083 (71.7)	750/1163 (64.5)	784/1750 (44.8)
Missing	144/4140 (3.5)	10/1093 (0.9)	7/1170 (0.6)	127/1877 (6.8)
Race				
Asian/Pacific Islander	178/3878 (4.6)	60/1072 (5.6)	56/1157 (4.8)	62/1649 (3.8)
Black	145/3878 (3.7)	50/1072 (4.7)	54/1157 (4.7)	41/1649 (2.5)
Native American/other	890/3878 (22.9)	182/1072 (17)	249/1157 (21.5)	459/1649 (27.8)
White	2273/3878 (58.6)	704/1072 (65.7)	692/1157 (59.8)	877/1649 (53.2)
Mixed	392/3878 (10.1)	76/1072 (7.1)	106/1157 (9.2)	210/1649 (12.7)
Missing	262/4140 (6.3)	21/1093 (1.9)	13/1170 (1.1)	228/1877 (12.2)
Parental education				
High school graduate or below	1424/3900 (36.5)	379/1068 (35.5)	385/1113 (34.6)	660/1719 (38.4)
Some college or above	2476/3900 (63.5)	689/1068 (64.5)	728/1113 (65.4)	1059/1719 (61.6)
Missing	240/4140 (5.8)	25/1093 (2.3)	57/1170 (4.9)	158/1877 (8.4)
Gas stove in home				
Yes	3153/3937 (80.1)	824/1067 (77.2)	860/1147 (75)	1469/1723 (85.3)
Play team sport				
Yes	2104/4042 (52.1)	542/1074 (50.5)	597/1136 (52.6)	965/1832 (52.7)
In utero exposure to smoking				
Yes	484/3939 (12.3)	187/1063 (17.6)	177/1143 (15.5)	120/1733 (6.9)
Secondhand smoke exposure <sup>a</sup>				
Yes	874/3880 (22.5)	302/1059 (28.5)	308/1116 (27.6)	264/1705 (15.5)
Parental history of asthma				
Yes	687/3922 (17.5)	175/1027 (17)	172/1084 (15.9)	340/1811 (18.8)
Spanish questionnaire				
Yes	654/4140 (15.8)	79/1093 (7.2)	158/1170 (13.5)	417/1877 (22.2)
Residential traffic-related pollution, mean (SD), parts per billion	19.6 (22.1)	27.5 (27.8)	20.5 (23.3)	14.9 (15.8)

<sup>a</sup> Exposure to secondhand smoke was classified based on a positive response to either of the following questions: "Does anyone living in this child's home currently smoke cigarettes, cigars or pipes on a daily basis inside the home?"

or "In the past, has anyone living in this child's home ever smoked cigarettes on a daily basis inside the home while the child was living there?"

those of the main analysis. Two pollutant models were fitted for both nitrogen dioxide and PM<sub>2.5</sub> with PM<sub>10</sub> (Pearson correlation coefficients were 0.50 and 0.55, respectively) and for nitrogen dioxide with ozone (correlation = 0.54). Results for nitrogen dioxide remained robust. Results for PM<sub>2.5</sub> were not statistically significant. Two pollutant models were not fitted for nitrogen dioxide with PM<sub>2.5</sub> (correlation = 0.60), nor PM<sub>2.5</sub> with ozone (correlation = 0.62). Results for nitrogen dioxide and PM<sub>2.5</sub> based on models with time-varying exposure remained statistically significant and point estimates

were similar, although attenuated, compared with the results for the main baseline exposure models (eTable 4 in the [Supplement](#)). Findings for nitrogen dioxide based on models adjusted for calendar time remained robust, while those for PM<sub>2.5</sub> did not and results for this model were null (eTable 4 in the [Supplement](#)). Overall in sensitivity analyses, associations for nitrogen dioxide remained statistically significant and generally similar in magnitude to those in the primary analysis. These analyses revealed, however, that the findings for PM<sub>2.5</sub> were not consistently robust.

**Figure 1. Annual Mean Air Pollutant Concentration During the Follow-up Period in 9 Communities of the Southern California Children's Health Study, 1993-2011**

Black, dark gray, and light gray horizontal bars represent follow-up periods for the 1993-2001, 1996-2004, and 2006-2014 cohorts, respectively. Follow-up for the 2006-2014 cohort is truncated on the graph at 2011, the last year with air pollution data. ppb indicates parts per billion.

## Discussion

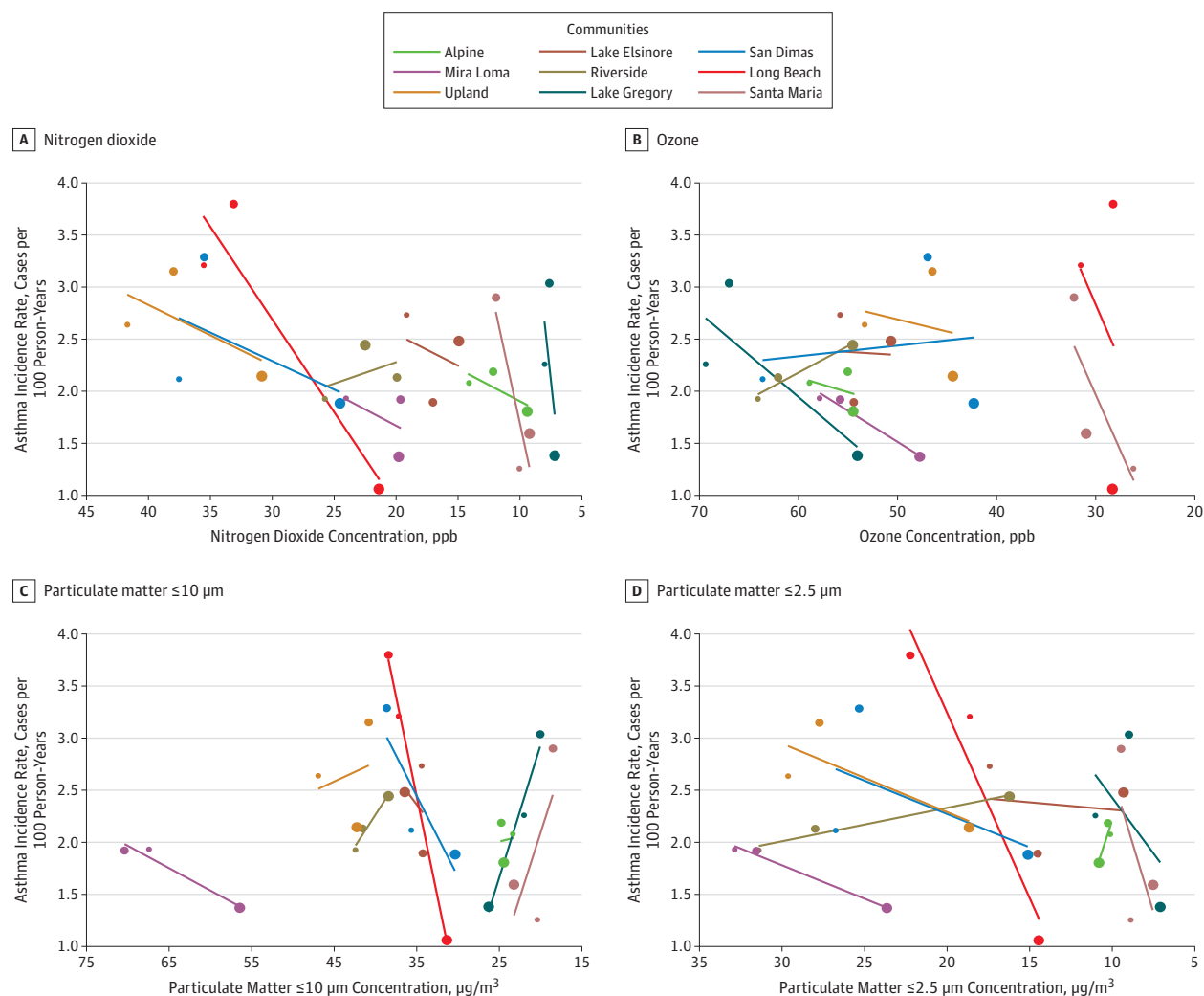
Reductions in levels of regional nitrogen dioxide from 1993 to 2014 were statistically significantly associated with improvements in asthma incidence rates in Southern Californian children. These results were independent of changes in exposure to near-roadway pollution. Findings from this study also suggested a potential association with regional  $\text{PM}_{2.5}$ ; these results, however, were less robust to sensitivity analyses. Nitrogen dioxide results remained robust in sensitivity analyses. Associations did not appear to be substantially influenced by a single community. The inclusion of prevalent asthma cases in the study population could have resulted in exacerbation in addition to incidence being captured in the

definition of the outcome. Sensitivity analyses excluding potential prevalent cases, including cough- and wheeze-variant potential asthmatic cases, did not show marked differences compared with main results, suggesting this source of bias was not of major concern. Findings for  $\text{PM}_{2.5}$  should be interpreted with caution because these appeared more sensitive to analytical choices, as demonstrated in sensitivity analyses. Point estimates for  $\text{PM}_{2.5}$  from sensitivity analyses were generally similar to those of the main models, but 95% CIs were wider, and several included the null.

This study provides evidence of a robust association between children's exposure to community-level nitrogen dioxide and development of asthma in childhood. Previous review of the development of childhood asthma and environmental exposures concluded that although several



**Figure 2. Asthma Incidence Rates and Air Pollutant Concentrations in 9 Communities During the 1993-2001, 1996-2004, and 2006-2014 Cohorts of the Southern California Children's Health Study, 1993-2014**



Symbol colors indicate community and the size—small, medium, and large—indicates the data are from the 1993-2001, 1996-2004, and 2006-2014 cohorts, respectively. Simple linear regression models based on asthma

incidence and air pollution concentration used to generate regression lines separately for each community. ppb indicates parts per billion.

ambient pollutants were associated with increased asthma, none were consistently identified.<sup>18</sup> Several meta-analysis studies, however, have concluded that nitrogen dioxide exposure was associated with asthma incidence among children.<sup>5,19-22</sup> A 2017 systematic review and meta-analysis reported an overall risk estimate of 1.05 (95% CI, 1.02-1.07) per 4  $\mu\text{g}/\text{m}^3$  (2.1 ppb) nitrogen dioxide. The IRR from the current study scaled to a 2.1-ppb change in nitrogen dioxide was 1.11 (95% CI, 1.05-1.18; fully adjusted model including near-roadway pollution).

It is unclear whether nitrogen dioxide is the causal agent or rather is serving as a marker for the traffic-related air pollution mixture. One CHS study, using data from the 2006-2014 cohort only, reported an association between exposure to nitrogen dioxide and asthma incidence.<sup>6,23</sup> Ambient nitrogen dioxide measured at community central sites was associ-

ated with increased asthma risk (hazard ratio, 2.18 [95% CI, 1.18-4.01]), although this association was attenuated after accounting for traffic-related pollutions (hazard ratio, 1.37 [95% CI, 0.69-2.71]). Results for nitrogen dioxide in the present study did not change when adjusted for near-roadway pollution. While positive associations were observed in both studies, the current study benefitted from a within-community, across-time design, which provided increased statistical power and allowed for the control of community-level unmeasured confounders. These results suggest that nitrogen dioxide is capturing effects of air pollution exposure beyond local near-roadway pollution.  $\text{PM}_{2.5}$  has also been found to be associated with asthma incidence,<sup>5,20,24</sup> and while  $\text{PM}_{2.5}$  was positively associated with incident asthma, the results were not consistently robust to sensitivity analyses.  $\text{PM}_{2.5}$  mass in the current study comprises traffic-related

**Table 2. Incidence Rate Ratios (IRRs) and Incidence Rate Differences (IRDs) per 100 Person-Years of Asthma Incidence Associated With Reduction in Regional Air Pollution, 1993-2014<sup>a</sup>**

Pollutant	Community-Only Adjusted Model (n = 4140) <sup>b</sup>				Fully Adjusted Model (n = 4140) <sup>c</sup>				Fully Adjusted Model With Near-Roadway Pollution (n = 3942) <sup>d</sup>			
	IRR (95% CI)	P Value	IRD (95% CI) <sup>e</sup>	P Value	IRR (95% CI)	P Value	IRD (95% CI) <sup>e</sup>	P Value	IRR (95% CI)	P Value	IRD (95% CI) <sup>e</sup>	P Value
Ozone	0.86 (0.72 to 1.02)	.08	-0.77 (-0.86 to -0.68)	<.001	0.85 (0.71 to 1.02)	.08	-0.78 (-1.44 to -0.12)	.02	0.86 (0.71 to 1.04)	.11	-0.76 (-1.41 to -0.11)	.02
Nitrogen dioxide	0.83 (0.74 to 0.92)	.001	-0.88 (-0.95 to -0.80)	<.001	0.80 (0.71 to 0.90)	<.001	-0.83 (-1.54 to -0.13)	.02	0.81 (0.72 to 0.91)	<.001	-0.82 (-1.52 to -0.12)	.02
PM <sub>10</sub>	0.92 (0.81 to 1.05)	.22	-0.47 (-0.67 to -0.28)	<.001	0.93 (0.82 to 1.07)	.32	-0.46 (-0.96 to 0.04)	.08	0.92 (0.81 to 1.04)	.17	-0.48 (-0.90 to -0.06)	.03
PM <sub>2.5</sub>	0.82 (0.69 to 0.98)	.03	-1.47 (-2.11 to -0.83)	<.001	0.81 (0.67 to 0.98)	.03	-1.53 (-2.95 to -0.11)	.04	0.82 (0.67 to 0.99)	.04	-1.48 (-2.88 to -0.07)	.04

Abbreviation: PM, particulate matter.

<sup>a</sup> IRR and IRD are per -8.9 ppb for ozone, -4.3 ppb for nitrogen dioxide, -4.0 µg/m<sup>3</sup> for PM<sub>10</sub>, and -8.1 µg/m<sup>3</sup> for PM<sub>2.5</sub> (median changes in air pollution concentrations observed among the 9 communities between 1993 and 2006).<sup>b</sup> Community-only adjusted model adjusted for community as a fixed effect.<sup>c</sup> Fully adjusted model additionally adjusted for age at baseline, sex, ethnicity,

race, gas stove in home, participation in sports, and community-level mean temperature for baseline year.

<sup>d</sup> Fully adjusted model with traffic additionally adjusted for local near-roadway pollution.<sup>e</sup> Models for IRD incorporated weights for communities, based on sample size contribution, to make results interpretable for the entire sample.

PM<sub>2.5</sub> and that from other sources, such as dust and ocean spray. Incorporating data on PM<sub>2.5</sub> mass sources may help specify the relation between PM<sub>2.5</sub> and incident asthma. From 1990 to 2012, a period corresponding to the period of study in the present analyses, levels of diesel PM have decreased by 68% in California<sup>25</sup> and this downward trend was observed in Los Angeles.<sup>26</sup> Furthermore, measures of diesel PM, such as elemental carbon and black carbon, are correlated with nitrogen dioxide particularly near major roadways.<sup>27,28</sup> Studies have reported positive associations between measures of diesel PM and incident childhood asthma,<sup>5</sup> but a lack of elemental carbon or black carbon data across all 3 cohorts precluded the evaluation of this relation in the present study.

There is evidence for the plausibility of a biological mechanism specifically for nitrogen dioxide. Studies indicate that at concentrations typical of high-income countries, exposure to nitrogen dioxide induces airway inflammation,<sup>29,30</sup> airway hyperresponsiveness,<sup>31</sup> and oxidative stress.<sup>32</sup> In healthy adult humans, controlled exposure to nitrogen dioxide produced enhanced pulmonary neutrophilic inflammation and the promotion of a Th2 phenotype.<sup>33</sup> The UK Committee on the Medical Effects of Air Pollutants identified 4 mechanisms for how air pollution might contribute to the pathogenesis of asthma: (1) oxidative stress and damage, including the depletion of antioxidants; (2) airway wall remodeling, leading to structural changes in the airways; (3) inflammatory pathways and immunological effects, including effects on the expression of inflammatory mediators; and (4) enhancement of respiratory sensitization to allergens.<sup>34</sup>

A benefit of the modeling framework used here was that communities were compared with themselves at 3 points in time, thus reducing the potential for confounding by spatial factors, under the assumption that contextual variables in the community did not change. A concern remains, however, for temporal confounding. Controlling for factors with trends across the 3 study cohorts (eg, health insurance, tobacco exposure, ethnicity) did not change results. Adjust-

ing for cohort or time-varying calendar year showed the nitrogen dioxide findings to be robust while the PM<sub>2.5</sub> findings were not. Furthermore, secular trends in asthma rates have been increasing over the study period,<sup>35</sup> which would bias the results toward the null. In addition, reductions in asthma incidence rates were larger in communities with greater reductions in nitrogen dioxide concentrations, further indicating results were not likely to be simply an artifact of secular trends in asthma diagnosis or other potential temporal confounders.

### Limitations

The study has several limitations. First, baseline, rather than time-varying, community-level annual average pollutant concentration was used as the exposure in the main models. Although a model with time-varying exposure was implemented using Cox proportional hazards regression in sensitivity analyses, this was not used as the main model because it was not possible to obtain estimates with the multilevel modeling approach (eg, when a fixed effect for community and a random effect of cohort nested within community was included). The sensitivity analyses using a Cox model with no random effect and time-varying air pollutant exposure generated results that were similar, although attenuated, compared with the Poisson models using baseline air pollutant exposure. These time-varying exposure data had more missing exposure data that necessitated imputation and, as such, may have resulted in greater exposure misclassification compared with the use of baseline exposure data. Additionally, these models may not capture the best time window of exposure because only a 1-year lag could be used given that no exposure data were available prior to the start of the study.

Second, the modeling framework, which controls for spatial confounding based on a fixed effect for community, remained susceptible to temporal confounding. This source of confounding was evaluated in sensitivity analyses, in which the findings for nitrogen dioxide were robust.

Third, the definition of asthma incidence depended on a questionnaire-based assessment of physician-diagnosed asthma, rather than a clinical evaluation of asthma (eg, methacholine challenge test). Studies examining the validity of questionnaire-based asthma diagnosis in children, using questions similar to those used in the current study, have reported a specificity of 96% compared with health claims as the reference standard<sup>36</sup> and a specificity of 87% compared with a clinical assessment as the standard.<sup>37</sup> Fourth, a lack of data on measures of diesel PM (eg, elemental carbon or black carbon) and PM<sub>2.5</sub> mass sources precluded the investigation

of temporal trends in concentrations of these air pollutants and the incidence of childhood asthma.

## Conclusions

Among children in Southern California, decreases in ambient nitrogen dioxide and PM<sub>2.5</sub> between 1993 and 2014 were significantly associated with lower asthma incidence. There were no statistically significant associations for ozone or PM<sub>10</sub>.

## ARTICLE INFORMATION

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**Author Contributions:** Drs Garcia and Gilliland had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

**Concept and design:** Garcia, Berhane, McConnell, Urman, Gilliland.

**Acquisition, analysis, or interpretation of data:** Garcia, Berhane, Islam, Urman, Chen, Gilliland.

**Drafting of the manuscript:** Garcia, Berhane, Islam. **Critical revision of the manuscript for important intellectual content:** All authors.

**Statistical analysis:** Garcia, Berhane, Islam, Urman, Chen.

**Obtained funding:** Gilliland.

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# AMERICAN THORACIC SOCIETY DOCUMENTS

## Outdoor Air Pollution and New-Onset Airway Disease An Official American Thoracic Society Workshop Report

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### Abstract

Although it is well accepted that air pollution exposure exacerbates preexisting airway disease, it has not been firmly established that long-term pollution exposure increases the risk of new-onset asthma or chronic obstruction pulmonary disease (COPD). This Workshop brought together experts on mechanistic, epidemiological, and clinical aspects of airway disease to review current knowledge regarding whether air pollution is a causal factor in the development of asthma and/or COPD. Speakers presented recent evidence in their respective areas of expertise related to air pollution and new airway disease incidence, followed by interactive discussions. A writing committee summarized their collective findings. The Epidemiology Group found that long-term exposure to air pollution, especially metrics of traffic-related air pollution such as nitrogen dioxide and black carbon, is associated with onset of

childhood asthma. However, the evidence for a causal role in adult-onset asthma or COPD remains insufficient. The Mechanistic Group concluded that air pollution exposure can cause airway remodeling, which can lead to asthma or COPD, as well as asthma-like phenotypes that worsen with long-term exposure to air pollution, especially fine particulate matter and ozone. The Clinical Group concluded that air pollution is a plausible contributor to the onset of both asthma and COPD. Available evidence indicates that long-term exposure to air pollution is a cause of childhood asthma, but the evidence for a similar determination for adult asthma or COPD remains insufficient. Further research is needed to elucidate the exact biological mechanism underlying incident childhood asthma, and the specific air pollutant that causes it.

**Keywords:** air pollution; asthma; COPD; new-onset airway disease

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## Overview

This workshop was convened to evaluate the evidence regarding outdoor air pollution as a causal factor in the development of new-onset asthma and/or chronic obstructive pulmonary disease (COPD). The available evidence on epidemiological associations, biological mechanisms, and clinical considerations was evaluated. Workshop participants presented the current state of the science in their respective fields, based on their expertise and review of the latest research available. Key conclusions and recommendations included the following:

- The weight of the evidence is consistent with a causal relationship between new onset of childhood asthma and long-term exposure to outdoor air pollution, especially metrics of traffic-related air pollution (TRAP), such as nitrogen dioxide (NO<sub>2</sub>) and black carbon (BC).
- It is unclear whether direct effects of NO<sub>2</sub> (the best-studied TRAP component in epidemiologic studies) or other components of TRAP, such as fuel combustion particles (implicated in toxicologic animal studies), explain the causal link with asthma.
- Further studies are needed to determine whether the relationships found in TRAP studies can be generalized to air pollution from other combustion sources, and to assess the impact of air pollution on the development of adult-onset asthma and/or COPD.
- The reduced incidence of new onset of childhood asthma should be

included in future assessments of the health and monetary benefits of lessening exposures to air pollution, especially TRAP.

## Introduction

Acute exacerbation of existing respiratory diseases by air pollution is well established and is commonly factored into the decision-making process of policymakers. For example, short-term outdoor air pollution exposures, including fine particulate matter (particulate matter less than or equal to 2.5  $\mu\text{m}$  in aerodynamic diameter [PM<sub>2.5</sub>]) (1), ozone (O<sub>3</sub>) (2), NO<sub>2</sub> (3), and sulfur dioxide (4) have been accepted by the U.S. Environmental Protection Agency as causally related to acute adverse respiratory health effects. The effects of air pollution on asthma include acute associations with increased symptoms (5), rescue medication use (6), school absences (7, 8), emergency department visits (9, 10), hospitalizations (11, 12), asthma lung function deficits (13–15), and airway hyperresponsiveness (16). Similarly, documented adverse COPD health associations with short-term PM<sub>2.5</sub> exposures include reduced pulmonary function (17) and increased emergency room visits (18), hospital admissions (19), and mortality (20).

Compared with acute exposures and health effects, it has been more challenging to study and evaluate the effects of long-term exposure on

incident disease; thus, less evidence has been available in the published literature. However, there is growing evidence that long-term outdoor air pollution exposures may also cause new onset of airway disease. This Workshop was convened to evaluate the evidence of outdoor air pollution as a causal factor in the development of new-onset asthma and/or COPD.

## Methods

At the annual American Thoracic Society (ATS) International Conference in May 2018, a cross-disciplinary group met to evaluate the evidence regarding the potential role of air pollution in the onset of new airway disease. The group included researchers experienced in the mechanistic aspects of airway disease development, air pollution epidemiologists, and clinicians with expertise in airway disease pathology/diagnosis. Participants presented the current state of the science in their respective fields, based on their expertise and a review of latest research available on their specific topics. This is a consensus document, rather than a formal systematic examination of all the evidence. Consensus was reached by majority vote. A writing committee summarized the Workshop findings, which all participants could review for an accurate reflection of the proceedings. Potential conflicts of interest were handled in accordance with the policies and procedures of the ATS.

We focused on the development of new-onset asthma or COPD related to outdoor air pollution exposure by addressing several key questions:

- Does the available epidemiologic evidence concerning long-term air pollution exposure support an association with new-onset asthma or COPD?
- Are there biological mechanisms by which air pollution could plausibly cause new asthma or COPD?
- Are the health effects of air pollution identified through epidemiologic and mechanistic studies consistent with the diagnosis of new asthma or COPD in a clinical setting?
- Is there sufficient overall evidence to conclude that long-term exposure to air pollution contributes to the induction of new asthma and/or COPD?

In this Workshop report, we first summarize the epidemiological associations found to date, and then assess whether these associations are biologically and/or clinically plausible.

## Epidemiological Evidence

### Air Pollution and New-Onset Asthma

Epidemiologic evidence linking exposure to air pollution with the development of new-onset asthma has grown in recent years. Many studies have focused on surrogate metrics of TRAP, as well as individual ambient air pollutants. Commonly studied TRAP components include nitrogen oxides, NO<sub>2</sub>, BC, PM<sub>2.5</sub>, and PM less than or equal to 10 μm in aerodynamic diameter (PM<sub>10</sub>).

### Nitrogen Oxides, BC, PM<sub>2.5</sub>, and Other Traffic-related Pollution

**TRAP studies in children.** TRAP exposures were previously evaluated as a cause of childhood or adult-onset asthma in Health Effects Institute (HEI) Special Report 17 (21). This 2010 publication concluded that living near busy roads is a risk factor for onset of childhood asthma, but the data were insufficient to conclude causality. Several studies on the topic have been published since the release of that report. For example, the Southern California Children's Health Study (CHS) found an increased risk of new-onset childhood asthma from TRAP at home residences (22). Khreis and colleagues subsequently synthesized 41 studies that focused on children's TRAP exposures as a potential cause for asthma development (23), and found associations with TRAP metrics, especially NO<sub>2</sub>. (Figure 1). A 2017

meta-analysis of 18 studies of prenatal air pollution exposures and childhood asthma similarly found associations for NO<sub>2</sub> and PM<sub>10</sub> (24). Findings were null for O<sub>3</sub> and PM<sub>2.5</sub> mass (perhaps indicating that effects varied by the PM<sub>2.5</sub> constituent or source). Other primary studies indicated that TRAP, including prenatal exposure, contributes to childhood asthma development (25–33).

In addition, in the United States, Latino and black populations disproportionately live in neighborhoods with higher air pollution levels (34). Puerto Ricans and black individuals have a higher prevalence of asthma than white individuals (35). The largest study of air pollution and incident childhood asthma in U.S. minorities found that early-life NO<sub>2</sub> exposure was associated with childhood asthma in Latinos and African Americans (36). Since this Workshop was convened, a multilevel longitudinal study drawn from three waves of the CHS over a decade of air pollution decline found that decreases in ambient NO<sub>2</sub> and PM<sub>2.5</sub> between 1993 and 2014 were significantly associated with lower asthma incidence (37). This study is consistent with an inference of causality in the association between air pollution and asthma incidence, as an intervention to reduce exposure was followed by a reduction in disease incidence.

**TRAP studies in adults.** A review and meta-analysis of cohort studies found a positive association between NO<sub>2</sub> exposure and asthma incidence in adults, but was based on only three studies (38). Another review found that the evidence was insufficient to support a causal role for ambient air pollution, but was qualitatively consistent with a role for TRAP in the development of adult-onset asthma (39). A Canadian study determined that living near a major road was associated with increased odds of new-onset asthma (40). In the U.S. Sister Study (a large cohort of U.S. women), incident asthma was positively associated with PM<sub>2.5</sub> and NO<sub>2</sub>, and both pollutants were significantly positively associated with incident wheeze, the cardinal symptom of asthma (41). The ESCAPE (European Study of Cohorts for Air Pollution Effects) study, a meta-analysis involving six European cohorts, reported positive associations between TRAP and adult-onset asthma (42), with several approaching statistical significance, including NO<sub>2</sub>, nitrogen oxides, and traffic intensity on the nearest road. Two meta-analyses of

adult-onset asthma reported positive associations with NO<sub>2</sub> (13, 23), but only one reached statistical significance (43). Since then, there have been four studies in adult populations (40, 44, 45). The largest of these adult studies found a significant hazard ratio for NO<sub>2</sub> (45).

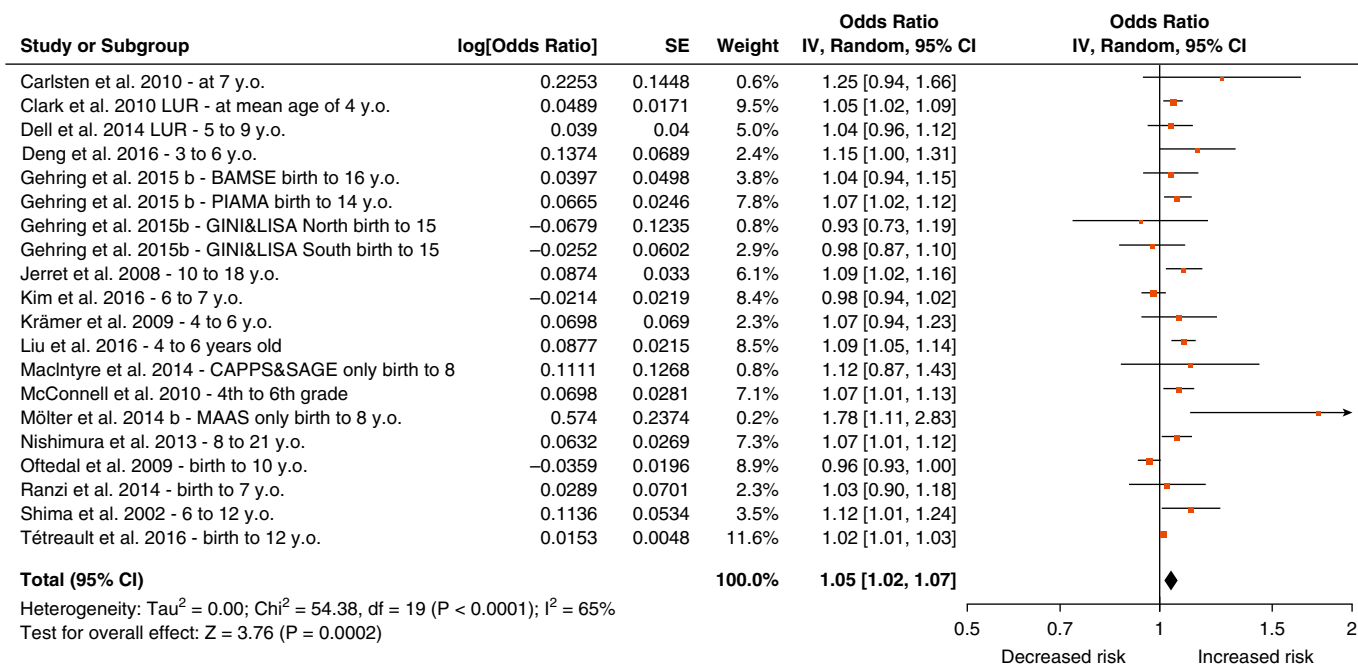
Overall, studies of new-onset asthma and TRAP pollutants indicate the most consistent positive relationship with NO<sub>2</sub> exposure among children, but it remains unclear whether NO<sub>2</sub> itself is the causal agent, simply has less measurement error than other TRAP components, and/or is simply a proxy for the combustion component of TRAP (e.g., fossil fuel combustion PM).

### Ozone

There is extensive evidence that O<sub>3</sub> exposure acutely exacerbates asthma, but less support for the hypothesis that long-term exposure causes incident asthma. In a study of long-term exposures, O<sub>3</sub> was associated with new-onset asthma in adult male Seventh-day Adventists (46). A study in Taiwan indicated an association between O<sub>3</sub> exposure and risk of childhood asthma (47). Also, the California CHS found that in communities with high O<sub>3</sub> concentrations, the relative risk of developing asthma was increased in children who played three or more sports as compared with children who played no sports (48). However, prenatal exposure to O<sub>3</sub> has not been associated with subsequent childhood asthma (24). Still, the ambient quenching of O<sub>3</sub> by traffic-emitted nitric oxide (49) can cause a negative correlation between O<sub>3</sub> and NO<sub>2</sub>, potentially confounding the relationships between O<sub>3</sub> and respiratory outcomes in epidemiologic models.

### Potential PM Composition Influences

Exposure to PM air pollution has been associated with chronic airway diseases, including asthma (23, 30, 41). In a study of TRAP and new-onset asthma in a high-risk cohort, Carlsten and colleagues found that, among the TRAP pollutants considered, PM<sub>2.5</sub> was the air pollutant most strongly associated with new-onset childhood asthma (50). PM, however, varies greatly in chemical composition as a function of its size and source (1). Also, traffic-related PM (indicated by BC) was found to be significantly related to an increased risk of new-onset asthma in children (23) (Figure 2). Although the investigators of the PIAMA (Prevention and



**Figure 1.** Meta-analysis of studies of nitrogen dioxide and new-onset asthma in children. Reprinted by permission from Reference 23. CI = confidence interval;  $I^2$  = percentage of variation across studies due to heterogeneity; IV = instrumental variable; SE = standard error.

Incidence of Asthma and Mite Allergy) birth cohort identified traffic as the major contributing factor in their study area,  $PM_{2.5}$  sulfur, a marker for fossil fuel combustion, generally had the largest relative risk for incident asthma among several PM constituents examined (51), so the  $PM_{2.5}$  and BC associations reported may not be specific to TRAP only.

### Conclusions Regarding the Epidemiology of New-Onset Asthma

- Overall, long-term exposure to air pollution, especially as represented by

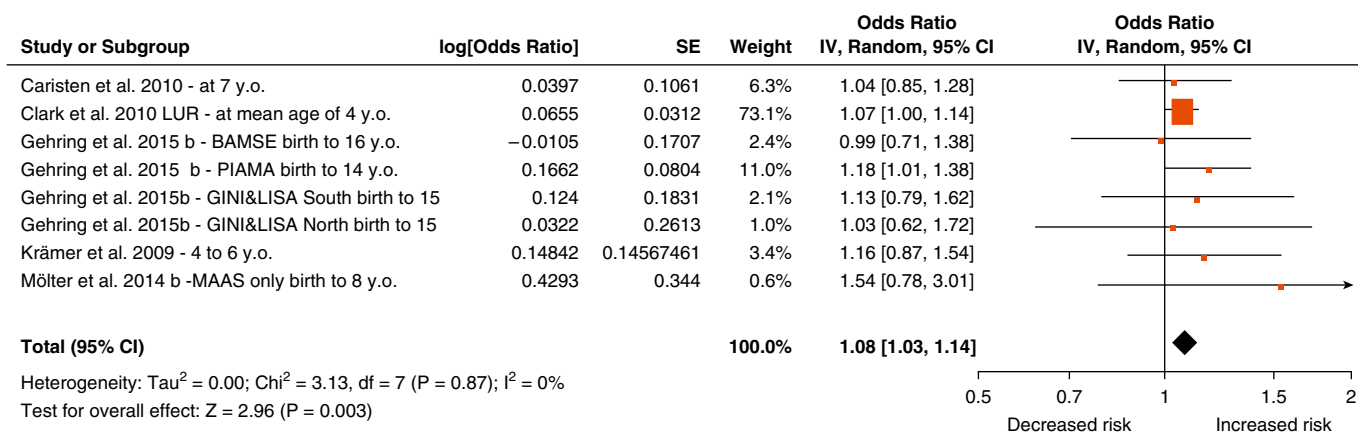
common metrics of TRAP exposure, is associated with onset of childhood asthma.

- The strongest epidemiologic evidence for a causal relationship with new-onset childhood asthma comes from studies that used  $NO_2$  as the TRAP metric.
- Evidence suggests that TRAP plays a role in adult-onset asthma, but it is not yet compelling.
- Greater effects likely occur in subgroups (e.g., genetically susceptible individuals and minorities).

- $NO_2$  may be acting as a marker for PM secondary to combustion of fossil fuels, other reactive gases, or other nontailpipe TRAP pollutants.

### Air Pollution and New-Onset COPD

The potential role of air pollution in COPD onset was addressed in a 2010 ATS review (52) and an HEI report (21). The ATS review concluded that there was limited/suggestive evidence, and the HEI report concluded that there was insufficient evidence of a causal association between TRAP and COPD.



**Figure 2.** Meta-analysis of black carbon soot associations with new-onset asthma. Reprinted by permission from Reference 23. CI = confidence interval;  $I^2$  = percentage of variation across studies due to heterogeneity; IV = instrumental variable; SE = standard error.



Since the publication of those reviews, a few new COPD studies have emerged. Some included new onset of chronic bronchitis symptoms and/or emphysema as COPD surrogates, and found positive (but not statistically significant) associations with air pollution (53, 54). An analysis of the European Community Respiratory Health Cohort yielded significant associations between NO<sub>2</sub> and chronic bronchitis (55). The ESCAPE study found significant associations between COPD incidence and TRAP among females (56). Most studies that assessed COPD using spirometry revealed positive associations with NO<sub>2</sub> and/or PM<sub>2.5</sub> (45, 57–60). One study investigated the development of asthma and COPD overlap syndrome (ACOS) in patients with asthma, and found a significant association between long-term PM<sub>2.5</sub> exposure and ACOS development (60). Associations found between indoor exposures to biomass pollution and increased risk of COPD, albeit at much higher than usual ambient PM<sub>2.5</sub> levels, are consistent with an association between fine PM and the development of COPD (61).

Only a limited number of studies have examined the associations between O<sub>3</sub> and COPD. A study of adults ≥40 years of age found no association between COPD development and O<sub>3</sub> (58). A survey-based study of 6,040 adults found that O<sub>3</sub> exposure was associated with the development of ACOS in adults with asthma (60), but the association was nonsignificant after adjustment for PM<sub>2.5</sub>. However, because the large hospital databases or survey cohorts used in these studies lacked important individual risk factors, the results should be interpreted with caution. Overall, there is little firm evidence that O<sub>3</sub> causes new-onset COPD.

### Conclusions Regarding the Epidemiology of New-Onset COPD

- Studies indicated that exposure to traffic has adverse effects on COPD, but were not conclusive. The strongest evidence comes from meta-analyses of COPD, and few longitudinal studies have been conducted.
- Overall, the available epidemiological evidence regarding an association between air pollution and new-onset COPD remains insufficient to indicate a causal relationship.

### Mechanistic Evidence

A key factor that should be considered in evaluating the causality of the above-discussed epidemiological associations is their biological plausibility (62, 63). The mechanistic literature regarding air pollution and asthma includes animal models and exposure paradigms, but only a few such studies have focused on COPD. Several mechanisms can plausibly explain how air pollution can induce new-onset airway disease with implications for both asthma and COPD, including 1) structural remodeling of lung components, predisposing to respiratory disease; 2) induced immune changes, promoting allergic sensitization or prolonged inflammation; 3) changes in innate cells (e.g., group 2 innate lymphoid cells [ILC2]) in nonatopic asthma; and 4) other modifiers of exposure, including genetics and stress.

Repeated inflammation and long-term air pollution exposure leads to airway remodeling. Early-life changes, including airway remodeling and oxidant stress, can be related to the onset of COPD or asthma, which may further progress to COPD (64). The conducting airways are an epithelial mesenchymal trophic unit (65) composed of airway epithelium, extracellular matrix, and fibroblasts, which interacts with nerves, smooth muscle, and immune cells. These elements grow interactively in a progressive fashion that may be disturbed by air pollution exposure. Alveolar growth and septation occur through young adulthood (66, 67), providing a substantial window of opportunity for air pollution-induced disruption.

### Asthma Development

Animal studies have demonstrated that early-life air pollution exposures alter conducting airway and alveolar growth (68–70). Air pollutants impact alveolar growth by pre- and postnatal exposures in mice (71), as well as by postnatal exposures in primates (72). Evidence strongly suggests that the cellular mechanism underlying this altered growth involves decreased cellular proliferation (73). In nonhuman primates, which have a postnatal maturation pattern and lung anatomy similar to those of humans, O<sub>3</sub> (70) and O<sub>3</sub> plus allergic sensitization to allergen induce substantial airway (74) and alveolar (75) remodeling

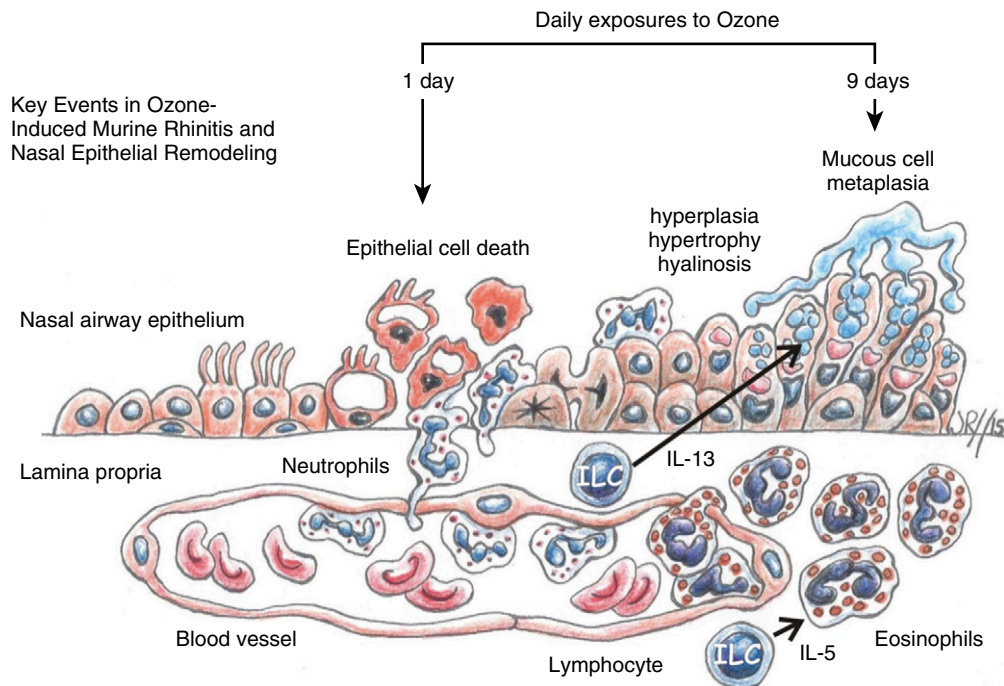
during the early postnatal period. These changes include alterations in smooth muscle, innervation, mucous cell abundance, and allergic sensitization linked to airway hyperresponsiveness (76). The most oxidizing particles, similarly to traffic combustion particles, change airway and/or lung size (68, 69). Thus, oxidant stress may be a common link with reduction in lung growth.

Numerous studies have demonstrated pulmonary responses to oxidant stress after exposure to air pollution. These responses occur in mice and rats with long-term exposure to particulate air pollution (77, 78), diesel exhaust (79–81), iron soot (82), and ambient PM (83), with changes in the antioxidant enzymes 8-hydroxydeoxyguanine (8-OHdG) and glutathione/oxidised glutathione (GSH/GSSG), and oxidation of lipids. Increases in tissue expression of antioxidant genes and proteins are a common response to long-term exposure. Treatment with antioxidants blunts the oxidant effects of particles (84, 85), but early-life responses to oxidant stress may differ from those observed in adults (86). In neonatal rats exposed to polycyclic aromatic hydrocarbon-laden ultrafine PM, which is similar to traffic PM, antioxidant gene and protein expression was not upregulated to levels similar to those observed in adults (87–89). There may be critical windows of time during postnatal lung development when antioxidant defenses are less able to upregulate.

### Early-Life Exposure Causes Immune Changes, Including Type 1/Type 2 Skewing

Early-life air pollution exposure promotes allergic sensitization. PM components, such as diesel emission particles (DEP) (90–93), ultrafine particles (94–96), and PM<sub>2.5</sub> (97–99), can act as allergen-like adjuvants. Such particles have redox-active metals, can induce inflammation and oxidative stress, shift immune function from a T-helper cell type 1 (Th1) to a Th2 response, and drive lymphocyte proliferation and IgE production.

Particle chemical composition is important to biologic potency (99, 100). Simultaneous intranasal administration of ultrafine carbon black particles (CBP)



**Figure 3.** Long-term ozone exposure effects consistent with a role for air pollution in airway inflammation and remodeling leading to asthma development. IL-5 = interleukin-5; IL-13 = interleukin-13; ILC = innate lymphoid cells. Image by Jack R. Harkema.

and allergen (95), has demonstrated increased adjuvant activity. Thus, CBP can directly stimulate dendritic cell maturation (94). DEP and residual oil fly ash (ROFA) can act as adjuvants to increase IgE, bronchoalveolar lavage eosinophilia, lymphocyte reactivity, Th2 cytokine (interleukin-5 and tumor necrosis factor) production, cholinergic airway responses, and allergen-induced bronchoconstriction (95), as can hydrocarbons and soluble transition metals present in DEP and residual oil fly ash, respectively. Thus, DEP and ultrafine particles (UFP) can act as adjuvants in the initial events of allergen sensitization, increasing cytokine production, inflammation, airway hyperresponsiveness (AHR), and airways obstruction.

The role of oxidant stress as a link between air pollution and asthma onset is also supported by studies showing susceptibility based on functional genetic variants in pathways predicted by mechanistic toxicology. For example, Salam and colleagues found that epoxide hydrolase 1 and glutathione-S-transferase variants contribute to the occurrence of childhood asthma and increase asthma susceptibility to pollution exposures from major roads (101). The

roles of these enzymes in asthma stem from their function in important xenobiotic metabolic pathways and the subsequent oxidant stress-mediated tissue damage that can contribute to the pathogenesis of asthma.

#### A Mechanism for Nonatopic Asthma

Consistent with air pollution-induced nonatopic asthma, mice repeatedly exposed to  $O_3$ , without allergen exposure, were found to develop nasal type 2 immunity and eosinophilic rhinitis with mucous cell metaplasia (Figure 3) (102). These  $O_3$ -induced airway alterations are mediated by ILC2s, rather than by the more classical T and B lymphoid cells that are important in adaptive immune responses typically associated with allergic rhinitis and allergic asthma (103). Furthermore, repeated exposure to  $O_3$  induces ILC2-mediated airway type 2 immunity, eosinophilic inflammation, and mucous cell metaplasia in the pulmonary airways (104, 105). Thus, repeated  $O_3$  exposures may induce a nonatopic asthma phenotype characterized by innate type 2 immunity, eosinophilic inflammation, and mucous cell metaplasia. These findings provide plausible paradigms for biological mechanisms underlying the epidemiologically identified associations

between airway eosinophilic inflammation and new onset of nonatopic asthma (106, 107). In addition, after this Workshop was conducted in May 2018, another study evaluated the current scientific evidence of a causal link between DEP and asthma, and found consistent evidence of physiological mechanisms by which DEPs can cause new asthma (108).

#### COPD Development

Relatively few toxicological studies have focused on COPD and air pollution, as most animal models replicate only a few COPD features, and are expensive, technologically challenging, and time-consuming (109, 110). One recent development is spontaneously hypertensive rats that require less time (14 wk vs. 6 mo) to induce COPD-like changes (111). A ferret model developed airway obstruction characteristic of bronchitis and bronchiolitis (112). Short-term PM exposures caused increased pulmonary injuries and attenuated lung antioxidant responses in spontaneously hypertensive rats, providing further evidence of this model's sensitivity to respiratory changes (113). Long-term exposures to  $O_3$  or diesel exhaust are known to induce

remodeling in distal airway regions, which are key to COPD airway obstruction (114–116).

### Modifiers of the Impact of Air Pollution on Airway Disease

Interindividual variation has been identified in susceptibility to the pulmonary effects of air pollution, via both extrinsic (environmental) and intrinsic (host) factors (117). Extrinsic factors include socioeconomic status, exposure to other environmental stimuli, nutrition, and coexposures/infections. Intrinsic factors include age, sex, preexisting disease, and genetic background. Other risk factors include host/maternal obesity (118, 119), diabetes and diet (120), childhood rhinovirus and respiratory syncytial virus infections, and psychosocial and maternal stressors (105).

### Gaps in the Evidence and Opportunities for Future Research

Various inbred strains and genetic models have been used to investigate

susceptibility to respiratory disease; however, these models do not reflect the genetic heterogeneity found in humans. Collaborative Cross and Diversity Outbred models more closely mimic human genetic variability (121). Furthermore, a number of promising animal models of COPD have been developed (111, 112) and used to study factors involved in tobacco smoke-induced COPD, but not air pollution. Because of the structural and immunologic similarities between humans and nonhuman primates, long-term studies in nonhuman primates would be fruitful (122).

### Mechanistic Conclusions

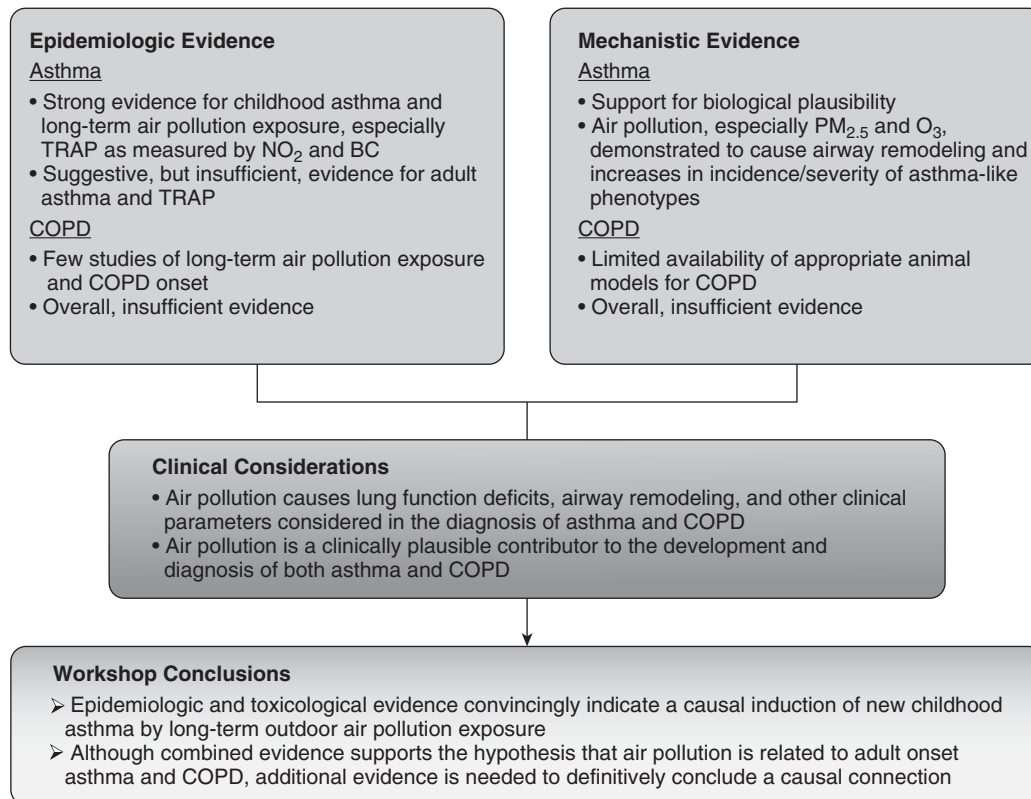
- There are asthma-like phenotypes that increase in incidence/severity with long-term exposure to air pollution, especially to PM and O<sub>3</sub>, consistent with the biological plausibility of air pollution as a causal factor in asthma development.
- Repeated and intermittent air pollution exposures can cause airway remodeling,

which leads to the development of asthma, and may also lead to COPD.

- Sufficient toxicological evidence for air pollution as a cause of COPD is still lacking.
- There remain multiple gaps in our knowledge about airway disease development, including a lack of validated mechanistic models for studies at environmentally relevant exposure levels, and evaluations of epigenetic and genetic influences.

### Clinical Considerations

Many of the clinical parameters considered in a diagnosis of asthma or COPD, such as symptoms of wheeze, cough and mucus production, dyspnea, airway hyperresponsiveness, reduced lung function, and airway remodeling, are also caused by long-term air pollution exposure (123). Air pollution is therefore a plausible contributor to the risk of a new clinical diagnosis of asthma or COPD. However, there are



**Figure 4.** Workshop findings and conclusions. BC = black carbon; COPD = chronic obstructive pulmonary disease; NO<sub>2</sub> = nitrogen dioxide; PM<sub>2.5</sub> = particulate matter less than or equal to 2.5 μm in aerodynamic diameter; TRAP = traffic-related air pollution.

challenges in translating epidemiological and toxicological findings to the clinical context. Large observational epidemiological studies often do not have the same information that may be incorporated into a clinician's diagnostic decision. Studies of exposures to air pollution and the risk of new-onset asthma or COPD have generally relied on self-reported physician diagnoses. For both asthma and COPD, a self-reported physician diagnosis is relatively specific but not sensitive, and cases may be missed or overreported in epidemiologic studies. Additional medical information, including medical history, response to therapeutic medication, physical examination, and lung function measurements, used for the diagnosis of asthma or COPD, may be lacking in epidemiologic studies. For example, pre- and postbronchodilator spirometry and/or methacholine challenge can contribute to a diagnosis, but may not always be included in large epidemiological studies.

Asthma is clinically defined by a history of intermittent respiratory symptoms (e.g., wheeze, shortness of breath, chest tightness, and cough) with reversible airways obstruction and/or hyperresponsiveness (124). Several phenotypes (e.g., allergic and nonallergic) and endotypes (e.g., with or without biomarkers of enhanced Th2 response) have been described (125), and air pollution may have differential effects on the risk for new-onset asthma depending on genetic susceptibility, the presence of allergy, coexposures, obesity, age, and sex. The etiology of asthma is likely multifactorial, and air pollution alone may rarely be the sole or even primary cause.

COPD is a condition characterized by more persistent respiratory symptoms (e.g., shortness of breath, chronic cough, and sputum production), defined by fixed airways obstruction that does not reverse with bronchodilator administration (126). COPD also has several phenotypes (e.g., chronic bronchitis and emphysema) and endotypes (e.g., sputum with or without eosinophils) (127). Spirometry is required for a COPD diagnosis (112), but many published observational studies of air pollution exposures and COPD have not used spirometry to define the outcome. It is well recognized that COPD is clinically underdiagnosed (128). Another challenge is inadequate data to adjust for possible confounding from smoking,

occupational exposures, or household air pollution from combustion of solid fuels for cooking and heating, and the long latency period for COPD development (52). COPD is likely multifactorial, and air pollution is often working in concert with other determinants of disease risk.

### Clinical Conclusions

Many of the clinical parameters considered in a diagnosis of asthma or COPD (e.g., lung function deficits and airway remodeling) are also caused by long-term air pollution exposure, as documented above, indicating that air pollution is a clinically plausible contributor to the development and diagnosis of both asthma and COPD.

### Workshop Conclusions and Recommendations

A summary of the Workshop findings and conclusions is presented in Figure 4. At the end of the Workshop, votes were taken on each of the overarching questions, and there was unanimous agreement that:

1. There are biological mechanisms by which air pollution could plausibly cause the induction of new asthma and/or COPD.
2. Air pollution's known effects on the lung and airways could plausibly contribute to a diagnosis of asthma or COPD in a clinical setting.
3. Epidemiologic and toxicological evidence convincingly indicates a causal link between long-term exposure to outdoor air pollution (especially TRAP) and new childhood asthma.
4. Based on the above, it is concluded that there is sufficient scientific evidence to conclude that long-term outdoor air pollution exposure causally contributes to the development of new childhood asthma.
5. Although combined toxicological and epidemiological evidence supports the hypothesis that long-term air pollution is related to adult-onset asthma and COPD onset, further investigations are needed to definitively conclude that there is a causal connection.

### Future Directions

1. Developing long-term, well-characterized mechanistic air pollution inhalation exposure models for asthma and COPD.
2. Gaining a better understanding of whether the epidemiological associations found for TRAP are due to direct effects of NO<sub>2</sub>, or to a component of the PM<sub>2.5</sub> mass with which NO<sub>2</sub> is commonly associated (e.g., fossil-fuel combustion fine particles).
3. Conducting further investigations of air pollution's impacts on the development of COPD and adult-onset asthma. ■

This official Workshop report was prepared by an *ad hoc* subcommittee of the ATS Assembly on Environmental, Occupational and Population Health.

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# Highly Acidic Ambient Particles, Soluble Metals, and Oxidative Potential: A Link between Sulfate and Aerosol Toxicity

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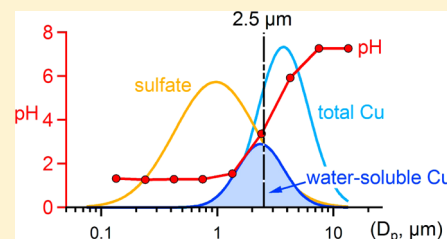
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## Supporting Information

**ABSTRACT:** Soluble transition metals in particulate matter (PM) can generate reactive oxygen species in vivo by redox cycling, leading to oxidative stress and adverse health effects. Most metals, such as those from roadway traffic, are emitted in an insoluble form, but must be soluble for redox cycling. Here we present the mechanism of metals dissolution by highly acidic sulfate aerosol and the effect on particle oxidative potential (OP) through analysis of size distributions. Size-segregated ambient PM were collected from a road-side and representative urban site in Atlanta, GA. Elemental and organic carbon, ions, total and water-soluble metals, and water-soluble OP were measured. Particle pH was determined with a thermodynamic model using measured ionic species. Sulfate was spatially uniform and found mainly in the fine mode, whereas total metals and mineral dust cations were highest at the road-side site and in the coarse mode, resulting in a fine mode pH < 2 and near neutral coarse mode. Soluble metals and OP peaked at the intersection of these modes demonstrating that sulfate plays a key role in producing highly acidic fine aerosols capable of dissolving primary transition metals that contribute to aerosol OP. Sulfate-driven metals dissolution may account for sulfate-health associations reported in past studies.



## INTRODUCTION

Although a substantial number of studies have supported the association between particulate matter (PM) and adverse health outcomes,<sup>1–3</sup> many questions remain on the underlying drivers of PM toxicity. Oxidative stress, an in vivo state of disequilibrium due to an imbalance between antioxidant defense capacity and reactive oxygen species (ROS), has been suggested as a mechanistic explanation for PM toxicity.<sup>4–6</sup> Oxidative potential (OP), referred to as the ability of particles to generate ROS by consumption of antioxidants and/or generation of oxidants, has been used as a health-based exposure measure of PM in several recent studies.<sup>7–11</sup> Methods to measure OP include cellular<sup>5,12</sup> and a-cellular assays.<sup>13–17</sup> Cellular assays involve culturing and exposing cells, whereas a-cellular assays generally involve more straightforward chemical analysis making them easier to perform and automate. The ascorbic acid (AA) (OP<sup>AA</sup>) and dithiothreitol (DTT) (OP<sup>DTT</sup>) assays are two commonly used a-cellular measures of aerosol OP. AA is a physiological antioxidant present in the lung and DTT is used as a chemical surrogate of antioxidants, such as glutathione and NADPH. Both assays measure the depletion of AA or DTT through oxidation under biological relevant temperature of 37 °C and pH of 7.4, mimicking the interaction

between PM and cellular antioxidants in vivo, providing an index of PM's ability to generate ROS. OP<sup>DTT</sup> has been widely used. It has been linked to airway inflammation markers,<sup>18</sup> cellular oxidative stress markers,<sup>19</sup> cellular cytotoxicity,<sup>20,21</sup> and more recently, cardiorespiratory health endpoints in epidemiological studies.<sup>7,8</sup> These results support the use of OP as a highly health relevant air quality parameter.

To mitigate adverse health effects, ambient particle OP sources, and atmospheric transformations that alter OP, need to be known. A number of aerosol components have been found to correlate with aerosol OP<sup>DTT</sup>. These include bulk water-soluble organic carbon (WSOC),<sup>22–24</sup> humic-like substances (HULIS)<sup>25,26</sup> and highly oxygenated organic aerosols,<sup>27</sup> and more specific aerosol components, such as polycyclic aromatic hydrocarbons (PAHs),<sup>22,28,29</sup> quinones,<sup>30,31</sup> and water-soluble transition metals (e.g., manganese (Mn) and copper (Cu)<sup>22–24,30,32</sup>). Source apportionment points to incomplete combustion from biomass and fossil fuels (gas and diesel

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engines), and sources associated with transition metals, such as mineral dust and resuspended road dust from tire or brake wear.<sup>23,24</sup> In contrast to  $OP^{DTT}$ , correlations suggest that transition metals (i.e., Cu) are the main aerosol component contributing to  $OP^{AA}$ , with road-traffic a major source.<sup>24,33</sup> In past studies, both  $OP^{DTT}$  and  $OP^{AA}$  source apportionment found a significant contribution from sources associated with sulfate. Since the AA and DTT assays do not respond to pure sulfate, these associations suggest that secondary processing related to sulfate sources is driving the correlations.

Mechanistic studies have linked increased OP to secondary atmospheric reactions. For example, although correlated with  $OP^{DTT}$ , PAHs are not DTT active, but PAHs can be oxidized to redox-active quinones or nitro-PAHs.<sup>15</sup> Highly oxygenated aromatic compounds, such as hydroxyquinones, make significant contributions to  $OP^{DTT}$ ,<sup>34</sup> consistent with  $OP^{DTT}$  correlations to WSOC and highly oxygenated OA. Aged diesel exhaust PM showed higher  $OP^{DTT}$  than fresh diesel exhaust emissions and the  $OP^{DTT}$  increased with time.<sup>35</sup> Secondary processing also applies to transition metals, which as noted are common drivers of both  $OP^{DTT}$  and  $OP^{AA}$ . Metals sources include industry,<sup>36</sup> vehicles,<sup>37,38</sup> mineral dust,<sup>38</sup> and ship engine emissions<sup>39,40</sup> which are emitted mainly in an insoluble form. Ambient aerosol water-soluble fractions of Cu and Mn range between 10 and 40%, and less than 10% for iron (Fe), even after atmospheric processing,<sup>41,42</sup> suggesting that concentrations of water-soluble forms are limited by the atmospheric conversion process. Thus, to contribute to OP, primary insoluble metals must undergo some form of atmospheric processing to become soluble, a state that could be arrived at by acidification<sup>43–45</sup> or complexation with an organic ligand.<sup>46,47</sup> Soluble metals, such as Fe and Cu participate in redox-cycling reactions, which may lead to enhanced lipid peroxidation, DNA damage, and altered calcium and sulfhydryl homeostasis.<sup>48–50</sup> Common mechanisms involve the Fenton reaction that catalytically converts hydrogen peroxide to the more toxic hydroxyl radicals.<sup>51,52</sup> Epidemiological studies have found associations of water-soluble transition metals with health endpoints, such as reductions in birth weight<sup>53</sup> and preterm birth.<sup>54</sup>

Sulfate, as a main component of  $PM_{2.5}$ , has shown a strong association with mortality in many studies.<sup>55–59</sup> Sulfate contributes to aerosol acidity<sup>60</sup> and there is a historical record of associations between so-called particle “strong acidity”<sup>61–64</sup> and adverse health effects.<sup>65–70</sup> Since fine particle OP has been linked to aerosol toxicity, and water-soluble transition metals and sulfate sources correlated with OP, it may be that sulfate linkages to health are largely through its role in acid dissolution of primary metals commonly found in ambient particles. Here we investigate metals dissolution and its effect on OP by measuring the size distributions of aerosol chemical species to predict particle pH and compare to water-soluble and total metals, and measures of OP.

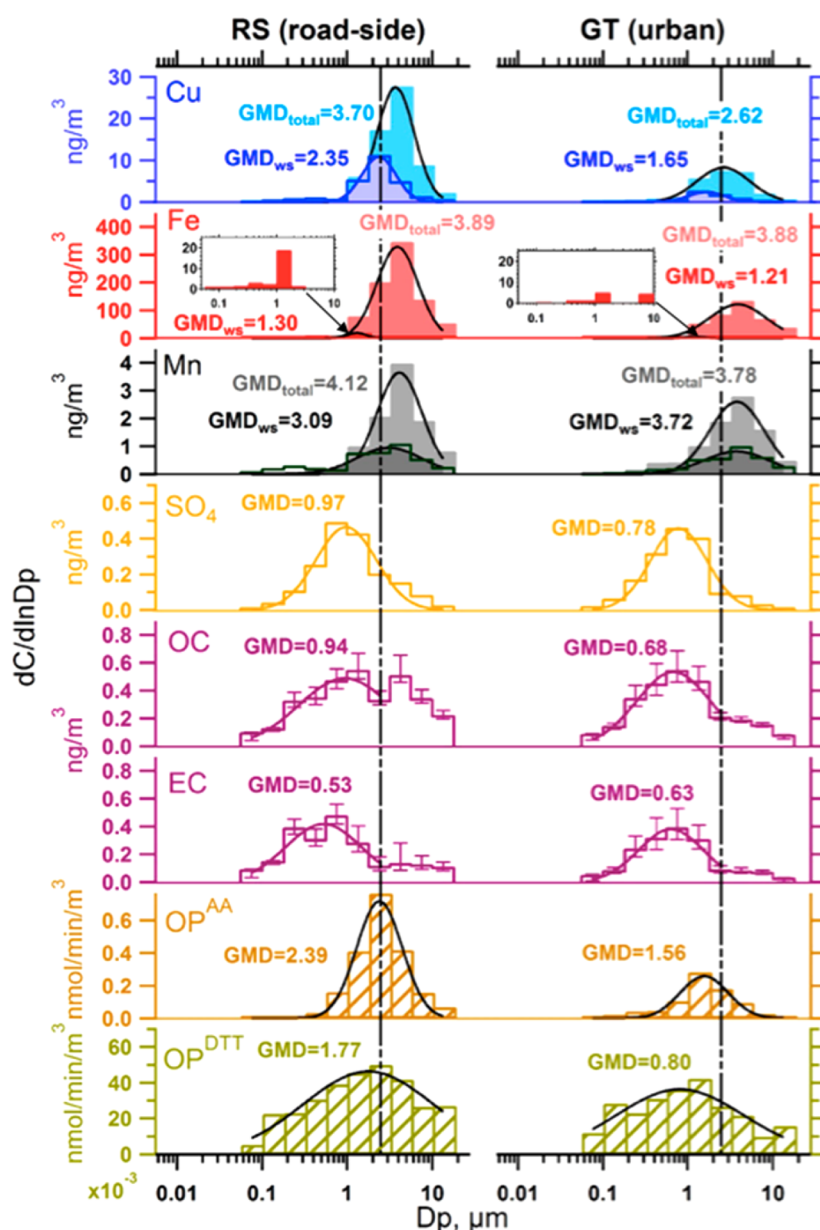
## MATERIALS AND METHODS

**Sample Collection.** Size-fractionated aerosol samples were collected at a road-side and urban site in Atlanta, GA using two Micro-Orifice Uniform Deposit Impactors (MOUDI, MSP Corp., Shoreview, MN). The road-side site (RS) was adjacent (within 5 m) to the interstate I75/85 highway that passes through downtown Atlanta and is highly utilized by light duty vehicles due to heavy duty vehicle (trucks with six wheels or more) restrictions. The 75/85 interstate-connector in Atlanta

had an annual average daily traffic of 301 000 vehicles in 2015, making it one of the busiest corridors in the U.S. (<http://geocounts.com/gdot/>). For contrast, the urban site (GT) was located on the rooftop of the Environmental Science and Technology building on the Georgia Tech campus roughly 420 m from the RS site. The MOUDIs collected samples in a nonrotating mode without a back-up filter and divided particles into ten different size bins under ambient conditions (e.g., RH and T). Aerodynamic particle diameters at 50% collection efficiency for the stages (so-called cutoff sizes) were 18, 10, 5.6, 3.2, 1.8, 1.0, 0.56, 0.32, 0.18, 0.1, and 0.056  $\mu\text{m}$ . Particles with sizes between these cutoffs are collected on separate stages. Two sets of samples were collected at each site, with one set using particle collection impaction substrates of prebaked quartz filters (47 mm Tissuquartz Filters, Pall Corp., Ann Arbor, MI) and the other Zeflur filters (47 mm, PTFE Membrane, 2  $\mu\text{m}$  pore size, Pall Corp., Ann Arbor, MI). For each MOUDI, sampling was conducted for approximately 7 days continuously. The GT set was collected on 3/16–3/23/2016 and RS set 3/28–4/4/2016. These samples were analyzed for various PM chemical components, including organic carbon (OC), elemental carbon (EC), anions, cations, and total and water-soluble Fe, Cu, and Mn, as well as OP. Four other sets of MOUDI samples were collected at each site for OP analysis only, and two out of these four sets were sampled at both sites simultaneously (see Supporting Information (SI) for results). Collected filter samples were immediately sealed in Petri dishes and stored at  $-18\text{ }^{\circ}\text{C}$  until analyzed, which typically occurred within 3 days following collection, except for metals analysis, which was done after a 2-month storage period.

**PM Chemical Components.** Each quartz filter from the MOUDI samples was cut into portions for determining various chemical components. A 1.5  $\text{cm}^2$  portion was used for OC and EC thermal optical determination using a Sunset OCEC Analyzer (Sunset Laboratory Inc., Tigard, OR, IMPROVE method). Another same size portion was extracted in DI water and filtered (0.45  $\mu\text{m}$  syringe filter) for ion measurement via ion chromatography with conductivity detection (Metrohm 761 Compact ICs, Riverside, FL). The remaining fraction of the filter was cut in half and analyzed for total and water-soluble metals using an Inductively Coupled Plasma Mass Spectrometry (ICP-MS) (Agilent 7500a series, Agilent Technologies, Inc., CA). Details on how the mass was determined on divided filters and how the OCEC analysis was done on MOUDI samples is provided in the SI.

For determining total metals, samples were digested in 1:3  $\text{HNO}_3$ :HCl solution, diluted in DI water, then filtered with 0.45  $\mu\text{m}$  syringe filters. For measuring water-soluble metals, filters were sonicated in DI water for 0.5 h with an Ultrasonic Cleanser (VWR International LLC, West Chester, PA, USA). After sonication, the extracts were filtered using 0.45  $\mu\text{m}$  syringe filters, then  $\text{HNO}_3$  was added to produce a final concentration of 2%. Here we focus on Fe, Cu, and Mn as they are common transition metals linked to aerosol toxicity.<sup>71–75</sup> A set of standards of these metals were treated following the same procedures as samples to establish filter mass concentrations from the ICP-MS responses.  $R^2$  of the standard calibration curves ranged from 0.9918 to 0.9995 ( $N = 8$ ) for various metals. A 25 ppb internal standard of scandium (Sc) was added to all samples and standards to monitor analytical drift. Overall uncertainty that included the precision of standards, variability in sample airflow rate, extraction procedure (assuming 5%), and blanks, (all one standard deviation), was estimated to be 8% for



**Figure 1.** Ambient size distributions of PM chemical components and water-soluble oxidative potential at a road-side site (left panel, RS, measurements 3/28–4/4/2016) and an urban background site (right panel, GT, measurements 3/16–3/23/2016) in Atlanta, GA. OC and EC were fitted with a log-normal curve (intercept forced to zero) for size ranges  $<2.5 \mu\text{m}$  while others were fitted for the whole size range. GMD is the geometric mean diameter ( $\mu\text{m}$ ). The vertical dotted line is aerodynamic diameter at  $2.5 \mu\text{m}$ , the so-called upper limit of  $\text{PM}_{2.5}$ . Water-soluble (denoted as ws) Fe had low concentrations relative to total. Water-soluble Fe with enlarged scale can be found in SI Figure S5.

Cu, 8% for Fe, and 6% for Mn. Limits of detection for the water-soluble metals method were  $0.0003 \text{ ng m}^{-3}$  for Cu,  $0.26 \text{ ng m}^{-3}$  for Fe, and  $0.003 \text{ ng m}^{-3}$  for Mn, and those for total metals method were  $0.098 \text{ ng m}^{-3}$  for Cu,  $0.95 \text{ ng m}^{-3}$  for Fe, and  $0.004 \text{ ng m}^{-3}$  for Mn.

**Oxidative Potential.** Oxidative potential (OP) was measured on a half portion of the MOUDI Zefluor filters. Sample preparation and the OP methods are described in detail in other publications.<sup>24,76</sup> Filters were extracted in DI water, filtered with  $0.45 \mu\text{m}$  syringe filters, then separated into two fractions. One was analyzed with the Ascorbic Acid (AA) method via a simplified approach to the AA analysis method that uses a synthetic respiratory tract lining fluid model<sup>77,78</sup> to determine water-soluble AA activity ( $\text{OP}_{\text{ws}}^{\text{AA}}$ ). The other filter fraction was measured for water-soluble DTT activity ( $\text{OP}_{\text{ws}}^{\text{DTT}}$ )

with the dithiothreitol (DTT) assay. These two assays measure the oxidative capacity of particles by monitoring the consumption rate of AA and DTT at pH 7.4 and  $T = 37^\circ\text{C}$ . Final OP data are reported as AA or DTT consumption per volume of sample air (units of  $\text{nmol min}^{-1} \text{ m}^{-3}$ ).

**Aerosol pH.** pH is defined as the logarithmic scale of the hydronium ion activity in an aqueous solution.

$$\text{pH} = -\log_{10} \gamma_{\text{H}^+} \text{H}_{\text{aq}}^+ \cong -\log_{10} \frac{1000 \gamma_{\text{H}^+} \text{H}_{\text{air}}^+}{W_i} \quad (1)$$

Where  $\gamma_{\text{H}^+}$  is the hydronium ion activity coefficient (assumed =1) and  $\text{H}_{\text{aq}}^+$  (mole  $\text{L}^{-1}$ ) is the hydronium ion concentration within the ambient particle liquid water.  $\text{H}_{\text{aq}}^+$  can be also be viewed as the hydronium ion concentration per volume of air



$H_{\text{air}}^+$  ( $\mu\text{g m}^{-3}$ ) divided by the concentration of particle liquid water,  $W_i$  ( $\mu\text{g m}^{-3}$ ). Since most particle water is associated with the highly hygroscopic inorganic species, such as sulfate, liquid water is often estimated from only the inorganic species concentrations, ignoring smaller contributions by organic aerosol components.<sup>79</sup> We follow that approach here.

There is no accurate way to directly measure the pH of ambient  $\text{PM}_{2.5}$ . Methods to infer pH based on ion balances or ratios of measured anion and cations (which does not include  $\text{OH}^-$  or  $\text{H}^+$ ) are not good surrogates for pH.<sup>79–82</sup> Measurements of “strong acidity”<sup>63</sup> often used in previous health studies, are also not an accurate measure of actual particle pH since in that approach  $H_{\text{aq}}^+$  is determined in a vastly more dilute solution than what exists in the ambient aerosol and so is essentially an ion balance approach. Currently, the most accurate way to determine particle pH is to run a thermodynamic model to predict pH based on measured gas and particle species that contribute to pH, assuming the thermodynamic system is in equilibrium. Here we use the model ISORROPIA-II,<sup>83,84</sup> which predicts both  $H_{\text{air}}^+$  and  $W_i$  to determine pH. Ideally, inputs to the model include total (gas plus particle) concentrations of all species that affect pH. The model then predicts equilibrium partitioning of species between the gas and particle phases. A number of studies show the equilibrium condition is met for fine particles,<sup>79,81,85</sup> which can occur fairly rapidly (15–30 min).<sup>86</sup> This is not true for the coarse mode due to kinetic limitations.<sup>86–88</sup> Thus, pH was calculated using different methods for fine and coarse modes. First, since no gas phase species were available, we determined pH in the fine mode through an iteration procedure that used the measured particulate species ( $\text{SO}_4^{2-}$ ,  $\text{NO}_3^-$ ,  $\text{Cl}^-$ ,  $\text{NH}_4^+$ ,  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Ca}^{2+}$ , and  $\text{Mg}^{2+}$ ) and ISORROPIA-II to predict gas species. Total fine mode aerosol concentrations for each ion were determined by summing measured concentrations for MOUDI stages below and including  $3.2\ \mu\text{m}$  cut size. Then, under the assumption that the fine mode ions were in equilibrium with the gas phase, ISORROPIA-II was run in forward mode and gas phase concentrations of  $\text{NH}_3$ ,  $\text{HNO}_3$ ,  $\text{HCl}$  were determined; predicted gas phase concentrations from the  $i - 1$  run were applied to the  $i$ th iteration, until the gas concentrations converged. The converged gas concentrations were similar to what has been observed in this region (e.g., ammonia, being the most important was predicted to be between  $0.6$  and  $0.7\ \mu\text{g m}^{-3}$ , similar to that recorded in the southeastern U.S. ( $0.1$ – $0.8\ \mu\text{g m}^{-3}$ )<sup>85</sup>). We have used this iterative method in a previous study.<sup>81</sup> With these gas phase concentrations, ISORROPIA-II was run for each MOUDI stage using that stage’s measured aerosol ion concentrations and estimated gas concentrations to determine pH for each stage. Since equilibrium is not expected between the gas and particles of the coarse mode due to kinetic limitations, and because these measurements were made fairly close to the source of the coarse mode particles, pH was determined by ignoring interaction with the gas phase. ISORROPIA-II was run in forward mode with zero gas concentrations. A similar result was found for ISORROPIA-II run in reverse mode for the coarse particles, although the numerical solution can be more unstable. (See [Supporting Information](#) for more discussion on coarse mode pH predictions). The fine mode pH predicted here (pH of 1 to 2) is similar to levels found in other studies in this region where more complete data sets (i.e., gases measured) were available. We assumed particles to be internally mixed (i.e., homogeneous pH within each size bin) when calculating pH for each size bin.

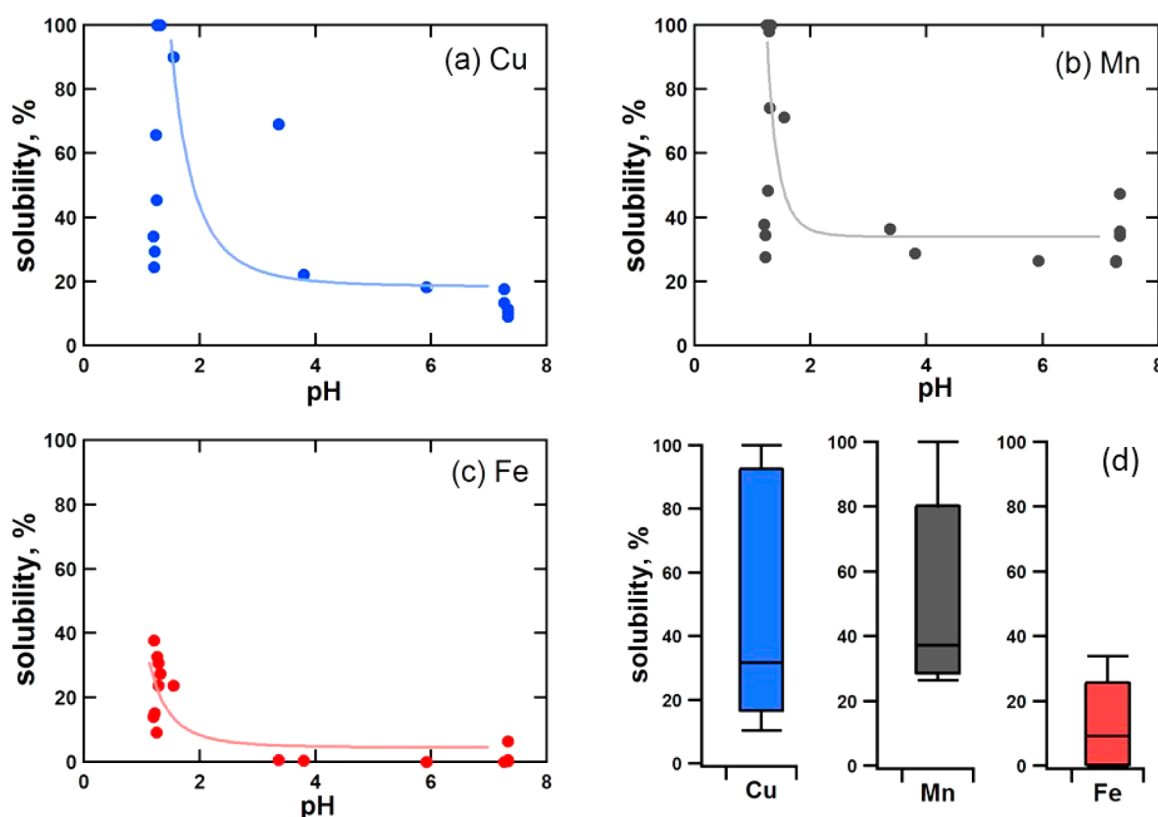
Freshly emitted particles and coarse mode particles may be largely external mixtures. A similar assumption has been made when calculating bulk pH of  $\text{PM}_1$  and  $\text{PM}_{2.5}$  in our past studies and good agreement was still found between observed and predicted partitioning of semivolatile species.<sup>79,81,85</sup> Insoluble ions are not an issue as their concentration is too low that they do not affect the equilibrium of  $\text{H}^+$  in the aqueous solution unless they impede dissolution or mass transport within the particle or between the particle and the gas phase.<sup>89</sup> The uncertainty of pH prediction based on known sources was estimated to be 13% in a previous study<sup>79</sup> and expected to be higher in this study considering the estimated gaseous concentrations and the lower inorganic mass loadings distributed in MOUDI stages. The accuracy of the pH prediction is predominantly judged by the reproduction of semivolatile components partitioning between gas and particle phases (i.e., how do predicted gas  $\text{NH}_3$  or  $\text{HNO}_3$  and particle  $\text{NH}_4^+$  or  $\text{NO}_3^-$  compare to the measurements of these species, as these predictions depend on other ions and RH, T. Details on ISORROPIA-II and verification of predicted pH for  $\text{PM}_{2.5}$  in both the southeastern and northeastern U.S. has been reported.<sup>79,81,85</sup>

## RESULTS

Figure 1 shows the measured size distributions of various PM components and oxidative potential at a road-side (RS) and urban (GT) site in Atlanta, GA. Measurements of OP from other sampling times show similar distributions ([SI Figure S1–S4](#)). The fitted distribution equation and parameters, (geometric mean diameters (GMD) and geometric standard deviation ( $\sigma_g$ )), and the associated uncertainties, can be found in [SI Table S1](#). Although the MOUDI samples at the two sites in [Figure 1](#) were collected at different times, given the samples were averaged over 7 days, and that both were collected close in time, the size distributions are taken to represent the general characteristics of the two sites. (Note that the comparisons of OP in [Figure 1](#) lead to similar observations to those from OP measured simultaneously at the two sites, see [SI Figure S2 and S4](#)).

**Size Distributions of Metals.** Cu, Fe, and Mn exhibited a single mode for both total (elemental) and water-soluble components. As expected for mechanically generated aerosols,<sup>90</sup> total metals were predominantly found in the coarse mode. Water-soluble metals, however, peaked at smaller sizes. Differences in particle GMD between total and water-soluble metals were more obvious for Cu and Fe than for Mn. At the two sites, the GMD for water-soluble Cu and Fe ranged from  $1.24$  to  $2.35\ \mu\text{m}$ , whereas for total Cu and Fe, GMDs were between  $2.63$  and  $3.90\ \mu\text{m}$ . Mn had more similar GMDs for water-soluble ( $3.17$  and  $3.78\ \mu\text{m}$ ) fractions versus total ( $3.83$  and  $4.13\ \mu\text{m}$ ).

Concentrations also differed between sites. The RS site had much higher levels of total Cu and Fe compared to the GT site, indicating a primary traffic emission source. Total Mn was more uniform between the two sites. This is consistent with known sources. Past studies have attributed Cu to brake/tire wear,<sup>91–93</sup> Fe to brake/tire wear<sup>91,94</sup> and mineral dust,<sup>95,96</sup> and some Mn to brake/tire wear<sup>41</sup> and a significant source from mineral dust.<sup>95,96</sup> For water-soluble comparisons between the two sites, Cu and Fe concentrations were also higher by the RS site, whereas Mn levels were similar. This is consistent with our previous findings for the Atlanta region where water-soluble Mn had a more regional characteristic, consistent with a



**Figure 2.** Metal solubility in relation to pH for (a) Cu, (b) Mn, and (c) Fe. (d) shows box plots of metals solubility across all size ranges. Top whisker, 90%; bottom whisker, 10%; line in the box, median; box top, third quartile; box bottom, first quartile. The finest stage ( $D_p = 0.056\text{--}0.1\ \mu\text{m}$ ) and the stage with  $D_p = 0.1\text{--}0.18\ \mu\text{m}$  (only for Fe) had metal concentrations below detection limit and not included in solubility calculations.

mineral dust source, while water-soluble Cu and Fe had a stronger traffic-related source.<sup>97</sup> Thus, comparisons between the two sites show that the RS metals were associated with generally larger particles, consistent with expectations that sedimentation would deplete larger particles as transported further from the source (i.e., metals in smaller particle sizes are enhanced relative to larger particles at the GT site).

Comparisons of the water-soluble to total fractions indicate the extent of their solubility. The average ( $\pm$ SD) water-soluble fraction for all size ranges from all data were  $13 \pm 14\%$ ,  $44 \pm 36\%$ , and  $50 \pm 30\%$  for Fe, Cu, and Mn, respectively. In general, Fe was the least soluble among the three metals, consistent with other studies.<sup>41,42</sup> A box plot showing the span of metal solubility over the whole size range is shown in Figure 2. For all three metals,  $\text{PM}_{2.5}$  had substantially higher soluble fractions than the coarse mode (see SI Figure S6).

**Size Distributions of Carbonaceous Particles and Sulfate.** Organic and elemental carbon (OC and EC) in ambient particles showed a typical aerosol bimodal distribution with a clear fine-mode ( $\leq \text{PM}_{2.5}$ ) and coarse-mode ( $\text{PM}_{2.5\text{--}10}$ ), with a minimum between modes at about  $2.5\ \mu\text{m}$ . At the RS site, OC had a more prominent coarse mode and EC was generally associated with smaller particles. In contrast, sulfate was most abundant in the fine mode. Although  $\text{SO}_2$  is mainly from point sources, such as coal-fired electric generating units, the conversion of  $\text{SO}_2$  to form secondary sulfate results in a regional characteristic for sulfate,<sup>98</sup> accounting for similar concentrations between the two sites (Figure 1).

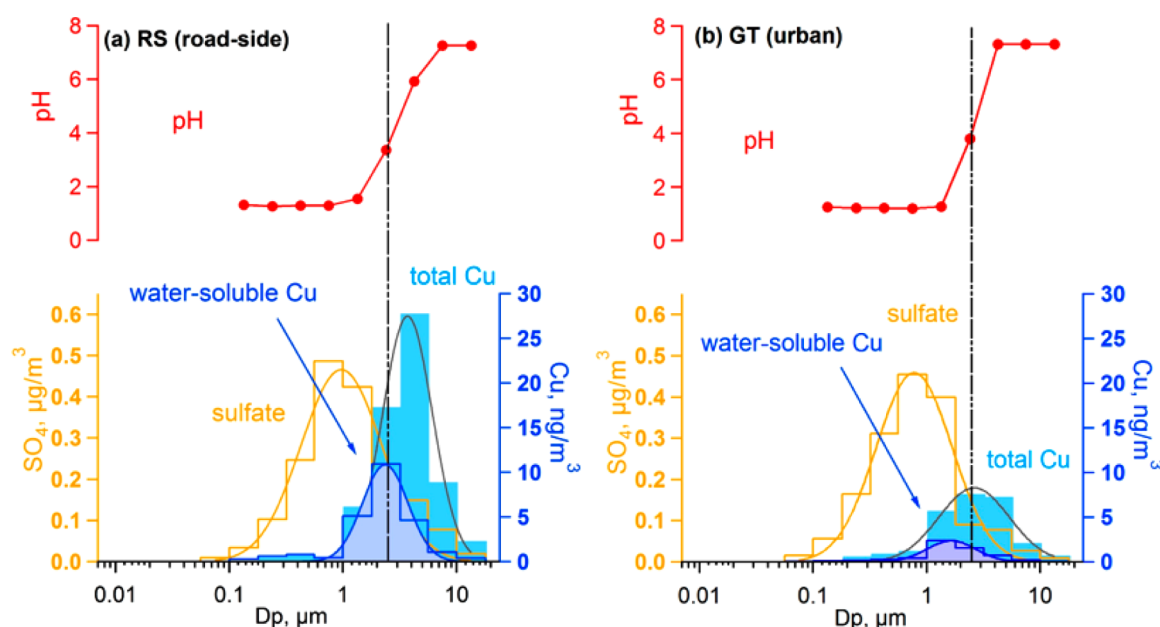
**Size Distributions of Water-Soluble Particulate Oxidative Potential.** Both  $\text{OP}_{\text{ws}}^{\text{AA}}$  and  $\text{OP}_{\text{ws}}^{\text{DTT}}$  had a monomodal distribution, which peaked near the separation between fine and

coarse modes (i.e.,  $2.5\ \mu\text{m}$ ). However, the peaks in the modes were at slightly higher sizes at the RS site, similar to what was observed for water-soluble Cu and Fe. In terms of OP magnitude,  $\text{OP}_{\text{ws}}^{\text{AA}}$  levels were substantially higher at the RS than the GT site, whereas  $\text{OP}_{\text{ws}}^{\text{DTT}}$  were similar at both sites, indicating, like sulfate, a more regional characteristic for  $\text{OP}_{\text{ws}}^{\text{DTT}}$ .  $\text{OP}_{\text{ws}}^{\text{AA}}$  appear mainly associated with roadway emissions. In comparing the two measures of OP,  $\text{OP}_{\text{ws}}^{\text{DTT}}$  peaked at a smaller size than  $\text{OP}_{\text{ws}}^{\text{AA}}$  at both sites and had a much broader distribution. Other studies have also reported OP size distributions, but only focused on differences between broad aerosol modes; for example, ultrafine, fine, and coarse. The more highly size-resolved data reported here are generally consistent with other studies (see SI Figure S7), but provide substantially more insight on the sources and processes leading to aerosol OP.

## DISCUSSION

The OP size distributions are unique in that they peak near  $2.5\ \mu\text{m}$ , which is the minimum separating the mass-based coarse and fine modes (see sulfate and OCEC distributions, for example). This affects where particles are deposited in the respiratory tract.<sup>99,100</sup> The cause for the OP distributions being largely centered near  $2.5\ \mu\text{m}$  can be explained by how the fine and coarse mode interaction contributes to water-soluble metals.

**Metals dissolution by acid processing.** There are two ways that a metal can become soluble, acid dissociation under low pH conditions,<sup>43–45</sup> or by forming a ligand with an organic species, such as oxalate, at higher pH.<sup>101,102</sup> A concentration of water-soluble metals peaking at the overlap between sulfate and



**Figure 3.** Metals (e.g., Cu) dissolution by sulfate under acidic conditions. The vertical dotted line is aerodynamic diameter ( $D_p$ ) at  $2.5\ \mu\text{m}$ , the upper limit of so-called  $\text{PM}_{2.5}$ . pH was estimated from ISORROPIA-II based on ionic species from MOUDI samples collected on 3/28–4/4/2016 and 3/16–3/23/2016 at road-side and urban site, respectively.

the total metals suggests an acidity-driven metals dissolution process. Taking Cu as an example, size distributions of sulfate, water-soluble and total Cu are plotted together in Figure 3 for the RS and GT site. The water-soluble Cu peak was within the overlap of the lower tail of the primary total Cu distribution in the coarse mode and upper tail of the secondary sulfate distribution in the fine mode. If in this overlap area, sulfate and total Cu were internally mixed (within a single particle), insoluble Cu may be mobilized over time by sulfuric acid, creating a soluble form of Cu.

Calculated particle pH for each MOUDI stage is also shown in Figure 3 and supports this hypothesis. Concentrations of the various ions measured on the MOUDI stages that went into the pH calculation are shown in SI Figure 8. Predominance of sulfate and lack of mineral cations in the fine mode ( $D_p$  less than approximately  $1.8\ \mu\text{m}$ ) results in a very low pH, ranging between 1 and 2, whereas low levels of sulfate and high levels of cations, such as  $\text{Ca}^{2+}$  and  $\text{Mg}^{2+}$  (see SI Figure S8), likely in the form of carbonates ( $\text{CaCO}_3$  and  $\text{MgCO}_3$ )<sup>103</sup> in the coarse mode leads to a more neutral coarse mode, with pH approximately near 7. The transition between these two modes is where the soluble metals are found. Metals solubility in relation to pH at each MOUDI stage from both sites are shown in Figure 2 (a–c). When pH is near neutral, metals solubility was low; as pH decreased, solubility substantially increased, further supporting the association of pH with metals solubility and the mechanism of acid processing. Longo et al.<sup>104</sup> also found increasing mineral dust Fe solubility with particle acidity in Saharan dust. The very low pH levels for the fine mode reported here are also consistent with more detailed calculations of pH we have reported for the same region<sup>79,85</sup> and in other locations,<sup>81,82</sup> indicating that this mechanism of metals solubility may apply to many regions.

The dissolution of metal oxides at low pH takes time, from hours up to weeks.<sup>44</sup> Meskhidze et al.<sup>45</sup> found that 2–5% Fe was mobilized after 4 days at pH 1, and that at low pH, the dissolution is much faster since the dissolution rate depends

exponentially on pH. Given that the sampling was done continuously for 7 days, particles collected on the filters were subject to possible postcollection dissolution for an average of 3.5 days. As shown in Figures 2 and 3, for fine particles, pH values were in the range of 1 to 2, therefore, the solubility of fine-mode Fe could be overestimated by up to roughly 5% compared to ambient aerosols, since the collected particles remained at ambient conditions for an extended period of time during the sampling period. As for coarse particles, the predicted pH was larger than 4 and so this effect will be negligible. In our study, metal analysis was done after a 2-month storage period in a freezer. Majestic et al.<sup>105</sup> showed that Fe particles collected on a filter and stored frozen did not change significantly over 3 months. Therefore, continuous dissolution during storage did not likely happen. The toxicity of aerosols is highly sensitive to pH since over their lifespan only a fraction of the total elemental metal is solubilized. Sulfate plays a key role; it is high hygroscopicity leads to formation of the aqueous drops and provides  $\text{H}^+$  that dissolves the metals, forming soluble forms of metal sulfates.

Our other studies in Atlanta support these findings. Correlations have been observed between water-soluble Fe and sulfate ( $r^2 = 0.62\text{--}0.76$ ,  $N = 181$ ) in summer and fall.<sup>97</sup> Single particle X-ray fluorescence (XRF) measurements has shown that Fe solubility was associated with particle sulfur content<sup>106</sup> and that approximately 50% of the sulfate within individual particles between 1 and  $2.5\ \mu\text{m}$  was associated with a metal cation, likely in the form of iron or copper sulfates.<sup>107</sup> These results are also consistent with our source apportionment analysis for aerosols in metropolitan Atlanta, where we found that in  $\text{PM}_{2.5}$ , roughly 50% of the water-soluble Fe and 40% of the water-soluble Cu were associated with secondary processing.<sup>97</sup> The remaining fraction of these metals was largely associated with a primary brake/tire wear source (32% of Fe and 51% of Cu) consistent with high levels at the RS site.

Because of water-soluble transition metals contribution to OP, these processes play an important role in shaping the size



distributions of  $OP_{ws}^{AA}$  and  $OP_{ws}^{DTT}$ . Our previous study showed that Cu is a common contributor to  $OP_{ws}^{AA}$  and  $OP_{ws}^{DTT}$  in  $PM_{2.5}$  ambient samples.<sup>24</sup> In fact, water-soluble Cu was nearly exclusively correlated with  $OP_{ws}^{AA}$  ( $r = 0.70-0.94$ ), consistent with identical size distributions for water-soluble Cu and  $OP_{ws}^{AA}$ , and  $OP_{ws}^{AA}$  higher at the RS than the GT site (Figure 1). In contrast, we have reported that  $OP_{ws}^{DTT}$  was sensitive to water-soluble Cu as well as certain organic species from combustion sources.<sup>23</sup> The combined contribution from these two different aerosol components to  $OP_{ws}^{DTT}$  can explain the differences between  $OP_{ws}^{AA}$  and  $OP_{ws}^{DTT}$  distributions; that is the  $OP_{ws}^{DTT}$  distribution peaking between the fine-mode OC peak and water-soluble Cu peak, resulting in a boarder distribution than  $OP_{ws}^{AA}$ .

**Role of Metals in OP and Health.** The size distribution results presented here, along with our previous single particle analysis, demonstrate that acid processing of metals by sulfate increased the metals solubility and particle OP, providing a linkage between sulfate and adverse health effects that may explain some of the past associations often found between sulfate or “strong acidity” and various health end-points.<sup>55–57,66,67,70,108–110</sup> Our results are consistent with the earlier findings of Ghio et al.,<sup>111</sup> who pointed out a linkage between ambient aerosol sulfate content, soluble metals and oxidant generation. However, they hypothesized Fe solubility was driven by ligand formation, and not acid-driven dissolution. Other processes involving sulfate may also adversely affect health, such as catalyzing the formation of secondary hazardous organic aerosols<sup>112</sup> and chemical reactions involving hydrogen peroxide.<sup>113</sup> In addition, other particle pH effects are possible, for example, pH below 5 can enhance the formation of ozonide at the air-lung surface, leading to ROS generation in vivo, inducing oxidative stress due to ozone.<sup>114</sup>

Changing emissions may increase the importance of aerosol pH health-effects in the future. Although particulate sulfate has substantially decreased in the southeast US over the past 15 years due to the reduction of sulfur dioxide emissions from electrical generating units,<sup>115</sup> we have shown that fine particle pH has not significantly changed, remaining highly acidic with pH between  $-1$  and  $2$ .<sup>85</sup> pH below nominally 3 is likely required to solubilize iron in ambient particles within a reasonable time,<sup>45</sup> (other transition metals, such as Cu, will solubilize at a higher pH), making it possible for this effect to be widespread given our observations of a ubiquitous low fine particle pH.<sup>79,81</sup> Traffic-related metals emissions are not expected to decrease substantially in the near future; traffic counts have increased over the past 10 years (<http://geocounts.com/gdot/>). Adaptation to electric powered vehicles will end tail-pipe emissions, but mechanically generated tire/brake wear emissions will continue. Metals mobilization by acidification is therefore likely to remain an important factor in future aerosol OP and the health effects of PM.

## ■ ASSOCIATED CONTENT

### ■ Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.6b06151.

Table S1, Figure S1–S11, equations to fit the distribution, comparison of  $OP^{DTT}$  to other studies, method for dividing deposits between cut filter portions and OCEC analysis, coarse mode particle pH (PDF)

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### Notes

The authors declare no competing financial interest.

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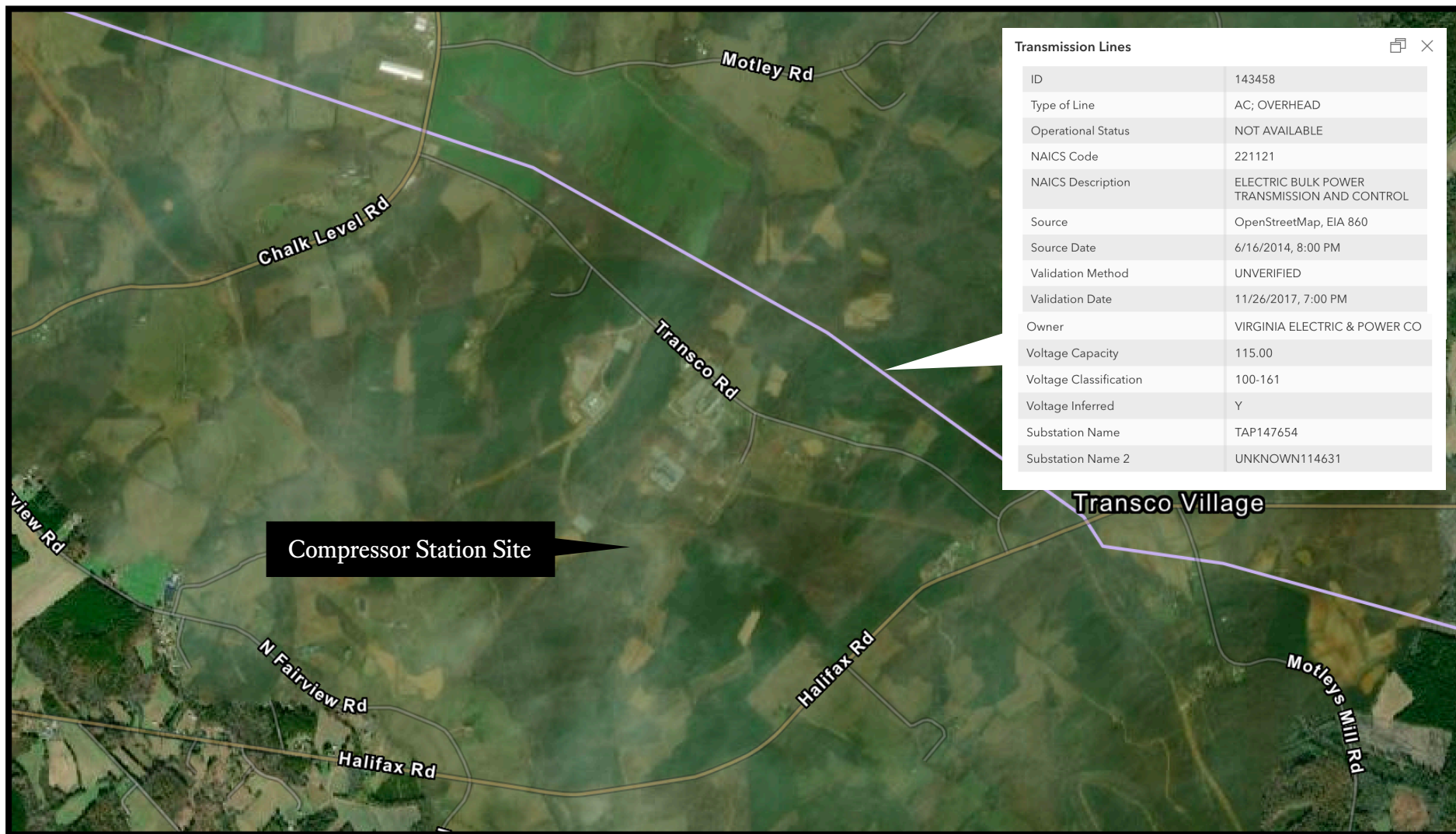
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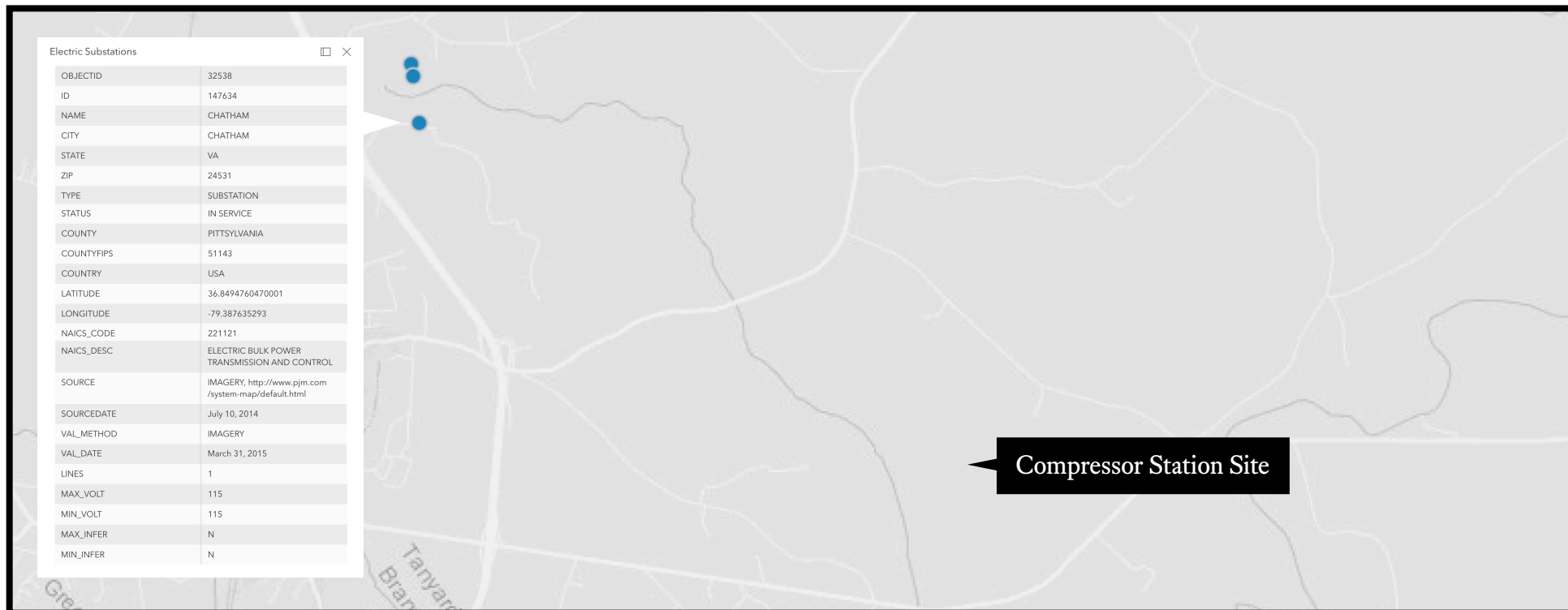
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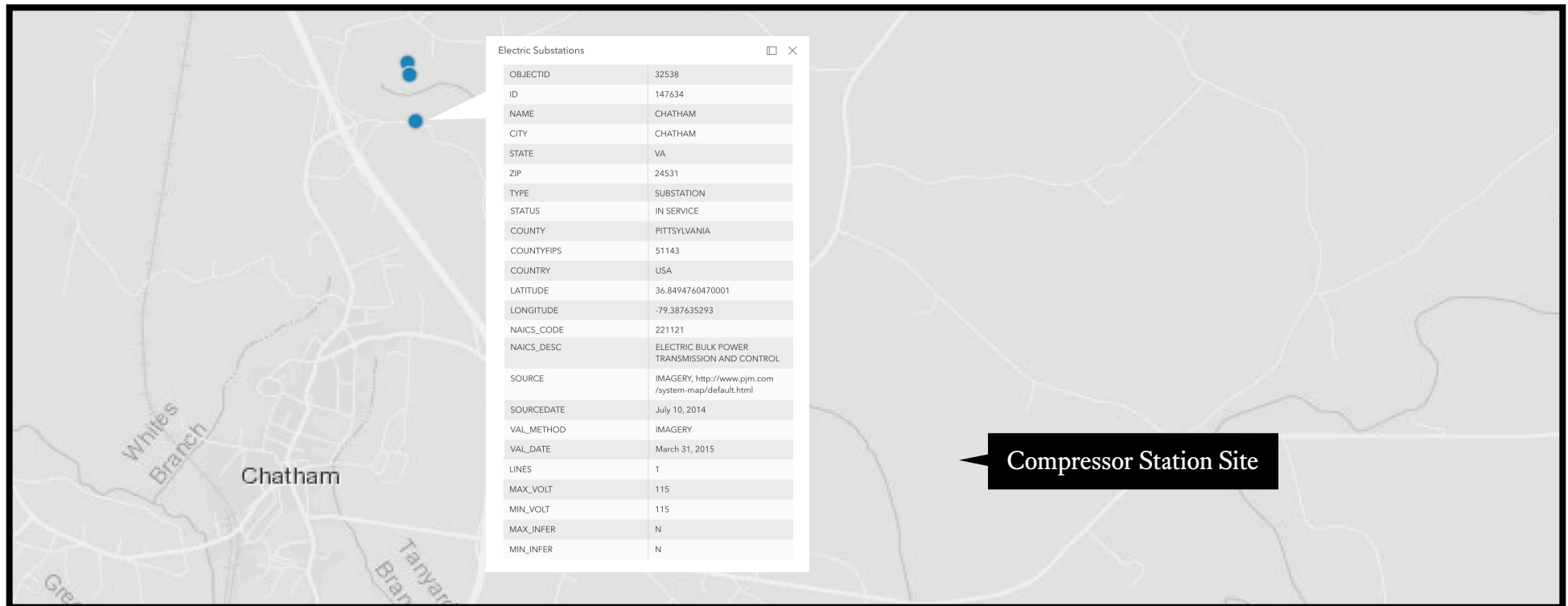




**Source:** UNITED STATES DEPARTMENT OF ENERGY, ENERGY INFORMATION ADMINISTRATION, *United States Energy Atlas: Electricity Energy Infrastructure & Resources* (accessed February 18, 2021), available at <https://atlas.eia.gov/app/895faaf79d744f2ab3b72f8bd5778e68>



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**IN RE DESERT ROCK ENERGY COMPANY, LLC**

PSD Appeal Nos. 08-03, 08-04, 08-05 &amp; 08-06

**REMAND ORDER**

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Decided September 24, 2009

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**Syllabus**

On July 31, 2008, United States EPA Region 9 ("Region") issued a final prevention of significant deterioration ("PSD") permit ("Permit") to Desert Rock Energy Company, LLC ("Desert Rock") pursuant to section 165 of the Clean Air Act ("CAA"), 42 U.S.C. § 7475. The Permit authorizes Desert Rock to construct a new 1,500-megawatt coal-fired electric generating facility approximately twenty-five miles southwest of Farmington, New Mexico.

In the fall of 2008, four different parties filed timely petitions for review of the Permit with the Environmental Appeals Board ("Board"). On April 27, 2009, the Region filed a motion for voluntary remand with the Board, requesting that the Board remand the entire Permit back to it so that it can reconsider its action on several issues that Petitioners raised. Three participants, including Desert Rock, oppose the motion.

Held: The Board remands the Permit on two independent grounds. The Board first concludes that it is appropriate to grant the Region's motion for voluntary remand. The Board also concludes that, based on the administrative record, the entire Permit should be remanded to the Region at this time with respect to one overarching issue related to the Region's best available control technology ("BACT") analysis.

(1) *Board's Determination Concerning the Region's Motion for Voluntary Remand.* The Board concludes that it is appropriate to grant the Region's motion for voluntary remand for several reasons. The Board first concludes that the Region's motion is not prohibited by the part 124 regulations because the regulations neither constrain a region from requesting a voluntary remand after the Board grants review nor proscribe the Board from granting a voluntary remand at any time. Moreover, a contrary result would unnecessarily hamper the Board in its adjudication of permit appeals. The Board further concludes that, under the facts and circumstances of this case, granting the Region's motion for voluntary remand at this time is warranted. The Region has shown good cause for its motion and granting the motion would best serve the interests of administrative and judicial efficiency. The Region asserts that some, if not all, issues it wishes to reconsider may result in changes to the Permit's conditions, including conditions that prompted the Board to grant review of the permit. Additionally, this Permit review is already bifurcated because of a prior stay of the carbon dioxide issue. Furthermore, because the Board has substantial concerns with the Region's approach to its Endangered Species Act compliance in this matter and because this is one of the issues the Region intends to revisit, the Board finds that voluntary remand is particularly appropriate in this case. Finally, as explained in (2) below, one of the issues

the Region wishes to reconsider is an issue on which the Board concludes, on independent grounds, that remand of the entire permit is appropriate.

The Board rejects Desert Rock's, Diné Power Authority's, and American Coalition for Clean Coal Electricity's arguments against remand, which include claims that the motion is made in bad faith, or at a minimum, is frivolous, claims that the motion violates CAA section 165(c), 42 U.S.C. § 7475(c), claims that the Region has violated its trust responsibilities, claims that the Region is denying Desert Rock equal protection, and claims that granting the motion would violate due process principles.

(2) *Board's Determination Concerning the Region's IGCC Analysis.* The Board concludes, based upon a review of the administrative record, that the Permit should be remanded in its entirety because the Region abused its discretion in declining to consider integrated gasification combined cycle ("IGCC") as a potential control technology in step 1 of its BACT analysis for the facility. Although the Region has broad discretion in determining whether imposition of a control technology would "redefine the source," the Board concludes that, based on the administrative record for this case, the Region's analysis is inadequate for two reasons. First, the Region did not provide a rational explanation of why IGCC would redefine the source, especially when the applicant itself had indicated in its initial application that IGCC was a technology that could be considered for the facility (i.e., could satisfy its business purpose), thereby suggesting that IGCC would not redefine the source. Second, the Region failed to adequately explain its conclusion in light of previously issued federal permits at similar facilities in which IGCC *had* been considered as a BACT step 1 production process and had not been considered a "redefinition of the source." The Board concludes that remand of the Permit in its entirety on this ground is warranted because reconsideration of the issue could have overarching impacts on the rest of the Region's analysis.

***Before Environmental Appeals Judges Kathie A. Stein, Charles J. Sheehan, and Anna L. Wolgast.***

***Opinion of the Board by Judge Stein:***

On April 27, 2009, Region 9 ("Region") of the United States Environmental Protection Agency ("EPA" or "Agency") filed a motion for voluntary remand of the final prevention of significant deterioration ("PSD") permit that is the subject of the above-captioned petition for review. *See generally* EPA Region 9's Motion for Voluntary Remand ("Mot. for Vol. Remand"). Several participants in this matter, including the permittee, oppose the motion, while several others support it. For the reasons articulated in Part III.A of this Order, the Environmental Appeals Board ("Board" or "EAB") concludes that it is appropriate to grant the Region's motion for voluntary remand. In addition, as described in Part III.B, the Board concludes, on independent grounds, that the entire Permit should be remanded to the Region at this time with respect to one overarching issue related to the Region's best available control technology ("BACT") analysis. Accordingly, the Board remands the entire Permit to the Region.



## I. CASE HISTORY

On July 31, 2008, pursuant to section 165 of the Clean Air Act (“CAA”), 42 U.S.C. § 7475, the Region issued a final PSD permit to Desert Rock Energy Company, LLC (“Desert Rock”)<sup>1</sup> for the construction of Desert Rock Energy Facility (“Facility”), a new 1,500-megawatt coal-fired electric generating facility proposed to be located approximately twenty-five miles southwest of Farmington, New Mexico. *See* Administrative Record (“A.R.”) 122, at 1 (U.S. EPA, Region 9, Prevention of Significant Deterioration Permit, Number AZP 04-01 (July 31, 2008)) [hereinafter Permit]. The Region serves as the permitting authority because the proposed facility will be located within the Navajo Indian Reservation, and the Navajo Nation lacks an EPA-approved tribal PSD permitting program.

In the fall of 2008, four different parties filed timely petitions for review of Desert Rock’s Permit under 40 C.F.R. § 124.19(a). Specifically, the Board received petitions from Diné Care, Environmental Defense Fund, Grand Canyon Trust, Natural Resources Defense Council, San Juan Citizens Alliance, Sierra Club, and WildEarth Guardians (“NGO Petitioners”); the State of New Mexico (“New Mexico”); Center for Biological Diversity (“CBD”); and Ms. Leslie Glus-trom. Together, the four petitions raise a significant number and a wide variety of issues.

During the course of this permit appeal, the Board granted several motions to participate and, pursuant to 40 C.F.R. § 124.19(c), provided a period in which any interested party could file an *amicus curiae* brief. *See* Order Granting Review, Staying the Carbon Dioxide BACT Issue, and Granting Motions to File Amicus/Nonparty Briefs and Motions to File Reply Briefs (“Order Granting Review”) at 7-8 (Jan. 22, 2009). Consequently, besides the four Petitioners and the Region, the following seven participants have also filed various response, *amicus curiae*, and/or nonparty briefs in this case: the Navajo Nation, Desert Rock (the permit-tee), the National Parks Conservation Association, the Diné Power Authority (“DPA”), the New Mexico Building and Construction Trades Council, Physicians for Social Responsibility, and the American Coalition for Clean Coal Electricity (“ACCCE”).<sup>2</sup>

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<sup>1</sup> STEAG Power, LLC (“Steag”) submitted the original PSD application proposing the Desert Rock Energy Facility. A.R. 120, at 2 (EPA Responses to Public Comments on the Proposed Prevention of Significant Deterioration Permit for the Desert Rock Energy Facility (July 31, 2008)). In September of 2004, Steag sold the rights to the project to Post Oak Power, LLC, a subsidiary of Sithe Global Power, LLC (“Sithe”). *Id.* Several years later, in 2007, Post Oak Power assigned the permit application and all other rights to the project to the current permittee, Desert Rock, another subsidiary of Sithe. *Id.*; *see also* Permit at 1.

<sup>2</sup> ACCCE filed its request to participate in response to the Region’s motion for voluntary remand.

On January 22, 2009, the Board granted review of the Permit pursuant to 40 C.F.R. § 124.19(c). In the Order Granting Review, the Board stayed one of the issues raised by two Petitioners – the question of whether or not to impose limitations on emissions of carbon dioxide. Order Granting Review at 4-5. Because the Region had withdrawn the portion of its permit decision related to carbon dioxide emissions, the Board stayed this issue pending the Region's final determination on it. *Id.* The Board also established a schedule for the filing of briefs on appeal, including the filing of surreply briefs by the Region, Desert Rock, and DPA. *See id.* at 7.

On April 27, 2009, the Region filed a motion for voluntary remand with the Board in lieu of filing its surreply brief. In its motion, the Region requests the Board grant it a voluntary remand, or alternatively, the Board withdraw or amend the Order Granting Review to enable the Region to unilaterally withdraw the Permit. *See* Mot. for Vol. Remand at 25-26. Desert Rock, DPA, and ACCCE filed oppositions to the Region's request. *See* Desert Rock's Response to EPA Region 9's Motion for Voluntary Remand ("DR Opp'n Br."); DPA's Opposition to EPA Region 9's Motion for Voluntary Remand ("DPA Opp'n Br."); ACCCE's Brief in Opposition to EPA Region 9's Motion for Voluntary Remand ("ACCCE Opp'n Br."). NGO Petitioners and CBD (collectively "Conservation Petitioners") filed a joint brief in support of the voluntary remand motion. *See* Conservation Petitioners' Response in Support of EPA's Motion for Voluntary Remand ("Cons. Pet'rs Resp."). In addition, the Region, New Mexico, and Conservation Petitioners filed reply briefs responding to the arguments Desert Rock, DPA, and ACCCE raised in their opposition briefs. *See* EPA Region 9's Reply to Oppositions to Motion for Voluntary Remand ("Reg. Reply") at 15; State of New Mexico's Reply in Support of EPA's Motion for Voluntary Remand ("NM Reply") at 3; Conservation Petitioners' Reply to Desert Rock and ACCCE Regarding the EPA's Motion for Voluntary Remand ("Cons. Pet'rs Reply"). Briefing on the remand motion concluded on June 29, 2009.<sup>3</sup>

## II. ISSUES

The first issue the Board must decide is whether it is appropriate to grant the Region's motion for voluntary remand. To do so, the Board looks at whether the Region has set forth good cause for granting its request.

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<sup>3</sup> Subsequent to the final briefing, ACCCE requested the Board to take notice of a recent Georgia decision. *See* ACCCE's Motion to Take Notice of Supplemental Authority at 1 & Ex.1 (attaching copy of *Longleaf Energy Assocs. v. Friends of the Chattahoochee, Inc.*, Nos. A09A0387 & A09A0388, 2009 WL 1929192 (Ga. Ct. App. July 7, 2009)). Conservation Petitioners responded to this motion with a brief of their own. *See* Conservation Petitioners' Response to ACCCE's Motion Regarding Supplemental Authority. The Board takes administrative notice of the decision.

In addition, the Board considers a second issue: whether it should remand the Permit on the ground that the Region should have considered integrated gasification combined cycle ("IGCC") as a potential control technology in step 1 of its BACT analysis. More specifically, the Board examines whether, based on the administrative record, the Region abused its discretion in concluding that IGCC "re-defines the source" and thus need not be included in BACT step 1.

### III. ANALYSIS

#### A. *The Board's Consideration of the Region's Motion for Voluntary Remand*

As noted, the first issue before the Board is whether it is appropriate to grant the Region's motion for voluntary remand. The Board first describes the Region's rationale for its request. The Board then considers Desert Rock's, DPA's, and ACCCE's arguments that the part 124 regulations prohibit the Region from filing and the Board from granting a motion for voluntary remand at this stage of the permit appeal. Finally, after concluding that the regulations do authorize the Region to file and the Board to entertain such a motion during this stage of the permit proceedings, the Board considers the merits of the Region's motion.

##### 1. *The Region's Rationale for Voluntary Remand*

In its motion, the Region requests that the Board remand the entire Permit back to it so that it can reconsider its action on several issues that Petitioners raised.<sup>4</sup> Mot. for Vol. Remand at 1. More specifically, the Region states that it seeks a remand because "the Administrator's office has requested that Region 9 reconsider its permitting decision with respect to" five issues: (1) using PM<sub>10</sub> (particulate matter with a diameter of 10 micrometers or less) as a surrogate to satisfy PSD requirements for PM<sub>2.5</sub> (particulate matter with a diameter of 2.5 micrometers or less); (2) issuing its final permit decision before completing consultation under section 7(a)(2) of the Endangered Species Act ("ESA"); (3) issuing its final permit decision before completing the case-by-case maximum achievable control technology ("MACT") analysis for hazardous air pollutants under CAA section 112(g); (4) failing to consider IGCC technology in step 1 of its analysis of BACT;

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<sup>4</sup> As noted above, *see supra* Part I, the Region requests, in the alternative, that the Board withdraw or amend the Order Granting Review to enable the Region to withdraw the Permit. Mot. for Vol. Remand at 25-26. The Region explains that part 124 authorizes unilateral withdrawal of a PSD permit prior to the Board's issuance of an order granting review. *Id.* at 25 (citing 40 C.F.R. § 124.19(c)). Thus, according to the Region, if the Board withdraws its Order Granting Review, the Region would then be able to unilaterally withdraw the permit. Because the Board is remanding the Permit, neither the Region's alternative request nor Desert Rock's arguments about this alternate process are addressed. *See* DR Opp'n Br. at 12-13.

and (5) heavily relying on a 1980 screening document in performing its additional impacts analysis for the Facility. *Id.* at 5, 23. The Region requests a remand of the entire Permit and associated administrative record for reconsideration, arguing that a complete, rather than partial, remand of the Permit “will promote efficiency in the Agency’s decision-making and potentially enable Region 9 to resolve several disputed issues.” *Id.* at 1.

The Region first explains that the Administrator recently issued a stay of the regulation addressing the PM<sub>2.5</sub> PSD requirements that Region 9 applied in this action.<sup>5</sup> *Id.* at 3; *see also id.* Ex. A (Letter from Lisa P. Jackson, Administrator, U.S. EPA, to Paul R. Cort, Earthjustice (Apr. 24, 2009)). The Administrator also has stated that the Agency intends to propose repealing the grandfather provision in the rule, which allows PM<sub>10</sub> to be used as a surrogate to comply with the PM<sub>2.5</sub> PSD requirements for certain permit applications that were pending when EPA issued the rule. Mot. for Vol. Remand at 4; *see also id.* Ex. A (mentioning plans to repeal the PM<sub>2.5</sub> grandfather provision). The Region argues that, because it based its final permit decision for PM<sub>2.5</sub> on this grandfathering provision, “it now appears unlikely that the current administrative record will be sufficient to establish compliance with the PSD requirements for PM<sub>2.5</sub>.”<sup>6</sup> Mot. for Vol. Remand at 9; *see also id.* at 4; A.R. 120, at 77 (EPA Responses to Public Comments on the Proposed Prevention of Significant Deterioration Permit for the Desert Rock Energy Facility (July 31, 2008)) [hereinafter RTC] (relying on 40 C.F.R. § 52.21(i)(1)(xi)).

The Region next explains its concerns about the ESA and MACT issues, which it argues are interconnected. First, the Region states that it issued the Permit “before the Agency had completed the consultation required under Section 7(a)(2) of the ESA.” Mot. for Vol. Remand at 9. To address this deficiency, the Region included a permit condition prohibiting construction at the Facility until the Region notifies the permittee that EPA has completed its consultation obligations under the ESA.<sup>7</sup> *Id.*; *see also* Permit at 2 (Condition II.A). According to the

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<sup>5</sup> The PM<sub>2.5</sub> regulation in question is found at 40 C.F.R. § 52.21(i)(1)(xi) (2008). *See* Implementation of the New Source Review (NSR) Program for Particulate Matter Less Than 2.5 Micrometers (PM<sub>2.5</sub>), 73 Fed. Reg. 28,321 (May 16, 2008). The Agency’s administrative stay of the grandfathering provision was published and became effective on June 1, 2009. *See* Implementation of the New Source Review Program for Particulate Matter Less Than 2.5 Micrometers (PM<sub>2.5</sub>), 74 Fed. Reg. 26,098, 26,098 (June 1, 2009). The Agency recently published a final rule staying the grandfathering provision for nine months. *See* Implementation of the New Source Review (NSR) Program for Particulate Matter Less Than 2.5 Micrometers (PM<sub>2.5</sub>); Final Rule to Stay the Grandfathering Provision for PM<sub>2.5</sub>, 74 Fed. Reg. 48,153 (Sept. 22, 2009).

<sup>6</sup> Significantly, two of the Petitioners challenged the Region’s PM<sub>2.5</sub> analysis. *See* NGO Petitioners’ Supplemental Brief at 201-15; NM Petition for Review and Supplemental Brief at 56-64.

<sup>7</sup> Three of the Petitioners challenged this condition. *See* NGO Pet’rs Suppl. Br. at 280-87; NM Suppl. Br. at 7-18; CBD Petition at 5-32.

Region, after issuance of its permit decision in July 2008, the federal agencies involved in permitting the Desert Rock project sent a Biological Assessment ("BA") to the United States Fish and Wildlife Service ("FWS") as part of the ongoing consultation process under ESA section 7(a)(2) regarding the Desert Rock project.<sup>8</sup> Mot. for Vol. Remand at 10. Recently, on February 26, 2009, presumably in response to the BA, FWS informed the Region that "its own analysis has led it to determine that mercury emissions may be adversely affecting the endangered Colorado pikeminnow, as well as contributing to numerous fish consumption advisories in the Four Corners area." *Id.* at 10; *see also id.* Ex. B (Letter from Wally Murphy, FWS New Mexico Ecological Services Field Office, to Deborah Jordan, EPA Region 9 Air Division Director (Feb. 26, 2009)). The Region states that "[m]ercury emissions therefore appear to be a significant concern to FWS in the context of the Desert Rock project ESA consultation." Mot. for Vol. Remand at 10. The Region asserts that the recent FWS concerns "have increased the likelihood that the ESA consultation will lead to an amendment to the permit application or a modification of the PSD permit terms" to address ESA concerns. *Id.*

Moreover, the Region explains that it plans to provide additional details about the mercury emissions to FWS, but that this additional information will be sent only after it receives an application from Desert Rock for a case-by-case MACT determination.<sup>9</sup> *Id.* at 10-11. The Region believes that these associated ESA and MACT issues "are of sufficient importance to reconsider [its] decision to conduct the PSD permit review, ESA consultation, and section 112(g) review on separate timetables." *Id.* at 11. Finally, the Region explains that "after further reviewing the EAB's *Indeck-Elwood* opinion and a more recent EAB Order in another matter, [it] believes it is no longer efficient or prudent under the circumstances surrounding this permit to request that the EAB proceed with its review of this permit prior to the conclusion of the ESA consultation covering the permit." *Id.* at 11. For these reasons, the Region requests that the Board remand the permit so it can "coordinate the completion of these processes." *Id.*

The Region also requests remand to reconsider the scope of its BACT analysis for the Facility. *Id.* at 18. More particularly, the Region seeks to reconsider its decision to issue the Permit without considering IGCC technology in the BACT

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<sup>8</sup> The BA was "prepared on behalf of the Bureau of Indian Affairs ('BIA')." Mot. for Vol. Remand at 10. BIA acts as the lead agency in the consultation process with FWS for the Desert Rock project. RTC at 169.

<sup>9</sup> Although Desert Rock previously provided estimates of the mercury emissions, it did not submit a detailed analysis with the PSD application. *See* Mot. for Vol. Remand at 9-10. The applicant typically calculates such estimates in connection with the MACT application, which, in this case, the Region had not required prior to issuance of the PSD permit. *See id.* at 9-10, 12, 14. Two Petitioners challenged the Region's decision not to require the case-by-case MACT analysis in conjunction with the PSD permit. NGO Pet'rs Suppl. Br. at 125-52; NM Suppl. Br. at 35-41.

analysis it performed.<sup>10</sup> *Id.*; see also RTC at 13-20 (explaining why IGCC was not considered). The Region states that the Administrator “does not support a policy that would preclude permitting authorities from exercising their discretion to evaluate this option.” Mot. for Vol. Remand at 18. Thus, the Region “prefers to reconsider the scope of its BACT analysis” for Desert Rock “rather than continue to contest this issue on appeal.” *Id.*

Lastly, the Region requests that the Board remand the Permit in order to give the Region an opportunity to reconsider its additional impacts analysis. *Id.* at 23-25. The Region explains that, in performing the analysis, it heavily relied on a 1980 Agency document entitled “A Screening Procedure for the Impacts of Air Pollution Sources on Plants, Soils, and Animals.” *Id.* at 23; see also RTC at 150 (discussing additional impacts analysis). The Region states that, “after further review of the EAB’s analysis of this document in the *Indeck-Elwood* matter, [it] has been persuaded that additional evaluation of site-specific conditions is warranted to strengthen compliance with section 52.21(o) of the applicable regulations.” Mot. for Vol. Remand at 23-24.

## 2. Part 124 Does Not Prohibit a Voluntary Remand

Several participants contend that EPA’s part 124 regulations prohibit permit issuers from requesting and/or the Board from granting motions for voluntary remand after the Board grants review, an argument the Region and Petitioners emphatically reject. Compare DR Opp’n Br. at 9-11 (“The Board cannot grant EPA Region 9 permission to do what 40 C.F.R. Part 124 prohibits.”), DPA Opp’n Br. at 1, 7-8,<sup>11</sup> and ACCCE Opp’n Br. at 4 (“EPA is prohibited by regulation from withdrawing the permit the agency previously issued.”) with Reg. Reply at 15 (“This regulation does not expressly permit or exclude the relief requested by [the Region] – leave of the EAB to reconsider disputed issues after the EAB has granted review.”), NM Reply at 3 (“A region’s inability to *unilaterally* withdraw the permit after review has been granted does not translate \* \* \* into a bar on a region’s ability to seek or the Board’s ability to grant leave to withdraw the permit.”), and Cons. Pet’rs Resp. at 5-8.

The participants’ dispute centers on section 124.19, which prescribes the procedures for PSD permit appeals. Notably, section 124.19 contains only a sole

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<sup>10</sup> At the time it issued the Permit, the Region concluded that IGCC would “redefine the source” and thus did not include it as a potentially available control technology in step 1 of the BACT analysis. See RTC at 13.

<sup>11</sup> DPA states that “it joins in the arguments set forth in the briefing herein by its co-developer, Desert Rock.” DPA Opp’n Br. at 1. Accordingly, where DPA does not specifically address an issue, the Board will assume without further citation that all arguments made by Desert Rock are also made by DPA.

reference to withdrawals, voluntary remands, and/or reconsiderations of a permit decision by a region after a petition has been filed. It states:

The Regional Administrator, at any time prior to the rendering of a [Board] decision \* \* \* to grant or deny review of a permit decision, may, upon notification to the Board and any interested parties, withdraw the permit and prepare a new draft permit under § 124.6 addressing the portions so withdrawn.

40 C.F.R. § 124.19(d).

As the participants acknowledge, this provision explicitly allows the Region to *unilaterally* withdraw a permit decision (or portion thereof) *prior* to the Board's grant of review. *See, e.g., In re San Jacinto River Auth.*, NPDES Appeal No. 07-19, at 3 (EAB Mar. 28, 2008) (Order Dismissing Petition for Review) (explaining, in an unpublished final order, that the region need only notify the Board and other parties prior to withdrawing all or a portion of the permit); *In re Wash. Aqueduct Water Treatment Plant*, NPDES Appeal No. 03-07, at 2 (EAB Dec. 15, 2003) (Order Dismissing Petition for Review) (explaining, in an unpublished final order, that motion for remand of permit conditions was unnecessary where region had withdrawn those portions of permit). The regulations, however, do not address a region's authority to request withdrawal, voluntary remand, and/or reconsideration *after* the Board issues an order granting review of the permit but *before* the Board issues a final decision.<sup>12</sup> *See* 40 C.F.R. pt. 124; *see also* EAB Practice Manual at 38 (June 2004), *available at* <http://www.epa.gov/eab/pmanual.pdf> ("There are no regulatory requirements for motions filed in permit proceedings under part 124 (except for the requirements in section 124.19(g) governing motions for reconsideration)."). Moreover, section 124.19(d) only addresses a region's authority to take action, not the Board's.

The participants interpret this part 124 regulatory silence differently. The Region, New Mexico, and Conservation Petitioners read the regulation to implicitly allow permit issuers to file a motion requesting voluntary remand after the Board has granted review, which the Board, in its discretion, may grant.<sup>13</sup> Mot. for Vol. Remand at 6-8, 25; Cons. Pet'rs Resp. at 5; NM Reply at 3. Desert Rock, DPA, and ACCCE, on the other hand, read the regulatory text in starker terms:

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<sup>12</sup> The Board addresses this issue solely in the context of a *grant* of review because, when the Board *denies* review of a permit, the permit decision becomes the final agency action. *See* 40 C.F.R. § 124.19(f)(1).

<sup>13</sup> *See, e.g., Cons. Pet'rs Resp.* at 5 ("Until the Board has made a final determination on a permit appeal, it has broad discretion within the administrative review process to remand permits, allow the Region to withdraw all or part of a permit, or to refer permit appeals to the Administrator.").



not only to prohibit the unilateral withdrawal of the permit by the region after a grant of review, but to prohibit any type of withdrawal, voluntary remand, or reconsideration, unilateral or otherwise by the Region. DR Opp'n Br. at 7-8, 9-11; ACCCE Opp'n Br. at 4-5. Thus, Desert Rock, ACCCE, and DPA all essentially contend that the Board's hands are tied, and it has no discretion to remand the Permit to the Region following a grant of review, short of rendering a decision on the merits. DR Opp'n Br. at 7; ACCCE Opp'n Br. at 4-5.

The Board disagrees with the interpretations advanced by Desert Rock, DPA, and ACCCE, which, if adopted, would unnecessarily hamper the Board in its adjudication of permit appeals. A limit on the Region's unilateral authority does not translate into a bar on the Board's exercise of discretion. More fundamentally, the regulations do not in any way prohibit the Board from granting a voluntary remand at any time. The Board reaches this conclusion based both on the regulatory text as well as several additional considerations.

First, the Board has broad discretion to grant a voluntary remand, and nothing in section 124.19(d) narrows its discretion. As the Board has previously explained, "[a] voluntary remand is generally available where the permitting authority has decided to make a substantive change to one or more permit conditions, or otherwise wishes to reconsider some element of the permit decision before reissuing the permit." *In re Indeck-Elwood, LLC*, PSD Appeal No. 03-04, at 6 (EAB May 20, 2004) (Order Denying Respondent's Motion for Voluntary Partial Remand and Petitioners' Cross Motion for Complete Remand, and Staying the Board's Decision on the Petition for Review) [hereinafter *Indeck-Elwood* 2004 Stay Order]. Indeed, the Board, "at it[s] discretion, has granted voluntary remands independent of Section 124.19(d)" on several occasions. *Id.* at 5 (citing *In re NE Hub Partners, L.P.*, 7 E.A.D. 561, 563 n.14 (EAB 1998); *In re GMC Delco Remy*, 7 E.A.D. 136, 138, 169, 170 (EAB 1997)); *see also In re City of Hollywood*, 5 E.A.D. 157, 170, 176-77 (EAB 1994) (granting region's remand request on two issues); *cf. In re Columbia Gulf Transmission Co.*, PSD Appeal No. 88-11, 1990 WL 324099 (Adm'r July 3, 1990) (Order on Motion for Stay) (granting permit issuer's motion for a stay following issuance of an order granting review).<sup>14</sup>

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<sup>14</sup> Desert Rock argues that *NE Hub*, *GMC*, and *Indeck* are not on point because, in those cases, "the permitting authority's withdrawal of the permit appears to have come before the Board rendered a decision granting or denying review, which is entirely consistent with section 124.19(d) and not at all the case here." DR Opp'n Br. at 10 n.3. The Board disagrees that these cases are irrelevant to the remand issue. Desert Rock's description of the three cases overlooks the critical facts. In *NE Hub*, while the remand occurred prior to the order denying review, the significant fact is that the permit issuer requested a voluntary remand, which the Board, in its discretion, granted. *See* 7 E.A.D. at 563-64 (describing case background); *see also In re NE Hub Partners, L.P.*, UIC Appeal Nos. 97-1 & 97-2, at 1-3 (EAB May 30, 1997) (Remand Order) (considering remand request) [hereinafter *NE Hub* Remand Order]. At that time, section 124.19(d) did not contain the language authorizing unilateral withdrawals, *see* discussion in text *infra*, nor did the regulations mention voluntary remands. *See* Continued



Part 124 does not contain any language proscribing the Board's general authority to grant voluntary remands, nor does section 124.19(d) limit the Board's discretion to consider a remand motion. Therefore, the mere fact that the permit issuer files a voluntary remand motion after the Board has issued an order granting review does not determine whether the motion can be granted.<sup>15</sup>

Second, the history of the section 124.19(d) language is consistent with the Board's reading of the permit regulations. This history suggests that the 2000 amendment to section 124.19 – which added the regulatory text at issue in this case – was solely intended to give regions unilateral authority to withdraw permits. In the preamble to the proposed rule, the Agency explained that: "In practice, EPA has withdrawn and reissued permits under all statutes prior to decisions of the EAB as well as prior to ALJ decisions."<sup>16</sup> Amendments to Streamline the

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40 C.F.R. § 124.19(d) (1997). The Board in no way suggested that a voluntary remand request was impermissible under the regulations because the regulations did not explicitly authorize such a request. Similarly, in *GMC*, the permit issuer requested a voluntary remand on one issue, which the Board granted. 7 E.A.D. at 169-70. Again, the Board did not in any way indicate that such a motion was impermissible even though the regulations did not explicitly authorize such a motion. Furthermore, the Board granted the voluntary remand request simultaneously with its grant of review, not before it, as Desert Rock suggests. *Id.* Finally, the fact that the participants in *Indeck* submitted their remand motions prior to the Board's order granting review is unimportant. The key points in that case are that the Board (1) specifically found that a voluntary remand "independent of Section 124.19(d)" was permissible and (2) considered the participants' remand motions, ultimately denying them *on their merits*. See *Indeck-Elwood* 2004 Stay Order at 5. Thus, just as the Board explains in the above text, the Board's order in *Indeck* indicates that the timing of a voluntary remand request is irrelevant to the Board's authority to entertain such a motion.

<sup>15</sup> Desert Rock argues that granting the Region's motion would essentially give the Board the authority to modify *any* of the procedures and requirements in part 124. DR Opp'n Br. at 11. Desert Rock's argument is flawed because the Board is not *modifying* any part 124 procedures or requirements here. The Board is merely interpreting section 124.19's silence on this issue in a manner consistent with the terms of part 124 and its purpose. See, e.g., *In re Heritage Envt'l Servs., Inc.*, RCRA Appeal No. 93-8, 1994 WL 544238 (EAB Aug. 3, 1994) (Order Dismissing Appeal) (summarizing, where regulation was silent on issue, case law interpreting the part 124 "filed by" date as meaning the date petition is received by Board rather than date it is postmarked by petitioner); see generally EAB Practice Manual at 26-42 (providing more detailed guidance for filing permit appeals than section 124.19 provides). As noted above, the Board has granted motions for voluntary remand in other cases, even though part 124 does not specifically address whether permit issuers may move for a voluntary remand or whether the Board may grant them. See, e.g., *NE Hub* Remand Order at 3; *GMC*, 7 E.A.D. at 136. Ironically, under Desert Rock's narrow reading of the regulations, the Board would be unable to review Desert Rock's opposition brief because the regulations do not explicitly allow opposition briefs to be filed. The Board does not believe Desert Rock's view of the regulations to be a fair reading or interpretation of section 124.19.

<sup>16</sup> The Agency added the language allowing unilateral withdrawal of permits by regions to section 124.19(d) in a final rule issued on May 15, 2000. See Amendments to Streamline the NPDES Program Regulations: Round Two, 65 Fed. Reg. 30,886, 30,911 (May 15, 2000). That rulemaking combined the National Pollutant Discharge Elimination System ("NPDES") permit procedural regula-

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NPDES Program Regulations: Round Two, 61 Fed. Reg. 65,268, 65,281 (Dec. 11, 1996) (proposed rule). The Agency therefore proposed to add the new regulatory text to “clarify” that regions “may withdraw and reissue any NPDES, RCRA, UIC, and PSD permit (or a contested condition thereof) prior to a decision of the EAB to grant or deny review.” *Id.* The preamble in no way suggests that this additional regulatory text was intended to limit or change the Board’s customary practice of allowing permit issuers to file motions either for remands or for stays of the proceedings. *E.g.*, *In re NE Hub Partners, L.P.*, UIC Appeal Nos. 97-1 & 97-2, at 3 (EAB May 30, 1997) (Remand Order) (1997 order granting region’s motion for voluntary remand); *GMC*, 7 E.A.D. at 170 & n.71 (1997 order granting voluntary remand); *cf. Columbia Gulf*, 1990 WL 324099 (1990 order granting motion requesting stay so that permit issuer could reexamine its analysis and/or supplement the record).<sup>17</sup>

Third, as the Board has often stated, Agency policy favors allowing the Region to make permit condition decisions rather than the Board. *E.g.*, *In re Dominion Energy Brayton Point, LLC*, 13 E.A.D. 407, 413 (EAB 2007), *appeal rendered moot by settlement*, No. 07-2059 (4th Cir. Dec. 17, 2007); *In re Teck Cominco Alaska Inc.*, 11 E.A.D. 457, 472 (EAB 2004); *In re Sutter Power Plant*, 8 E.A.D. 680, 687 (EAB 1999). The preamble to the part 124 regulations articulates this principle, stating that “most permit conditions should be finally determined at the Regional level.” Consolidated Permit Regulations, 45 Fed. Reg. 33,290, 33,412 (May 19, 1980); *accord In re BP Cherry Point*, 12 E.A.D. 209, 217 (EAB 2005); *In re Cardinal FG Co.*, 12 E.A.D. 153, 160 (EAB 2005). This is one of the reasons the Board often remands a permit to the permit issuer rather than making a decision on the merits when the Board finds error in the permit decision. *See, e.g.*, *Teck Cominco*, 11 E.A.D. at 496 (remanding a second issue to the permit issuer rather than reaching its merits where the Board had already decided to remand the permit on other grounds); *In re Knauf Fiber Glass, GmbH*, 8 E.A.D. 121, 140-41, 175 (EAB 1999) (remanding one issue to allow permit issuer to further develop its rationale and a second issue to place rationale in administrative record); *City of Hollywood*, 5 E.A.D. at 166-68 (remanding several additional issues for further consideration by the permit issuer in light of remand

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tions with the procedural regulations under other environmental permit programs administered by EPA, including the PSD program. Because the Agency did not receive comments on the proposed regulatory text, the Agency finalized the language with no further explanation of the provision. *See id.* at 30,901. Thus, the proposed rule preamble discussion provides the Agency’s only explanation for the regulation.

<sup>17</sup> ACCCE claims that allowing remand would establish “new grounds” for permit issuers to reconsider permits and thus “harm” ACCCE’s members. ACCCE Opp’n Br. at 14. As the Board has already concluded, granting the Region’s request would not break new ground. Thus, ACCCE’s claim of harm is unpersuasive.

on another issue); *see also In re Dominion Energy Brayton Point, LLC*, 12 E.A.D. 490, 508-09 (EAB 2006) (explaining that Board typically either sustains a permit decision or remands it to the permit issuer). Granting a permit issuer's request for a voluntary remand so it may amend its permit decision is clearly consistent with this policy. Moreover, allowing for remand requests makes sense in light of the purpose of the administrative appeals process, which is to ensure that the agency fully considers the relevant issues and makes a sound, reasoned final decision.

Finally, requiring a permit issuer to request a voluntary remand from the Board after the Board has granted review but before it issues a final decision makes sense from a judicial and administrative efficiency standpoint. It allows the Board to decide whether, after the Board has granted review and performed a substantial review of the case, it would be more appropriate for the Board to issue a final decision on the merits or grant the voluntary remand request. Thus, for example, in cases where significant time has passed following the submission of final briefs by all the parties, the Board may be in a position to issue a final decision at the time of a request for voluntary remand. *See Indeck-Elwood 2004 Stay Order* at 9 & n.16 (noting that a stay – rather than a remand – was appropriate where the Board had already “made considerable headway in its examination of the record”). On the other hand, where the request is made by the permit issuer shortly after the grant of review, the Board may determine it more appropriate to grant the motion for voluntary remand.

From a procedural standpoint, requiring the Region to seek permission from the Board for a voluntary remand in cases where the Board has already granted review is similar to the practice in federal courts. If a federal agency seeks to reconsider an action that has been appealed to a federal court, the agency cannot unilaterally withdraw its decision but must instead move the court to either remand the matter or stay the case pending the agency's reconsideration. *E.g., B.J. Alan Co. v. ICC*, 897 F.2d 561, 563 n.1 (D.C. Cir. 1990); *Anchor Line Ltd. v. Fed. Mar. Comm'n*, 299 F.2d 124, 125 (D.C. Cir.), *cert. denied*, 370 U.S. 922 (1962); *see also SKF USA, Inc. v. United States*, 254 F.3d 1022, 1029-30 (Fed. Cir. 2001) (listing three scenarios in which an agency may want to reconsider its decision and thus seek remand). The federal courts have recognized the wisdom of granting remand motions because it allows an agency to correct its mistakes, thereby promoting good government and judicial efficiency. *See, e.g., Citizens Against the Pellissippi Parkway Extension, Inc. v. Mineta*, 375 F.3d 412, 418 (6th Cir. 2004) (allowing agency to reconsider and reissue relevant NEPA documents would conserve resources of the judiciary and the parties); *SKF*, 254 F.3d at 1029-30 (noting that, where an agency requests voluntary remand in connection with a change in agency policy or interpretation, while the “court need not necessarily grant such a remand request, remand may conserve judicial resources”); *B.J. Alan*, 897 F.2d at 563 n.1 (explaining that the D.C. Circuit has “recognized that “[a]dministrative reconsideration is a more expeditious and efficient means of achieving an adjustment of agency policy than is resort to federal courts”” (quoting *Pennsylvania v.*

ICC, 590 F.2d 1187, 1194 (D.C. Cir. 1978))). Similarly, it would be highly inefficient for the Board to issue a final ruling on a permit when the Agency is contemplating changes to that permit. *See Indeck-Elwood 2004 Stay Order* at 8; *see also In re Multitrade Ltd. P'ship*, 3 E.A.D. 773, 777 (Adm'r 1992) (remanding matter to permit issuer rather than reviewing petitions because it was the "more responsible (and hopefully expeditious) course" where permittee planned to request permit amendments).

In sum, the Board concludes that the part 124 regulations do not prohibit the Region from requesting a voluntary remand following the Board's grant of review. Nor do they prohibit the Board from granting a voluntary remand motion. To the contrary, such authority advances the Board's task of fairly and efficiently adjudicating permit appeals. The Board next examines whether it is appropriate to grant the Region's request in this case.

### 3. *It Is Appropriate to Grant the Region's Motion*

Desert Rock, DPA, and ACCCE also challenge the appropriateness of granting the Region's motion for voluntary remand. Their arguments against remand range from asserting that the Region fails to show cause for its motion, ACCCE Opp'n Br. at 7-12, to claiming that the motion is made in bad faith, or at a minimum, is frivolous, DR Opp'n Br. at 16-26; ACCCE Opp'n Br. at 12-13, to raising other issues, such as due process and equal protection claims, statutorily-based claims under CAA section 165(c), and claims of trust responsibility violations, *e.g.*, DR Opp'n Br. at 11-42; ACCCE Opp'n Br. at 7-13; DPA Opp'n Br. at 4-8. As discussed in more detail below, the Board disagrees with these three participants and concludes, in light of the Region's rationale for requesting the remand, the Board's analysis of the Region's ESA compliance activities, and the current posture of this permit appeal, that a remand is appropriate.

#### a. *The Region's Motion is Meritorious, Not Frivolous or in Bad Faith*

The Region's rationale for its motion justifies granting remand in this case. As a general matter, the Board typically grants a motion where the movant shows good cause for its request and/or granting the motion makes sense from an administrative or judicial efficiency standpoint. *Compare, e.g., In re Desert Rock Energy Co.*, PSD Appeal Nos. 08-03 to -06, at 3-5 (EAB Aug. 21, 2008) (Order Granting Desert Rock's Motion to Participate, Granting a 30-Day Extension of Time, and Denying a Stay of Briefing on Certain Issues) (discussing merits of extension of time motion and judicial efficiency considerations) *and Columbia Gulf*, 1990 WL 324099 (granting joint motion of permit authority and applicant for stay of proceedings rather than region's request for remand because movants' argument was rational and conducive to administrative efficiency) *with Indeck-Elwood 2004 Stay Order* at 6, 16 (denying remand where basis for request

was flawed and judicially inefficient). More specifically, the Board generally grants voluntary remand motions “where the permitting authority has decided to make a substantive change to one or more permit conditions, or otherwise wishes to reconsider some element of the permit decision before reissuing the permit.” *Indeck-Elwood* 2004 Stay Order at 6; *see also NE Hub* Remand Order at 2 (noting that the region was proposing to issue new permit decisions if the remand motion was granted); *GMC*, 7 E.A.D. at 169 (explaining that the region would incorporate new language into the permit on remand).

Similarly, the federal courts tend to liberally grant agency motions for remand where an agency seeks to reconsider its prior decision. *See Pellissippi Parkway*, 375 F.3d at 417 (“[V]oluntary remand is appropriate even without a change in the law or new evidence \* \* \*.”); *SKF*, 254 F.3d at 1029-30 (explaining that “an agency may request a remand (without confessing error) in order to reconsider its previous position” or “because it believes that its original decision is incorrect on the merits and wishes to change the result” and that federal court has discretion over whether or not to grant either type of request); *Sw. Bell Tel. Co. v. FCC*, 10 F.3d 892, 896 (D.C. Cir.) (noting that court had granted agency’s request for voluntary remand “to permit FCC to give further consideration to the matters addressed”), *cert. denied*, 512 U.S. 1204 (1993); *Wilkett v. ICC*, 710 F.2d 861, 863 (D.C. Cir. 1983) (noting that federal court had granted an agency request for remand “for the purpose of reconsideration” and that agency ultimately reached same conclusion); *Trujillo v. Gen. Elec. Co.*, 621 F.2d 1084, 1086 (10th Cir. 1980) (explaining that agencies have “inherent authority to reconsider their own decisions” and noting that such reconsideration may, in some instances, lead to a different result). As *Desert Rock* and *ACCCE* note, however, federal courts may deny remand motions where the request is frivolous or in bad faith. *SKF*, 254 F.3d at 1029; *see e.g., Lutheran Church-Mo. Synod v. FCC*, 141 F.3d 344, 349 (D.C. Cir. 1998). Likewise, there is ample room within the Board’s standard for the Board to deny a motion should it conclude that bad faith or frivolousness were the driving force for the Region’s request.

In its motion, the Region discusses several issues it proposes to reconsider on remand. *See Mot. for Vol. Remand* at 8-25. The Region also indicates that its reconsideration of some, if not all, of these issues may necessitate changes in some terms of the Permit. *Id.* For instance, the Region requests remand so that “it may coordinate the completion of” the PSD permit review, ESA, and section 112(g) MACT determination. *Id.* at 11. The Region represents that there is a likelihood that the Permit’s terms will change as a result of FWS’s concerns about mercury emissions.<sup>18</sup> *Id.* at 14-15. Likewise, the Region explains that it is requesting remand to “reconsider its decision not to evaluate IGCC as a BACT option for this project.” *Id.* at 21. This, too, may lead to reissuance of the permit, issuance of

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<sup>18</sup> The Board discusses the ESA issue further *infra* Part III.A.3.c.

an amended response to comments document, and/or issuance of an amended draft permit.

Based on the above statements, which indicate that the Region indeed “wishes to reconsider some element[s] of the permit decision before reissuing the permit” and may “make a substantive change to one or more permit conditions,” the Board finds that, contrary to Desert Rock’s and ACCCE’s arguments, the Region has shown good cause for requesting a remand. *Indeck-Elwood 2004 Stay Order* at 6. Thus, for these reasons alone, the Board concludes that remand is appropriate.

Furthermore, the Board granted review in this case because it had substantial concerns with several conditions of the Permit, concerns with some of the very issues the Region is planning to reconsider on remand. The Board provides an analysis of one of these problematic issues – the Region’s compliance with the ESA and its reliance on Condition II.A to do so – in Part III.A.3.c of this opinion. The Board also concludes, based on its own review of the administrative record, that it is appropriate to remand the case at this time on one ground: the Region’s failure to consider IGCC in step 1 of the BACT analysis. *See infra* Part III.B. For these two reasons, the Board disagrees with Desert Rock’s and ACCCE’s arguments that the Region has no real cause to request the remand and that the Region’s request is in bad faith, or at the very least, is frivolous.<sup>19</sup> DR Opp’n Br. at 16-26; ACCCE Opp’n Br. at 5-13.

Finally, the already partially bifurcated status of the case lends further support for remand. In January, the Board stayed one issue raised by Petitioners so that the Region could “‘prepare a new statement of basis addressing the issue of whether the permit should contain an emissions limitation for carbon dioxide,’ provide notice of the revised statement, and provide an opportunity for comment.” Order Granting Review at 3 (quoting Region’s Notice of Partial Withdrawal of Permit at 3); *see also* discussion of procedural history *supra* Part I. Judicial and administrative efficiency considerations weigh on the side of remanding the entire case so that, if the Region concludes that permit reissuance is necessary on multiple grounds, it may reissue the permit only once. Furthermore, it is important for

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<sup>19</sup> ACCCE’s arguments against remand, at least in part, appear to rely on an assumption that the Permit “was properly issued” by the Region. *See, e.g.*, ACCCE Opp’n Br. at 3, 6, 12; *see also* DR Opp’n Br. at 3, 21, 25 (arguing that there was no error in the permitting decision). In light of our discussion above and in Part III.A.3.c, it is obvious that the Board has concerns with the Permit. Moreover, as the Board also mentions above and discusses in Part III.B *infra*, the Board has found the Permit to be inadequate. The Board also interprets the Region’s statements that it is requesting remand to reconsider its ESA obligations and its additional impacts analysis after “further reviewing” *Indeck* to indicate, at least in part, that the Region believes its original decision was incorrect on the merits. *See* Mot. for Vol. Remand at 11, 23. ACCCE’s, and Desert Rock’s, arguments on this point are therefore unpersuasive.



the Region to have the opportunity on remand to consider the permit as a whole so that it may evaluate the impact of changing one permit condition on any other impacted conditions.

b. *The Board Rejects Other Grounds for Denying Remand*

Before turning to the Board's concerns with the ESA issue, the Board first considers and rejects the other arguments DPA, Desert Rock, and ACCCE raise against granting the Region's motion.

(i) *DPA's Trust Responsibility Argument*

DPA argues that the Region should have consulted the tribe prior to requesting a remand and has therefore "flouted" its trust responsibilities. DPA Opp'n Br. at 7; *see also id.* at 4 ("Denial of th[e] motion is further compelled by EPA's utter disregard of its government-to-government obligations to consult on such matters with the tribal interests in this proceeding."). DPA further argues that the Agency has failed to follow various Agency policies and procedures concerning interactions with tribal governments, which, for example, require the Agency to "coordinate and consult meaningfully with [t]ribes to the greatest extent practicable for agency actions that may affect the tribes." *Id.* at 6 (quoting Office of Policy, Economics, and Innovation, U.S. EPA, EPA-233-B-03-002, *Public Involvement Policy of the U.S. Environmental Protection Agency* 5 (May 2003)), available at <http://www.epa.gov/publicinvolvement/pdf/policy2003.pdf>. Although DPA does not identify a standard against which the Board should review its argument in the context of the Region's request for remand, the Board reads DPA's claim as suggesting, akin to the arguments the Board addressed in the previous section, that the Region's conduct somehow constitutes grounds for denying the Region's motion. *See supra* Part III.A.3.a.

While it is far from clear that the Board even has jurisdiction to review DPA's claim, without deciding this question, the Board concludes that, based on the facts and circumstances described here, DPA has not shown conduct on the part of the Region that could constitute grounds for denying the motion.<sup>20</sup> While DPA claims that EPA "filed its motion with no prior tribal consultation whatsoever," DPA Opp'n Br. at 5,<sup>21</sup> the Region states that there *has* been an ongoing dialogue between the Agency and the Navajo Nation about the Permit. Reg. Reply at 8-10. The Region's brief documents at least two conversations during the rele-

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<sup>20</sup> As noted in Part III.A.3.a, the Board reviews all motions, such as the Region's motion, to see whether the movant shows good cause. Here, the Board considers whether DPA's allegations somehow deprive the Region of the good cause the Board found it had demonstrated.

<sup>21</sup> *See also* DPA Opp'n Br. at 7 ("[H]igh-level political appointees (as well as one EPA staff attorney in the case), have been meeting with various Petitioners – and *not* the Navajo \* \* \*").

vant time period between the Administrator and the President of the Navajo Nation. *See id.* Ex. B (calendar printout of scheduled meetings, talking points for meeting), Ex. C (Letter from Dr. Joe Shirley, President, Navajo Nation, to Lisa Jackson, Adm'r, EPA 1 (Apr. 28, 2009) (mentioning prior conversation in April 2009)). Thus, any suggestion by DPA that the Region failed to consult with the tribe at all on this issue is inconsistent with the Region's documentary evidence. Rather than evincing any bad faith or inappropriate conduct on the part of the Region, DPA's arguments, at most, suggest that a disagreement exists between the participants about the scope of the consultation and not about whether consultation in fact occurred: the Region believes the Agency's discussions with the Navajo Nation President that included mention of the "possibility that Region 9 might change one or more of its positions in the appeal" and its call to the tribe on the date the remand request was submitted were sufficient, *id.* at 10, whereas DPA believes the Region should have provided the Navajo Nation with advance notice of the Region's plan to file a motion for remand, DPA Opp'n Br. at 4-5, 7. For the foregoing reasons, DPA has failed to demonstrate that the Region's actions provide grounds for denying the Region's motion. The Board emphasizes that it respects the government-to-government relationship between the Navajo Nation and EPA and is confident that the Region will continue to appropriately include the tribe during the remand stage.<sup>22</sup>

(ii) *Desert Rock's Section 165(c) Argument*

Desert Rock argues that the Region's motion for a voluntary remand is a "clear violation" of section 165(c) of the CAA and that the Region should not be allowed to "snatch the PSD permit away from the Board right before a decision on the merits." DR Opp'n Br. at 13. Section 165(c) of the Act states that "[a]ny completed permit application under section 7410 of this title for a major emitting facility in any area to which this part applies shall be granted or denied not later than one year after the date of filing of such completed application." 42 U.S.C. § 7475(c).

As an initial matter, nothing in section 165(c) prohibits the Board from granting a motion for voluntary remand. To the extent Desert Rock is arguing that the Region's actions are barred by section 165(c), it is not clear from this record that the application is, in fact, "completed" within the meaning of section 165(c).<sup>23</sup>

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<sup>22</sup> Of course, the Board itself cannot individually meet with a tribe during the pendency of a case as this would be a prohibited *ex parte* communication.

<sup>23</sup> The time frame in section 165(c) runs from the date the Region receives a "completed application." 42 U.S.C. § 7475(c). The Region contends that the application "is not currently complete under regulations currently in effect." *See* Reg. Reply at 16. The Region may also find that additional ESA-MACT or IGCC information is necessary to ensure it has sufficient information to make a final  
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Even if Desert Rock is challenging the Region's failure to act as set forth in section 165(c), the Board would not have jurisdiction to adjudicate the claim. *See* CAA § 304(a), 42 U.S.C. § 7604(a) (granting district courts of the United States the jurisdiction to compel nondiscretionary agency action unreasonably delayed).

Moreover, as described in this decision, the Board has concluded, *on the merits*, that at least one critical aspect of the Region's permit decision *was* an abuse of discretion, and it is therefore remanding the Permit on this ground. *See infra* Part III.B; *see also supra* Part III.A.3.c. The Board is doing so at this time to speed up the process so that the parties will have the benefit of the Board's analysis on remand. The Board therefore does not find it necessary to address this argument further.

### (iii) *Desert Rock's Constitutional Challenges*

Desert Rock also challenges the Region's request for remand on both equal protection and due process grounds. DR Opp'n Br. at 35-42 (equal protection), 42-45 (due process). Desert Rock first asserts that the Region's motion for voluntary remand "constitutes an attempt to intentionally administer a facially neutral statute – the Clean Air Act – unequally against Desert Rock" in violation of the equal protection principles inherent in the due process clause of the Fifth Amendment of the U.S. Constitution. *Id.* at 35. Desert Rock also claims that a remand would effectively withdraw its Permit without hearing or review in violation of due process. DR Opp'n Br. at 42.

As a preliminary matter, constitutional challenges to statutes and Agency regulations are rarely entertained in the context of a permit appeal. *See In re USGen New England, Inc.*, 11 E.A.D. 525, 560-61 (EAB 2004) (Interlocutory Order Dismissing Motion for Evidentiary Hearing), *appeal dismissed for lack of juris. sub nom. Dominion Energy Brayton Point, LLC, v. Johnson*, 443 F.3d 12 (1st Cir. 2006); *In re City of Irving*, 10 E.A.D. 111, 124 (EAB 2001); *see also In re Ocean State Asbestos Removal, Inc.*, 7 E.A.D. 522, 557-58 (EAB 1998) (explaining that Board rarely considers constitutional challenges in penalty enforcement context); *In re B.J. Carney Indus.*, 7 E.A.D. 171, 194 (EAB 1997) (same). The Board, however, will consider constitutionally-based challenges to the *manner* in which a statute or regulation has been applied. *Ocean State*, 7 E.A.D. at 558; *In re Gen. Elec. Co.*, 4 E.A.D. 615, 627-36 (EAB 1993); *see also Irving*, 10 E.A.D. at 124 (acknowledging general rule). Because Desert Rock is essentially questioning the manner in which the Region applied the CAA and the applicable regulations in the context of this permit decision rather than challenging the

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permit decision. *See* Mot. for Vol. Remand, Ex. B at 1 (letter from FWS to Region stating that "source attribution data" are needed for ESA analysis).

constitutionality of the statutes or regulations themselves, the Board considers Desert Rock's two constitutional claims in turn below.

(a) *The Region Has Not Denied Desert Rock  
Equal Protection*

First, according to Desert Rock, the Region is unequally administering the CAA, treating Desert Rock differently than other "similarly situated" PSD applicants with no rational basis. DR Opp'n Br. at 36. Specifically, Desert Rock asserts that the Region's motion for voluntary remand "constitutes intentionally unequal treatment of Desert Rock" as compared to three other prospective (or recent) coal-fired power plant PSD applicants: one that received a final PSD permit from the Georgia Department of Natural Resources, one that received a permit from the Louisiana Department of Environmental Quality, and one that received a permit from the Florida Department of Environmental Protection.<sup>24</sup> *Id.* at 37-38. Although Desert Rock admits that "the permitting agencies in the three permitting cases are not EPA," it argues that, because all the permits were issued under the CAA (by these three approved states), EPA could "force equal treatment" by seeking a "SIP Call" under another provision of the statute.<sup>25</sup> *Id.* at 38 (citing 42 U.S.C.

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<sup>24</sup> Under the CAA and associated regulations, a PSD program, or portions thereof, may be administered within a state (not including Indian Reservations) in one of three ways. *In re Milford Power Plant*, 8 E.A.D. 670, 673 (EAB 1999). First, EPA may run the program pursuant to a "Federal Implementation Plan" under part 52. *See* CAA §§ 109-110, 165, 168, 42 U.S.C. §§ 7409-7410, 7475, 7478; 40 C.F.R. part 52; *Milford*, 8 E.A.D. at 673. Second, EPA can delegate its authority to operate the PSD program to the state. *Milford*, 8 E.A.D. at 674. In such cases, the state issues PSD permits as federal permits on behalf of the Agency. 40 C.F.R. § 52.21(u); *see also* discussion of Illinois delegated program *infra* Part III.B.3.a. Third, if a state PSD program meets certain applicable (generally minimum) requirements of federal law, EPA can approve the state's program and such program is incorporated into the state's overall State Implementation Plan ("SIP"). *See* CAA §§ 110, 116, 161, 42 U.S.C. §§ 7410, 7416, 7471; 40 C.F.R. § 51.166; *Virginia v. EPA*, 108 F.3d 1397, 1406-10 (D.C. Cir.) (containing lengthy history of SIP provision and explaining federal and state roles and responsibilities in SIP process), *modified on reh'g*, 116 F.3d 499 (1997); *Milford*, 8 E.A.D. at 673. In this last circumstance, the state conducts PSD permitting under its own authority, and its PSD requirements, although similar to the federal requirements, may differ. Office of Air Quality Planning & Standards, U.S. EPA, *New Source Review Workshop Manual* 1 (draft Oct. 1990) ("NSR Manual"); *see also Virginia v. EPA*, 108 F.3d at 1406-10; *Milford*, 8 E.A.D. at 673; *In re Carlton, Inc.*, 9 E.A.D. 690, 692-93 (EAB 2001) (noting that state-issued permits, and even state requirements in a federal PSD permit, may only be challenged under state law) (citing cases); *In re Sutter Power Plant*, 8 E.A.D. 680, 690 (EAB 1999) (explaining that the Board may only review permit conditions implementing the federal PSD program, not those related to state or local requirements); *In re Knauf Fiber Glass, GmbH*, 8 E.A.D. 121, 161 (EAB 1999) (same). The third scenario applies to the Georgia, Louisiana, and Florida PSD permits that Desert Rock references.

<sup>25</sup> Under section 110 of the CAA, EPA may make what is known as a "SIP Call," where it requires a state to revise its program to correct a "substantially inadequate" SIP. CAA § 110(k)(5), 42 U.S.C. § 7410(k)(5); *accord Sierra Club v. Ga. Power Co.*, 443 F.3d 1346, 1348 (11th Cir. 2006); *see also In re Newmont Nev. Energy Inv., LLC*, 12 E.A.D. 429, 457 n.9 (discussing a SIP Call issued

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§ 7410(k)). Notably, Desert Rock does not mention or compare itself to any recent federal PSD applicants or refer to any recent federally issued PSD permits.<sup>26</sup>

Desert Rock's claim is essentially a "class of one" equal protection claim, in other words, a claim that it "has been 'irrationally singled out,' without regard to any group affiliation, for discriminatory treatment." *United States v. Moore*, 543 F.3d 891, 896 (7th Cir. 2008) (quoting *Engquist v. Ore. Dep't of Agric.*, 128 S.Ct. 2146, 2153 (2008)). Generally, under equal protection jurisprudence, in order to establish a "class of one" claim, a party must show that it has intentionally been treated differently than others with whom it is "similarly situated."<sup>27</sup> *E.g.*, *Engquist*, 128 S.Ct. at 2153; *Vill. of Willowbrook v. Olech*, 528 U.S. 562, 564 (2000); *Leib v. Hillsborough County Pub. Transp. Comm'n*, 558 F.3d 1301, 1306-07 (11th Cir. 2009); *Moore*, 543 F.3d at 896. Desert Rock fails to make such a showing.

First of all, as Desert Rock admits, EPA did not issue the other three permits; instead, those permits were issued by states operating under approved programs. DR Opp'n Br. at 37. Thus, Desert Rock's "class of one" equal protection claim is atypical in that, although its claim does contain an underlying comparison between different sovereigns's actions, it does not per se challenge and compare decisions made by one governmental entity. Desert Rock's claim instead primarily relies upon the rather unique premise that it may challenge one governmental entity's failure to require other sovereigns to make identical decisions and/or exercise their discretion in the same manner as the first, where the laws and regulations of the sovereigns are not necessarily identical and the decisions involve the exercise of discretion. *See supra* note 24. Desert Rock has not cited any authority to support its argument. Notably, comparing two different decisionmakers's actions has generally been found to be inappropriate in the equal protection context. *E.g.*, *Moore*, 543 F.3d at 897 (concluding that comparison between decisions of

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by EPA). Notably, Desert Rock does not specify precisely what the SIP Call it believes the Agency should have issued would have entailed, except that it would have "force[d] equal treatment of these issues throughout the United States." DR Opp'n Br. at 38. Without an explanation of the contents of such a SIP call, Desert Rock's vague arguments lack force.

<sup>26</sup> Ironically, the Region's statements that it wants to reconsider its Permit decision in light of the *Indeck* permit decision, *see* Mot. for Vol. Remand at 11-13, 23-24, which was a *federally* issued PSD permit, *see In re Indeck-Elwood, LLC*, 13 E.A.D. 126, 128 (EAB 2006), suggests the reverse of Desert Rock's claim: that the Region may seek to treat Desert Rock equally to other similarly situated coal-fired power plants. In addition, as the Board discusses in Part III.B *infra*, reconsideration of IGCC in step 1 of the BACT analysis would be consistent with two federally issued PSD permits.

<sup>27</sup> In addition, a party must show that there is no rational basis for the government's differential treatment. *E.g.*, *Engquist*, 128 S.Ct. at 2153; *Olech*, 528 U.S. at 564; *Leib*, 558 F.3d at 1306-07. Because the Board concludes that Desert Rock fails to make the required showing that it has been intentionally treated differently from others similarly situated, the Board does not address the second issue.

federal and state prosecutors “simply does not raise equal protection concerns”); *Purze v. Vill. of Winthrop Harbor*, 286 F.3d 452, 455 (7th Cir. 2002) (finding no demonstration of similarly-situated individuals where comparison was, among other things, between decisions of two different zoning Board panels); *Harvey v. Anheuser-Busch, Inc.*, 38 F.3d 968, 972 (8th Cir. 1994) (“When different decision-makers are involved, two decisions are rarely ‘similarly situated in all relevant respects.’”). Consequently, to the extent Desert Rock attempts to challenge the Region’s PSD decision on equal protection grounds merely because it is different than the decisions of the three state permitting authorities, the Board rejects it.

Furthermore, the Supreme Court has explained that, with respect to government actions “which by their nature involve discretionary decisionmaking based on a vast array of subjective, individualized assessments,” the principles underlying equal protection are “not violated when one person is treated differently from others.” *Engquist*, 128 S. Ct. at 2154; *accord Leib*, 558 F.3d at 1307; *see also Moore*, 543 F.3d at 897-98. This is because “treating like individuals differently is an accepted consequence of the discretion granted.” *Engquist*, 128 S. Ct. at 2154. “In such situations, allowing a challenge based on the arbitrary singling out of a particular person would undermine the very discretion that [government] officials are entrusted to exercise.” *Id.* Such is the case here. The very nature of the analyses required by the PSD permitting process necessitates that permit issuers – EPA Regions and other approved governmental entities – make numerous subjective individualized assessments and discretionary decisions in their consideration and issuance of PSD permits. *See, e.g.*, 40 C.F.R. § 52.21 (c)-(p) (containing requirements for various analyses to be performed, including the analysis of ambient air increments, source impacts, additional impacts, and visibility). Thus, PSD permitting decisions clearly fall within the category of government actions that the Supreme Court has concluded do not trigger equal protection concerns. For this reason alone, Desert Rock’s claim must fail.

Moreover, even if the Board were to accept Desert Rock’s underlying premise that a “class of one” equal protection claim may successfully be raised in the context of EPA’s failure to require states issuing permits under somewhat different frameworks acting within their own discretion to make identical determinations to EPA’s, Desert Rock has failed to demonstrate how the three applicants it cites are indeed “similarly situated.” While Desert Rock baldly asserts that these applicants are similar, it has identified no factual evidence in the record to support its claim.<sup>28</sup> Thus, Desert Rock’s “class-of-one challenge never gets off the

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<sup>28</sup> Some limited information about the Georgia proposed facility and permit is included in the *Longleaf Energy Associates v. Friends of the Chattahoochee, Inc.*, Nos. A09A0387 & A09A0388, 2009 WL 1929192 (Ga. Ct. App. July 7, 2009), decision discussed *infra* Part III.B.3.e. This information  
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ground.” *Moore*, 543 F.3d at 898 (dismissing defendant’s “class of one” claim where defendant failed to provide any detailed factual information comparing himself with his coconspirators); *see also, e.g., Leib*, 558 F.3d at 1307 (dismissing class-of-one claim where the “complaint makes only the barest conclusory assertion” and “complete[ly] lacks factual detail regarding the ‘similarly situated’ requirement”).

(b) *A Voluntary Remand Does Not Violate Due Process*

Desert Rock’s due process claim – that a voluntary remand would effectively withdraw the Permit without hearing or review in violation of due process principles – is equally unavailing. Desert Rock’s arguments seemingly rely on its erroneous belief that the permit is already “final.”<sup>29</sup> *See* DR Opp’n Br. at 44 & n.6 (arguing that EPA “has already issued a ‘final’ PSD permit to Desert Rock,” thereby implicating the due process clause). The regulations state that a Region’s final permit decision is not “final agency action” where a petition for review has been filed with the Board. *See* 40 C.F.R. § 124.19(f)(1); *accord In re J&L Specialty Prods. Corp.*, 5 E.A.D. 31, 66 (EAB 1994). In fact, when a Region’s final permit decision is appealed, the permit does not become final agency action until either (1) the Board denies review, (2) the Board issues a decision on the merits that does not include a remand, or (3) the remand procedures are completed and the remand order did not require appeal of the remand decision to exhaust administrative remedies. 40 C.F.R. § 124.19(f)(1)(i)-(iii). Here, none of these three circumstances have occurred; thus, the permit is not yet final. Consequently, any arguments that rely on the “final” nature of the permit – such as Desert Rock’s due process arguments – are inapposite.<sup>30</sup> Desert Rock, therefore, has not demon-

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tion is in no way sufficient to determine whether the Georgia facility, as a factual matter, is similarly situated.

<sup>29</sup> As Desert Rock explains, to establish a due process claim, a petitioner must establish three things: (1) it has “a life, liberty, or property interest protected by the Due Process Clause”; (2) it was deprived of that protected interest within the meaning of the Due Process Clause; and (3) the government did not afford it adequate procedural rights prior to depriving it of that protected interest. DR Opp’n Br. at 42 (relying on *Hahn v. Star Bank*, 190 F.3d 708, 716 (6th Cir. 1999), *cert. denied*, 529 U.S. 1020 (2000)). Therefore, in order to successfully make its argument, Desert Rock must first demonstrate that it, in fact, has a property interest protected by the Due Process Clause. Desert Rock’s arguments thus hinge on its assertion that the Region’s (non-final) permit decision is that constitutionally protected property interest and that it was deprived of that interest.

<sup>30</sup> Desert Rock’s reliance on *In re General Electric Co.*, 4 E.A.D. 615 (EAB 1993), is groundless. That case focused on the proper procedures to handle future revisions to a RCRA permit that would be final at the time of the revisions. *See id.* at 628-29. Here, any potential revisions will be made before the permit is final.

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strated that the Region's motion deprives it of an interest protected by the Due Process Clause.

(iv) *Desert Rock's and ACCCE's "New Policy" Claims*

Finally, the Board notes that, in several places, Desert Rock argues that the Region may not change the Permit based on new, or future, policy. *See, e.g.*, DR Opp'n Br. at 8-9, 11-12, 18-20, 29-35. ACCCE raises similar concerns about the Region's rationale for requesting remand to reconsider PM<sub>2.5</sub> and IGCC. ACCCE Opp'n Br. at 8-10. At this stage, however, the Board cannot predict what the Region may, or may not, do on remand nor is it appropriate for the Board to provide a legal opinion on the merits of these theoretical outcomes. As the Board has noted in similar situations, "[t]o do so before the Region has actually relied on the theory in issuing the permit would, in effect, be offering an advisory opinion." *In re Mille Lacs Wastewater Treatment Fac. & Vineland Sewage Lagoons*, NPDES Appeal No. 01-16, at 12 (EAB Sept. 3, 2002) (Order Denying Review in Part and Remanding in Part); *In re Cavenham Forest Indus., Inc.*, 5 E.A.D. 722, 731 n.15 (EAB 1995) (declining to provide advisory opinion); *In re Multitrade Ltd. P'ship*, 3 E.A.D. 773, 777 (Adm'r 1992) (declining to speculate on outcome of planned permit changes that had not yet been made). Consequently, these arguments do not persuade the Board to deny the Region's remand request.

c. *The Region's ESA Compliance Strategy Raises Concerns the Board Cautioned Against in Indeck*

As the Board stated above, *see supra* Part III.A.3.a, it has serious concerns with the Region's past ESA compliance strategy for the Desert Rock Permit. The Region issued the Desert Rock Permit prior to completing the consultation required by ESA section 7(a)(2).<sup>31</sup> *See supra* Part III.A.1. In an attempt to address

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Several of ACCCE's arguments also appear to rely on its belief that the permit is "final." *See, e.g.*, ACCCE Opp'n Br. at 13 (referring to the permit as "final"). Consequently, these arguments are baseless as well. In a similar vein, ACCCE also mistakenly analogizes the permit process to a rulemaking. *See* ACCCE Opp'n Br. at 7-12. There are significant differences between the two administrative processes. The most important difference is the fact that, again, the Region's final permit decision is not final agency action where, as here, that permit is pending review by the Board. *See* 40 C.F.R. § 124.19(f)(1). Thus, arguments that the Region, in reconsidering its non-final permit decision, "should be held to same standard of review that any agency is when it decides to rescind a [final] rule," ACCCE Opp'n Br. at 7, are unconvincing.

<sup>31</sup> It is unclear from the participants' briefs whether the Region (or lead agency BIA) had truly even "initiated" consultation, as that term is meant under the ESA and its implementing regulations, at the time the Region issued the Permit. *Compare* NGO Suppl. Br. at 284 and CBD Petition at 5, 7 with Region's Response at 114-15; *see also* A.R. 80, at 1 (Letter from Timothy DeAsis, Acting Regional Director, BIA, to Jennifer Fowler-Propst, Field Supervisor, FWS (Apr. 30, 2007) (requesting formal

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this deficiency, the Region included a condition in the Permit that, among other things, prohibits Desert Rock from beginning construction at the Facility until the Region notifies the permittee that the Region has met its ESA responsibilities. *See* Permit at 2 (Condition II.A); *see also* Mot. for Vol. Remand at 9. Specifically, the Condition states:

Construction under this permit may not commence until EPA notifies the Permittee that it has satisfied any consultation obligations under Section 7(a)(2) of the Endangered Species Act with respect to issuance of the permit. EPA shall have the power to reopen and amend the permit, or request that the Permittee amend its permit application, to address any alternatives, conservation measures, reasonable and prudent measures, or terms and conditions deemed by EPA to be appropriate as a result of the ESA consultation process.

Permit at 2 (Condition II.A); *see also* Mot. for Vol. Remand at 9. In its most recent motion, the Region admits that FWS has recently concluded that the Permit may “adversely affect” at least one endangered species, indicating that the required ESA consultation is still ongoing. Mot. for Vol. Remand at 10. Based on these facts and in light of ESA requirements and Board precedent, the Board has significant concerns about the Region’s inclusion of Condition II.A in the Permit. The Board therefore believes the Region’s action requesting remand on this ground is well-taken. Because of the significance and complexity of this issue, the Board reviews it in some detail below to assist the Agency on remand and in other permit cases.

(i) *Relevant ESA Statutory and Regulatory Provisions*

Congress enacted the ESA in 1973 to provide for the conservation of endangered and threatened fish, wildlife, and plants and their natural habitats. ESA § 2, 16 U.S.C. § 1531. In order to accomplish this goal, the statute requires the Secretaries of the Interior and Commerce to determine which species are endangered or threatened – i.e., to make a “list” of such species – and to designate the critical

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consultation)); A.R. 82, at 1 (Letter from Wally Murphy, Supervisor, N.M. Ecological Field Services Field Office, FWS, to Regional Director, Navajo Regional Office, BIA (July 2, 2007) (stating that the FWS had not yet received all the necessary information to initiate formal consultation)). Whether or not consultation had begun at the time the permit was issued does not affect our discussion, especially now that it appears that some form of consultation has been initiated.



habitat for such listed species.<sup>32</sup> ESA § 4(a), 16 U.S.C. § 1533(a).

The ESA also imposes a number of substantive and procedural obligations on all federal agencies, including EPA. *See, e.g.*, ESA § 7(a)(1), (a)(2), 9(a)(1), (a)(2), 16 U.S.C. § 1536(a)(1), (a)(2), 1538(a)(1), (a)(2); *see also* 50 C.F.R. § 402.06(a). Of particular relevance is section 7(a)(2), which requires that:

Each federal agency shall, in consultation with and with assistance of the Secretary, insure that any action authorized, funded, or carried out by such agency \* \* \* is not likely to jeopardize the continued existence of any endangered species or threatened species or result in the destruction or adverse modification of [critical] habitat of such species \* \* \* .

ESA § 7(a)(2), 16 U.S.C. § 1536(a)(2). Significantly, the definition of agency “action” is broad and includes “the granting of licenses, contracts, leases, easements, rights-of-way, [or] permits.” 50 C.F.R. § 402.02 (emphasis added); *accord Env'tl. Prot. Info. Ctr. (“EPIC”) v. Simpson Timber Co.*, 255 F.3d 1073, 1075 (9th Cir. 2001); *In re Indeck-Elwood, LLC*, 13 E.A.D. 126, 195 (EAB 2006); *In re Ash Grove Cement Co.*, 7 E.A.D. 387, 428 & n.34 (EAB 1997); *In re Dos Republicas Res. Co.*, 6 E.A.D. 643, 649 (EAB 1996). Thus, section 7(a)(2) imposes a substantive duty on federal agencies to ensure that none of their actions – including the issuance of a permit – is likely to jeopardize listed species or destroy or adversely modify the critical habitat of such species. *See* 51 Fed. Reg. at 19,926; *see also Indeck*, 13 E.A.D. at 195-96; *In re Phelps Dodge Corp.*, 10 E.A.D. 460, 485 (EAB 2002); *Dos Republicas*, 6 E.A.D. at 649, 666.

To assure that agencies meet this substantive obligation, section 7(a)(2) also imposes a procedural duty on federal agencies – to consult with FWS prior to engaging in a discretionary action that “may affect listed species or critical habitat.”<sup>33</sup> 50 C.F.R. § 402.14(a); *Sierra Club v. Babbitt*, 65 F.3d 1502, 1504-05

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<sup>32</sup> The two secretaries generally share responsibilities under the ESA. *See* ESA § 3(15), 16 U.S.C. § 1532(15) (definition of “Secretary”); 50 C.F.R. § 402.01(b); ESA Consultation Regulations, 51 Fed. Reg. 19,926, 19,926 (June 3, 1986). More particularly, the Secretary of the Interior acts through the U.S. Fish and Wildlife Service (“FWS”) to implement ESA requirements with respect to terrestrial species, whereas the Secretary of Commerce, through the National Oceanic and Atmospheric Administration’s National Marine Fisheries Service, handles responsibilities for marine species. 50 C.F.R. § 401.01(b); 51 Fed. Reg. at 19,926. Because the species at issue in this case are not marine species, this opinion will use the term “FWS” or “Service” hereinafter when referring to the duties or responsibilities of the “Secretary” or the “Service[s].”

<sup>33</sup> As the Board explained in *Indeck*, “[t]he term ‘may affect’ is broadly construed by FWS to include ‘[a]ny possible effect, whether beneficial, benign, adverse, or of an undetermined character,’

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(9th Cir. 1995); *Indeck*, 13 E.A.D. at 196-97. If the agency determines that its proposed action, such as issuing a permit, may affect a listed species or its critical habitat, then formal consultation is required, with limited exceptions seemingly not relevant here.<sup>34</sup> 50 C.F.R. § 402.14(a); *Indeck*, at 196. For certain types of projects, the agency engaging in a federal "action" must prepare a biological assessment ("BA") and submit it to FWS, although agencies may voluntarily prepare a BA even when it is not required. ESA § 7(c)(1), 16 U.S.C. § 1536(c)(1); 50 C.F.R. § 402.12, .14(c)(5); *Phelps Dodge*, 10 E.A.D. at 486 & n.23; *Dos Republicas*, 6 E.A.D. at 666 & n.68.

Upon conclusion of the agencies' formal consultation, FWS prepares a biological opinion evaluating the potential effect of the action on the protected species. ESA § 7(b)(3)(A), 16 U.S.C. § 1536(b)(3)(A); 50 C.F.R. § 402.14(l); *Phelps Dodge*, 10 E.A.D. at 487; *Dos Republicas*, 6 E.A.D. at 653 n.40, 666. If FWS finds jeopardy or adverse modification to critical habitat, it recommends reasonable and prudent alternatives to the action agency's proposed action that can be taken by the action agency or applicant and that would not violate section 7(a)(2). ESA § 7(b)(3)(A), 16 U.S.C. § 1536(b)(3)(A); *Phelps Dodge*, 10 E.A.D. at 487; *see also Dos Republicas*, 6 E.A.D. at 654 & n.43. On the other hand, if the Service's biological opinion concludes that the proposed activity is not likely to jeopardize an endangered or threatened species or adversely modify critical habitat, the proposed action is generally permitted. *E.g.*, *EPIC*, 255 F.3d at 1076; *see also Dos Republicas*, 6 E.A.D. at 653 & n.40, 668-69. Even in the case of a "no jeopardy" biological opinion by the Service, FWS still may provide discretionary, non-binding conservation recommendations, which the action agency may consider and implement in its final action.<sup>35</sup> 50 C.F.R. § 402.14(g)(6), (j), .15; *Natural Res. Def. Council v. Houston*, 146 F.3d 1118, 1129 (9th Cir. 1998). Finally

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and is thus easily triggered." 13 E.A.D. at 196 (quoting 51 Fed. Reg. at 19,926). Additionally, as the Board emphasized in *Indeck*, the ESA implementing regulations indicate that an agency should review its actions "'at the earliest possible time' to determine whether the low 'may affect' threshold is met," thereby triggering the need "to initiate some type of consultation." *Indeck*, 13 E.A.D. at 197 (quoting 50 C.F.R. § 402.14(a)); *see also* 50 C.F.R. § 402.11(b) (mentioning "early consultation").

<sup>34</sup> The regulations list several exceptions, including the possibility that, through the informal consultation process or as a result of the preparation of a biological assessment, the federal agency may, with the written concurrence of the Service, conclude that its action will not likely adversely affect listed species or critical habitat. 50 C.F.R. § 402.14(b)(1)-(2); *see also Indeck*, 13 E.A.D. at 197 n.136; *Ash Grove*, 7 E.A.D. at 429. In addition, if the agency determines that its proposed action will have "no effect" on any federally-listed species or critical habitat, the federal agency need not formally consult with the Service, and the section 7 process terminates. 50 C.F.R. § 402.14(a); *Indeck*, 13 E.A.D. at 197 n.134; *Phelps Dodge*, 10 E.A.D. at 486. From the Region's recent motion, it appears that none of these options is applicable here. *See* Mot. for Vol. Remand at 10 (noting that a Biological Assessment has been prepared and that the FWS has stated that there may be adverse effects).

<sup>35</sup> For a more detailed discussion of the consultation and post-consultation process, *see Phelps Dodge*, 10 E.A.D. at 485-88, and *Dos Republicas*, 6 E.A.D. at 649, 652-58 & nn.40-43, 666-74.

and most importantly, “[a]fter meaningful consultation” with the Service, it is the federal agency who “possesses the ultimate decisionmaking authority to determine whether it may proceed with an action.” *Pac. Rivers Council v. Thomas*, 936 F.Supp. 738, 744 (D. Idaho 1996); *accord* 50 C.F.R. § 402.15; *Roosevelt Campobello Int’l Park Comm’n v. EPA*, 684 F.2d 1041, 1049 (1st Cir. 1982); *Phelps Dodge*, 10 E.A.D. at 487; *Dos Republicas*, 6 E.A.D. at 666 n.69.

Significantly, once consultation with FWS is initiated, ESA section 7(d) also applies to the federal action agency and the permit applicant. Section 7(d) prohibits both entities from “mak[ing] any irreversible or irretrievable commitment of resources with respect to the agency action which has the effect of foreclosing the formulation or implementation of any reasonable and prudent alternative measures.” ESA § 7(d), 16 U.S.C. § 1536(d); *accord* 50 C.F.R. § 402.09; *Indeck*, 13 E.A.D. at 197.

(ii) *Indeck and the Question of the Appropriate Timing of Consultation*

In 2004, before the final Desert Rock Permit was issued, the Board, in *Indeck*, considered several ESA issues in the PSD permitting context, including the proper timing of any required consultation. 13 E.A.D. at 205-11. In that case, petitioners claimed that Region 5 had failed to comply with the ESA by initiating consultation with FWS after the Region had issued a final decision. *See id.* 13 E.A.D. at 201 & nn.143-44. While the permit was on appeal before the Board, Region 5 and FWS initiated and completed an informal consultation,<sup>36</sup> and no action was taken with respect to the permit as a result of the ESA consultation process. 13 E.A.D. at 209. Notably, the Permit at issue in *Indeck* did not contain a condition similar to that in the present case.

In considering the *Indeck* Petitioners’ ESA issues, the Board discussed, at length, the question of *when* the Agency must comply with ESA requirements. The Board stated:

[W]hile neither the ESA nor its implementing regulations specify when the consultation process needs to be completed vis-a-vis the associated agency action, the statute does prohibit an agency from, “mak[ing] any irreversible or irretrievable commitment of resources with respect to the agency action which has the effect of foreclosing the formulation or implementation of any reasonable and pru-

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<sup>36</sup> The Region concluded that *Indeck*’s permit was “not likely to adversely affect” any federally-listed species or the designated habitat of such species. *Indeck*, 13 E.A.D. at 199. FWS concurred in writing with this conclusion, thereby completing the informal consultation. *Id.*

dent alternative measures,” after consultation with the Service is initiated. ESA § 7(d), 16 U.S.C. § 1536(d). In the ordinary course, the issuance of a final PSD permit would appear to be the point at which the permitting agency has irretrievably committed itself with respect to the discrete act of permitting a given activity. Accordingly, to avoid violating this requirement, the Agency should complete the ESA process prior to the issuance of the final permit. This ensures that, if FWS recommends any changes to the permit during the consultation process or, alternatively, if EPA decides to add or amend permit conditions based on any information or findings that arise during the ESA consultation process, such changes may be implemented in the final PSD permit.

*Indeck*, 13 E.A.D. at 206-07 (footnotes and citations omitted). Consequently, the Board concluded that it would “expect ESA consultation [to] ordinarily be completed, *at the very latest, prior to issuance of the permit* and, optimally, prior to the comment period on the permit, where the flexibility to address ESA concerns is the greatest.” *Id.* 13 E.A.D. at 209 (emphasis added); *see also Ash Grove*, 7 E.A.D. at 429. In other words, *the ESA process should be completed at the time a region issues its final permit decision.*<sup>37</sup>

The Board in *Indeck*, however, did determine that there was one exception to this general timing rule. Because the permitting regulations effectively postpone “final agency action” when a final permit decision is appealed, *id.* at 111 n.150, 112-13; *see* 40 C.F.R. § 124.19(f)(1), if the ESA process is completed during the appeal, “there [still] remains legal capacity to adjust the terms of the permit.”<sup>38</sup> *Indeck*, 13 E.A.D. at 208. Thus, in this special situation, as a “technical matter,” the completion of an ESA consultation during an appeal “me[ets] minimum standards.” *Id.* at 112.

While recognizing that this exception applied under the circumstances in *Indeck*, the Board pointed out that “[b]y all appearances, had an appeal not been taken, and consultation not been undertaken during the pendency of this appeal, this permit would have gone final in dereliction of legally binding ESA requirements.” *Indeck*, 13 E.A.D. at 209; *see also Ash Grove*, 7 E.A.D. at 429. The Board

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<sup>37</sup> The Board emphasizes this statement because permit conditions have been included in more than one recently issued final permit suggesting that regions have not consistently followed the Board’s *Indeck* decision. The Region also made this observation in its motion. *See* Mot. for Vol. Remand at 13-14.

<sup>38</sup> In other words, if changes are necessary based on the consultation, the permit can be remanded to the region to implement the needed modifications.

emphasized that the Region's approach to meeting its ESA requirements was problematic, stating that "an ESA compliance strategy that acknowledges ESA only in the event of an appeal is not a compliance strategy at all, in that it would tolerate an ESA violation whenever an appeal is not taken." *Indeck*, 13 E.A.D. at 209; *see also id.*, 13 E.A.D. at 208 n.154 ("[W]aiting to consult as late as during the pendency of a PSD appeal \* \* \* is prudentially inadvisable.").

(iii) *The Region's Approach to ESA Compliance Here*

In this case, in issuing the final permit, the Region appears to have taken one more step down the slippery slope the Board cautioned against in *Indeck*. Not only did the Region issue its permit decision without completing consultation, it issued the Permit with a condition essentially declaring that ESA requirements had not been met at the time the permit was issued, with the intention of relying on future permit modifications to "fix" or "re-do" the Permit, if changes were found to be necessary. The Board believes the Region's reliance upon this condition and its past ESA compliance strategy for the Desert Rock permit in general raise significant concerns.

The Board concludes that a condition like the one included in the Desert Rock Permit does not obviate the concerns the Board highlighted in *Indeck*. In *Indeck*, the Board specifically stated that reliance on the permit modification process to change an already-issued permit is problematic because "[t]he fact that a permit once issued may subsequently be amended does not diminish the irretrievable nature of the decision to issue the permit as amendments are discrete actions independent from the decision to issue the permit in the first instance." *Indeck*, 13 E.A.D. at 207 n.151. This statement strongly cautioned against relying on a later permit amendment to meet the ESA requirements for the permit's initial issuance – the very strategy the Region planned to follow in this case.

Second, by deferring its ESA compliance until some uncertain time after permit issuance and relying on a permit condition to allow it to "redo" the permit later to meet any ESA requirements found to be necessary, the Region arguably turned the statute on its head. Although the federal courts' approach to after-the-fact ESA compliance is not entirely consistent,<sup>39</sup> the Ninth Circuit, in two cases with facts and circumstances similar to those in the present case, found a strategy like the Region's to be flawed and violative of the ESA.

In *Conner v. Burford*, 848 F.2d 1441, 1454-55 (9th Cir. 1988), *cert. denied sub nom. Sun Exploration & Prod. Co. v. Lujan*, 489 U.S. 1012 (1989), the Bureau of Land Management issued leases prior to the FWS's preparation of a comprehensive biological opinion covering the effects of leasing and post-leasing ac-

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<sup>39</sup> *See infra* note 40.

tivities, but included stipulations in the leases that essentially provided that future restrictions might be necessary based on the federal agency's future examination of ESA impacts. The Ninth Circuit concluded that this strategy – which it termed an “incremental-step consultation” – was an attempt “to carve out a judicial exception to ESA’s clear mandate that a comprehensive biological opinion \* \* \* be completed before initiation of the agency action.” *Id.* at 1455. The court declined “this invitation to amend the ESA.” *Id.* The Court also noted that “[s]ection 7(d) does not amend section 7(a) to read that a comprehensive biological opinion is not required before the initiation of agency action so long as there is no irreversible or irretrievable commitment of resources. Rather, section 7(d) clarifies the requirements of 7(a), ensuring that the status quo will be maintained during the consultation process.” *Id.* at 1455 n.34 (citation omitted); *see also Pac. Rivers Council v. Thomas*, 30 F.3d 1050, 1056 (9th Cir. 1994), *cert. denied*, 514 U.S. 1082 (1995) (reaffirming statements made in *Conner*); *Pac. Rivers Council v. Thomas*, 873 F.Supp. 365, 371 (D. Idaho 1995) (reiterating *Conner*).

Similarly, in *Natural Resources Defense Council v. Houston*, 146 F.3d 1118, 1127 (9th Cir. 1998), a case even more analogous to the situation here, the Bureau of Reclamation issued water contracts that contained a clause allowing “contract modification pursuant to environmental review.” Defendants argued that even if the contracts constituted an “irreversible and irretrievable commitment of resources,” the contractual savings clause “prevented the foreclosure of reasonable and prudent alternatives, and, therefore, § 7(d) was not violated.” *Id.* at 1128. The Ninth Circuit disagreed, concluding: “We do not think an agency should be permitted to skirt the procedural requirements of § 7(d) by including such a catchall savings clause in illegally executed contracts.” *Id.* Consequently, the Court held that rescission of the contracts was an appropriate remedy.<sup>40</sup> *Id.* at 1129; *see also*

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<sup>40</sup> Other courts have concluded, also in a non-PSD context, that a delayed ESA strategy did not violate sections 7(a) and/or 7(d) of the ESA. *See, e.g., N. Slope Borough v. Andrus*, 642 F.2d 589, 610-11 (D.C. Cir. 1980) (allowing oil and gas lease sales to proceed under the Outer Continental Shelf Lands Act (“OCSLA”) despite incomplete consultation over all future impacts), *aff’ing in part, rev’ing in part*, 486 F.Supp. 332 (D.D.C. 1980); *Wyo. Outdoor Council v. Bosworth*, 284 F.Supp.2d 81, 90-93 (D.D.C. 2003) (concluding that consultation need not be initiated, and thus challenge was not yet ripe, where agency issued oil and gas lease but retained authority under agency regulations and lease stipulations to preclude partial or full use of leased property if required by agency’s later ESA consideration; lessee, in next stage of process, was required to submit application to conduct surface-disturbing activity on property); *No Oilport! v. Carter*, 520 F.Supp. 334, 364-66 (W.D. Wash. 1981) (concluding that issuance of right-of-way permit prior to completion of biological assessment did not violate the ESA where permit restricted initiation of construction until the agency issued a Notice to Proceed and the notice was conditioned on compliance with the ESA). Significantly, several courts have suggested that the reasoning in *North Slope* and other OCSLA cases was based on the nature of the statute under which the agency was operating, which itself included an incremental step approach. *Conner v. Burford*, 848 F.2d at 1455-57; *Nat’l Wildlife Fed’n v. Brownlee*, 402 F.Supp.2d 1, 10 n.15 (D.D.C. 2005). Thus, the relevance of OCSLA-based cases, and any other cases in which the underlying statute and regulations require the agency to take an incremental step approach, in the CAA/PSD context is ques-

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*Pac. Rivers Council v. Thomas*, 936 F.Supp. 738, 746-51 (D. Idaho 1996) (declining to allow the U.S. Forest Service to take an action for which it was currently in consultation with the Service based on the agency's 7(d) conclusion that it would comply with 7(d)). *But see Sw. Ctr. for Biological Diversity v. U.S. Forest Serv.*, 82 F.Supp.2d 1070, 1080 (D. Ariz. 2000) (explicitly disagreeing with the *Pacific Rivers* decision).

A third concern the Board raises about a permit with a condition like Condition II.A is that, should the permit indeed become "final agency action" prior to completion of consultation as the terms of the condition intend,<sup>41</sup> the very fact that the permit is "final" will likely impact the consultation process with the Service, who may unsurprisingly assume that modifications to the permit would be difficult to implement.<sup>42</sup> The *Houston* court remarked on this very problem when it held that rescission was appropriate even though the FWS had ultimately issued a 'no jeopardy' Biological Opinion after the issuance of the contracts, stating that "if the Biological Opinion had been rendered before the contracts were executed, the FWS would have had more flexibility to make, and the [action agency] to implement, suggested modifications to the proposed contracts." 146 F.3d at 1129; *cf. In re Phoenix Constr. Servs., Inc.*, 11 E.A.D. 379, 407 n.63 (EAB 2004) ("We do not believe that after-the-fact permits always reflect what the [agency] would have initially granted \* \* \* because the after-the-fact permit may have been issued as a part of a negotiation or settlement between the regulatory agencies and the 'permittee.'"). The Ninth Circuit further explained: "Even where there is a 'no jeopardy' Biological Opinion, the Service may make non-binding conservation recommendations. 50 C.F.R. § 402.14(g)(6), (j). The failure to respect the process

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tionable. Moreover, because the Board is part of the agency, it is in a different position than the federal courts and can obviate the problem of mooted issues and remedies by ensuring that ESA obligations are completed prior to permit issuance and that any necessary consultation is meaningful.

<sup>41</sup> Such a permit becoming "final agency action" presupposes that either (1) the permit is not appealed or (2) the Board denies review of the permit despite the inclusion of the condition in the permit.

<sup>42</sup> *Indeck* presented different facts. In *Indeck*, the Board disagreed with Petitioners' arguments that "FWS's ability to suggest modifications to the permit was curtailed because the consultation occurred after the permit had been issued and that the integrity of the consultation process was thus compromised." *Indeck*, 13 E.A.D. at 208 n.156. In that case, however, the Service had explicitly stated that it stood by both the informal consultation process that had taken place and the conclusions that had been made during that process. *Id.* Moreover, in *Indeck* the consultation occurred while the appeal was ongoing and before the permit became final agency action. Additionally, the consultation in *Indeck* was an informal one, whereas the present consultation is apparently formal, *see supra* note 31, which increases the likelihood that FWS may provide the Agency with reasonable and prudent alternatives, or at least non-binding conservation measures. Finally, our rationale here is also based on Condition II.A's underlying premise that the permit will be *final agency action* at the time the biological opinion is drafted and modifications to the permit are implemented. The current situation, therefore, more closely resembles the circumstances in *Houston* rather than those in *Indeck*.

mandated by law cannot be corrected with post-hoc assessments of a done deal.” *Houston*, 146 F.3d at 1129.

Based on the aforementioned reasons, the Board wholeheartedly agrees that the Region should reconsider its ESA compliance strategy for the Permit, including its reliance on Condition II.A.<sup>43</sup> In light of this conclusion, granting the Region’s voluntary remand request is more than appropriate here. The Board acknowledges, however, that it does have the discretion to instead stay the case and await the Region’s completion of its ESA compliance activities, as was essentially done in *Indeck*.<sup>44</sup> *Indeck-Elwood* 2004 Stay Order at 6-8; cf. *Anchor Line Ltd. v. Fed. Mar. Comm’n*, 299 F.2d 124, 125 (D.C. Cir.) (explaining that agency may either move for a remand or request a stay when it seeks to reconsider its action), *cert. denied*, 370 U.S. 922 (1962). The Board declines to stay the case rather than remand for two reasons.

First, *Indeck* was based on exceptional circumstances that explained, in large part, the belated ESA compliance: in that case the Region had not initiated consultation prior to IEPA’s issuance of the permit because there had been a question about whether, as a legal matter, the ESA requirements even applied to a permit issued by a delegated state. See *Indeck*, 13 E.A.D. at 209. After IEPA’s issuance of *Indeck*’s permit, the Agency concluded that they did apply. *Id.* at 102, 105. Here, there is no such exceptional reason for failure to complete consultation in a timely fashion, and *Indeck* was decided long before the Region issued the Desert Rock Permit.<sup>45</sup> In this case, the Region – and the applicant<sup>46</sup> – have had

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<sup>43</sup> The Board also notes that it is far from clear how, or under what authority, the Region would accomplish an uncharted and after-the-fact PSD permit modification such as that envisioned by Condition II.A or, moreover, whether any such permit modification would trigger the need for public comment.

<sup>44</sup> Because any amendments to the Permit that the Region deems necessary as a result of the consultation and compliance with its ESA obligations could potentially impact any aspect of the Permit, it is appropriate to grant a remand of the entire Permit on ESA grounds. See *Indeck-Elwood* 2004 Stay Order at 8 (explaining that it is impossible to predict which conditions of the permit might change as a result of the ESA consultation process).

<sup>45</sup> The participants acknowledge that Desert Rock filed a complaint in federal district court alleging that the Region had failed to make a timely PSD permit decision. *E.g.*, DR Opp’n Br. at 2; Cons. Pet’rs Reply at 5 n.8. Under some circumstances, the fact that an applicant filed a complaint in federal district court alleging improper delay in issuing the permit might be considered an exceptional circumstance. The Board, however, declines to so conclude under the facts of this particular case. As indicated by our discussion above, it is perplexing why the ESA process took so long here and why neither the Agency nor the applicant moved the formal consultation process along earlier. See *infra* note 46 and accompanying text.

<sup>46</sup> While responsibility for ESA compliance rests on the Agency’s shoulders, as the Board noted in *Indeck*, the statute and regulations authorize the applicant to play a proactive role in the process. For example, the regulations provide that “[i]f a prospective applicant has reason to believe  
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several years to initiate and conclude the ESA process.

In addition, here, unlike in *Indeck*, FWS has indicated that there may well be adverse effects, apparently resulting in a formal consultation, not an informal one.<sup>47</sup> As noted earlier, in *Indeck*, the FWS did not recommend changes to the permit. See 13 E.A.D. at 206. The fact that the Region and FWS are undergoing formal consultation in this case renders it more likely that the present consultation will result in modifications to the Permit. Should the Permit be stayed and should modifications be needed, the Board would have to remand the Permit at a later date anyway. Thus, the Board, in its discretion, believes it is appropriate to grant the Region's remand request at this time.

#### *4. Summary of Conclusions Regarding Voluntary Remand Motion*

In sum, the Board concludes that 40 C.F.R. § 124.19(d) neither constrains a region from requesting a voluntary remand after the Board grants review nor proscribes the Board from granting a voluntary remand at any time. Consequently, the Region's motion for voluntary remand is not prohibited. The Board further concludes that, under the facts and circumstances of this case, granting the Region's motion for voluntary remand at this time is warranted. The Region has shown good cause for its motion, explaining that it wishes to reconsider some elements of its permit decision and representing that it may make changes to one or more permit conditions. Moreover, because the Board has substantial concerns with the Region's approach to ESA compliance and because this is one of the issues the Region intends to revisit, the Board believes voluntary remand is particularly appropriate in this case. Additionally, as explained below, one of the issues the Region wishes to reconsider is an issue on which the Board concludes, on independent grounds, that remand of the entire permit is appropriate. Based on these factors, the Board concludes that granting the motion would best serve the interests of administrative and judicial efficiency.

#### *B. Independent Grounds for Remand of the Entire Permit: the Region's IGCC Analysis*

In addition to the Board's determination that granting the Region's motion for voluntary remand is appropriate, the Board finds independent grounds for remanding the entire Permit. The Board granted review in this matter, in part, because upon a preliminary review of the issues, the Board had very significant concerns about certain aspects of the Permit. The Region's IGCC analysis was

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that the prospective action may affect listed species or critical habitat, it may request the Federal agency to enter into early consultation with the Service." 50 C.F.R. § 402.11(b).

<sup>47</sup> See *supra* note 31, referring to BIA letter to FWS requesting formal consultation.



one of the issues about which the Board was most concerned.<sup>48</sup> Upon review of the administrative record, the Board concludes that the record inadequately supports the Region's decision not to consider IGCC in step 1 of its BACT analysis. Neither additional briefing nor further argument would resolve the problems the Board has identified in the record.<sup>49</sup>

Furthermore, because the Region's IGCC determination is essentially a BACT step 1 issue, reconsideration of the issue could have overarching impacts on the rest of the Region's BACT analysis and consequently on a number of the Permit conditions.<sup>50</sup> While the Board could require the Region to file its final surreply brief, hold oral argument, complete final review of all approximately thirteen issues raised by Petitioners, and *then* remand the permit, the Board believes it appropriate in this case to remand the permit at this time based on this critical issue. Such a step should ultimately provide a speedier resolution of the Desert Rock permitting process. Moreover, because the Board's review of the carbon dioxide issue has been stayed pursuant to the Board's January 22, 2009 Order and because of the direction on remand related to the ESA issue highlighted in Part III.A.3.c, judicial efficiency would best be served in this case by remanding the entire permit rather than sending it back in a piecemeal fashion or alterna-

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<sup>48</sup> Three Petitioners – New Mexico, NGO Petitioners, and Ms. Glustrom – challenged the Region's BACT analysis, contending that the Region made numerous errors in setting the BACT limits for several pollutants at the Facility. *E.g.*, N.M. Pet. for Review and Suppl. Br. at 18-30; NGO Suppl. Br. at 72-124; Glustrom Pet. for Review at 11-37. The first two, New Mexico and NGO Petitioners, specifically questioned the Region's failure to consider IGCC under step 1 of the BACT analysis. N.M. Suppl. Br. at 18-22; NGO Suppl. Br. at 72, 75-78; Cons. Pet'rs Reply Br. at 1-5. More specifically, New Mexico and NGO Petitioners claimed that IGCC would provide "the maximum degree of emissions reductions for several of the air pollutants emitted by [the Desert Rock Facility]." NGO Suppl. Br. at 102; *see also* N.M. Suppl. Br. at 17. They argued that sections 165(a)(4) and 169(3) of the Act require EPA to consider "production processes and available methods" including "fuel cleaning" and "innovative fuel combustion techniques" in the BACT analysis and that IGCC falls squarely within the meaning of those terms. NGO Suppl. Br. at 72, 75-78; N.M. Suppl. Br. at 18-22. Petitioners pointed to the legislative history of the term "innovative fuel combustion process," in support of their arguments. N.M. Suppl. Br. at 19-20; 21-23; NGO Suppl. Br. at 94-97; Cons. Pet'rs Reply Br. at 2. Petitioners also challenged the Region's application of the "redefinition of the source" policy in this case. New Mexico argued that the Region's determination that IGCC would redefine the source is clearly erroneous and "bad policy," N.M. Suppl. Br. at 20, and stretches the "redefine the source" principle beyond Board precedent, *id.* at 24. NGO Petitioners similarly asserted that the Region's position is contrary to law and "disregards the statutory limits on EPA's discretion as affirmed by the courts." NGO Suppl. Br. at 78; *accord* Cons. Pet'rs Reply Br. at 1-5.

<sup>49</sup> Two parties requested oral argument. *See* State of New Mexico's Petition for Review and Request for Oral Argument at 2, 4-5; Desert Rock's Response to Petitions for Review at 275.

<sup>50</sup> Notably, Petitioners raised a number of other BACT-related issues. *See, e.g.*, NGO Suppl. Br. at 112-24, 152-90.

tively issuing stays in a piecemeal fashion.<sup>51</sup>

In considering this issue, the Board first outlines its standard of review in permit appeal cases. The Board next describes the statutory and regulatory requirements for BACT as well as the method permit issuers often use to determine BACT. The Board then generally describes IGCC. Next, the Board describes the history of the “redefining the source policy,” which the Region relied on to exclude IGCC from further consideration as BACT. Finally, the Board analyzes the Region’s consideration of IGCC under the statutes, regulations, policy, and Board precedent.

### 1. *Standard of Review*

Part 124 contains the procedures governing both the Agency’s processing of permit applications and appeals of those permitting decisions. *See generally* 40 C.F.R. pt. 124. In reviewing a permit under part 124 for which it has granted review, the Board looks at whether the permit issuer based the permit on a clearly erroneous finding of fact or conclusion of law. 40 C.F.R. § 124.19(a)(1); *In re Deseret Power Elec. Coop.*, 14 E.A.D. 212, 226; *In re Dominion Energy Brayton Point, LLC*, 12 E.A.D. 490, 509 (EAB 2006); *In re Inter-Power of N.Y., Inc.*, 5 E.A.D. 130, 144 (EAB 1994). In addition, in its discretion, the Board may evaluate whether the permit issuer abused its discretion or may review important policy considerations. 40 C.F.R. § 124.19(a)(2); *Dominion*, 12 E.A.D. at 509; *Deseret*, 14 E.A.D. at 226; *see also, e.g., In re GSX Servs. of S.C., Inc.*, 4 E.A.D. 451, 454 (EAB 1992) (remanding permit based on abuse of discretion); *In re Chem. Waste Mgmt.*, 2 E.A.D. 575, 577 (Adm’r 1988) (granting review and remanding case to region based on policy considerations on issue involving region’s exercise of discretion). As a preliminary procedural matter, the Board requires that a petitioner describe each objection it is raising and explain why the permit issuer’s response to the petitioner’s comments during the comment period is clearly erro-

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<sup>51</sup> The Board emphasizes that its action should not be read to suggest that the Board has concluded that there are no other problems with the Permit. Instead, because resolution of this particular issue – the consideration of IGCC in the Region’s BACT analysis – could impact multiple Permit conditions, the Board considered it first.

Mindful of the time-sensitive nature of PSD permitting and in order to expedite any future review of the Permit, the Board encourages the Region on remand to reexamine several other aspects of its permitting decision to ensure that the administrative record adequately supports its decision. In particular, the Region may want to examine the basis for its determination that emissions from the facility will not cause or contribute to an exceedance of the ozone NAAQS. The Board suggests that the Region ensure that it adequately responds to comments about the actual monitored ozone levels in the area as well as comments regarding the flaws in the model EPA used and that it clearly explains its rationale for relying on a model that appears inconsistent with actual monitoring data. The Region may also want to reexamine the record supporting its visibility determination to ensure that the Federal Land Managers did not make any findings of adverse impacts and to ensure that any permit conditions the Region relies upon to support its visibility determinations are enforceable.

neous or otherwise warrants consideration (e.g., is an abuse of discretion). *E.g.*, *Deseret*, 14 E.A.D. at 226; *In re Peabody W. Coal Co.*, 12 E.A.D. 22, 33 (EAB 2005); *Indeck*, 13 E.A.D. 126, 143.

A petitioner challenging an issue that is fundamentally technical in nature bears a particularly heavy burden because the Board generally defers to the permit issuer on questions of technical judgment. *E.g.*, *Dominion*, 12 E.A.D. at 510; *Peabody*, 12 E.A.D. at 33. Nevertheless, the Board has stated that BACT determinations, which are generally technical in nature, are one of the most critical elements in the PSD permitting process and thus “should be well documented in the record, and any decision to eliminate a control option should be adequately explained and justified.” *Indeck*, 13 E.A.D. at 134 (citing *In re Knauf Fiber Glass GmbH*, 8 E.A.D. 121, 131(EAB 1999)); accord *In re Newmont Nev. Energy Inv., LLC*, 12 E.A.D. 429, 442 (EAB 2005); *In re Gen. Motors, Inc.*, 10 E.A.D. 360, 363 (EAB 2002). Consequently, in evaluating a BACT determination on appeal, the Board looks at whether the determination “reflects ‘considered judgment’ on the part of the permitting authority,” as documented in the record. *Knauf*, 8 E.A.D. at 132; accord *In re Masonite Corp.*, 5 E.A.D. 551, 566-69 (EAB 1994) (analyses incomplete); *In re Austin Powder Co.*, 6 E.A.D. 713, 720 (EAB 1997); *GSX Servs.*, 4 E.A.D. at 454. The Board has remanded permits where the permit issuer’s BACT analyses were incomplete or the rationale was unclear. *E.g.*, *Knauf*, 8 E.A.D. at 134, 140 (BACT rationale unclear); *Masonite*, 5 E.A.D. at 566-69 (BACT analyses incomplete); see also *In re NE Hub Partners, L.P.*, 7 E.A.D. 561, 568 (EAB 1998) (noting that the Board will not hesitate to order a remand on a technical issue “when a Region’s decision \* \* \* is illogical or inadequately supported by the record”); *In re Ash Grove Cement Co.*, 7 E.A.D. 387, 417-19 (EAB 1977) (remanding RCRA permit limits where region’s ultimate choice did not follow logically from its chosen method, a method which region had discretion in selecting); *Austin Powder*, 6 E.A.D. at 720 (remanding RCRA permit where rationale unclear); *GSX Servs.*, 4 E.A.D. at 454 (remanding RCRA permit because, even though establishing the permit term was an exercise of discretion, record did not “reflect the ‘considered judgment’ necessary to support the region’s determination”).

## 2. Overview of PSD Legal Requirements and the “Top Down” Method

Congress enacted the PSD provisions of the CAA as part of the 1977 amendments to the Act. *In re BP Cherry Point*, 12 E.A.D. 209, 213 (EAB 2005). The PSD provisions govern air pollution in certain areas, called “attainment” areas, where the air quality meets or is cleaner than the national ambient air quality standards, as well as in unclassifiable areas that are neither attainment or “non-attainment.” CAA §§ 160-69, 42 U.S.C. §§ 7470-79; see *In re Rockgen Energy Ctr.*, 8 E.A.D. 536, 541 (EAB 1999). The statutory PSD provisions are largely carried out through a regulatory process that requires new major stationary

sources in attainment (or unclassifiable) areas, such as Desert Rock, to obtain preconstruction permits pursuant to CAA § 165, 42 U.S.C. § 7475. *See* 40 C.F.R. § 52.21; *Rockgen*, 8 E.A.D. at 541; *Knauf*, 8 E.A.D. at 123.

The CAA and Agency PSD regulations require, as part of the preconstruction review process, that new major stationary sources and major modifications of such sources employ the “best available control technology,” or BACT, to minimize emissions of regulated pollutants. 42 U.S.C. § 7475(a)(4); 40 C.F.R. § 52.21(j)(2). The statute defines the BACT requirements as follows:

The term “best available control technology” means an emission limitation based on the maximum degree of reduction of each pollutant subject to regulation under this chapter emitted from or which results from any major emitting facility, which the permitting authority, on a case-by-case basis, taking into account energy, environmental, and economic impacts and other costs, determines is achievable for such facility through application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment or innovative fuel combustion techniques for control of each such pollutant.

CAA § 169(3), 42 U.S.C. § 7479(3); *accord* 40 C.F.R. § 52.21(b)(12) (similar regulatory definition). As the Board recently explained in *In re Northern Michigan University* (“*NMU*”), the BACT definition requires permit issuers to “proceed[] on a case-by-case basis, taking a careful and detailed look, attentive to the technology or methods appropriate for the particular facility, [] to seek the result tailor-made for that facility and that pollutant.” 14 E.A.D. 284, 292 (EAB 2009) (citations and quotations omitted). BACT is therefore a site-specific determination that results in the selection of an emission limitation representing application of control technology or methods appropriate for the particular facility. *In re Prairie State Generating Co.*, 13 E.A.D. 1, 12 (EAB 2006), *aff’d sub. nom Sierra Club v. U.S. EPA*, 499 F.3d 653 (7th Cir. 2007); *In re Cardinal FG Co.*, 12 E.A.D. 153, 161 (EAB 2005); *In re Three Mountain Power, L.L.C.*, 10 E.A.D. 39, 47 (EAB 2001); *Knauf*, 8 E.A.D. at 128-29; *see also In re Christian County Generation, LLC*, 13 E.A.D. 449, 454 (EAB 2008).

In determining BACT emission limits for the Desert Rock Permit, the Region utilized the “top-down method,” *see* RTC at 13-21, which is described in an EPA manual that provides guidance to permit issuers reviewing new sources under the CAA. *See* Office of Air Quality Planning & Standards, U.S. EPA, *New Source Review Workshop Manual 1* (draft Oct. 1990) (“NSR Manual”). Permit issuers often use the NSR Manual’s “top-down” method to perform their BACT analyses, as the Region did in this case. Notably, the NSR Manual is not a binding

Agency regulation and consequently strict application of the methodology described in it is not mandatory nor is it the required vehicle for making BACT determinations. *E.g.*, *NMU*, 14 E.A.D. at 293; *Prairie State*, 13 E.A.D. at 6 n.2; *Knauf*, 8 E.A.D. at 129 n.13. Nevertheless, because it provides a framework for determining BACT that assures adequate consideration of the statutory and regulatory criteria, it has guided state and federal permit issuers, as well as PSD permit applicants, on PSD requirements and policy for years. *E.g.*, *NMU*, 14 E.A.D. at 293; *Cardinal*, 12 E.A.D. at 162; *see also In re Steel Dynamics, Inc.*, 9 E.A.D. 165, 183 (EAB 2000) ("This top-down analysis is not a mandatory methodology, but it is frequently used by permitting authorities to ensure that a defensible BACT determination, involving consideration of all requisite statutory and regulatory criteria, is reached."). The NSR Manual summarizes the top-down method for determining BACT as follows:

[T]he top-down process provides that all available control technologies be ranked in descending order of control effectiveness. The PSD applicant first examines the most stringent – or “top” – alternative. That alternative is established as BACT unless the applicant demonstrates, and the permitting authority in its informed judgment agrees, that technical considerations, or energy, environmental, or economic impacts justify a conclusion that the most stringent technology is not “achievable” in that case.

NSR Manual at B.2; *accord Prairie State*, 13 E.A.D. at 1, 13; *see also NMU*, 14 E.A.D. at 293.

The NSR Manual's recommended top-down analysis employs five steps. NSR Manual at B.5-9; *see also NMU*, 14 E.A.D. at 292-94 (summarizing steps); *Prairie State*, 13 E.A.D. at 13-14 (same); *In re Haw. Elec. Light Co.*, 8 E.A.D. 66, 84 (EAB 1998) (same). Of particular relevance here is step 1, in which the applicant (and the permitting authority) initially identifies all potentially available control alternatives, or in more specific terms, “all control options with *potential* application to the source and pollutant under evaluation.” NSR Manual at B.10 (emphasis added). The NSR Manual lists three general categories of potentially applicable control alternatives: (1) inherently lower emitting processes and/or practices; (2) add-on controls; and (3) combinations of the two. *Id.* The BACT analysis should include a consideration of potentially applicable control techniques from all three. *Id.*

Regarding the scope of the step 1 analysis, as the Manual explains, “[a]pplicants are expected to identify *all demonstrated and potentially applicable* control technology alternatives.” *Id.* at B.11 (emphasis added). Thus, “[t]he control alternatives should include not only existing controls for the source category in question, but also (through technology transfer) controls applied to similar

source categories and gas streams, and innovative control technologies.” *Id.* at B.5. “Technologies employed outside the United States” should also be considered. *Id.* The Manual lists a number of information resources that applicants should consider in performing the BACT step 1 analysis, including other federal, state, and local new source review permits. *Id.* at B.11. Thus, the BACT step 1 analysis is intended to be very broad, leading to the development of a comprehensive list of control options. *In re ConocoPhillips Co.*, 13 E.A.D. at 768; *Knauf*, 8 E.A.D. at 130. The Board has previously held that failure to consider all potentially applicable control options is grounds for remand. *See, e.g., Knauf*, 8 E.A.D. at 140-41; *In re Hibbing Taconite Co.*, 2 E.A.D. 838, 842-43 (Adm’r 1989); *see also Prairie State*, 13 E.A.D. at 15-28 (applying step 1).

In the second step, the permit issuer eliminates “technically infeasible” options from those identified as potentially available at step 1. NSR Manual at B.7. This step involves first determining for each technology whether it is “demonstrated,” in other words, whether it has been installed and operated successfully elsewhere on a similar facility. *Id.* at B.17. If it has not been demonstrated, the permit issuer then performs a somewhat more difficult analysis: whether the technology is both “available” and “applicable.” *Id.* at B.17-22. Technologies identified in step 1 as “potentially” available, but that are neither demonstrated nor found after careful review to be both available and applicable, are eliminated under step 2 from further analysis. *Id.*; *see e.g., Prairie State*, 13 E.A.D. at 34-38 (reviewing step 2 analysis); *Cardinal*, 12 E.A.D. at 163-168 (same); *Steel Dynamics*, 9 E.A.D. at 199-202 (same).

In step 3, the permit issuer ranks the remaining control technologies and then lists them in order of control effectiveness for the pollutant in question, with the most effective alternative at the top. NSR Manual at B.7, .22. A step 3 analysis includes making determinations about comparative control efficiency among control techniques employing different emission performance levels and different units of measure of their effectiveness. *Id.* at B.22-25; *Newmont*, 12 E.A.D. at 459-64 (evaluating challenge to step 3 analysis).

In the fourth step of the analysis, the permitting authority considers energy, environmental, and economic impacts and confirms the top alternative as appropriate or determines it to be inappropriate. NSR Manual at B.8-.9, .26-.53. Thus, it is in this step that issues surrounding the relative cost effectiveness of the alternative technologies are considered. *Id.* at B.31-46. The purpose of step 4 is to either validate the suitability of the top control option identified or provide a clear justification as to why that option should not be selected as BACT. *Id.* at B.26; *see also Prairie State*, 13 E.A.D. at 38-45 (considering the application of step 4); *Three Mountain Power*, 10 E.A.D. at 42 n.3 (evaluating environmental impacts); *Steel Dynamics*, 9 E.A.D. at 202-07, 212-13 (remanding permit because cost-effectiveness analysis under step 4 was incomplete).



Finally, under step 5, the permit issuer selects the most effective control alternative not eliminated in step 4. NSR Manual at B.9, .53. BACT is set as an emissions limit for a specific pollutant that is appropriate for the selected control method. *Id.* at B.53-.54; *see also NMU*, 14 E.A.D. at 292-94 (explaining five-step process); *Prairie State*, 13 E.A.D. at 38-51 (same).

### 3. *The Region Abused Its Discretion in Concluding that IGCC "Redefines the Source"*

In its final determination for the Desert Rock Permit, the Region did not consider an integrated gasification combined cycle or, as previously defined, "IGCC," system as a potentially available control technology in step 1 of its BACT analysis. *See RTC* at 13 (specifically stating that the Region declined to perform a detailed evaluation of IGCC "at or beyond step 1 of the top-down BACT process"). Instead, the Region considered the technology as an "alternative" under another PSD provision, section 165(a)(2), 42 U.S.C. § 7475(a)(2).<sup>52</sup> *See id.* at 10-11, 13-21 & app. A. The Region explained its rationale for considering IGCC under the alternatives provision rather than the BACT provision in its Response to Comments document, stating that it retains discretion not to list options in step 1 of the BACT analysis that it believes would fundamentally "redefine" the proposed source and that IGCC would "redefine the source" proposed by the applicant. *Id.* at 13-20.

As an initial matter, in order to determine whether the Region appropriately declined to consider IGCC under its BACT analysis for the Desert Rock Permit, it is important to understand two underlying concepts: (1) how IGCC generally works and (2) what is meant by "redefining the source."

#### a. *Description of IGCC and History of Its Applicability*

In a typical pulverized coal ("PC") combustion-based electric generating facility, such as that proposed for the Facility, coal is burned to create heat, which is used to boil water, creating steam that drives a steam turbine power generator. *See A.R.* 120.10, at 2-10 to -154 (U.S. EPA, EPA-430/R-06-006, *Final Report, Environmental Footprints and Costs of Coal-Based Integrated Gasification Combined Cycle and Pulverized Coal Technologies* (2006)) [hereinafter *EPA 2006 Report on IGCC and PC Technologies*]; *DR Resp.* at 55. IGCC, on the other hand, is a dual electric-power-generating system. *See EPA 2006 Report on IGCC and PC Technologies* at 2-4. It too uses coal, but in an initial "gasification" part of the

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<sup>52</sup> CAA section 165(a)(2) requires that the proposed permit be subject to a public hearing "with opportunity for interested persons \* \* \* to appear and submit written or oral presentations on the air quality impact of such source, *alternatives thereto*, control technology requirements, and other appropriate considerations[.]" CAA § 165(a)(2), 42 U.S.C. § 7475(a)(2) (emphasis added).

process, the coal is chemically converted into a synthetic gas (“syngas”). *Id.*; *Christian County*, 13 E.A.D. at 449, 451. The syngas is cleaned to remove various pollutants, such as particulate matter, mercury, sulfur compounds, ammonia, and other acid gases, and is then burned in a gas turbine to generate electric power.<sup>53</sup> *EPA 2006 Report on IGCC and PC Technologies* at 2-4; *Christian County*, 13 E.A.D. at 451. Heat is recovered from the gas turbine and the gasification process and is then used to produce additional power using a steam turbine. *EPA 2006 Report on IGCC and PC Technologies* at 2-4; *Christian County*, 13 E.A.D. at 451. Thus, as the Board explained in *Prairie State*, “IGCC is not simply an add-on emissions control technology,” but instead requires a differently designed power block. 13 E.A.D. at 27.

IGCC has been considered a potentially applicable control technique under step 1 of BACT for coal-fired electric generating plants in at least two PSD permits that the Board has reviewed. In 2005, the Illinois Environmental Protection Agency (“IEPA”) – which issues PSD permits under a delegation of authority from Region 5 – found IGCC to be a potentially applicable control technique for two pollutants, SO<sub>2</sub> and NO<sub>x</sub>, for a proposed mine-mouth, coal-fuel powered generating plant. *See id.* at 5-6, 45, 13 E.A.D. at 5, 35. In its permit determination for the Prairie State Generating Station, IEPA explained that it had considered IGCC as a potentially applicable control technique under step 1 of BACT because it had concluded “that IGCC is a production process that can be used to produce electricity from coal, that IGCC is a technically feasible production process, and that \* \* \* it qualifies as an alternative emission control technique that must be fully addressed in the BACT demonstration for the proposed plant.” *Id.* 13 E.A.D. at 27 n.30 (citation and quotations omitted). Ultimately, however, because IEPA concluded that IGCC had not been shown to achieve greater emission reductions than the technology proposed by the applicant, it did not select IGCC as BACT for the Prairie State Generating Station. *Id.* 13 E.A.D. at 27. Thus, in that case, IGCC was included in the BACT analysis but was dismissed from further BACT consideration at step 2.

In the second case, *In re Christian County Generation, LLC*, IEPA – again acting under a delegation of authority from Region 5 – once more considered IGCC as a potentially applicable control technology in BACT step 1 for a proposed coal-fired generating plant, the Taylorville Energy Center. *See* 13 E.A.D. at 450-51. In fact, in that case, after consideration of IGCC in all five steps of the

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<sup>53</sup> Notably, the EPA Report states that “it is generally accepted that the IGCC system, by removing most pollutants from the syngas prior to combustion, is capable of meeting more stringent emission standards than PC technologies.” *EPA 2006 Report on IGCC and PC Technologies* at 2-4; *see also Christian County*, 13 E.A.D. at 462-63 (comparing emissions for sulfur dioxide). The Report further remarks that “[i]t is also generally accepted that IGCC costs are higher and more uncertain than for PC plants, because PC technology has been demonstrated at many more facilities.” *EPA 2006 Report on IGCC and PC Technologies* at 2-4.



BACT analysis, IGCC was ultimately *selected as BACT* for the facility. *See id.* 13 E.A.D. at 450, 462-63.

Because IEPA issues PSD permits under a delegation of authority from EPA, these two permits are considered EPA-issued under federal law.<sup>54</sup> As the preamble to the Agency's permitting regulations explains, "[f]or the purposes of Part 124, a delegate State stands in the shoes of the Regional Administrator \* \* \* [and] must follow the procedural requirements of Part 124. \* \* \* A permit issued by a delegate is still an 'EPA-issued permit' \* \* \*." *Consolidated Permit Regulations*, 45 Fed. Reg. 33,290, 33,413 (May 19, 1980); *accord Prairie State*, 13 E.A.D. 1, 4 n.1 (EAB 2006) ("Permits issued by states acting with delegated authority are considered EPA-issued permits."); *Indeck*, 13 E.A.D. at 128 ("Where EPA delegates administration of the federal PSD program, the delegate state implements the substantive and procedural aspects of the federal PSD regulations on behalf of EPA \* \* \* [thereby] stand[ing] in the shoes of EPA, and the permit remains a federal action \* \* \*." (quoting EPA's Offices of Air and Radiation and of General Counsel)); *In re Zion Energy, L.L.C.*, 9 E.A.D. 701, 701 n.1 (EAB 2001); *In re W. Suburban Recycling & Energy Ctr., L.P.*, 6 E.A.D. 692, 695 n.4 (EAB 1996); *see also* 40 C.F.R. § 124.41 (definitions applicable to federal PSD permits).

b. "Redefinition of the Source"

"Redefining the source' is a term of art described in the NSR Manual," *Knauf*, 8 E.A.D. at 136, although the concept predates the 1990 manual, *see, e.g., Hibbing*, 2 E.A.D. at 843 & n.12; *In re Pennsauken County*, 2 E.A.D. 667, 673 (Adm'r 1988). As the Board explained in *Knauf*, "[t]he Manual states that it is legitimate to look at inherently lower-polluting processes in the BACT analysis, but EPA has not generally required a source to change (i.e., redefine) its basic design." 8 E.A.D. at 136 (citing NSR Manual at B.13). The Board further explained that, while "it is not EPA's policy to require a source to employ a different design, redefinition of the source is not always prohibited. This is a matter for the permitting authority's discretion." *Id.* The NSR Manual explains the concept as follows:

Historically, EPA has not considered the BACT requirement as a means to redefine the design of the source when considering available control alternatives. \* \* \* However, there may be instances where, in the permit author-

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<sup>54</sup> Significantly, as explained in Part III.B.2 *supra*, the NSR Manual suggests that applicants review recently issued federal PSD permits, such as the permit at issue in *Christian County*, when "identify[ing] all demonstrated and potentially applicable control technology alternatives." NSR Manual at B.11.

ity's judgment, the consideration of alternative production processes is warranted and appropriate for consideration in the BACT analysis. \* \* \* In such cases, the permit agency may require the applicant to include the inherently lower-polluting process in the list of BACT candidates.

In some cases, a given production process or emissions unit can be made to be inherently less polluting \* \* \*. In such cases the ability of design considerations to make the process inherently less polluting must be considered as a control alternative for the source.

NSR Manual at B.13-14; *see also* *Prairie State*, 13 E.A.D. at 17-18, 25-26 (discussing same provisions).

In the earliest case referring to the “redefinition of the source” concept, the Administrator denied a petition urging the Agency to require use of existing power plants in lieu of the proposed source, a municipal waste combustor, because the Administrator concluded petitioner was essentially “redefining the source.” *Pennsauken*, 2 E.A.D. at 673. The Administrator stated that, while “imposition of the conditions may, among other things, have a profound effect on the viability of the proposed facility as conceived by the applicant, the conditions themselves are not intended to redefine the source.” *Id.* Consequently, he concluded that “permit conditions defining the emissions control systems ‘are imposed on the source *as the applicant has defined it*’ and [] ‘the source itself is not a condition of the permit.’” *Prairie State*, 13 E.A.D. at 23 (quoting *Pennsauken*, 2 E.A.D. at 673 (emphasis added)). As the Administrator further elaborated in a later case: “[t]raditionally, EPA has not required a PSD applicant to redefine the fundamental scope of its project.” *Hibbing*, 2 E.A.D. at 843 (citing *Pennsauken*); *accord In re Old Dominion Elec. Coop.*, 3 E.A.D. 779, 793 n.38 (Adm’r 1992).

More recently, the Board has discussed the application and scope of the “redefining the source” policy in two cases: *Prairie State* and *NMU*. In fact, in *Prairie State* – a case in which participants’ arguments bear a marked resemblance to the ones raised here – the Board painstakingly analyzed the history, basis, and application of the “policy”<sup>55</sup> and its relationship to the statutory BACT provisions. 13 E.A.D. at 14-28. Rather than repeat the entire analysis here, the Board merely summarizes its relevant key points.

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<sup>55</sup> While often referred to as a “policy,” as discussed below, it is clear from the description in *Prairie State* both before the Board and on appeal to the Seventh Circuit that the policy is really an agency interpretation of ambiguous statutory provisions.

In *Prairie State*, as in this case, petitioners challenged the permit issuer's failure to consider an alleged potential control option in step 1 of the BACT analysis. Petitioners there argued that IEPA's failure to consider low-sulfur coal at step 1 violated the statutory BACT definitional requirement that "clean fuels" be considered. *Id.* at 16-17.<sup>56</sup> In response, IEPA took the same position the Region is taking here – that "it did not abuse its discretion in relying upon the 'redefining the source doctrine' when it concluded that consideration of [the option at issue] would redefine the proposed source and, therefore, may be eliminated from further consideration at step 1." *Id.* at 23, 13 E.A.D. at 17-18.

In *Prairie State*, the Board provided a lengthy discussion of the basis behind the Agency's longstanding "redefining the source policy," explaining that the "policy" resolves ambiguity found in the statutory text of CAA sections 165 and 169.<sup>57</sup> *See id.* at 23-30, 13 E.A.D. at 25-28. This ambiguity arises from several statutory words and phrases, including but not limited to the fact that the BACT definition's requirement to consider the "application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment of innovative fuel combustion techniques" cannot be read in isolation from the requirement that the "proposed facility" be "subject to" BACT.<sup>58</sup> *Id.* at 22; *see also id.* at 18-23 & nn.15, 19, 22. The Board also noted that Congress designed the PSD program as a permitting program in which the permit applicant initiates

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<sup>56</sup> As noted above, the statute defines BACT as "an emission limitation" achievable by "application of production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment or innovative fuel combustion techniques for control of each such pollutant." CAA § 169(3), 42 U.S.C. § 7479(3) (emphasis added). Notably, "clean fuels" is one of the terms listed in the BACT definition's "production processes and available methods, systems, and techniques" along with "innovative fuel combustion techniques" and "fuel cleaning," the statutory terms New Mexico and Conservation Petitioners relied upon in their petitions. Replacing *Prairie State* Petitioners' "clean fuels" references with the other two listed terms – "fuel cleaning" and "innovative fuel combustion techniques" – would essentially yield the same arguments raised by New Mexico and NGO Petitioners.

<sup>57</sup> Consequently, Petitioners' argument that certain terms in sections 165 and 169 "require" consideration of a specific technology under BACT, *e.g.*, NGO Suppl. Br. at 88; NM Suppl. Br. at 18, is somewhat misplaced because such an argument implicitly fails to recognize the fact that those terms are subject to the Agency's interpretation, which refines their meaning (*i.e.*, the redefining the source policy). *See Sierra Club*, 499 F.3d at 655. For this reason, rather than debating the meaning of the ambiguous terms, the discussion in the text focuses on the policy itself and its applicability here.

<sup>58</sup> Other sources of ambiguity in the CAA include section 165(a)(2)'s separate listing of "alternatives" and "control technology requirements," which indicates a distinction between the two concepts. *See Prairie State*, 13 E.A.D. at 18-22 & nn.15, 22; *see also Sierra Club*, 499 F.3d at 655 (noting that requiring the consideration of certain hypothetical "clean fuels" under BACT, such as the redesign of a coal-fired plant into a nuclear one, would "stretch the term 'control technology' beyond the breaking point and collide with the 'alternatives' provision of the statute"); *RTC* at 14-16 (same). Additionally, the BACT definition explicitly requires a "case-by-case" determination, suggesting that an across-the-board application of a control technology would not be appropriate. CAA § 169(3), 42 U.S.C. § 7479(3).

the process. *See id.* at 28-29, 13 E.A.D. at 22. The Board concluded that the heart of the parties' debate in *Prairie State* was not whether "Congress intended the permit applicant to have the prerogative to define certain aspects of the proposed facility that may not be redesigned through application of BACT," but where the "proper demarcation between those aspects of a proposed facility that are subject to modification through the application of BACT and those that are not" should be drawn. *Id.* at 20-21. In other words, the question the Board decided in *Prairie State* was not whether the Agency may interpret the CAA PSD provisions to contain a limit on redefining the source, but rather how such an interpretation should properly be applied.<sup>59</sup>

On appeal, the Seventh Circuit generally agreed with this analysis. The Court acknowledged the ambiguity in the statute, particularly referring to the CAA's requirement that a "proposed facility" must have the "best available control technology" and that "clean fuels" be considered and also noted that a separate provision from the one requiring adoption of BACT directs EPA to consider "alternatives" suggested by interested persons. *Sierra Club v U.S. EPA*, 499 F.3d 653, 654-55 (7th Cir. 2007). Based on this ambiguity, the Court deferred to the Agency, stating that "[r]efining the statutory definition of 'control technology' – 'production processes and available methods, systems, and techniques, including fuel cleaning, clean fuels, or treatment of innovative fuel combustion techniques' – to exclude redesign is the kind of judgment by an administrative agency to which a reviewing court should defer." *Id.* at 655. The Court thus concluded that "the crucial question [is] where control technology ends and a redesign of the 'proposed facility' begins."<sup>60</sup> *Id.*

### c. The Proper Test for Redesign

In this case, the real debate centers around the same fundamental question raised in *Prairie State*: when does the imposition of a control technology require enough of a redesign of the proposed facility that it strays over the dividing line to become an impermissible redefinition of the source? More specifically, did the Region correctly conclude that imposition of IGCC would so substantially alter

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<sup>59</sup> Thus, in *Prairie State*, the Board did not explicitly address the statutory interpretation debate over the meaning of "clean fuels" in the BACT definition.

<sup>60</sup> The Seventh Circuit concluded that EPA, as the author of the underlying distinction, should draw the dividing line "within reason," but also implied that an interpretation that would read "clean fuels" entirely out of the statute would be questionable. *Sierra Club*, 499 F.3d at 655-56; *see also NMU*, at 302 ("Clean fuels may not be 'read out' of the Act merely because their use requires 'some adjustment' to the proposed technology." (quoting *Sierra Club*, 499 F.3d at 656)). The Seventh Circuit also noted that this question "require[s] an expert judgment." *Sierra Club*, 499 F.3d at 656. The Court further observed that such a question is "one of degree and the treatment of differences of degree in a technically complex field with limited statutory guidance is entrusted to the judgment of the agency that administers the regulatory scheme rather than to courts of generalist judges." *Id.*

the purpose or basic design of Desert Rock's proposed facility that it should be considered a redefinition of the source?

The Board articulated the proper test to be used to answer that question in *Prairie State*. As the Board explained there, the permit applicant initially "defines the proposed facility's end, object, aim, or purpose – that is the facility's basic design,"<sup>61</sup> although the applicant's definition must be "for reasons independent of air permitting."<sup>62</sup> *Prairie State*, 13 E.A.D. at 23 n.23; *accord NMU*, 14 E.A.D. at 303 & n.28. The inquiry, however, does not end there. The permit issuer (here, the Region) should take a "hard look" at the applicant's determination in order to discern which design elements are inherent for the applicant's purpose and which design elements "may be changed to achieve pollutant emissions reductions without disrupting the applicant's basic business purpose for the proposed facility," while keeping in mind that BACT, in most cases, should not be applied to regulate the applicant's purpose or objective for the proposed facility. *Prairie State*, 13 E.A.D. at 23, 25-26; *accord NMU*, 14 E.A.D. at 302-04.

To determine whether the Region properly concluded that IGCC would redefine the source in this case, keeping in mind that the Region has broad discretion on this issue, the Board first looks at the administrative record to see how the applicant defined its "goal, objectives, purpose, or basic design" for the proposed Facility in its application. The Board then looks at whether the Region took a "hard look" at the applicant's stated purpose to determine which design elements were inherent to the applicant's basic purpose or objective and which elements could be changed to achieve pollutant emissions reductions without disrupting the purpose. Based on the current administrative record, the Board concludes that the Region abused its discretion in declining to consider IGCC in step 1 of the BACT analysis for the Desert Rock Facility.

#### d. *Treatment of IGCC in the Administrative Record*

Looking at the initial application, it is clear, and telling, that the applicant itself believed that IGCC was consistent with the proposed facility's purpose, objective, or basic design. In its 2004 application, the then-applicant Steag stated

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<sup>61</sup> Regarding the meaning of the term "design," the Board in *Prairie State* explained that "[a]s a practical matter, 'design,' understood as a schematic drawing showing the means to an end, and 'design,' used to identify the end, object, or purpose, are inherently intertwined." 13 E.A.D. at 21. Thus, "[t]he permit applicant's schematic design can be presumed to be directed at accomplishing the permit applicant's purpose or basic design for the proposed facility." *Id.*

<sup>62</sup> Thus considerations such as cost savings or avoidance of risks associated with new, innovative, or transferable technologies would generally not justify treating a proposed facility's design element as basic or fundamental. *Prairie State*, 13 E.A.D. at 23 n.23; *NMU*, 14 E.A.D. at 303 & n.28. These factors, however, could be considered elsewhere in the BACT analysis, for example at step 2 or 4. *See Prairie State*, 13 E.A.D. at 23 n.23 (citing examples); *see also* further discussion *infra*.

that its proposed project was the construction of “a mine-mouth coal-fired power plant on Navajo Nation land.” A.R. 6, at 2-2. It further stated that “[f]our technologies may be considered for a new large coal fueled power plant \* \* \* : pulverized coal combustion (sub-critical steam production); pulverized coal combustion (supercritical steam production); circulating fluidized bed (CFB) combustion; and *Integrated Gasification Combined Cycle (IGCC)*.” *Id.* at 2-2 (emphasis added). A few pages later, the applicant rejected IGCC as an option because it “is not currently an available or commercially viable technology for a 1,500 MW commercial coal-fired power plant.” *Id.* at 2-4. Thus, at the time of the initial application in 2004, the applicant’s rationale for not considering IGCC appears to have been its “unavailability” and its lack of commercial viability.<sup>63</sup> Significantly, the applicant does not suggest that IGCC would somehow be outside the fundamental scope of its project; in fact, by listing IGCC as a possible technology to implement its project, it actually indicates the reverse.<sup>64</sup> While the applicant may have backtracked on these initial statements at some point,<sup>65</sup> this does not change the fact that it originally listed IGCC as a potential technology that could be used to meet the proposed facility’s basic business objective.<sup>66</sup>

The Region, in its Ambient Air Quality Impact Report (“AAQIR”) – the document the Region developed as the statement of basis and fact sheet for the proposed permit and which included the Region’s initial BACT analysis for the Facility, *see* A.R. 46, at 6-35 – similarly noted that the applicant proposed to construct a “1,500 [MW] mine-mouth, coal-fired power plant,” *id.* at 1. The Region explained that the proposed permit would allow use of two supercritical pulverized coal boilers for the Facility. *Id.* at 2. As part of its BACT analysis, the Region first considered a number of add-on control technologies to the supercritical pulverized coal boilers for each regulated pollutant. *See, e.g., id.* at 8-15 (considering four potentially applicable add-on control technologies for NO<sub>x</sub>). In addition, the Region separately considered whether an alternate technology for combusting

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<sup>63</sup> The former, if supported by the administrative record and withstanding the permit issuer’s “hard look,” would be a legitimate reason to exclude IGCC from BACT step 1. *See* NSR Manual at B.5; *Prairie State*, at 29-34. The latter, however, is more properly considered a BACT step 2 or 4 issue, depending on whether its viability is questionable from a technical feasibility standpoint or an economic/cost standpoint. *See* NSR Manual at B.7-9; *see also* discussion *infra*.

<sup>64</sup> Although the Board does not understand how, based on such statements in the application, the Agency found that IGCC would be redefining the source, the Board continues its analysis in the interest of completeness.

<sup>65</sup> It is unclear when Desert Rock first took its revised position that IGCC would redefine the source; at a minimum, however, Desert Rock has consistently taken this position during the appeal process. *E.g.,* DR Opp’n Br. at 21; DR Resp. to Petitions at 49, 51.

<sup>66</sup> As noted above, the new rationale is subject to scrutiny to determine whether it is “independent of air quality permitting.”

coal – CFB combustion – was potentially applicable.<sup>67</sup> *Id.* at 32-35. The Region concluded that CFB was not “an appropriate technology for the project” and would “result in higher emissions than the Facility as proposed.” <sup>68</sup> *Id.* at 35. Finally, the Region noted that it had not included IGCC as an alternate technology to a pulverized coal boiler in its BACT step 1 analysis because IGCC “would fundamentally change the basic design of the proposed source” and “would be redefining the source.” *Id.* The Region did not, however, address either of the reasons the applicant had relied on to ultimately exclude IGCC from consideration, i.e., its availability (or lack thereof) or its commercial viability, nor did the Region explain why IGCC would be redefining the source when the application had suggested the reverse.

In response to the proposed permit and AAQIR, several commenters questioned the Region’s failure to consider IGCC as part of the BACT analysis. *See* RTC at 12 (listing numerous comments on this issue). Some commenters noted that the technology was now “available.” RTC at 12. In fact, a group of environmental organizations, including the seven NGO Petitioners, submitted a comment that pressed for the use of IGCC and “provided [its] own BACT evaluation of the availability, feasibility, cost, emission rates, and other environmental impacts of IGCC.” RTC at 21. Moreover, that same commenter argued that the Region’s determination that IGCC redefined the source at the Desert Rock facility ran counter to the Board’s “favorable consideration” of IGCC in *Prairie State*. A.R. 66, at 21-22 & n.38.

Responding to these comments in its Final Permit determination, the Region stated that it “does not agree that the [CAA] requires a detailed evaluation of IGCC for the proposed facility, at or beyond step 1 of the top-down BACT process.” RTC at 13. Thus, as noted earlier, instead of analyzing IGCC under BACT step 1, the Region continued to consider IGCC as an “alternative” under section 165(a)(2), 42 U.S.C. § 7475(a)(2).<sup>69</sup> *Id.*; *see also id.* app. A at 220, 224-26 (Re-

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<sup>67</sup> The Region explained that it had not included the CFB analysis in the pollutant-by-pollutant portion of its BACT assessment “because an applicant must choose either a pulverized coal boiler or CFB for all pollutants.” A.R. 46, at 32.

<sup>68</sup> The Region also looked at, to some degree, sub-critical pulverized coal combustion. *See* A.R. 46, at 32 tbl.12 (including emissions for sub-critical PC). Therefore, in the BACT step 1 analysis in its AAQIR, the Region considered three of the four technologies the applicant listed in its application as potential technologies, at least to some degree. IGCC was the only technology the applicant listed that the Region failed to consider.

<sup>69</sup> The level of analysis in a permit issuer’s consideration of a technology under the alternatives provision, CAA section 165(a)(2), is not necessarily identical to the level of analysis that the permit issuer would undertake for the same technology under the BACT provision, CAA section 165(a)(4). For example, while the consideration of a technology as part of the BACT analysis may be quite extensive under the NSR Manual guidelines, under the PSD alternatives provision, “the extent of the  
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gion's consideration of IGCC as an "alternative"). The Region stated that it had not "been persuaded to change [its] view that this alternative process would redefine the source proposed by the applicant and thus need not be listed as a potentially applicable control option at step 1." *Id.* at 13. The Region explained that, in its view, IGCC "would fundamentally change the nature of the proposed major source as it would change the basic design of the equipment Sithe proposed to install." *Id.* at 19. The Region also analogized the design changes that would be necessitated by IGCC to those in previous Board and Administrator cases in which "redefining the source" was relied upon to exclude consideration of the use of a different type of electric generating facility as BACT. *Id.* at 19 (referring to *In re SEI Birchwood, Inc.*, 5 E.A.D. 25 (EAB 1994) (noting in dicta that petitioner's preference for natural gas over coal did not demonstrate clear error by the delegated state permitting authority); *In re Old Dominion Elec. Coop.*, 3 E.A.D. 779 (Adm'r 1992)). Finally, the Region argued that "the core process of gasification at an IGCC facility is fundamentally different than operating a boiler" and thus would require "different types of expertise to operate."<sup>70</sup> *Id.* at 19-20.

Significantly, the Region failed to address several critical questions in its consideration of IGCC and its BACT step 1 analysis. First, the Region did not take a "hard look" to see how Desert Rock defined its project in order to discern which design elements were inherent to that purpose and which design elements could be changed to achieve pollutant emission reductions without disrupting Desert Rock's basic business purpose. If it had followed the analytical framework the Board outlined in *Prairie State*, it would have seen that, at least in its initial application, Desert Rock admitted that IGCC was a "technolog[y] that may be considered for a coal fueled power plant," such as its proposed facility.<sup>71</sup> A.R. 6, at 2-2.

Second, the Region did not explain in its BACT analysis how IGCC could be considered as a "potentially available control technology" under step 1 of the BACT analysis for two other EPA-issued permits (i.e., federal permitting decisions) at similar facilities – the Christian County coal-fired electric generating plant and the Prairie State mine-mouth, coal-fired electric generating station – but

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permitting authority's consideration and analysis of alternatives need be no broader than the analysis supplied in public comments." *Prairie State*, 13 E.A.D. at 30 (quotation omitted); *see also id.* at 31-32 (discussing petitioner's argument about the permit issuer's alternatives discussion of "need" for facility).

<sup>70</sup> This latter argument is particularly weak in the PSD context. Even where add-on control technologies are required, such technology may require different expertise than the applicant originally planned in its proposed facility. The mere fact that different expertise may be required does not eliminate a technology from BACT step 1. Indeed, if such a factor is considered in the BACT analysis, it may be best considered in step 4.

<sup>71</sup> Again, at that time, the applicant took the position that IGCC, while theoretically feasible, was not currently available. A.R. 6, at 2-2, 2-4.



was not likewise considered by the Agency at the Desert Rock Facility, which is proposed to be a mine-mouth, coal-fired electric generating station.<sup>72</sup> Nor did the Region explain why use of IGCC was considered “redefining the source” at the coal-fired electric generating plant proposed for Desert Rock when it had not been a “redefinition of the source” at two earlier EPA-permitted coal-fired electric generating plants.

While it is true that each BACT analysis is a case-by-case determination, when a technology has been considered a “potentially available control technology” at otherwise seemingly similar facilities in previous permitting actions, one would expect some explanation as to why the previously “potentially available control technology” is no longer potentially available at the latest facility. *See* NSR Manual at B.11 (stating that “[a]pplicants are expected to identify all demonstrated and potentially applicable control technology alternatives,” including federal new source review permits), B.35 (“Consistency in the approach to decision-making is a primary objective of the top-down BACT approach.”); *Indeck*, 13 E.A.D. at 183 (“[T]he existence of a similar facility with a lower emissions level creates an obligation for [the permit applicant] to consider or document whether that same emissions limit can be achieved at [the] proposed facility.”); *In re Inter-Power of NY, Inc.*, 5 E.A.D. 130, 135 (EAB 1994) (“In determining the most stringent control option, the proposed source is required to look at other recently permitted sources.”); *see also NMU*, 14 E.A.D. at 209 (questioning the permit issuer’s passing over BACT emission limits from the most similarly situated facility without any justification).<sup>73</sup> This is particularly so since, at the time the Region issued the permit, IGCC had actually been selected as the emission

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<sup>72</sup> The Region did attempt to distinguish its determination from that of other states that have concluded that IGCC is a “potentially available control technology” for coal-fired steam electric generating facilities by arguing that, because the decision of where to draw the line is discretionary, “[s]tate decisions as to how to conduct a BACT analysis do not necessarily set the bar for EPA.” RTC at 20. The Region, however, did not provide any factual information in its Response to Comments that would distinguish the various coal-fired power plants. *See id.* Interestingly, the Region went on to note that “because Illinois administers the Federal PSD program under a delegation agreement with EPA Region V, Illinois must act in a manner consistent with EPA’s interpretation of the Clean Air Act and controlling regulations.” *Id.* at 20-21. The Region has not asserted that Illinois’s actions regarding the Prairie State and Christian County facilities were inconsistent with the CAA and applicable regulations.

<sup>73</sup> This should not be read to imply or suggest an absolute rule that once a technology is considered BACT, it always must be BACT. Typically, however, once a technology qualifies as “a potentially applicable control option” at a certain type of facility, it should remain “potentially applicable” thereafter for similar facilities without some distinguishing rationale otherwise. *See* NSR Manual at B.11 (expecting applicants to identify in step 1 all demonstrated and potentially applicable control technology alternatives, including those in federal, state, and local new source review permits). Moreover, the fact that a technology is considered in BACT step 1 does not mean that it would ultimately be considered BACT for that facility.

control technology to be implemented at the Christian County facility. *See Christian County*, 13 E.A.D. at 450, 462-63.

Similarly, while a permit issuer has broad discretion in determining whether or not an alternative production process would “redefine the source,” where a permit issuer concludes that a particular technology is not a “redefinition of the source” at one facility, if it later decides that the particular technology does “redefine the source” at a similar facility, it should provide some rational explanation justifying the differential treatment. As the Board has stated on a number of occasions, the BACT analysis is one of the most critical elements of the PSD permitting process and thus must be well documented in the administrative record. *Indeck*, 13 E.A.D. at 184-85; *Knauf*, 8 E.A.D. at 131; *Steel Dynamics*, 9 E.A.D. at 224. There may be a factual distinction between the three facilities justifying the different outcomes, but such distinction is not articulated in the record at all, much less to the standard required. *See Steel Dynamics*, 9 E.A.D. at 224 (requiring a greater degree of explanation, clearly documented in the record, where limits proposed to be imposed on a facility differ from fifteen other comparative facilities).

Furthermore, arguments about the technical viability or the economics of IGCC at the proposed facility are inapplicable at stage 1 of the BACT analysis.<sup>74</sup> *See, e.g.,* DR Resp. 65-69. As the Board noted in *Prairie State*, neither of these factors justify treating a design element as basic or fundamental. 13 E.A.D. at 23 n.23; *see also NMU*, 14 E.A.D. at 303 n.28. The business objective of avoiding risk associated with new, innovative or transferable control technologies and the technical feasibility of such technologies should instead be considered under step 2 of the top-down method. NSR Manual at B.18 (“A source would not be required to experience extended time delays or resource penalties to allow research to be conducted on a technique. Neither is it expected that an applicant would be required to experience extended trials to learn how to apply a technology on a totally new or dissimilar source type.”). Similarly, cost is generally considered later, at step 4. NSR Manual at B.8, B.26-45; *Steel Dynamics*, 9 E.A.D. at 202-07; *see also In re Masonite Corp.*, 5 E.A.D. 551, 564 (EAB 1994); *Inter-Power*, 5 E.A.D. at 135-36, 145-50 & n.33 (considering cost effectiveness issue after all control options selected); *Hibbing*, 2 E.A.D. at 843 (requiring consideration of burning natural gas, rather than petroleum coke, in the BACT analysis notwithstanding the greater cost of natural gas). A permit issuer, therefore, when evaluating whether an applicant’s purpose or design of a facility would be substantially altered by

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<sup>74</sup> In its response to the Petitions, Desert Rock contended that Petitioners’ arguments “are not material to the outcome” of a BACT determination for the Facility “because IGCC is not a feasible business venture and would be worse for the environment.” DR Resp. at 65. Desert Rock explained that it, as well as the original parent company Steag, submitted a number of documents to the Region regarding IGCC’s technical feasibility at the site, *id.* at 65-57 & n.34, and that these documents demonstrate that IGCC would be infeasible, *id.* at 67-69.

application of a particular technology, should consider whether the facts underlying such assertion are better considered within the framework of steps 2 through 5 of the top-down method, rather than grounds for excluding redesign at step 1.

*e. Other Court Decisions Concerning IGCC*

In connection with this IGCC BACT issue, several participants cite recent cases in which state courts have also looked at whether IGCC should be considered in the BACT analysis. *See, e.g.,* D.R. Surreply at 4 (referring to *Blue Skies Alliance v. Tex. Comm'n on Env'tl. Quality*, 283 S.W.3d 525 (Tex. App. 2009)); Cons. Reply at 3 n.6 (same);<sup>75</sup> ACCCE's Mot. to Take Notice of Suppl. Authority at 1 & Ex.1 (attaching copy of *Longleaf Energy Assocs. v. Friends of the Chattahoochee, Inc.*, Nos. A09A037 & A09A0388, 2009 WL 1929192 (Ga. Ct. App. July 7, 2009)); Cons. Pet'r's Resp. to ACCCE's Mot. Regarding Suppl. Authority at 1 (same). Both cases involve state court review of state-issued permits. As such, these cases are not binding on the Board. The Board did, however, consider both courts' analyses in reviewing this issue but did not find that those courts' rejection of IGCC in a BACT step 1 evaluation persuaded it to change its view.

In *Blue Skies*, the Court of Appeals for the Seventh District of Texas analyzed the Texas statutory definition of BACT, which, because the federal definition is incorporated by reference into the state definition, is identical to the CAA BACT definition. 283 S.W.3d at 534 & n.7. The Texas Court of Appeals concluded that "the BACT definition clearly provides that only those control technologies that can be *applied* to the *proposed* major source be considered in the BACT analysis." *Id.* at 535 (emphasis in original). According to the court, "the only control technologies that must be considered in a BACT analysis are those control technologies that can be *incorporated into or added to* the facility as proposed by the applicant," *id.* (emphasis added), and because the court found that there was no evidence that IGCC is "a process that could be applied to the pulverized coal power plant proposed" by the applicant, the court concluded that IGCC need not be considered as BACT, *id.* at 537. In so concluding, the court relied on an extremely narrow definition of the terms "applied" and "application."<sup>76</sup> *See id.*

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<sup>75</sup> The participants actually cited *Blue Skies Alliance v. Tex. Comm'n on Env'tl. Quality*, No. 07-07-0306-CV (Tex. App. Jan. 29, 2009). On April 14, 2009, however, after the participants had filed their briefs citing the case, the Texas Court of Appeals withdrew its January 29, 2009 opinion and issued an opinion in its place. *Blue Skies*, 283 S.W.3d at 528. The discussion below refers solely to the second opinion.

<sup>76</sup> The term "application" has several definitions, including "employment as a means: specific use" as in "the [application] of certain new techniques" as well as "the act of laying on or of bringing into contact." Webster's Third New International Dictionary at 105 (1993). The word "apply" similarly has several definitions, including "to make use of as suitable, fitting, or relevant," "to put to use esp[ecially] for some practical purpose," "to use for a particular purpose or in a particular case," "to put  
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at 534-37. In fact, under the Texas Court of Appeal's reading of the statute, only add-on controls – because, according to the court, only these could be *applied* to the proposed source – could be considered BACT.<sup>77</sup> This reading is inconsistent with the language, purpose, and legislative history of the CAA as well as EPA's longstanding interpretation and practice. Thus, the Board respectfully disagrees with the Texas Court of Appeal's statutory analysis and its conclusion based on its interpretation of the statute.<sup>78</sup>

More recently, the Georgia Court of Appeals held that the Superior Court had “erred by ruling *as a matter of law* that the CAA required consideration of IGCC technology in the BACT analysis” for a proposed coal-fired power plant.<sup>79</sup> *Longleaf Energy*, 2009 WL 1929192, at \*6 (emphasis added). The Superior Court had determined, based on its interpretation of the CAA and a regulation describing types of electric utility steam generating units, that “the CAA *mandated* consideration of IGCC technology in the BACT analysis.”<sup>80</sup> *Id.* at \*5 (emphasis ad-

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into effect,” and “to place in contact: \* \* \* lay or spread on: overlay \* \* \* : superpose.” *Id.* at 105. While the Texas court appears to have relied on the latter definitions of these words (i.e., “the laying on” or “superposing”), which, notably, are the much narrower definitions, the Board believes it more appropriate to rely on the other, broader definitions (i.e., “employment as a means” and “specific use” and “make use of as suitable, fitting, or relevant”). This broader reading is more consistent with the Agency's longstanding interpretation of the statutory term “application,” as evidenced by the 1990 NSR Manual's description of BACT step 1 as including both “inherently lower emitting processes and/or practices” and “add-on controls.” *See supra* Part III.B.2 (citing NSR Manual at B.10); *Knauf*, 8 E.A.D. at 129 (explaining that BACT analysis involves considering add-on control technology as well as inherently lower polluting processes).

<sup>77</sup> *See supra* note 76.

<sup>78</sup> The court also placed significant emphasis on the *proposed* source. *See, e.g., Blue Skies*, 283 S.W.3d at 534, 535. In so doing, the Texas court appears to implicitly rely on the applicant's planned design without taking a “hard look” at which design elements are truly inherent for the applicant's purpose and which elements may be changed. *See id.* at 534-37. Thus, the Texas court's analysis is also inconsistent with the approach the Board outlined in *Prairie State* regarding permissible redesigns of facilities. 13 E.A.D. at 23, 25-28; *see also* NMU, 14 E.A.D. at 301-03. The Board respectfully disagrees with the court's analysis for this additional reason as well.

<sup>79</sup> The Superior Court had invalidated an air quality permit issued by the Environmental Protection Division (“EPD”) of the Georgia Department of Natural Resources pursuant to the Georgia SIP and upheld by an administrative law judge (“ALJ”) following an evidentiary hearing under the state's Administrative Procedure Act. *Longleaf Energy*, 2009 WL 1929192, at \*2. The EPD and the ALJ had not considered IGCC because both had determined that IGCC would redefine the design of the proposed PC power plant. *Id.* at \*5. The permit at issue in this case is one of the permits to which Desert Rock refers in its equal protection argument. *See supra* Part III.A.3.b(iii)(a).

<sup>80</sup> Based on language contained in a former EPA regulation, 40 C.F.R. § 60.41Da (2008), the Superior Court had concluded that the proposed power plant was the same type of “major emitting facility” within the meaning of the CAA no matter whether it was a PC plant or an IGCC plant, and thus, according to the court, the CAA mandated that IGCC be considered in the BACT analysis. *See* 2009 WL 1929192, at \*5-6.

ded). In its decision, the Georgia Court of Appeals rejected the Superior Court's statutory and regulatory interpretation because the lower court had ignored the "redefining the source" policy and because EPA, in 2009, had amended the regulation upon which it had based its interpretation, eliminating the "relied-upon portion of the regulation." *Id.* at \*6. Significantly, the Georgia Court of Appeals noted that the Superior Court had not reviewed the administrative record evidence concerning the redesign of the power plant that would be necessitated by IGCC. *Id.* at \*5.

Because the Georgia Court of Appeal's holding on the IGCC BACT issue is based on a statutory interpretation question that was not raised in the present matter, that court's analysis is inapplicable here. Furthermore, neither the Georgia Court of Appeals nor the Superior Court specifically focused on whether the administrative record supported the agency's and the ALJ's determination that IGCC would redefine the source.<sup>81</sup> *See id.* at \*5-6. The Board's analysis in *Prairie State*, *NMU*, and today's decision emphasize that such an analysis of the underlying administrative record is an essential component of a supportable BACT decision that a proposed control technology redefines the source.

#### 4. *Summary of Conclusions Regarding the Region's IGCC Analysis*

In sum, while the Region has broad discretion in determining whether imposition of a control technology would "redefine the source," the Board concludes that, based on the administrative record for this case, the Region's analysis is inadequate for two reasons. First, the Region did not provide a rational explanation of why IGCC would redefine the source, especially when the applicant itself had indicated in its initial application that IGCC was a technology that could be considered for such a facility (i.e., could satisfy its business purpose), thereby suggesting that IGCC would not redefine the source. *See Prairie State*, 13 E.A.D. at 23-28 (describing proper analysis for concluding that a redesign is impermissible); *Knauf*, 8 E.A.D. at 139-42 (remanding permit because permit issuer had failed to take sufficiently hard look at design issues). Second, the Region failed to adequately explain its conclusion in light of previously issued federal permits at similar facilities in which IGCC had been considered as a BACT step 1 production process and had not been considered a "redefinition of the source." *See NMU*, 14 E.A.D. at 331 ("[A]ny contention that particular fuel choices or related factors would improperly 'redefine the source' must be thoroughly explained and supported \* \* \* ."); *see also Knauf*, 8 E.A.D. at 140 (remanding BACT issue where Board could not tell, "based on the record information and arguments made on appeal," whether a particular control technology and associated limit selected

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<sup>81</sup> It is possible that, on remand, the Superior Court may reexamine the IGCC question to determine whether the administrative record supports the conclusion that IGCC was an impermissible redesign of the proposed source. *See id.* at \*5-6, 11.

truly qualified as BACT); *Masonite*, 5 E.A.D. at 566-69 (remanding PSD permit because region's BACT analyses were incomplete); *In re Austin Powder Co.*, 6 E.A.D. 713, 720 (EAB 1997) (remanding permit because region's rationale was unclear); *In re GSX Servs. of S.C., Inc.*, 4 E.A.D. 451, 454 (EAB 1992) (remanding permit because, even though establishing the permit term was an exercise of discretion, record did not "reflect the 'considered judgment' necessary to support the region's determination"). The Board therefore finds that the Region abused its discretion here, based on the current administrative record, and concludes that the Permit should be remanded on this ground so that the Region may either provide a further explanation for its determination that IGCC would "redefine the source" consistent with this decision or reconsider IGCC under step 1 of its BACT analysis. See, e.g., *Austin Powder*, 6 E.A.D. at 720 (remanding permit so that region could either clarify its basis or change permit condition). Because a new step 1 BACT determination could have widespread impacts on the entire Permit, the Board concludes it is appropriate to remand the Permit in its entirety on this ground.<sup>82</sup>

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<sup>82</sup> Although it is not necessary in this Remand Order to reach the issue of whether the CAA section 169 statutory language requires consideration of IGCC, a question that was raised by New Mexico and the NGO Petitioners, the Board notes that the legislative history of the "innovative fuel combustion techniques" language suggests there may be some outer limits to the "redefining the source policy." During the 1977 debate, Senator Huddleston proposed additional, clarifying language to the committee's proposed section 169 language – the insertion of "innovative combustion techniques" after the word "treatment." He stated:

The definition in the committee bill of best available control technology indicates a consideration for various control strategies by including the phrase "through application of production processes and available methods systems, and techniques, including fuel cleaning or treatment." And I believe it is likely that the concept of BACT is intended to include such technologies as low Btu gasification and fluidized bed combustion. But, this intention is not explicitly spelled out, and I am concerned that without clarification, the possibility of misinterpretation would remain.

*It is the purpose of this amendment to leave no doubt that in determining best available control technology, all actions taken by the fuel user are to be taken into account - be they the purchasing or production of fuels which may have been cleaned or up-graded through chemical treatment, gasification, or liquefaction; use of combustion systems such as fluidized bed combustion which specifically reduce emissions and/or the post-combustion treatment of emissions with cleanup equipment like stack scrubbers.*

123 Cong. Rec. S9421, 9435 (1977) (statement of Sen. Huddleston) (emphasis added), *reprinted in* Comm. on Env't and Pub. Works, 95th Cong., Legislative History of the Clean Air Act Amendments of 1977, at 1054 (1978). This clarification of the statutory terms was accepted.

Based on Senator Huddleston's clarification and his explanation of the addition of the language "innovative combustion techniques" to CAA section 169, it appears that the amendments were intended to broaden the definition of BACT so that actions such as the production of gas from coal via

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#### IV. CONCLUSION

Based on the foregoing, the Board remands the Permit on two independent grounds. The Board first concludes that it is appropriate to grant the Region's motion for voluntary remand. The Board also concludes that, based upon a review of the administrative record, the entire Permit should be remanded to the Region because the Region abused its discretion in declining to consider IGCC in step 1 of the BACT analysis for the Facility. Accordingly, the Board REMANDS the Permit to the Region in its entirety, and PSD Appeal Nos. 08-03, 08-04, 08-05, and 08-06 are DISMISSED. The dismissal of Appeal Nos. 08-03 through 08-06 is without prejudice to the filing of new petitions for review with the Board pursuant to 40 C.F.R. § 124.19 by Petitioners following the Region's issuance of a final permit decision on remand.<sup>83</sup> An appeal of the Region's decision on remand is required to exhaust administrative remedies. 40 C.F.R. § 124.19(f)(1)(iii).

So ordered.

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gasification would generally be considered in the BACT analysis. While the "redefining the source policy" may play a role in determining on a case-by-case basis what technologies should be considered in a BACT analysis for a facility, as the Seventh Circuit intimated in *Sierra Club v. EPA*, an interpretation that would completely read a statutory term out of the BACT definition would be questionable. *Sierra Club*, 499 F.3d at 656; *see also NMU*, slip op. at 27 (acknowledging the Seventh Circuit's language in *Sierra Club*).

<sup>83</sup> In any petitions for review filed after the Region's issuance of a new permit decision, Petitioners will be able both to reassert objections already raised in their current petitions and to assert objections based on any changes made to the permit decisions on remand. Persons other than Petitioners, on the other hand, will only be able to petition the Board for review of the new permit decision to the extent of any changes made on remand. *See* 40 C.F.R. § 124.19(a).



# Enforcement Alert

Publication no. EPA 325-N-20-001

November 2020

## EPA Reminder About Inappropriate Use of AP-42 Emission Factors

### Purpose

This purpose of this Enforcement Alert is to remind permitting agencies, consultants, and regulated entities that improperly using AP-42 emission factors can be costly to their businesses, inefficient, and in some circumstances, can subject regulated entities to enforcement and penalties. The Environmental Protection Agency (EPA) is concerned that some permitting agencies, consultants, and regulated entities may incorrectly be using AP-42 emission factors in place of more representative source-specific emission values for Clean Air Act permitting and compliance demonstration purposes.

### Consequences of Using AP-42 Factors

Permitting agencies, consultants, and regulated entities should be aware that even emission factors with more highly rated AP-42 grades of “A” or “B” are only based on averages of data from multiple, albeit similar, sources (See the Attachment for an overview of the history of AP-42 emission factors and the AP-42 emission factor rating system). Accordingly, these factors are not likely to be accurate predictors of emissions from any one specific source, except in very limited scenarios. While emission factors are helpful in making emission estimates for area-wide inventories for specific source types, AP-42 provides the following warning:

“Use of these factors as source-specific permit limits and/or as emission regulation compliance determinations is not recommended by EPA. Because emission factors essentially represent an average of a range of emission rates, approximately half of the subject sources will have emission rates greater than the emission factor and the other half will have emission rates less than the factor. As such, a permit limit using an AP-42 emission factor would result in half of the sources being in noncompliance.”<sup>1</sup>

With the advent of 1-hour and short-term National Ambient Air Quality Standards (NAAQS), permit limits must be able to account for short term fluctuations. AP-42 emission factors also do not account for short term variation in emissions as the emission factors are intended for use in developing area-wide annual or triannual inventories. In developing emission factors, test data are typically taken from normal operating conditions and generally avoid conditions that can cause short-term fluctuations in emissions. These short-term fluctuations in emissions can stem from variations in process conditions, control device conditions, raw materials, ambient conditions, or other similar factors. This means that if facilities use AP-42 emission factors as permit limits, facilities increase their chances of violating their short-term permit limits. It also increases the likelihood of a geographic area’s non-compliance with the NAAQS.

**DISCLAIMER:** This document aims to explain the application of certain EPA regulatory provisions using plain language. Nothing in this Alert revises or replaces any regulatory provisions, any other part of the Code of Federal Regulations, the Federal Register, or the Clean Air Act. Following the approaches for determining a single storage vessel’s potential for VOC emissions and attempting to comply with the closed vent system requirements as discussed in this Alert do not equate to or guarantee compliance with the Clean Air Act, its implementing regulations, and associated state/local requirements. For more information, visit: [www.epa.gov/compliance](http://www.epa.gov/compliance).

<sup>1</sup> AP-42, Fifth Edition Compilation of Air Pollutant Emissions Factors, Volume 1: Stationary Point and Area Sources. Introduction, p. 2 (emphasis added).



It is also important to understand that there is a great deal of variability in the emissions data that are used to generate the emission factors. This variability is not necessarily reflected in the emission factor. AP-42 describes this as follows:

“The extent of between-source variability that exists, even among similar individual sources, can be large depending on process, control system, and pollutant. Although the causes of this variability are considered in emission factor development, this type of information is seldom included in emission test reports used to develop AP-42 factors. As a result, some emission factors are derived from tests that may vary by an order of magnitude or more. Even when the major process variables are accounted for, the emission factors developed may be the result of averaging source tests that differ by factors of five or more.”<sup>2</sup>

In addition to potential permit noncompliance, or increased risk of area noncompliance with the NAAQS, using an emission factor as an emission limit could have monetary implications for an individual source or permitting agency. For example, many permitting agencies collect permitting fees based on the amount of pollution emitted. If a facility uses an emission factor to estimate and report emissions, but the actual emission rate is lower than the emission factor, then the facility will report more emissions and consequently pay more in fees. On the other hand, if a facility emits at a rate above the emission factor, not only is the source violating its permit limit and the Clean Air Act, it is also not paying the appropriate amount in fees.

Another potential monetary implication for facilities is an enforcement action assessing penalties for violating the Clean Air Act. As described in a 2006 report issued by the EPA Inspector General:

“...according to EPA enforcement records, three industries – petroleum refineries, wood products, and ethanol production – operated with insufficient control equipment primarily because emission limits were significantly underestimated due to the emission factors used. EPA, through separate enforcement actions, required companies in these industries to install additional emission controls, resulting in the combined reduction of over 1,000,000 tons of pollutants.”<sup>3</sup>

For example, the EPA Inspector General’s 2006 report documented an EPA investigation in the Wood Products industry that found a nationwide pattern of Clean Air Act violations by one company. EPA found that the company had used an AP-42 emission factor designated as “poor” for volatile organic compound (VOC) emissions that resulted in the company underestimating such emissions and claiming that its facilities were not subject to permitting requirements. To resolve the violations, the company entered into a consent decree with the United States, which required the company to pay a civil penalty of \$1.1 million and to install air pollution control equipment at a cost of \$70 million.<sup>4</sup>

One example of a present-day concern is the use of a default vapor pressure value for estimating VOC emissions from heated tanks that store heavy refinery liquids such as No. 6 fuel oil. The true vapor pressure (TVP) of a stored liquid is important when calculating the emissions from tanks using the equations in AP-42, Chapter 7, Liquid Storage Tanks. The default vapor pressure is only an estimate and may not be correct for every blend of No. 6 fuel oil. Direct emissions testing of No. 6 fuel oil tanks and TVP testing in 2012 and 2013, suggested that in those cases the use of the default vapor pressure in AP-42 had resulted in emissions estimates that were understated by a factor of 100 for permitting and reporting purposes. Reliance on the default vapor pressure in AP-42 and the resulting emission factors, instead of directly measuring VOC emissions and vapor pressure, can be very costly for businesses as shown by two recently concluded cases, summarized in the following two boxes.

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<sup>2</sup> AP-42, Fifth Edition Compilation of Air Pollutant Emissions Factors, Volume 1: Stationary Point and Area Sources. Introduction, p. 3 (emphasis added).

<sup>3</sup> U.S. EPA Office of Inspector General, *EPA Can Improve Emissions Factors Development and Management*, Report No. 2006-P-00017, March 22, 2006.

<sup>4</sup> Id.

**Sprague Resources LP** operates heated asphalt and No. 6 fuel oil storage tanks at seven facilities across New England. Applying VOC testing results rather than AP-42 estimates, EPA found that Sprague had unpermitted facilities that required permits, and also had facilities with permits that failed to fully account for VOC emissions. Sprague entered into a settlement with the United States and the Commonwealth of Massachusetts that required the company to pay \$350,000 civil penalties, obtain revised state air pollution control permits, limit the amount of asphalt and No. 6 fuel oil stored in and passed through the tanks at six facilities, and provide odor controls on tanks at two facilities.

**Global Partners LP** operates heated asphalt and No. 6 fuel oil storage tanks at a facility in South Portland, Maine. Applying VOC testing results rather than AP-42 estimates, EPA found that Global's permit failed to fully account for VOC emissions. Global entered into a settlement with the United States that required the company to obtain a revised state air pollution control permit, limit the amount of asphalt and No. 6 fuel oil stored in and passed through the tanks at the facility, install odor controls on tanks, pay a \$40,000 penalty, and invest \$150,000 in a local wood-stove replacement project.

Regulated entities of any size who voluntarily discover, promptly disclose, expeditiously correct, and take steps to prevent recurrence of potential violations may be eligible for a reduction or elimination of any civil penalties that otherwise might apply. Most violations can be disclosed and processed via EPA's automated online "eDisclosure" system (see <https://www.epa.gov/compliance/epas-edisclosure>). To learn more about the EPA's violation disclosure policies, including conditions for eligibility, please review EPA's Audit Policy website at <https://www.epa.gov/compliance/epas-audit-policy>. Many states also offer incentives for self-policing; please check with the appropriate state agency for more information.

## What Can Be Done?

Consultants and facility owners/operators should obtain and use the most representative emissions data, which in many cases may be source-specific emissions data, when determining applicability, applying for a permit, or demonstrating compliance with permit limits.

Various EPA publications (e.g., <https://www.epa.gov/emc>) describe the benefits and limitations of different ways to quantify source-specific emissions. These techniques in order of accuracy are:

- **Continuous Emissions Monitoring System (CEMS)** – CEMS offers a highly accurate source-specific method that continuously monitors the emissions coming out of a particular stack; however, although the accuracy of this method is high, the cost is also the highest at \$20,000-\$50,000 per year.
- **Stack Testing** – Like a CEMS, source-specific data are generated at a particular stack but emissions are only measured for a specific time, typically for a few hours during normal operations. Costs for stack testing typically run \$20,000, but testing may only be necessary every 2 to 5 years.
- **Vendor Guarantees and Stack Test Data from Similar Facilities** – If representative source-specific data cannot be obtained, emissions information from equipment vendors, particularly emission performance guarantees or actual test data from similar equipment, is a better source of information for permitting decisions than an AP-42 emission factor.
- **Material Balance Calculations** – While the material balance calculations are not generally considered as accurate as direct measurements, they may provide more reliable average emission estimates for certain sources where a high percentage of material is lost to the atmosphere (e.g., solvent VOC emissions). The costs for recordkeeping are approximately \$2,000-\$10,000 per year. This method works well for materials and processes that are well understood.
- **Optical Remote Sensing** – Measurement techniques involving differential absorption light detection and ranging (known as DIAL) and solar occultation flux or SOF can be used to measure emissions from sources such as coke ovens, storage tanks, wastewater treatment plants, and process units that are otherwise difficult to measure by other means. Measurement bias on the order of  $\pm 30$  percent is expected but the data can be more accurate than engineering estimates or emission factors.
- **Emission Factors** – When source-specific emissions or other more reliable approaches are unavailable, AP-42 emission factors may be the only way to estimate emissions. Again, the limitations of the factor in accurately representing the facility's emissions and the environmental/financial risk of using the emission factor for a particular situation should be carefully considered. **Remember, AP-42 emission factors should only be used as a last resort. Even then the facility assumes all risk associated with their use!**

## Attachment – History of AP-42

Before the EPA existed, the U.S. Public Health Service (PHS) published “A Compilation of Air Pollutant Emission Factors” in 1968.\* The purpose of the report was to assist the various agencies responsible for compiling air pollution emission inventories for communities across the nation by providing them with relevant data. PHS recognized that

\* The PHS assigned the number 999-AP-42 to this publication. 999 was the series number, AP was an abbreviation for air pollution, and 42 was the document number. Thus, the origin of today’s AP-42!

measuring each individual source of air pollution in a particular airshed was impractical, and so, to simplify the airshed emission inventory process, while still maintaining a reasonably accurate inventory, PHS developed emission factors based on the technical literature and a limited number of source-specific tests. The resulting emission factors were simple averages of the rate at which pollutants were emitted from the burning or processing of a given quantity of material. In some cases, emission factors were based on only one or two data points.

With the creation of the EPA, publication of the emission factors was continued with “Compilation of Air Pollutant Emission Factors, Second Edition,” by the EPA Office of Air Quality Planning and Standards in 1973.

The 3<sup>rd</sup> and 4<sup>th</sup> editions of AP-42 were released in 1977 and 1985. EPA published the most recent AP-42, the 5<sup>th</sup> edition in 1995<sup>5</sup>, and has published multiple supplements and updates since. Currently, AP-42 contains more than 21,500 emission factors for over 200 air pollutants. Within AP-42, each emission factor is given a rating between “A” (excellent) and “E” (poor) (see Table 1 below). It is important to note that half of the emission factors are rated “D” or “E” and one-fifth are unrated. This means that less than one-third of the emission factors are rated between “Excellent” and “Average.”

As we work to improve our ability to estimate emissions nationally, the grading in AP-42 helps us better understand the quality of the data. But even factors that are rated “A” or “B” are not designed to be used by a single source where other, more reliable, site-specific, data are available.

**Table 1: Explanation of AP-42 Emission Factor Quality Ratings**

Rating	Explanation
<b>“A” – Excellent</b>	Emission factor is developed from tests conducted with sound, or generally sound, methodology. Test data are from many randomly chosen facilities and the source category population is sufficiently specific to minimize variability. Data may, or may not, be reported in enough detail for adequate validation.
<b>“B” – Above Average</b>	Same as “A,” but test data are from a “reasonable number” of facilities. Although no specific bias is evident, it’s not clear if the facilities represent a random sample of the industry. The source category population is sufficiently specific to minimize variability.
<b>“C” – Average</b>	Same as “B,” but the factor can be developed from an unproven or new methodology. Test data may be lacking a significant amount of background information. Although no specific bias is evident, it’s not clear if the facilities tested represent a random sample of the industry. The source category population is specific enough to minimize variability.
<b>“D” – Below Average</b>	Same as “C,” but test data are from a small number of facilities, and there may be reason to suspect the facilities do not represent a random sample of the industry. There may also be evidence of variability within the source population.
<b>“E” – Poor</b>	Factor is developed from: (1) tests based on an unproven or new methodology, or tests that may be lacking a significant amount of background information, or (2) tests based on a generally unacceptable method, but the method may provide an “order of magnitude” value for the source. Facilities tested may not represent a random sample of the industry and there is evidence of variability within the source category population.

<sup>5</sup> AP-42, Fifth Edition Compilation of Air Pollutant Emissions Factors, Volume 1: Stationary Point and Area Sources. Introduction, pp. 9-10.

FEDERAL INFORMATION  
PROCESSING STANDARDS PUBLICATION

1979 AUGUST 15

U.S. DEPARTMENT OF COMMERCE / National Bureau of Standards

STANDARD  
INDUSTRIAL  
CLASSIFICATION  
(SIC) CODESFEDERAL GENERAL DATA STANDARD  
REPRESENTATIONS AND CODESJK  
468  
.A3A8  
NO. 66  
1979

**U.S. DEPARTMENT OF COMMERCE, Juanita M. Kreps, *Secretary***

**Jordan J. Baruch, *Assistant Secretary* for Science and Technology**

**NATIONAL BUREAU OF STANDARDS, Ernest Ambler, *Director***

**Foreword**

Federal Information Processing Standards Publications of the National Bureau of Standards are adopted and promulgated under the provisions of Public Law 89-306, and Part 6 of Title 15 Code of Federal Regulations. These standards are required for implementation by Federal agencies in the acquisition, development and use of automated information systems and in the interchange of data between and among agencies and with the public. The use of such standards which are adopted after extensive review by Federal agencies, industry and the public are intended to reduce government costs and improve the effectiveness of government services.

Comments concerning Federal Information Processing Standards Publications are welcomed, and should be addressed to the Director, Institute for Computer Sciences and Technology, National Bureau of Standards, Washington, D.C. 20234.

Ernest Ambler, *Director*

**Abstract**

This standard provides classifications, short titles, and codes for representing industries. The general concept of an industry is one of a group of establishments with similar economic activities. The SIC codes, initially developed by the Office of Management and Budget, are currently being maintained and published by the DOC Office of Federal Statistical Policy and Standards, the organization to which this function has been transferred. Their implementation in Federal ADP systems will avoid unnecessary and costly incompatibilities in the collection, processing and dissemination of data.

Key words: Computers; data processing; Federal Information Processing Standards Publication; industries; representations and codes; Standard Industrial Classification.

Nat. Bur. Stand. (U.S.) Fed. Info. Process. Stand. Publ. (FIPS PUB) 66, 23 pages.

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## Federal Information Processing Standards Publication 66

1979 AUGUST 15

ANNOUNCING THE STANDARD FOR

### STANDARD INDUSTRIAL CLASSIFICATION (SIC) CODES



FIPS PUB 66

Federal Information Processing Standards Publications are issued by the National Bureau of Standards pursuant to the Federal Property and Administrative Services Act of 1949, as amended, Public Law 89-306 (79 Stat. 1127), Executive Order 11717 (38 FR 12315, dated May 11, 1973) and Part 6 of Title 15 Code of Federal Regulations (CFR).

**Name of Standard.** Standard Industrial Classification (SIC) Codes, (FIPS PUB 66).

**Category of Standard.** Federal General Data Standard, Representations and Codes.

**Explanation.** This standard provides classifications, short titles, and codes for representing industries. The general concept of an industry is one of a group of establishments with similar economic activities. The SIC codes, initially developed by the Office of Management and Budget, are currently being maintained and published by the DOC Office of Federal Statistical Policy and Standards, the organization to which this function has been transferred.

**Approving Authority.** Secretary of Commerce.

**Maintenance Agency.** Department of Commerce, Office of Federal Statistical Policy and Standards, Washington, D.C. 20230.

Questions concerning the list of industries and codes should be addressed to the Maintenance Agency. The Maintenance Agency will provide to the National Bureau of Standards changes relating to SIC industries, their definitions and codes. Users of this FIPS PUB who need to be notified of changes to this standard can complete the change request form included on page 23 of this publication and return it as indicated to the National Bureau of Standards. Change notices will be issued by NBS based upon the information provided by the Office of Federal Statistical Policy and Standards.

**Cross Index.** The SIC codes adopted by this standard have been taken from the Standard Industrial Classification Manual dated 1972, published by the Office of Management and Budget, and the 1977 Supplement, published by the Office of Federal Statistical Policy and Standards, the organization to which this function has been transferred. Inquiries relating to these publications should be directed to the Maintenance Agency indicated above.

**Applicability.** This standard is prescribed for the interchange of data among agencies and between agencies and the public including industry and State and local governments. Use within agency data systems is encouraged when such use contributes to operational benefits, efficiency, or economy.

This classification should be used whenever data for domestic establishments need to be classified industrially. For international comparisons, the SIC can be converted to the International Standard Industrial Classification (ISIC) with minor exceptions where the U.S. has a different classification structure.

**Implementation Schedule.** Provisions of this standard become effective six months after publication of the FIPS PUB.

Where data interchange is based upon reporting requirements in Federal or departmentwide programs, the implementation of this standard needs to be coordinated by the organization having

the authority to impose the reporting requirement. Exceptions, deferments and revisions of this standard will be considered under the provisions of section 6.8, Part 6 of Title 15 CFR.

**Specifications.** Federal Information Processing Standard 66 (FIPS 66), Standard Industrial Classification (SIC) Codes (affixed).

**Waiver Procedure.** Heads of agencies may request the provisions of this standard be waived in instances where its use would seriously affect the capability of the agency in performing its operational mission. Such waiver requests will be reviewed and approved by the Secretary of Commerce. Correspondence setting forth the reasons and justification for the waiver should be included in the waiver request.

Forty-five days should be allowed for review and response by the Secretary of Commerce. Waiver requests shall be submitted to the Secretary of Commerce, Washington, D.C. 20230, and labeled as a Request for Waiver to a Federal Information Processing Standard. No action will be taken by the agency to deviate from the standard prior to the receipt of a waiver approval response from the Secretary of Commerce.

**Qualifications.** None.

**Where To Obtain Copies.** Copies of this publication are for sale by the National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia 22161. When ordering, refer to Federal Information Processing Standards Publication 66 (NBS-FIPS-PUB-66), and title. When microfiche is desired, this should be specified. Copies of the Standard Industrial Classification Manual 1972 and the 1977 Supplement are for sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402, Stock Nos. 4101-0066 and 003-005-00176-0, respectively. Payment may be made by check, money order, or deposit account.

Magnetic tape copies of the official titles, short titles and index items are also for sale by the National Technical Information Service, U.S. Department of Commerce, Springfield, Virginia 22161.

**Special Information.** In order to provide a better understanding of the principles of the SIC, a portion of the Introduction to the 1972 Manual follows. The SIC Manual has been scheduled for a revision to be published in 1982.

## Introduction

# THE STANDARD INDUSTRIAL CLASSIFICATION OF ESTABLISHMENTS

### Purpose and Scope of the Classification

The Standard Industrial Classification was developed for use in the classification of establishments by type of activity in which they are engaged; for purposes of facilitating the collection, tabulation, presentation, and analysis of data relating to establishments; and for promoting uniformity and comparability in the presentation of statistical data collected by various agencies of the United States Government, State agencies, trade associations, and private research organizations. The Standard Industrial Classification for establishments differs from a classification for enterprises or companies. An Enterprise Standard Industrial Classification related to the Standard Industrial Classification has been developed for use in classifying enterprises. Other classifications have been developed for use in the classification of commodities or products and also for occupations.

The Standard Industrial Classification is intended to cover the entire field of economic activities: agriculture, forestry, fishing, hunting, and trapping; mining; construction; manufacturing; transportation, communications, electric, gas, and sanitary services; wholesale and retail trade; finance, insurance, and real estate; personal, business, repair, and other services; and public administration.

### Principles of the Classification

In preparing the Classification, the Technical Committee on Industrial Classification was guided by the following general principles:

- (1) The Classification should conform to the existing structure of American industry.
- (2) Each establishment is to be classified according to its primary activity.
- (3) To be recognized as an industry, the group of establishments constituting the proposed classification must be statistically significant in the number of persons employed, the volume of business done, and other measures of economic activity.

**Definition of Establishments.** For purpose of this classification, an establishment is an economic unit, generally at a single physical location where business is conducted or where services or industrial operations are performed. (For example: a factory, mill, store, hotel, movie theater, mine, farm, ranch, bank, railroad depot, airline terminal, sales office, warehouse, or central administrative office.)

Where distinct and separate economic activities are performed at a single physical location (such as construction activities operated out of the same physical location as a lumber yard), each activity should be treated as a separate establishment wherever: (1) no one industry description in the classification includes such combined activities; (2) the employment in each such economic activity is significant; and (3) reports can be prepared on the number of employees, their wages and salaries, sales or receipts, and other establishment type data.

For activities such as construction, transportation, communications, electric, gas and sanitary services, and similar physically dispersed operations, establishments are represented by those relatively permanent main or branch offices, terminals, stations, etc., which are either (1) directly responsible for supervising such activities, or (2) the base from which personnel operate



to carry out these activities. Hence, the individual sites, projects, fields, networks, lines or systems of such dispersed activities are not ordinarily considered to be establishments.

An establishment is not necessarily identical with the enterprise or company which may consist of one or more establishments. Also, it is to be distinguished from subunits, departments, or divisions. Supplemental interpretations of the definition of an establishment are included in the industry descriptions of the Standard Industrial Classification where appropriate. Central administrative offices and auxiliary units also further described in the manual are recognized as special types of establishments.

### **Structure of the Classification.**

The structure of the classification makes it possible to tabulate, analyze, and publish establishment data on a division, a two-digit, a three-digit, or a four-digit industry code basis, according to the level of industrial detail considered most appropriate. An agency may use additional subdivisions within specific four-digit industries in adopting this classification for its own use, while still retaining comparability with the classifications used by other agencies.

It should be noted that the digit "9" that usually appears in the third or fourth digit position of the classification code designates miscellaneous three-digit groups or four-digit industries covering establishments "not elsewhere classified." These residual establishments do not usually constitute homogeneous primary activity groups; for purposes of this classification system they are grouped together and treated as a separate industry to retain the homogeneity of the other industries in the group.

**Change in the Treatment of Ownership Characteristics.** The classification has been changed so that all establishments primarily engaged in the same kind of economic activity are classified in the same four-digit industry, regardless of their types of ownership; hence, their owners may include such diverse legal organizations, as corporations, partnerships, individual proprietors, government agencies, joint ventures, etc.

This change from the 1967 edition removes "Government" as an industry division, per se, and treats it as an ownership characteristic. Government establishments, therefore, are now classified by their primary economic activity, rather than by type of owner. The ownership classification system shown in the 1972 SIC Manual can provide continuity between the 1967 SIC and the present one. Where applicable, at least summary data should be published separately for the private and Government establishments constituting an industry or industry group.

### **Basis of Code Assignment**

Each establishment is assigned an industry code on the basis of its primary activity, which is determined by its principal product or group of products produced or distributed, or services rendered. Ideally, the principal product or service should be determined by its relative share of "value added" at the establishment. In practice, however, it is rarely possible to obtain this measure for individual products or services; typically, it is necessary to adopt some other criterion which may be expected to give approximately the same results in determining the primary activity of an establishment.



**Federal Information  
Processing Standards Publication 66**

1979 August 15

SPECIFICATIONS FOR



**STANDARD INDUSTRIAL CLASSIFICATION (SIC) CODES**

**Name of Standard.** Standard Industrial Classification (SIC) Codes.

**Category of Standard.** Federal General Data Standard, Representations and Codes.

**Explanation.** This standard provides classifications, short titles, and codes for representing industries. The general concept of an industry is one of a group of establishments with similar economic activities.

**Specifications.** Listed in the Table are the short titles of the Standard Industrial Classification with their assigned numeric codes.

In applications where this standard is implemented in fixed length fields, the most significant characters of the code should be left justified. In these instances when an SIC code is less than the specified field length, the remaining character positions will be filled with a space character.

SIC codes are structured based upon classification significance. Accordingly, the least significant digits of the code (to the right) are dependent upon the more significant digits (to the left) for their meaning. Consequently, the least significant digits of the code cannot be used separate and apart from their associated more significant digits.

## TABLE

### List of SIC Codes and Their Short Titles

The official SIC titles of the divisions and the two-, three-, and four-digit industries are those shown in the SIC Manual. For various reasons, including presentation of statistical tables, it is desirable to have a standard list of short SIC titles so that all agencies may use the same short titles for the same codes as long as the titles fit the space requirements of the publication.

The standard short titles have been limited to 36 spaces for four-digit SIC codes and 38 spaces for two-digit and three-digit codes. Where a two-digit or three-digit group contains only a single four-digit industry, then the two-digit or three-digit titles are allowed 36 rather than 38 spaces.

It is understood, of course, that just as a title itself is not sufficient to define an industry, so too a short title may not appear to represent the same content as the official title. Content can only be defined by reference to the official title and description.

In view of the fact that the standard short titles are intended to apply to publication copy, abbreviations of individual words are avoided if at all possible. However in a number of instances, abbreviations are necessary, as follows:

admin .....	administration	Intl .....	International
& .....	and	misc .....	miscellaneous
exc. or ex .....	except	nec .....	not elsewhere
Fed .....	Federal		classified
incorp .....	incorporated	Res .....	Reserve

## List of SIC Codes and Their Short Titles

**A. AGRICULTURE, FORESTRY, AND FISHING**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
<b>01</b>	<b>AGRICULTURAL PRODUCTION— CROPS</b>	<b>0272</b>	Horses and other equines
<b>011</b>	Cash Grains	<b>0279</b>	Animal specialties, nec
<b>0111</b>	Wheat	<b>029</b>	General Farms, Primarily Livestock
<b>0112</b>	Rice	<b>0291</b>	General farms, primarily livestock
<b>0115</b>	Corn	<b>07</b>	<b>AGRICULTURAL SERVICES</b>
<b>0116</b>	Soybeans	<b>071</b>	Soil Preparation Services
<b>0119</b>	Cash grains, nec	<b>0711</b>	Soil preparation services
<b>013</b>	Field Crops, Except Cash Grains	<b>072</b>	Crop Services
<b>0131</b>	Cotton	<b>0721</b>	Crop planting and protection
<b>0132</b>	Tobacco	<b>0722</b>	Crop harvesting
<b>0133</b>	Sugar crops	<b>0723</b>	Crop preparation services for market
<b>0134</b>	Irish potatoes	<b>0724</b>	Cotton ginning
<b>0139</b>	Field crops, except cash grains, nec	<b>0729</b>	General crop services
<b>016</b>	Vegetables and Melons	<b>074</b>	Veterinary Services
<b>0161</b>	Vegetables and melons	<b>0741</b>	Veterinary services, farm livestock
<b>017</b>	Fruits and Tree Nuts	<b>0742</b>	Veterinary services, specialties
<b>0171</b>	Berry crops	<b>075</b>	Animal Services, Except Veterinary
<b>0172</b>	Grapes	<b>0751</b>	Livestock services, exc. specialties
<b>0173</b>	Tree nuts	<b>0752</b>	Animal specialty services
<b>0174</b>	Citrus fruits	<b>076</b>	Farm Labor and Management Services
<b>0175</b>	Deciduous tree fruits	<b>0761</b>	Farm labor contractors
<b>0179</b>	Fruits and tree nuts, nec	<b>0762</b>	Farm management services
<b>018</b>	Horticultural Specialties	<b>078</b>	Landscape and Horticultural Services
<b>0181</b>	Ornamental nursery products	<b>0781</b>	Landscape counseling and planning
<b>0182</b>	Food crops grown under cover	<b>0782</b>	Lawn and garden services
<b>0189</b>	Horticultural specialties, nec	<b>0783</b>	Ornamental shrub and tree services
<b>019</b>	General Farms, Primarily Crop	<b>08</b>	<b>FORESTRY</b>
<b>0191</b>	General farms, primarily crop	<b>081</b>	Timber Tracts
<b>02</b>	<b>AGRICULTURAL PRODUCTION— LIVESTOCK</b>	<b>0811</b>	Timber tracts
<b>021</b>	Livestock, exc. Dairy, Poultry, etc.	<b>082</b>	Forest Nurseries and Seed Gathering
<b>0211</b>	Beef cattle feedlots	<b>0821</b>	Forest nurseries and seed gathering
<b>0212</b>	Beef cattle, except feedlots	<b>084</b>	Gathering of Misc. Forest Products
<b>0213</b>	Hogs	<b>0843</b>	Extraction of pine gum
<b>0214</b>	Sheep and goats	<b>0849</b>	Gathering of forest products, nec
<b>0219</b>	General livestock, nec	<b>085</b>	Forestry Services
<b>024</b>	Dairy Farms	<b>0851</b>	Forestry services
<b>0241</b>	Dairy farms	<b>09</b>	<b>FISHING, HUNTING, AND TRAPPING</b>
<b>025</b>	Poultry and Eggs	<b>091</b>	Commercial Fishing
<b>0251</b>	Broiler, fryer, and roaster chickens	<b>0912</b>	Finfish
<b>0252</b>	Chicken eggs	<b>0913</b>	Shellfish
<b>0253</b>	Turkeys and turkey eggs	<b>0919</b>	Miscellaneous marine products
<b>0254</b>	Poultry hatcheries	<b>092</b>	Fish Hatcheries and Preserves
<b>0259</b>	Poultry and eggs, nec	<b>0921</b>	Fish hatcheries and preserves
<b>027</b>	Animal Specialties	<b>097</b>	Hunting, Trapping, Game Propagation
<b>0271</b>	Fur-bearing animals and rabbits	<b>0971</b>	Hunting, trapping, game propagation



## List of SIC Codes and Their Short Titles

**B. MINING**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
<b>10</b>	<b>METAL MINING</b>	<b>1321</b>	<b>Natural gas liquids</b>
<b>101</b>	<b>Iron Ores</b>	<b>138</b>	<b>Oil and Gas Field Services</b>
<b>1011</b>	<b>Iron ores</b>	<b>1381</b>	<b>Drilling oil and gas wells</b>
<b>102</b>	<b>Copper Ores</b>	<b>1382</b>	<b>Oil and gas exploration services</b>
<b>1021</b>	<b>Copper ores</b>	<b>1389</b>	<b>Oil and gas field services, nec</b>
<b>103</b>	<b>Lead and Zinc Ores</b>	<b>14</b>	<b>NONMETALLIC MINERALS, EXCEPT FUELS</b>
<b>1031</b>	<b>Lead and zinc ores</b>	<b>141</b>	<b>Dimension Stone</b>
<b>104</b>	<b>Gold and Silver Ores</b>	<b>1411</b>	<b>Dimension stone</b>
<b>1041</b>	<b>Gold ores</b>	<b>142</b>	<b>Crushed and Broken Stone</b>
<b>1044</b>	<b>Silver ores</b>	<b>1422</b>	<b>Crushed and broken limestone</b>
<b>105</b>	<b>Bauxite and Other Aluminum Ores</b>	<b>1423</b>	<b>Crushed and broken granite</b>
<b>1051</b>	<b>Bauxite and other aluminum ores</b>	<b>1429</b>	<b>Crushed and broken stone, nec</b>
<b>106</b>	<b>Ferroalloy Ores, Except Vanadium</b>	<b>144</b>	<b>Sand and Gravel</b>
<b>1061</b>	<b>Ferroalloy ores, except vanadium</b>	<b>1442</b>	<b>Construction sand and gravel</b>
<b>108</b>	<b>Metal Mining Services</b>	<b>1446</b>	<b>Industrial sand</b>
<b>1081</b>	<b>Metal mining services</b>	<b>145</b>	<b>Clay and Related Minerals</b>
<b>109</b>	<b>Miscellaneous Metal Ores</b>	<b>1452</b>	<b>Bentonite</b>
<b>1092</b>	<b>Mercury ores</b>	<b>1453</b>	<b>Fire clay</b>
<b>1094</b>	<b>Uranium-radium-vanadium ores</b>	<b>1454</b>	<b>Fuller's earth</b>
<b>1099</b>	<b>Metal ores, nec</b>	<b>1455</b>	<b>Kaolin and ball clay</b>
<b>11</b>	<b>ANTHRACITE MINING</b>	<b>1459</b>	<b>Clay and related minerals, nec</b>
<b>111</b>	<b>Anthracite Mining</b>	<b>147</b>	<b>Chemical and Fertilizer Minerals</b>
<b>1111</b>	<b>Anthracite</b>	<b>1472</b>	<b>Barite</b>
<b>1112</b>	<b>Anthracite mining services</b>	<b>1473</b>	<b>Fluorspar</b>
<b>12</b>	<b>BITUMINOUS COAL AND LIGNITE MINING</b>	<b>1474</b>	<b>Potash, soda, and borate minerals</b>
<b>121</b>	<b>Bituminous Coal and Lignite Mining</b>	<b>1475</b>	<b>Phosphate rock</b>
<b>1211</b>	<b>Bituminous coal and lignite</b>	<b>1476</b>	<b>Rock salt</b>
<b>1213</b>	<b>Bituminous &amp; lignite mining services</b>	<b>1477</b>	<b>Sulfur</b>
<b>13</b>	<b>OIL AND GAS EXTRACTION</b>	<b>1479</b>	<b>Chemical and fertilizer mining, nec</b>
<b>131</b>	<b>Crude Petroleum and Natural Gas</b>	<b>148</b>	<b>Nonmetallic Minerals Services</b>
<b>1311</b>	<b>Crude petroleum and natural gas</b>	<b>1481</b>	<b>Nonmetallic minerals services</b>
<b>132</b>	<b>Natural Gas Liquids</b>	<b>149</b>	<b>Miscellaneous Nonmetallic Minerals</b>
		<b>1492</b>	<b>Gypsum</b>
		<b>1496</b>	<b>Talc, soapstone, and pyrophyllite</b>
		<b>1499</b>	<b>Nonmetallic minerals, nec</b>

**C. CONSTRUCTION**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
<b>15</b>	<b>GENERAL BUILDING CONTRACTORS</b>	<b>162</b>	<b>Heavy Construction, Except Highway</b>
<b>152</b>	<b>Residential Building Construction</b>	<b>1622</b>	<b>Bridge, tunnel, &amp; elevated highway</b>
<b>1521</b>	<b>Single-family housing construction</b>	<b>1623</b>	<b>Water, sewer, and utility lines</b>
<b>1522</b>	<b>Residential construction, nec</b>	<b>1629</b>	<b>Heavy construction, nec</b>
<b>153</b>	<b>Operative Builders</b>	<b>17</b>	<b>SPECIAL TRADE CONTRACTORS</b>
<b>1531</b>	<b>Operative builders</b>	<b>171</b>	<b>Plumbing, Heating, Air Conditioning</b>
<b>154</b>	<b>Nonresidential Building Construction</b>	<b>1711</b>	<b>Plumbing, heating, air conditioning</b>
<b>1541</b>	<b>Industrial buildings and warehouses</b>	<b>172</b>	<b>Painting, Paper Hanging, Decorating</b>
<b>1542</b>	<b>Nonresidential construction, nec</b>	<b>1721</b>	<b>Painting, paper hanging, decorating</b>
<b>16</b>	<b>HEAVY CONSTRUCTION CONTRACTORS</b>	<b>173</b>	<b>Electrical Work</b>
<b>161</b>	<b>Highway and Street Construction</b>	<b>1731</b>	<b>Electrical work</b>
<b>1611</b>	<b>Highway and street construction</b>	<b>174</b>	<b>Masonry, Stonework, and Plastering</b>
		<b>1741</b>	<b>Masonry and other stonework</b>

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
1742	Plastering, drywall and insulation	178	Water Well Drilling
1743	Terrazzo, tile, marble, mosaic work	1781	Water well drilling
175	Carpentering and Flooring	179	Misc. Special Trade Contractors
1751	Carpentering	1791	Structural steel erection
1752	Floor laying and floor work, nec	1793	Glass and glazing work
176	Roofing and Sheet Metal Work	1794	Excavating and foundation work
1761	Roofing and sheet metal work	1795	Wrecking and demolition work
177	Concrete Work	1796	Installing building equipment, nec
1771	Concrete work	1799	Special trade contractors, nec

## D. MANUFACTURING

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
20	<b>FOOD AND KINDRED PRODUCTS</b>	208	Beverages
201	Meat Products	2082	Malt beverages
2011	Meat packing plants	2083	Malt
2013	Sausages and other prepared meats	2084	Wines, brandy, and brandy spirits
2016	Poultry dressing plants	2085	Distilled liquor, except brandy
2017	Poultry and egg processing	2086	Bottled and canned soft drinks
202	Dairy Products	2087	Flavoring extracts and sirups, nec
2021	Creamery butter	209	Misc. Foods and Kindred Products
2022	Cheese, natural and processed	2091	Canned and cured seafoods
2023	Condensed and evaporated milk	2092	Fresh or frozen packaged fish
2024	Ice cream and frozen desserts	2095	Roasted coffee
2026	Fluid milk	2097	Manufactured ice
203	Preserved Fruits and Vegetables	2098	Macaroni and spaghetti
2032	Canned specialties	2099	Food preparations, nec
2033	Canned fruits and vegetables		
2034	Dehydrated fruits, vegetables, soups	21	<b>TOBACCO MANUFACTURES</b>
2035	Pickles, sauces, and salad dressings	211	Cigarettes
2037	Frozen fruits and vegetables	2111	Cigarettes
2038	Frozen specialties	212	Cigars
204	Grain Mill Products	2121	Cigars
2041	Flour and other grain mill products	213	Chewing and Smoking Tobacco
2043	Cereal breakfast foods	2131	Chewing and smoking tobacco
2044	Rice milling	214	Tobacco Stemming and Redrying
2045	Blended and prepared flour	2141	Tobacco stemming and redrying
2046	Wet corn milling		
2047	Dog, cat, and other pet food	22	<b>TEXTILE MILL PRODUCTS</b>
2048	Prepared feeds, nec	221	Weaving Mills, Cotton
205	Bakery Products	2211	Weaving mills, cotton
2051	Bread, cake, and related products	222	Weaving Mills, Synthetics
2052	Cookies and crackers	2221	Weaving mills, synthetics
206	Sugar and Confectionery Products	223	Weaving and Finishing Mills, Wool
2061	Raw cane sugar	2231	Weaving and finishing mills, wool
2062	Cane sugar refining	224	Narrow Fabric Mills
2063	Beet sugar	2241	Narrow fabric mills
2065	Confectionery products	225	Knitting mills
2066	Chocolate and cocoa products	2251	Women's hosiery, except socks
2067	Chewing gum	2252	Hosiery, nec
207	Fats and Oils	2253	Knit outerwear mills
2074	Cottonseed oil mills	2254	Knit underwear mills
2075	Soybean oil mills	2257	Circular knit fabric mills
2076	Vegetable oil mills, nec	2258	Warp knit fabric mills
2077	Animal and marine fats and oils	2259	Knitting mills, nec
2079	Shortening and cooking oils		

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
226	<b>Textile Finishing, Except Wool</b>	2387	Apparel belts
2261	Finishing plants, cotton	2389	Apparel and accessories, nec
2262	Finishing plants, synthetics	239	<b>Misc. Fabricated Textile Products</b>
2269	Finishing plants, nec	2391	Curtains and draperies
227	<b>Floor Covering Mills</b>	2392	House furnishings, nec
2271	Woven carpets and rugs	2393	Textile bags
2272	Tufted carpets and rugs	2394	Canvas and related products
2279	Carpets and rugs, nec	2395	Pleating and stitching
228	<b>Yarn and Thread Mills</b>	2396	Automotive and apparel trimmings
2281	Yarn mills, except wool	2397	Schiffli machine embroideries
2282	Throwing and winding mills	2399	Fabricated textile products, nec
2283	Wool yarn mills		
2284	Thread mills	24	<b>LUMBER AND WOOD PRODUCTS</b>
229	<b>Miscellaneous Textile Goods</b>	241	<b>Logging Camps &amp; Logging Contractors</b>
2291	Felt goods, exc. woven felts & hats	2411	Logging camps & logging contractors
2292	Lace goods	242	<b>Sawmills and Planing Mills</b>
2293	Paddings and upholstery filling	2421	Sawmills and planing mills, general
2294	Processed textile waste	2426	Hardwood dimension and flooring
2295	Coated fabrics, not rubberized	2429	Special product sawmills, nec
2296	Tire cord and fabric	243	<b>Millwork, Plywood &amp; Structural Members</b>
2297	Nonwoven fabrics	2431	Millwork
2298	Cordage and twine	2434	Wood kitchen cabinets
2299	Textile goods, nec	2435	Hardwood veneer and plywood
23	<b>APPAREL AND OTHER TEXTILE PRODUCTS</b>	2436	Softwood veneer and plywood
231	<b>Men's and Boys' Suits and Coats</b>	2439	Structural wood members, nec
2311	Men's and boys' suits and coats	244	<b>Wood Containers</b>
232	<b>Men's and Boys' Furnishings</b>	2441	Nailed wood boxes and shooks
2321	Men's and boys' shirts and nightwear	2448	Wood pallets and skids
2322	Men's and boys' underwear	2449	Wood containers, nec
2323	Men's and boys' neckwear	245	<b>Wood Buildings and Mobile Homes</b>
2327	Men's and boys' separate trousers	2451	Mobile homes
2328	Men's and boys work clothing	2452	Prefabricated wood buildings
2329	Men's and boys' clothing, nec	249	<b>Miscellaneous Wood Products</b>
233	<b>Women's and Misses' Outerwear</b>	2491	Wood preserving
2331	Women's & misses' blouses & waists	2492	Particleboard
2335	Women's and misses' dresses	2499	Wood products, nec
2337	Women's and misses' suits and coats	25	<b>FURNITURE AND FIXTURES</b>
2339	Women's and misses' outerwear, nec	251	<b>Household Furniture</b>
234	<b>Women's and Children's Undergarments</b>	2511	Wood household furniture
2341	Women's and children's underwear	2512	Upholstered household furniture
2342	Brassieres and allied garments	2514	Metal household furniture
235	<b>Hats, Caps, and Millinery</b>	2515	Mattresses and bedsprings
2351	Millinery	2517	Wood TV and radio cabinets
2352	Hats and caps, except millinery	2519	Household furniture, nec
236	<b>Children's Outerwear</b>	252	<b>Office Furniture</b>
2361	Children's dresses and blouses	2521	Wood office furniture
2363	Children's coats and suits	2522	Metal office furniture
2369	Children's outerwear, nec	253	<b>Public Building &amp; Related Furniture</b>
237	<b>Fur Goods</b>	2531	Public building & related furniture
2371	Fur goods	254	<b>Partitions and Fixtures</b>
238	<b>Miscellaneous Apparel and Accessories</b>	2541	Wood partitions and fixtures
2381	Fabric dress and work gloves	2542	Metal partitions and fixtures
2384	Robes and dressing gowns	259	<b>Miscellaneous Furniture and Fixtures</b>
2385	Waterproof outer garments	2591	Drapery hardware & blinds & shades
2386	Leather and sheep lined clothing	2599	Furniture and fixtures, nec



## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
26	<b>PAPER AND ALLIED PRODUCTS</b>	2813	Industrial gases
261	Pulp Mills	2816	Inorganic pigments
2611	Pulp mills	2819	Industrial inorganic chemicals, nec
262	Paper Mills, Except Building Paper	282	Plastics Materials and Synthetics
2621	Paper mills, except building paper	2821	Plastics materials and resins
263	Paperboard Mills	2822	Synthetic rubber
2631	Paperboard mills	2823	Cellulosic man-made fibers
264	Misc. Converted Paper Products	2824	Organic fibers, noncellulosic
2641	Paper coating and glazing	283	Drugs
2642	Envelopes	2831	Biological products
2643	Bags, except textile bags	2833	Medicinals and botanicals
2645	Die-cut paper and board	2834	Pharmaceutical preparations
2646	Pressed and molded pulp goods	284	Soap, Cleaners, and Toilet Goods
2647	Sanitary paper products	2841	Soap and other detergents
2648	Stationery products	2842	Polishes and sanitation goods
2649	Converted paper products, nec	2843	Surface active agents
265	Paperboard Containers and Boxes	2844	Toilet preparations
2651	Folding paperboard boxes	285	Paints and Allied Products
2652	Set-up paperboard boxes	2851	Paints and allied products
2653	Corrugated and solid fiber boxes	286	Industrial Organic Chemicals
2654	Sanitary food containers	2861	Gum and wood chemicals
2655	Fiber cans, drums & similar products	2865	Cyclic crudes and intermediates
266	Building Paper and Board Mills	2869	Industrial organic chemicals, nec
2661	Building paper and board mills	287	Agricultural Chemicals
27	<b>PRINTING AND PUBLISHING</b>	2873	Nitrogenous fertilizers
271	Newspapers	2874	Phosphatic fertilizers
2711	Newspapers	2875	Fertilizers, mixing only
272	Periodicals	2879	Agricultural chemicals, nec
2721	Periodicals	289	Miscellaneous Chemical Products
273	Books	2891	Adhesives and sealants
2731	Book publishing	2892	Explosives
2732	Book printing	2893	Printing ink
274	Miscellaneous Publishing	2895	Carbon black
2741	Miscellaneous publishing	2899	Chemical preparations, nec
275	Commercial Printing	29	<b>PETROLEUM AND COAL PRODUCTS</b>
2751	Commercial printing, letterpress	291	Petroleum Refining
2752	Commercial printing, lithographic	2911	Petroleum refining
2753	Engraving and plate printing	295	Paving and Roofing Materials
2754	Commercial printing, gravure	2951	Paving mixtures and blocks
276	Manifold Business Forms	2952	Asphalt felts and coatings
2761	Manifold business forms	299	Misc. Petroleum and Coal Products
277	Greeting Card Publishing	2992	Lubricating oils and greases
2771	Greeting card publishing	2999	Petroleum and coal products, nec
278	Blankbooks and Bookbinding	30	<b>RUBBER AND MISC. PLASTICS PRODUCTS</b>
2782	Blankbooks and looseleaf binders	301	Tires and Inner Tubes
2789	Bookbinding and related work	3011	Tires and inner tubes
279	Printing Trade Services	302	Rubber and Plastics Footwear
2791	Typesetting	3021	Rubber and plastics footwear
2793	Photoengraving	303	Reclaimed Rubber
2794	Electrotyping and stereotyping	3031	Reclaimed rubber
2795	Lithographic platemaking services	304	Rubber and Plastics Hose and Belting
28	<b>CHEMICALS AND ALLIED PRODUCTS</b>	3041	Rubber and plastics hose and belting
281	Industrial Inorganic Chemicals	306	Fabricated Rubber Products, nec
2812	Alkalies and chlorine	3069	Fabricated rubber products, nec



## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
307	Miscellaneous Plastics Products	3295	Minerals, ground or treated
3079	Miscellaneous plastics products	3296	Mineral wool
31	<b>LEATHER AND LEATHER PRODUCTS</b>	3297	Nonclay refractories
311	Leather Tanning and Finishing	3299	Nonmetallic mineral products, nec
3111	Leather tanning and finishing	33	<b>PRIMARY METAL INDUSTRIES</b>
313	Boot and Shoe Cut Stock and Findings	331	Blast Furnace and Basic Steel Products
3131	Boot and shoe cut stock and findings	3312	Blast furnaces and steel mills
314	Footwear, Except Rubber	3313	Electrometallurgical products
3142	House slippers	3315	Steel wire and related products
3143	Men's footwear, except athletic	3316	Cold finishing of steel shapes
3144	Women's footwear, except athletic	3317	Steel pipe and tubes
3149	Footwear, except rubber, nec	332	Iron and Steel Foundries
315	Leather Gloves and Mittens	3321	Gray iron foundries
3151	Leather gloves and mittens	3322	Malleable iron foundries
316	Luggage	3324	Steel investment foundries
3161	Luggage	3325	Steel foundries, nec
317	Handbags and Personal Leather Goods	333	Primary Nonferrous Metals
3171	Women's handbags and purses	3331	Primary copper
3172	Personal leather goods, nec	3332	Primary lead
319	Leather Goods, nec	3333	Primary zinc
3199	Leather goods, nec	3334	Primary aluminum
32	<b>STONE, CLAY, AND GLASS PRODUCTS</b>	3339	Primary nonferrous metals, nec
321	Flat Glass	334	Secondary Nonferrous Metals
3211	Flat glass	3341	Secondary nonferrous metals
322	Glass and Glassware, Pressed or Blown	335	Nonferrous Rolling and Drawing
3221	Glass containers	3351	Copper rolling and drawing
3229	Pressed and blown glass, nec	3353	Aluminum sheet, plate, and foil
323	Products of Purchased Glass	3354	Aluminum extruded products
3231	Products of purchased glass	3355	Aluminum rolling and drawing, nec
324	Cement, Hydraulic	3356	Nonferrous rolling and drawing, nec
3241	Cement, hydraulic	3357	Nonferrous wire drawing & insulating
325	Structural Clay Products	336	Nonferrous Foundries
3251	Brick and structural clay tile	3361	Aluminum foundries
3253	Ceramic wall and floor tile	3362	Brass, bronze, and copper foundries
3255	Clay refractories	3369	Nonferrous foundries, nec
3259	Structural clay products, nec	339	Miscellaneous Primary Metal Products
326	Pottery and Related Products	3398	Metal heat treating
3261	Vitreous plumbing fixtures	3399	Primary metal products, nec
3262	Vitreous china food utensils	34	<b>FABRICATED METAL PRODUCTS</b>
3263	Fine earthenware food utensils	341	Metal Cans and Shipping Containers
3264	Porcelain electrical supplies	3411	Metal cans
3269	Pottery products, nec	3412	Metal barrels, drums, and pails
327	Concrete, Gypsum, and Plaster Products	342	Cutlery, Hand Tools, and Hardware
3271	Concrete block and brick	3421	Cutlery
3272	Concrete products, nec	3423	Hand and edge tools, nec
3273	Ready-mixed concrete	3425	Hand saws and saw blades
3274	Lime	3429	Hardware, nec
3275	Gypsum products	343	Plumbing and Heating, Except Electric
328	Cut Stone and Stone Products	3431	Metal sanitary ware
3281	Cut stone and stone products	3432	Plumbing fittings and brass goods
329	Misc. Nonmetallic Mineral Products	3433	Heating equipment, except electric
3291	Abrasive products	344	Fabricated Structural Metal Products
3292	Asbestos products	3441	Fabricated structural metal
3293	Gaskets, packing and sealing devices	3442	Metal doors, sash, and trim

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
3443	Fabricated plate work (boiler shops)	3552	Textile machinery
3444	Sheet metal work	3553	Woodworking machinery
3446	Architectural metal work	3554	Paper industries machinery
3448	Prefabricated metal buildings	3555	Printing trades machinery
3449	Miscellaneous metal work	3559	Special industry machinery, nec
345	Screw Machine Products, Bolts, etc.	356	General Industrial Machinery
3451	Screw machine products	3561	Pumps and pumping equipment
3452	Bolts, nuts, rivets, and washers	3562	Ball and roller bearings
346	Metal Forgings and Stampings	3563	Air and gas compressors
3462	Iron and steel forgings	3564	Blowers and fans
3463	Nonferrous forgings	3565	Industrial patterns
3465	Automotive stampings	3566	Speed changers, drives, and gears
3466	Crowns and closures	3567	Industrial furnaces and ovens
3469	Metal stampings, nec	3568	Power transmission equipment, nec
347	Metal Services, nec	3569	General industrial machinery, nec
3471	Plating and polishing	357	Office and Computing Machines
3479	Metal coating and allied services	3572	Typewriters
348	Ordnance and Accessories, nec	3573	Electronic computing equipment
3482	Small arms ammunition	3574	Calculating and accounting machines
3483	Ammunition, exc. for small arms, nec	3576	Scales and balances, exc. laboratory
3484	Small arms	3579	Office machines, nec
3489	Ordnance and accessories, nec	358	Refrigeration and Service Machinery
349	Misc. Fabricated Metal Products	3581	Automatic merchandising machines
3493	Steel springs, except wire	3582	Commercial laundry equipment
3494	Valves and pipe fittings	3585	Refrigeration and heating equipment
3495	Wire springs	3586	Measuring and dispensing pumps
3496	Misc. fabricated wire products	3589	Service industry machinery, nec
3497	Metal foil and leaf	359	Misc. Machinery, Except Electrical
3498	Fabricated pipe and fittings	3592	Carburetors, pistons, rings, valves
3499	Fabricated metal products, nec	3599	Machinery, except electrical, nec
35	MACHINERY, EXCEPT ELECTRICAL	36	ELECTRIC AND ELECTRONIC EQUIPMENT
351	Engines and Turbines	361	Electric Distributing Equipment
3511	Turbines and turbine generator sets	3612	Transformers
3519	Internal combustion engines, nec	3613	Switchgear and switchboard apparatus
352	Farm and Garden Machinery	362	Electrical Industrial Apparatus
3523	Farm machinery and equipment	3621	Motors and generators
3524	Lawn and garden equipment	3622	Industrial controls
353	Construction and Related Machinery	3623	Welding apparatus, electric
3531	Construction machinery	3624	Carbon and graphite products
3532	Mining machinery	3629	Electrical industrial apparatus, nec
3533	Oil field machinery	363	Household Appliances
3534	Elevators and moving stairways	3631	Household cooking equipment
3535	Conveyors and conveying equipment	3632	Household refrigerators and freezers
3536	Hoists, cranes, and monorails	3633	Household laundry equipment
3537	Industrial trucks and tractors	3634	Electric housewares and fans
354	Metalworking Machinery	3635	Household vacuum cleaners
3541	Machine tools, metal cutting types	3636	Sewing machines
3542	Machine tools, metal forming types	3639	Household appliances, nec
3544	Special dies, tools, jigs & fixtures	364	Electric Lighting and Wiring Equipment
3545	Machine tool accessories	3641	Electric lamps
3546	Power driven hand tools	3643	Current-carrying wiring devices
3547	Rolling mill machinery	3644	Noncurrent-carrying wiring devices
3549	Metalworking machinery, nec	3645	Residential lighting fixtures
355	Special Industry Machinery	3646	Commercial lighting fixtures
3551	Food products machinery	3647	Vehicular lighting equipment



## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
3648	Lighting equipment, nec	38	<b>INSTRUMENTS AND RELATED PRODUCTS</b>
365	<b>Radio and TV Receiving Equipment</b>	381	<b>Engineering &amp; Scientific Instruments</b>
3651	Radio and TV receiving sets	3811	Engineering & scientific instruments
3652	Phonograph records	382	<b>Measuring and Controlling Devices</b>
366	<b>Communication Equipment</b>	3822	Environmental controls
3661	Telephone and telegraph apparatus	3823	Process control instruments
3662	Radio and TV communication equipment	3824	Fluid meters and counting devices
367	<b>Electronic Components and Accessories</b>	3825	Instruments to measure electricity
3671	Electron tubes, receiving type	3829	Measuring & controlling devices, nec
3672	Cathode ray television picture tubes	383	<b>Optical Instruments and Lenses</b>
3673	Electron tubes, transmitting	3832	Optical instruments and lenses
3674	Semiconductors and related devices	384	<b>Medical Instruments and Supplies</b>
3675	Electronic capacitors	3841	Surgical and medical instruments
3676	Electronic resistors	3842	Surgical appliances and supplies
3677	Electronic coils and transformers	3843	Dental equipment and supplies
3678	Electronic connectors	385	<b>Ophthalmic Goods</b>
3679	Electronic components, nec	3851	Ophthalmic goods
369	<b>Misc. Electrical Equipment &amp; Supplies</b>	386	<b>Photographic Equipment and Supplies</b>
3691	Storage batteries	3861	Photographic equipment and supplies
3692	Primary batteries, dry and wet	387	<b>Watches, Clocks, and Watchcases</b>
3693	X-ray apparatus and tubes	3873	Watches, clocks, and watchcases
3694	Engine electrical equipment		
3699	Electrical equipment & supplies, nec	39	<b>MISCELLANEOUS MANUFACTURING INDUSTRIES</b>
37	<b>TRANSPORTATION EQUIPMENT</b>	391	<b>Jewelry, Silverware, and Plated Ware</b>
371	<b>Motor Vehicles and Equipment</b>	3911	Jewelry, precious metal
3711	Motor vehicles and car bodies	3914	Silverware and plated ware
3713	Truck and bus bodies	3915	Jewelers' materials & lapidary work
3714	Motor vehicle parts and accessories	393	<b>Musical Instruments</b>
3715	Truck trailers	3931	Musical instruments
3716	Motor Homes*	394	<b>Toys and Sporting Goods</b>
372	<b>Aircraft and Parts</b>	3942	Dolls
3721	Aircraft	3944	Games, toys, and children's vehicles
3724	Aircraft engines and engine parts	3949	Sporting and athletic goods, nec
3728	Aircraft equipment, nec	395	<b>Pens, Pencils, Office and Art Supplies</b>
373	<b>Ship and Boat Building and Repairing</b>	3951	Pens and mechanical pencils
3731	Ship building and repairing	3952	Lead pencils and art goods
3732	Boat building and repairing	3953	Marking devices
374	<b>Railroad Equipment</b>	3955	Carbon paper and inked ribbons
3743	Railroad equipment	396	<b>Costume Jewelry and Notions</b>
375	<b>Motorcycles, Bicycles, and Parts</b>	3961	Costume jewelry
3751	Motorcycles, bicycles, and parts	3962	Artificial flowers
376	<b>Guided Missiles, Space Vehicles, Parts</b>	3963	Buttons
3761	Guided missiles and space vehicles	3964	Needles, pins, and fasteners
3764	Space propulsion units and parts	399	<b>Miscellaneous Manufactures</b>
3769	Space vehicle equipment, nec	3991	Brooms and brushes
379	<b>Miscellaneous Transportation Equipment</b>	3993	Signs and advertising displays
3792	Travel trailers and campers	3995	Burial caskets
3795	Tanks and tank components	3996	Hard surface floor coverings
3799	Transportation equipment, nec.	3999	Manufacturing industries, nec

## List of SIC Codes and Their Short Titles

**E. TRANSPORTATION AND PUBLIC UTILITIES**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
<b>40</b>	<b>RAILROAD TRANSPORTATION</b>	<b>4452</b>	<b>Ferries</b>
<b>401</b>	<b>Railroads</b>	<b>4453</b>	<b>Lighterage</b>
<b>4011</b>	<b>Railroads, line-haul operating</b>	<b>4454</b>	<b>Towing and tugboat service</b>
<b>4013</b>	<b>Switching and terminal services</b>	<b>4459</b>	<b>Local water transportation, nec</b>
*		<b>446</b>	<b>Water Transportation Services</b>
<b>41</b>	<b>LOCAL AND INTERURBAN PASSENGER TRANSIT</b>	<b>4463</b>	<b>Marine cargo handling</b>
<b>411</b>	<b>Local and Suburban Transportation</b>	<b>4464</b>	<b>Canal operation</b>
<b>4111</b>	<b>Local and suburban transit</b>	<b>4469</b>	<b>Water transportation services, nec</b>
<b>4119</b>	<b>Local passenger transportation, nec</b>	<b>45</b>	<b>TRANSPORTATION BY AIR</b>
<b>412</b>	<b>Taxicabs</b>	<b>451</b>	<b>Certificated Air Transportation</b>
<b>4121</b>	<b>Taxicabs</b>	<b>4511</b>	<b>Certificated air transportation</b>
<b>413</b>	<b>Intercity Highway Transportation</b>	<b>452</b>	<b>Noncertificated Air Transportation</b>
<b>4131</b>	<b>Intercity highway transportation</b>	<b>4521</b>	<b>Noncertificated air transportation</b>
<b>414</b>	<b>Transportation Charter Service</b>	<b>458</b>	<b>Air Transportation Services</b>
<b>4141</b>	<b>Local passenger charter service</b>	<b>4582</b>	<b>Airports and flying fields</b>
<b>4142</b>	<b>Charter service, except local</b>	<b>4583</b>	<b>Airport terminal services</b>
<b>415</b>	<b>School Buses</b>	<b>46</b>	<b>PIPE LINES, EXCEPT NATURAL GAS</b>
<b>4151</b>	<b>School buses</b>	<b>461</b>	<b>Pipe Lines, Except Natural Gas</b>
<b>417</b>	<b>Bus Terminal and Service Facilities</b>	<b>4612</b>	<b>Crude petroleum pipe lines</b>
<b>4171</b>	<b>Bus terminal facilities</b>	<b>4613</b>	<b>Refined petroleum pipe lines</b>
<b>4172</b>	<b>Bus service facilities</b>	<b>4619</b>	<b>Pipe lines, nec</b>
<b>42</b>	<b>TRUCKING AND WAREHOUSING</b>	<b>47</b>	<b>TRANSPORTATION SERVICES</b>
<b>421</b>	<b>Trucking, Local and Long Distance</b>	<b>471</b>	<b>Freight Forwarding</b>
<b>4212</b>	<b>Local trucking, without storage</b>	<b>4712</b>	<b>Freight forwarding</b>
<b>4213</b>	<b>Trucking, except local</b>	<b>472</b>	<b>Arrangement of Transportation</b>
<b>4214</b>	<b>Local trucking and storage</b>	<b>4722</b>	<b>Passenger transportation arrangement</b>
<b>422</b>	<b>Public Warehousing</b>	<b>4723</b>	<b>Freight transportation arrangement</b>
<b>4221</b>	<b>Farm product warehousing and storage</b>	<b>474</b>	<b>Rental of Railroad Cars</b>
<b>4222</b>	<b>Refrigerated warehousing</b>	<b>4742</b>	<b>Railroad car rental with service</b>
<b>4224</b>	<b>Household goods warehousing</b>	<b>4743</b>	<b>Railroad car rental without service</b>
<b>4225</b>	<b>General warehousing and storage</b>	<b>478</b>	<b>Miscellaneous Transportation Services</b>
<b>4226</b>	<b>Special warehousing and storage, nec</b>	<b>4782</b>	<b>Inspection and weighing services</b>
<b>423</b>	<b>Trucking Terminal Facilities</b>	<b>4783</b>	<b>Packing and crating</b>
<b>4231</b>	<b>Trucking terminal facilities</b>	<b>4784</b>	<b>Fixed facilities for vehicles, nec</b>
<b>43</b>	<b>U.S. POSTAL SERVICE</b>	<b>4789</b>	<b>Transportation services, nec</b>
<b>431</b>	<b>U.S. Postal Service</b>	<b>48</b>	<b>COMMUNICATION</b>
<b>4311</b>	<b>U.S. Postal Service</b>	<b>481</b>	<b>Telephone Communication</b>
<b>44</b>	<b>WATER TRANSPORTATION</b>	<b>4811</b>	<b>Telephone communication</b>
<b>441</b>	<b>Deep Sea Foreign Transportation</b>	<b>482</b>	<b>Telegraph Communication</b>
<b>4411</b>	<b>Deep sea foreign transportation</b>	<b>4821</b>	<b>Telegraph communication</b>
<b>442</b>	<b>Deep Sea Domestic Transportation</b>	<b>483</b>	<b>Radio and Television Broadcasting</b>
<b>4421</b>	<b>Noncontiguous area transportation</b>	<b>4832</b>	<b>Radio broadcasting</b>
<b>4422</b>	<b>Coastwise transportation</b>	<b>4833</b>	<b>Television broadcasting</b>
<b>4423</b>	<b>Intercoastal transportation</b>	<b>489</b>	<b>Communication Services, nec</b>
<b>443</b>	<b>Great Lakes Transportation</b>	<b>4899</b>	<b>Communication services, nec</b>
<b>4431</b>	<b>Great Lakes transportation</b>	<b>49</b>	<b>ELECTRIC, GAS, AND SANITARY SERVICES</b>
<b>444</b>	<b>Transportation on Rivers and Canals</b>	<b>491</b>	<b>Electric Services</b>
<b>4441</b>	<b>Transportation on rivers and canals</b>	<b>4911</b>	<b>Electric services</b>
<b>445</b>	<b>Local Water Transportation</b>		

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
492	Gas Production and Distribution	4941	Water supply
4922	Natural gas transmission	495	Sanitary Services
4923	Gas transmission and distribution	4952	Sewerage systems
4924	Natural gas distribution	4953	Refuse systems
4925	Gas production and/or distribution	4959	Sanitary services, nec
493	Combination Utility Services	496	Steam Supply
4931	Electric and other services combined	4961	Steam supply
4932	Gas and other services combined	497	Irrigation Systems
4939	Combination utility services, nec	4971	Irrigation systems
494	Water Supply		

## F. WHOLESALE TRADE

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
50	WHOLESALE TRADE—DURABLE GOODS	51	WHOLESALE TRADE—NONDURABLE GOODS
501	Motor Vehicles & Automotive Equipment	511	Paper and Paper Products
5012	Automobiles and other motor vehicles	5111	Printing and writing paper
5013	Automotive parts and supplies	5112	Stationery supplies
5014	Tires and tubes	5113	Industrial & personal service paper
502	Furniture and Home Furnishings	512	Drugs, Proprietarys, and Sundries
5021	Furniture	5122	Drugs, proprietarys, and sundries
5023	Home furnishings	513	Apparel, Piece Goods, and Notions
503	Lumber and Construction Materials	5133	Piece goods
5031	Lumber, plywood and millwork	5134	Notions and other dry goods
5039	Construction materials, nec	5136	Men's clothing and furnishings
504	Sporting Goods, Toys, and Hobby Goods	5137	Women's and children's clothing
5041	Sporting and recreational goods	5139	Footwear
5042	Toys and hobby goods and supplies	514	Groceries and Related Products
5043	Photographic equipment and supplies	5141	Groceries, general line
505	Metals and Minerals, Except Petroleum	5142	Frozen foods
5051	Metals service centers and offices	5143	Dairy products
5052	Coal and other minerals and ores	5144	Poultry and poultry products
506	Electrical Goods	5145	Confectionery
5063	Electrical apparatus and equipment	5146	Fish and seafoods
5064	Electrical appliances, TV and radios	5147	Meats and meat products
5065	Electronic parts and equipment	5148	Fresh fruits and vegetables
507	Hardware, Plumbing & Heating Equipment	5149	Groceries and related products, nec
5072	Hardware	515	Farm-Product Raw Materials
5074	Plumbing & hydronic heating supplies	5152	Cotton
5075	Warm air heating & air conditioning	5153	Grain
5078	Refrigeration equipment and supplies	5154	Livestock
508	Machinery, Equipment, and Supplies	5159	Farm-products raw materials, nec
5081	Commercial machines and equipment	516	Chemicals and Allied Products
5082	Construction and mining machinery	5161	Chemicals and allied products
5083	Farm machinery and equipment	517	Petroleum and Petroleum Products
5084	Industrial machinery and equipment	5171	Petroleum bulk stations & terminals
5085	Industrial supplies	5172	Petroleum products, nec
5086	Professional equipment and supplies	518	Beer, Wine, and Distilled Beverages
5087	Service establishment equipment	5181	Beer and ale
5088	Transportation equipment & supplies	5182	Wines and distilled beverages
509	Miscellaneous Durable Goods	519	Miscellaneous Nondurable Goods
5093	Scrap and waste materials	5191	Farm supplies
5094	Jewelry, watches, & precious stones	5194	Tobacco and tobacco products
5099	Durable goods, nec	5198	Paints, varnishes, and supplies
		5199	Nondurable goods, nec



## List of SIC Codes and Their Short Titles

**G. RETAIL TRADE**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
<b>52</b>	<b>BUILDING MATERIALS &amp; GARDEN SUPPLIES</b>	<b>5571</b>	<b>Motorcycle dealers</b>
<b>521</b>	<b>Lumber and Other Building Materials</b>	<b>559</b>	<b>Automotive Dealers, nec</b>
<b>5211</b>	<b>Lumber and other building materials</b>	<b>5599</b>	<b>Automotive dealers, nec</b>
<b>523</b>	<b>Paint, Glass, and Wallpaper Stores</b>	<b>56</b>	<b>APPAREL AND ACCESSORY STORES</b>
<b>5231</b>	<b>Paint, glass, and wallpaper stores</b>	<b>561</b>	<b>Men's &amp; Boys' Clothing &amp; Furnishings</b>
<b>525</b>	<b>Hardware Stores</b>	<b>5611</b>	<b>Men's &amp; boys' clothing &amp; furnishings</b>
<b>5251</b>	<b>Hardware stores</b>	<b>562</b>	<b>Women's Ready-to-Wear Stores</b>
<b>526</b>	<b>Retail Nurseries and Garden Stores</b>	<b>5621</b>	<b>Women's ready-to-wear stores</b>
<b>5261</b>	<b>Retail nurseries and garden stores</b>	<b>563</b>	<b>Women's Accessory and Specialty Stores</b>
<b>527</b>	<b>Mobile Home Dealers</b>	<b>5631</b>	<b>Women's accessory and specialty stores</b>
<b>5271</b>	<b>Mobile home dealers</b>	<b>564</b>	<b>Children's and Infants' Wear Stores</b>
<b>53</b>	<b>GENERAL MERCHANDISE STORES</b>	<b>5641</b>	<b>Children's and infants' wear stores</b>
<b>531</b>	<b>Department Stores</b>	<b>565</b>	<b>Family Clothing Stores</b>
<b>5311</b>	<b>Department stores</b>	<b>5651</b>	<b>Family clothing stores</b>
<b>533</b>	<b>Variety Stores</b>	<b>566</b>	<b>Shoe Stores</b>
<b>5331</b>	<b>Variety stores</b>	<b>5661</b>	<b>Shoe stores</b>
<b>539</b>	<b>Misc. General Merchandise Stores</b>	<b>568</b>	<b>Furriers and Fur Shops</b>
<b>5399</b>	<b>Misc. general merchandise stores</b>	<b>5681</b>	<b>Furriers and fur shops</b>
<b>54</b>	<b>FOOD STORES</b>	<b>569</b>	<b>Miscellaneous Apparel &amp; Accessories</b>
<b>541</b>	<b>Grocery Stores</b>	<b>5699</b>	<b>Miscellaneous apparel &amp; accessories</b>
<b>5411</b>	<b>Grocery stores</b>	<b>57</b>	<b>FURNITURE AND HOME FURNISHINGS STORES</b>
<b>542</b>	<b>Meat Markets and Freezer Provisioners</b>	<b>571</b>	<b>Furniture and Home Furnishings Stores</b>
<b>5422</b>	<b>Freezer and locker meat provisioners</b>	<b>5712</b>	<b>Furniture stores</b>
<b>5423</b>	<b>Meat and fish (seafood) markets</b>	<b>5713</b>	<b>Floor covering stores</b>
<b>543</b>	<b>Fruit Stores and Vegetable Markets</b>	<b>5714</b>	<b>Drapery and upholstery stores</b>
<b>5431</b>	<b>Fruit stores and vegetable markets</b>	<b>5719</b>	<b>Misc. home furnishings stores</b>
<b>544</b>	<b>Candy, Nut, and Confectionery Stores</b>	<b>572</b>	<b>Household Appliance Stores</b>
<b>5441</b>	<b>Candy, nut, and confectionery stores</b>	<b>5722</b>	<b>Household appliance stores</b>
<b>545</b>	<b>Dairy Products Stores</b>	<b>573</b>	<b>Radio, Television, and Music Stores</b>
<b>5451</b>	<b>Dairy products stores</b>	<b>5732</b>	<b>Radio and television stores</b>
<b>546</b>	<b>Retail Bakeries</b>	<b>5733</b>	<b>Music stores</b>
<b>5462</b>	<b>Retail bakeries—baking and selling</b>	<b>58</b>	<b>EATING AND DRINKING PLACES</b>
<b>5463</b>	<b>Retail bakeries—selling only</b>	<b>581</b>	<b>Eating and Drinking Places</b>
<b>549</b>	<b>Miscellaneous Food Stores</b>	<b>5812</b>	<b>Eating places</b>
<b>5499</b>	<b>Miscellaneous food stores</b>	<b>5813</b>	<b>Drinking places</b>
<b>55</b>	<b>AUTOMOTIVE DEALERS &amp; SERVICE STATIONS</b>	<b>59</b>	<b>MISCELLANEOUS RETAIL</b>
<b>551</b>	<b>New and Used Car Dealers</b>	<b>591</b>	<b>Drug Stores and Proprietary Stores</b>
<b>5511</b>	<b>New and used car dealers</b>	<b>5912</b>	<b>Drug stores and proprietary stores</b>
<b>552</b>	<b>Used Car Dealers</b>	<b>592</b>	<b>Liquor Stores</b>
<b>5521</b>	<b>Used car dealers</b>	<b>5921</b>	<b>Liquor stores</b>
<b>553</b>	<b>Auto and Home Supply Stores</b>	<b>593</b>	<b>Used Merchandise Stores</b>
<b>5531</b>	<b>Auto and home supply stores</b>	<b>5931</b>	<b>Used merchandise stores</b>
<b>554</b>	<b>Gasoline Service Stations</b>	<b>594</b>	<b>Miscellaneous Shopping Goods Stores</b>
<b>5541</b>	<b>Gasoline service stations</b>	<b>5941</b>	<b>Sporting goods and bicycle shops</b>
<b>555</b>	<b>Boat Dealers</b>	<b>5942</b>	<b>Book stores</b>
<b>5551</b>	<b>Boat dealers</b>	<b>5943</b>	<b>Stationery stores</b>
<b>556</b>	<b>Recreation &amp; Utility Trailer Dealers</b>	<b>5944</b>	<b>Jewelry stores</b>
<b>5561</b>	<b>Recreation &amp; utility trailer dealers</b>	<b>5945</b>	<b>Hobby, toy, and game shops</b>
<b>557</b>	<b>Motorcycle Dealers</b>	<b>5946</b>	<b>Camera &amp; photographic supply stores</b>

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
5947	Gift, novelty, and souvenir shops	5982	Fuel and ice dealers, nec
5948	Luggage and leather goods stores	5983	Fuel oil dealers
5949	Sewing, needlework, and piece goods	5984	Liquefied petroleum gas dealers
596	Nonstore Retailers	599	Retail Stores, nec
5961	Mail order houses	5992	Florists
5962	Merchandising machine operators	5993	Cigar stores and stands
5963	Direct selling organizations	5994	News dealers and newsstands
598	Fuel and Ice Dealers	5999	Miscellaneous retail stores, nec

**H. FINANCE, INSURANCE, AND REAL ESTATE**

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
60	<b>BANKING</b>	6149	Misc. personal credit institutions
601	Federal Reserve Banks	615	Business Credit Institutions
6011	Federal Reserve banks	6153	Short-term business credit
602	Commercial and Stock Savings Banks	6159	Misc. business credit institutions
6022	State banks, Federal Reserve	616	Mortgage Bankers and Brokers
6023	State banks, not Fed. Reserve, FDIC	6162	Mortgage bankers and correspondents
6024	State banks, not Fed. Res., not FDIC	6163	Loan brokers
6025	National banks, Federal Reserve	62	<b>SECURITY, COMMODITY BROKERS &amp; SERVICES</b>
6026	National banks, not Fed. Res., FDIC	621	Security Brokers and Dealers
6027	National banks, not FDIC	6211	Security brokers and dealers
6028	Private banks, not incorp., not FDIC	622	Commodity Contracts Brokers, Dealers
603	Mutual Savings Banks	6221	Commodity contracts brokers, dealers
6032	Mutual savings banks, Federal Reserve	623	Security and Commodity Exchanges
6033	Mutual savings banks, nec	6231	Security and commodity exchanges
6034	Mutual savings banks, not FDIC	628	Security and Commodity Services
604	Trust Companies, Nondeposit	6281	Security and commodity services
6042	Nondeposit trusts, Federal Reserve	63	<b>INSURANCE CARRIERS</b>
6044	Nondeposit trusts, not FDIC	631	Life Insurance
605	Functions Closely Related to Banking	6311	Life insurance
6052	Foreign exchange establishments	632	Medical Service and Health Insurance
6054	Safe deposit companies	6321	Accident and health insurance
6055	Clearinghouse associations	6324	Hospital and medical service plans
6056	Corporations for banking abroad	633	Fire, Marine, and Casualty Insurance
6059	Functions related to banking, nec	6331	Fire, marine, and casualty insurance
61	<b>CREDIT AGENCIES OTHER THAN BANKS</b>	635	Surety Insurance
611	Rediscount and Financing Institutions	6351	Surety insurance
6112	Rediscounting, not for agricultural	636	Title Insurance
6113	Rediscounting, for agricultural	6361	Title insurance
612	Savings and Loan Associations	637	Pension, Health, and Welfare Funds
6122	Federal savings & loan associations	6371	Pension, health, and welfare funds
6123	State associations, insured	639	Insurance Carriers, nec
6124	State associations, noninsured, FHLB	6399	Insurance carriers, nec
6125	State associations, noninsured, nec	64	<b>INSURANCE AGENTS, BROKERS &amp; SERVICE</b>
613	Agricultural Credit Institutions	641	Insurance Agents, Brokers & Service
6131	Agricultural credit institutions	6411	Insurance agents, brokers & service
614	Personal Credit Institutions	65	<b>REAL ESTATE</b>
6142	Federal credit unions	651	Real Estate Operators and Lessors
6143	State credit unions	6512	Nonresidential building operators
6144	Nondeposit industrial loan companies	6513	Apartment building operators
6145	Licensed small loan lenders		
6146	Installment sales finance companies		

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
6514	Dwelling operators, exc. apartments	67	<b>HOLDING AND OTHER INVESTMENT OFFICES</b>
6515	Mobile home site operators	671	<b>Holding Offices</b>
6517	Railroad property lessors	6711	Holding offices
6519	Real property lessors, nec	672	<b>Investment Offices</b>
653	<b>Real Estate Agents and Managers</b>	6722	Management investment, open-end
6531	Real estate agents and managers	6723	Management investment, closed-end
654	<b>Title Abstract Offices</b>	6724	Unit investment trusts
6541	Title abstract offices	6725	Face-amount certificate offices
655	<b>Subdividers and Developers</b>	673	<b>Trusts</b>
6552	Subdividers and developers, nec	6732	Educational, religious, etc. trusts
6553	Cemetery subdividers and developers	6733	Trusts, nec
66	<b>COMBINED REAL ESTATE, INSURANCE, ETC.</b>	679	<b>Miscellaneous Investing</b>
661	Combined Real Estate, Insurance, etc	6792	Oil royalty traders
6611	Combined real estate, insurance, etc	6793	Commodity traders
		6794	Patent owners and lessors
		6798	Real Estate Investment Trusts*
		6799	Investors, nec

## I. SERVICES

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
70	<b>HOTELS AND OTHER LODGING PLACES</b>	7261	Funeral service and crematories
701	<b>Hotels, Motels, and Tourist Courts</b>	729	<b>Miscellaneous Personal Services</b>
7011	Hotels, motels, and tourist courts	7299	Miscellaneous personal services
702	<b>Rooming and Boarding Houses</b>	73	<b>BUSINESS SERVICES</b>
7021	Rooming and boarding houses	731	<b>Advertising</b>
703	<b>Camps and Trailering Parks</b>	7311	Advertising agencies
7032	Sporting and recreational camps	7312	Outdoor advertising services
7033	Trailering parks for transients	7313	Radio, TV, publisher representatives
704	<b>Membership-Basis Organization Hotels</b>	7319	Advertising, nec
7041	Membership-basis organization hotels	732	<b>Credit Reporting and Collection</b>
72	<b>PERSONAL SERVICES</b>	7321	Credit reporting and collection
721	<b>Laundry, Cleaning, &amp; Garment Services</b>	733	<b>Mailing, Reproduction, Stenographic</b>
7211	Power laundries, family & commercial	7331	Direct mail advertising services
7212	Garment pressing & cleaners' agents	7332	Blueprinting and photocopying
7213	Linen supply	7333	Commercial photography and art
7214	Diaper service	7339	Stenographic and reproduction, nec
7215	Coin-operated laundries and cleaning	734	<b>Services to Buildings</b>
7216	Dry cleaning plants, except rug	7341	Window cleaning
7217	Carpet and upholstery cleaning	7342	Disinfecting and exterminating
7218	Industrial launderers	7349	Building maintenance services, nec
7219	Laundry and garment services, nec	735	<b>News Syndicates</b>
722	<b>Photographic Studios, Portrait</b>	7351	News syndicates
7221	Photographic studios, portrait	736	<b>Personnel Supply Services</b>
723	<b>Beauty Shops</b>	7361	Employment agencies
7231	Beauty shops	7362	Temporary help supply services
724	<b>Barber Shops</b>	7369	Personnel supply services, nec
7241	Barber shops	737	<b>Computer and Data Processing Services</b>
725	<b>Shoe Repair and Hat Cleaning Shops</b>	7372	Computer programming and software
7251	Shoe repair and hat cleaning shops	7374	Data processing services
726	<b>Funeral Service and Crematories</b>	7379	Computer related services, nec
		739	<b>Miscellaneous Business Services</b>
		7391	Research & development laboratories



## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
7392	Management and public relations	79	<b>AMUSEMENT &amp; RECREATION SERVICES</b>
7393	Detective and protective services	791	<b>Dance Halls, Studios, and Schools</b>
7394	Equipment rental and leasing	7911	Dance halls, studios, and schools
7395	Photofinishing laboratories	792	<b>Producers, Orchestras, Entertainers</b>
7396	Trading stamp services	7922	Theatrical producers and services
7397	Commercial testing laboratories	7929	Entertainers & entertainment groups
7399	Business services, nec	793	<b>Bowling and Billiard Establishments</b>
75	<b>AUTO REPAIR, SERVICES, AND GARAGES</b>	7932	Billiard and pool establishments
751	<b>Automotive Rentals, Without Drivers</b>	7933	Bowling alleys
7512	Passenger car rental and leasing	794	<b>Commercial Sports</b>
7513	Truck rental and leasing	7941	Sports clubs and promoters
7519	Utility trailer rental	7948	Racing, including track operation
752	<b>Automobile Parking</b>	799	<b>Misc. Amusement, Recreational Services</b>
7523	Parking lots	7992	Public golf courses
7525	Parking structures	7993	Coin-operated amusement devices
753	<b>Automotive Repair Shops</b>	7996	Amusement parks
7531	Top and body repair shops	7997	Membership sports & recreation clubs
7534	Tire retreading and repair shops	7999	Amusement and recreation, nec
7535	Paint shops	80	<b>HEALTH SERVICES</b>
7538	General automotive repair shops	801	<b>Offices of Physicians</b>
7539	Automotive repair shops, nec	8011	Offices of physicians
754	<b>Automotive Services, Except Repair</b>	802	<b>Offices of Dentists</b>
7542	Car washes	8021	Offices of dentists
7549	Automotive services, nec	803	<b>Offices of Osteopathic Physicians</b>
76	<b>MISCELLANEOUS REPAIR SERVICES</b>	8031	Offices of osteopathic physicians
762	<b>Electrical Repair Shops</b>	804	<b>Offices of Other Health Practitioners</b>
7622	Radio and television repair	8041	Offices of chiropractors
7623	Refrigeration service and repair	8042	Offices of optometrists
7629	Electrical repair shops, nec	8049	Offices of health practitioners, nec
763	<b>Watch, Clock, and Jewelry Repair</b>	805	<b>Nursing and Personal Care Facilities</b>
7631	Watch, clock, and jewelry repair	8051	Skilled nursing care facilities
764	<b>Reupholstery and Furniture Repair</b>	8059	Nursing and personal care, nec
7641	Reupholstery and furniture repair	806	<b>Hospitals</b>
769	<b>Miscellaneous Repair Shops</b>	8062	General medical & surgical hospitals
7692	Welding repair	8063	Psychiatric hospitals
7694	Armature rewinding shops	8069	Specialty hospitals, exc. psychiatric
7699	Repair services, nec	807	<b>Medical and Dental Laboratories</b>
78	<b>MOTION PICTURES</b>	8071	Medical laboratories
781	<b>Motion Picture Production &amp; Services</b>	8072	Dental laboratories
7813	Motion picture production, except TV	808	<b>Outpatient Care Facilities</b>
7814	Motion picture production for TV	8081	Outpatient care facilities
7819	Services allied to motion pictures	809	<b>Health and Allied Services, nec</b>
782	<b>Motion Picture Distribution and Services</b>	8091	Health and allied services, nec
7823	Motion picture film exchanges	81	<b>LEGAL SERVICES</b>
7824	Film or tape distribution for TV	811	<b>Legal Services</b>
7829	Motion picture distribution services	8111	Legal services
783	<b>Motion Picture Theaters</b>	82	<b>EDUCATIONAL SERVICES</b>
7832	Motion picture theaters, ex drive-in	821	<b>Elementary and Secondary Schools</b>
7833	Drive-in motion picture theaters	8211	Elementary and secondary schools
		822	<b>Colleges and Universities</b>
		8221	Colleges and universities, nec

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>
8222	Junior colleges
823	<b>Libraries and Information Centers</b>
8231	Libraries and information centers
824	<b>Correspondence and Vocational Schools</b>
8241	Correspondence schools
8243	Data processing schools
8244	Business and secretarial schools
8249	Vocational schools, nec
829	<b>Schools &amp; Educational Services, nec</b>
8299	Schools & educational services, nec
83	<b>SOCIAL SERVICES</b>
832	<b>Individual and Family Services</b>
8321	Individual and family services
833	<b>Job Training and Related Services</b>
8331	Job training and related services
835	<b>Child Day Care Services</b>
8351	Child day care services
836	<b>Residential Care</b>
8361	Residential care
839	<b>Social Services, nec</b>
8399	Social services, nec
84	<b>MUSEUMS, BOTANICAL, ZOOLOGICAL GARDENS</b>
841	<b>Museums and Art Galleries</b>
8411	Museums and art galleries
842	<b>Botanical and Zoological Gardens</b>
8421	Botanical and zoological gardens

<i>Code</i>	<i>Short Title</i>
86	<b>MEMBERSHIP ORGANIZATIONS</b>
861	<b>Business Associations</b>
8611	Business associations
862	<b>Professional Organizations</b>
8621	Professional organizations
863	<b>Labor Organizations</b>
8631	Labor organizations
864	<b>Civic and Social Associations</b>
8641	Civic and social associations
865	<b>Political Organizations</b>
8651	Political organizations
866	<b>Religious Organizations</b>
8661	Religious organizations
869	<b>Membership Organizations, nec</b>
8699	Membership organizations, nec
88	<b>PRIVATE HOUSEHOLDS</b>
881	<b>Private Households</b>
8811	Private households
89	<b>MISCELLANEOUS SERVICES</b>
891	<b>Engineering &amp; Architectural Services</b>
8911	Engineering & architectural services
892	<b>Noncommercial Research Organizations</b>
8922	Noncommercial research organizations
893	<b>Accounting, Auditing &amp; Bookkeeping</b>
8931	Accounting, auditing & bookkeeping
899	<b>Services, nec</b>
8999	Services, nec

## J. PUBLIC ADMINISTRATION

<i>Code</i>	<i>Short Title</i>
91	<b>EXECUTIVE, LEGISLATIVE, AND GENERAL</b>
911	<b>Executive Offices</b>
9111	Executive offices
912	<b>Legislative Bodies</b>
9121	Legislative bodies
913	<b>Executive and Legislative Combined</b>
9131	Executive and legislative combined
919	<b>General Government, nec</b>
9199	General government, nec
92	<b>JUSTICE, PUBLIC ORDER, AND SAFETY</b>
921	<b>Courts</b>
9211	Courts
922	<b>Public Order and Safety</b>
9221	Police protection
9222	Legal counsel and prosecution
9223	Correctional institutions
9224	Fire protection
9229	Public order and safety, nec

<i>Code</i>	<i>Short Title</i>
93	<b>FINANCE, TAXATION &amp; MONETARY POLICY</b>
931	<b>Finance, Taxation &amp; Monetary Policy</b>
9311	Finance, taxation & monetary policy
94	<b>ADMINISTRATION OF HUMAN RESOURCES</b>
941	<b>Admin. of Educational Programs</b>
9411	Admin. of educational programs
943	<b>Admin. of Public Health Programs</b>
9431	Admin. of public health programs
944	<b>Admin. of Social &amp; Manpower Programs</b>
9441	Admin. of social & manpower programs
945	<b>Administration of Veterans' Affairs</b>
9451	Administration of veterans' affairs
95	<b>ENVIRONMENTAL QUALITY AND HOUSING</b>
951	<b>Environmental Quality</b>
9511	Air, water & solid waste management
9512	Land, mineral, wildlife conservation

## List of SIC Codes and Their Short Titles

<i>Code</i>	<i>Short Title</i>	<i>Code</i>	<i>Short Title</i>
953	Housing and Urban Development	964	Regulation of Agricultural Marketing
9531	Housing programs	9641	Regulation of agricultural marketing
9532	Urban and community development	965	Regulation Misc. Commercial Sectors
96	ADMINISTRATION OF ECONOMIC PROGRAMS	9651	Regulation misc. commercial sectors
961	Admin. of General Economic Programs	966	Space Research and Technology
9611	Admin. of general economic programs	9661	Space research and technology
962	Regulation, Admin. of Transportation	97	NATIONAL SECURITY AND INTL. AFFAIRS
9621	Regulation, admin. of transportation	971	National Security
963	Regulation, Admin. of Utilities	9711	National security
9631	Regulation, admin. of utilities	972	International Affairs
		9721	International affairs

## K. NONCLASSIFIABLE ESTABLISHMENTS

<i>Code</i>	<i>Short Title</i>
99	NONCLASSIFIABLE ESTABLISHMENTS
999	Nonclassifiable Establishments
9999	Nonclassifiable establishments

\*New codes in 1977 (3716 and 6798). Codes 404 and 4041 were deleted.

**Announcement of Changes  
to  
FIPS PUB 66**

**STANDARD INDUSTRIAL CLASSIFICATION (SIC) CODES**

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Institute for Computer Sciences  
and Technology  
National Bureau of Standards  
Washington, D.C. 20234

Gentlemen :

Please add my name to your mailing list for changes to FIPS PUB 66. I understand that I will receive information relating to changes in SIC industries, their definitions and codes .

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## FIPS PUBLICATION CHANGE NOTICE

PUBLICATION TITLE

### FIPS PUB 66, STANDARD INDUSTRIAL CLASSIFICATION (SIC) CODES.

THIS OFFICE HAS A RECORD OF YOUR INTEREST IN RECEIVING CHANGES TO THE ABOVE FIPS PUBLICATION. THE CHANGE(S) INDICATED BELOW HAVE BEEN PROVIDED BY THE MAINTENANCE AGENCY FOR THIS PUBLICATION AND WILL BE INCLUDED IN THE NEXT PUBLISHED REVISION TO THIS FIPS PUBLICATION. QUESTIONS OR REQUESTS FOR ADDITIONAL INFORMATION SHOULD BE ADDRESSED TO THE MAINTENANCE AGENCY:

**Mr. Paul Bugg, Office of Information and Regulatory Affairs, Office of Management and Budget, Washington, DC 20503, 202/395-3093. Address Corrections/changes to: Computer Systems Laboratory, Bldg. 225, Rm. B64, NIST, Gaithersburg, MD 20899.**

CHANGE ITEM(S)

This change notice incorporates Change Notice 1 for FIPS 66 and provides information on the maintenance, applicability, and implementation of FIPS 66.

\*\*\*\*\*

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**Name of Standard.** Standard Industrial Classification (SIC) Codes, (FIPS PUB 66).

**Category of Standard.** Representations and Codes.

**Explanation.** This standard provides classifications, short titles, and codes for representing industries. The general concept of an industry is one of a group of establishments with similar economic activities. The SIC codes are developed and maintained by the Office of Management and Budget. This revised text for FIPS 66 reflects editorial changes and updates. This revised text and the Standard Industrial Classification Manual for 1987 comprise FIPS PUB 66 and supersede in its entirety FIPS PUB 66 dated 1979 August 15.

**Approving Authority.** Secretary of Commerce.

**Maintenance Agency.** Office of Management and Budget, Office of Information and Regulatory Affairs, Washington, DC 20503.

**Applicability.** This standard is prescribed for the interchange of data among agencies and between agencies and the public including industry and State and local governments. Use within agency data systems is encouraged when such use contributes to operational benefits, efficiency, or economy.

This classification should be used whenever data for domestic establishments need to be classified industrially. For international comparisons, the SIC can be converted to the International Standard Industrial Classification (ISIC) with minor exceptions where the U.S. has a different classification structure.

**Effective date.** February 15, 1980.

-contd.-



**Specifications.** The Standard Industrial Classification (SIC) Manual for 1987 issued by the Office of the Management and Budget in the Executive Office of the President should be used as the source of classifications, short titles, and codes for representing industries as prescribed by FIPS PUB 66. This Manual supersedes in its entirety the classifications, short titles and codes that were published in FIPS PUB 66, dated 1979 August 15.

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- a. compliance with a standard would adversely affect the accomplishment of the mission of an operator of a Federal computer system, or
- b. cause a major adverse financial impact on the operator which is not offset by Governmentwide savings.

Agency heads may act upon a written waiver request containing the information detailed above. Agency heads may also act without a written waiver request when they determine that conditions for meeting the standard cannot be met. Agency heads may approve waivers only by a written decision which explains the basis on which the agency head made the required finding(s). A copy of each such decision, with procurement sensitive or classified portions clearly identified, shall be sent to: National Institute of Standards and Technology; ATTN: FIPS Waiver Decisions, Technology Building, Room B-154; Gaithersburg, MD 20899.

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When the determination on a waiver applies to the procurement of equipment and/or services, a notice of the waiver determination must be published in the Commerce Business Daily as a part of the notice of solicitation for offers of an acquisition or, if the waiver determination is made after that notice is published, by amendment to such notice.

A copy of the waiver, any supporting documents, the document approving the waiver and any supporting and accompanying documents, with such deletions as the agency is authorized and decides to make under 5 U.S.C. Section 552(b), shall be part of the procurement documentation and retained by the agency.

**Where to Obtain Copies.** Copies of the Standard Industrial Classification (SIC) Manual for 1987 are for sale by the National Technical Information Service, U.S. Department of Commerce, Springfield, VA 22161.

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A computer tape version, including documentation, (9-Track, 1600 or 6250 bpi) may be ordered as PB87-100020, \$240. Also available on high density diskettes (PB87-199576, \$195.) by special request and a computerized SIC index for microcomputers which has search retrieval (PB91-507947, \$149). Add \$3 handling fee per order.

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**COMMONWEALTH OF VIRGINIA**  
**Department of Environmental Quality**

**Intra-Agency Memorandum**

**DATE:** April 30, 2013

**SUBJECT:** Engineering Evaluation of a Nonattainment Area Major New Source Review (NAA-MNSR) and Prevention of Significant Deterioration (PSD) Permit Application Submitted by Green Energy Partners / Stonewall, LLC Registration No. 73826

**TO:** Thomas A Faha, Director, Northern Regional Office

**FROM:** Thomas Valentour, Environmental Specialist Senior, Northern Regional Office

**AIR PERMIT MANAGER REVIEW:**

*James B. LeBarth* 4/30/13

**REGIONAL DIRECTOR REVIEW:**

*Thomas A. Faha* 4.30.13

**I. Executive Summary**

Green Energy Partners / Stonewall, LLC has proposed to construct and operate a combined-cycle electric power generating facility in Loudoun County with a nominal generating capacity of 750 megawatts (MW) at ISO (International Organization for Standardization) conditions. Both Major Nonattainment New Source Review (NAA-MNSR) permitting and Prevention of Significant Deterioration (PSD) permitting are applicable because, the facility is a fossil fuel-fired steam electric plant of more than 250 million British thermal units (Btus) heat input capacity, and is locating in an ozone nonattainment area and a PM-2.5 nonattainment area but an attainment area for the other criteria pollutants. The proposed facility has the potential to emit (PTE) more than 100 tons per year (tpy) of nitrogen oxides (NO<sub>x</sub>) and over 50 tpy of volatile organic compounds (VOC) which trigger the requirements of Major Nonattainment permitting under Article 9 of 9 VAC 5 Chapter 80. The facility also has the PTE of more than 100 tpy of carbon monoxide (CO) triggering the PSD requirements under Article 8 of 9 VAC 5 Chapter 80. Other pollutants for which the facility has the PTE in significant amounts include NO<sub>x</sub>, PM (TSP), PM-10, PM-2.5 and greenhouse gasses (CO<sub>2</sub>e) all of which are also subject to PSD Review. The facility's PTE for all other regulated NSR pollutants is not significant for PSD purposes.

Both the Nonattainment NSR and PSD regulations provide reviewing authority to Federal Land Managers (FLMs) of Class I areas that may be affected by emissions from the proposed facility. In accordance with Memoranda of

Understanding (MOU) between the Virginia Department of Environmental Quality (DEQ) and the respective FLMs, both the National Park Service (NPS) and the National Forest Service (NFS) are given a 60-day review and comment period once provided notification that the application is considered complete. Within the first 30 days of the review period, the FLMs are asked whether or not they will provide a finding of an adverse impact on visibility and other applicable Air Quality Related Values (AQRVs) as a result of the proposed facility. FLMs may comment on any aspect of permit processing, but are specifically charged with protecting the AQRVs within the Class I areas.

The following table shows the distances between the proposed plant site and the closest Class I areas:

*Table 1. Distance of proposed plant from Class I areas (km)*

<b>Class I area</b>	<b>Distance from proposed plant (km)</b>
Shenandoah National Park (SNP)	57
Dolly Sods Wilderness Area (West Virginia)	152
Otter Creek Wilderness Area (West Virginia)	175
James River Face Wilderness Area	227
Brigantine National Wildlife Refuge	271

Sources located in nonattainment areas must apply the Lowest Achievable Emission Rates (LAER) to pollutants for which the area is in nonattainment. For this permit action, LAER was evaluated for both NO<sub>x</sub> and VOCs. Also, as a requirement of MNSR, the source must obtain offsets of nonattainment pollutants. Pollutants for which the area is in attainment are subject to a Best Available Control Technology (BACT) analysis. This involves a “top down” analysis of all technically feasible control technologies and the utilization of the most stringent level of control that can be demonstrated to be either technically or economically feasible. Economic feasibility takes into consideration the cost of controls required at similar recently permitted facilities. Pollutants for which the facility’s PTE is not significant may undergo a state BACT determination.

Green Energy Partners / Stonewall, LLC originally submitted an application in May 2010, but did not complete the application process. In July 2012 they resubmitted the application that revised the plan to eliminate the two simple cycle combustion turbines. The application was treated as an amended application.

## **II. Introduction and Background**

On July 24, 2012, the Northern Regional Office of the Department of Environmental Quality (NRO-DEQ) received an application dated July 19, 2012, from Green Energy Partners / Stonewall, LLC (GEP/S) for a NAA-MNSR/PSD/Minor NSR permit to construct and operate a combined-cycle electric generating facility in Loudoun County. GEP/S has requested that the proposed permit allow two optional plant configurations, each having a different

combustion turbine manufacturer. The two combustion turbine configuration options currently being considered are the General Electric GE7FA.05 and Siemens SGT6-5000F5 units. GEP/S will submit a letter requesting the withdrawal of one of the two options at the time when their final decision is made.

#### **A. Site Information**

The proposed site for Green Energy Partners / Stonewall, LLC (GEP/S) is a 101-acre parcel, approximately south-southeast of the Town of Leesburg airport and north of the Dulles Toll Road, and adjacent Gant Lane and Cochran Mill Road.

The address for the facility is 20077 Gant Lane, Leesburg, Virginia 20175. The UTM coordinates of the proposed site are 279.7435 kilometers (km) Easting and 4326.0578 km Northing. The project will be located at a base elevation of 320 feet above mean sea level.

There is gently rolling terrain with wetlands, forest and undeveloped land around the proposed site.

#### **B. Site Suitability**

In accordance with Section 10.1-1307 E of the Air Pollution Control Law of Virginia, consideration has been given to the following facts and circumstances relevant to the reasonableness of the activity involved:

1. *The character and degree of injury to, or interference with safety, health, or the reasonable use of property which is caused or threatened to be caused:*

The activities regulated in this permit have been evaluated consistent with 9 VAC5-80-1750 (PSD BACT), 9VAC5-80-2050 (LAER), 9 VAC 5-50-260 (State BACT) and 9 VAC 5-60-320 (Toxics Rule) and have been determined to meet these standards where applicable. Please see Section IV.D.2 for a description of the Lowest Achievable Emission Rate, and see Section IV.D.3 for Best Available Control Technology standards included in the permit. Please refer to Section IV.B for more information on the applicability of the Toxics Rule to the proposed facility.

As a fossil fuel-fired steam electric generating plant having heat input greater than 250 million British thermal units per hour, the proposed facility is a major stationary source according to Article 8, 9 VAC 5-80-1615 C for carbon monoxide (CO), nitrogen oxides (NOx), PM-10, and greenhouse gasses, and a major stationary source according to

Article 9, 9 VAC 5-80-2000 for oxides of nitrogen (NO<sub>x</sub>). If the facility chooses the Siemens model combustion turbines, it will also be subject to 9 VAC 5-80-2000, *et seq.* for volatile organic compounds (VOC) air pollutant emissions. In accordance with Article 8 and 9, Permits for Major Stationary Sources and Major Modifications Locating in a Prevention of Significant Deterioration Areas and Major Sources Locating in Nonattainment Areas or the Ozone Transport Region, air quality modeling was conducted to predict the maximum ambient impacts of criteria pollutants emitted by the proposed source. The modeling results for NO<sub>2</sub> (annual averaging period), PM-2.5 and CO (8-hour averaging period) were less than the applicable Significant Impact Levels (SILs) for both turbine options. Also, the modeling results for CO (1-hour averaging period) for the Siemens turbine option only were less than the applicable SIL. Therefore, a full impact analysis for these pollutants and averaging periods was not required. Furthermore, the additional pollution from this facility would not cause or contribute to a violation of any applicable National Ambient Air Quality Standard (NAAQS) or PSD increment for all pollutants and averaging periods with impacts below the applicable SILs.

A full impact analysis for CO (1-hour averaging period, General Electric turbine option only), NO<sub>2</sub> (1-hour averaging period), and PM-2.5 (24-hour averaging period) was conducted because the preliminary modeling analysis results exceeded the applicable SILs. Additionally, a full impact analysis was conducted for PM-2.5 (annual averaging period) at the request of DEQ even though the facility's predicted impact was below the SIL. This was done to provide additional assurance of NAAQS compliance in the Washington, D.C. Metropolitan Statistical Area (MSA). The results of the full impact analysis demonstrated compliance with the applicable NAAQS.

GEP/S's project is proposed to be sited at a distance of 57 kilometers from SNP, a protected Class I area. Based on the level of emissions from the proposed facility, the FLMs determined an AQRV analysis is not required because the project is not expected to show any significant additional impacts to AQRVs. Therefore, only a Class I area analysis to assess compliance with the Class I PSD increments was required. The analysis demonstrated that the proposed facility does not cause or significantly contribute to a predicted violation of any applicable Class I area PSD increment. The modeling results are discussed in Attachment C.

The emissions of toxic pollutants from electric generating units such as those proposed by GEP/S are subject to the standards in 9 VAC 5-60-300 *et seq.* GEP/S calculated the emissions of toxic pollutants from all

of the emission units proposed for the site. An analysis was conducted in accordance with the regulations for permitting applicability and the predicted concentrations for each toxic pollutant were below their respective Significant Ambient Air Concentrations (SAACs). Modeling demonstrated that proposed emissions of acrolein, formaldehyde, cadmium, chromium, and nickel are well below (less than 1 %) the associated SAACs.

Since Loudoun County is part of the Northern Virginia Ozone Nonattainment area and is part of the Ozone Transport Region, GEP/S is required to obtain NO<sub>x</sub> emissions offsets at a 1.15:1.00 ratio. If GEP/S chooses Siemens combustion turbines, a VOC emission offset in the ratio of 1.15:1.00 will also be required. GEP/S had not yet identified the source of the offsets but is required to make them federally enforceable and enforceable as a practicable matter prior to the initial start up of the combined cycle combustion turbines.

Results of modeling conducted for emissions from the proposed facility show compliance with the health-based NAAQS for all applicable pollutants. Furthermore, single source and cumulative modeling analyses indicate that the proposed project will not result in a violation of any PSD increment. Accordingly, approval of the proposed permit is not expected to cause injury to or interference with safety, health, or reasonable use of property.

2. *The social and economic value of the activity involved:*

The social and economic value of the facility submitting the application has been evaluated relative to local zoning requirements. The local official has deemed this activity not inconsistent with local ordinances. The signed Local Government Form is included in Attachment E.

The proposed GEP/S facility will generate electricity using natural gas. The availability of clean fuel electric generation facilities is necessary if operation of conventional coal-fired power plants is to be reduced or replaced. Although it is not guaranteed that regional coal-powered generation will be reduced if clean-burning plants such as the GEP/S project are built, if they are not built, it is certain that electricity demand will continue to be met through use of the older, dirtier facilities. Construction of clean-burning, efficient generation plants such as the proposed GEP/S facility creates the potential for regional SO<sub>2</sub> and NO<sub>x</sub> reductions resulting from displacement of older, more polluting forms of electricity generation.

3. *The suitability of the activity to the area in which it is located:*

The activities regulated in this permit are deemed suitable as follows:

- (i) *Air Quality characteristics and performance requirements defined by SAPCB regulations:*

This permit is written consistent with existing applicable regulations. The proposed facility will emit toxics and the modeling shows compliance with the applicable SAACs. The emissions for criteria pollutants associated with this permit have likewise been modeled and have been shown to not cause or contribute to a violation of the ambient air quality standards or allowable increments within any Class I or Class II areas.

The PSD Regulations require that GEP/S conduct modeling analyses to determine potential impacts of the proposed facility on visibility and other applicable AQRVs in Class I areas. However, based on the level of emissions from the proposed facility, the FLMs determined an AQRV analysis is not required because the project is not expected to show any significant additional impacts to AQRVs. The Class I and Class II area modeling results are discussed in Attachment C.

- (ii) *The health impact of air quality deterioration which might reasonably be expected to occur during the grace period allowed by the Regulations or the permit conditions to fix malfunctioning air pollution control equipment:*

The permit requires the facility to notify the Regional Office within four business hours of discovery of any malfunction of pollution control equipment.

- (iii) *Anticipated impact of odor on surrounding communities or violation of the SAPCB Odor Rule:*

No violation of Odor requirements is anticipated as a result of the proposed project.

4. *The scientific and economic practicality of reducing or eliminating the discharge resulting from the activity:*

The state NSR program as well as the PSD and nonattainment programs require consideration of levels of control technology that are written into regulation to define the level of scientific and



economic practicality for reducing or eliminating emissions. By properly implementing the Regulations through the issuance of the proposed permit, the staff has addressed the scientific and economic practicality of reducing or eliminating emissions associated with this project.

The permit requires numerous pollution control strategies (e.g., BACT, LAER, etc.) that will result in reduction of emissions. LAER is the most stringent emissions limitation which is achieved in practice by such class or category of stationary sources. These include pollution prevention techniques such as use of clean fuels, good combustion practices, and clean burning "low-NO<sub>x</sub>" lean premix burners as well as post-combustion controls (SCR for NO<sub>x</sub> removal and an Oxidation Catalyst for CO, VOC, and VOC toxic pollutant control). Pollution prevention measures have been included in the draft permit, such as a requirement to use ultra-low sulfur (no more than 0.0015 % by weight) oil in emergency equipment, and a limit on ammonia emissions (not currently a regulated pollutant)

### **C. Project Summary**

Green Energy Partners / Stonewall, LLC applied for a permit to construct and operate a combined-cycle electric power generating facility with a nominal generating capacity of 750 megawatts (MW). The proposed facility is comprised of two combustion turbine (CT) generators, each having a heat recovery steam generator (HRSG) driving a common steam turbine (ST) for additional electricity generation. Each HRSG has a duct burner (DB) for supplemental firing. The CT-HRSG arrangement is commonly called combined cycle. The proposed facility also includes an auxiliary boiler, an emergency firewater pump, an emergency generator, a fuel gas heater, and two turbine air inlet conditioners.

The CTs, HRSG DBs, the auxiliary boiler and fuel gas heater will only combust pipeline quality natural gas. The emergency firewater pump and emergency generator will utilize ultra low sulfur diesel fuel oil.

GEP/S has requested that the proposed permit allow two optional plant configurations, each having a different combustion turbine manufacturer. The two combustion turbine configuration options currently being considered are the General Electric GE7FA.05 and Siemens SGT6-5000F5 units. GEP/S will submit a letter requesting the withdrawal of one of the two options at the time when their final decision is made. Therefore, the proposed CT generators will either be General Electric



GE7FA.05 or Siemens SGT6-5000F5 units. Both scenarios were evaluated.

The proposed facility is capable of operating in either a gas (simple cycle) or steam cycle (combined cycle). In the simple cycle only the electric generators connected to the combustion turbine are used to produce electricity. The steam cycle provides increased efficiency by employing the HRSGs to recover otherwise lost heat from the CT exhaust and using it to create steam and drive the ST generator to produce additional electricity. The steam that exhausts the ST generator is cooled and condensed via the ten cell mechanical draft cooling tower for reuse in the steam cycle. The combined cycle system will provide approximately 750 MW of nominal power output.

Proposed annual mass emission rates from the GEP/S project are presented in Table 2.

*Table 2. Proposed Maximum Mass Emission rates (tons/yr) from the Green Energy Partners / Stonewall project.*

Pollutant	Emissions (tons/yr)	
	GE F7FA.05 Combustion Turbines & HRSGs with DBs On	Siemens SGT6- 5000F5 Combustion Turbines & HRSGs with DBs On
NO <sub>x</sub>	159.0	164.9
CO	205.6	143.6
SO <sub>2</sub>	5.44	5.37
VOC	37.6	51.9
PM-10	105.2	106.2
PM-2.5	98.1	99.1
Greenhouse Gases (GHG) Carbon Dioxide Equivalent (CO <sub>2</sub> e)	2,468,467	2,464,490
Sulfuric acid mist	2.87	2.81
Acrolein	8.76E-01	8.88E-02
Cadmium	2.25E-02	2.25E-02
Chromium	2.86E-02	2.86E-02
Formaldehyde	3.09	3.11
Nickel	4.29E-02	4.29E-02

Note: Emissions of regulated toxic pollutants other than formaldehyde, acrolein, cadmium, chromium, and nickel are below permitting annual exemption thresholds and were therefore not included in Table 2. Tables 8 and 9 below have all HAPS listed.

The following permitting regulations apply to the proposed facility:

- 9 VAC 5 Chapter 80 Article 9 Permits for Major Stationary Sources and Major Modifications Locating in Nonattainment Areas or the Ozone Transport Region NAA-NSR for either NO<sub>x</sub>, or NO<sub>x</sub> and VOC depending on the combustion turbine model chosen.
- 9 VAC 5 Chapter 80 Article 8 Permits for Major Stationary Sources and Major Modifications Locating in Prevention of Significant Deterioration Areas PSD permitting regulations for emissions of CO, NO<sub>x</sub>, PM, PM<sub>10</sub>, and GHG.
- 9 VAC 5 Chapter 80 Article 6 Permits for New and Modified Stationary Sources - Minor NSR for PM-2.5, PM-10, CO, NO<sub>x</sub> and VOC
- 9 VAC Chapter 80 Article 1 Federal (Title V) Operating Permits for Stationary Sources (application must be submitted within one year of commencing operation)

The following regulations also apply to the proposed facility:

- New Source Performance Standard (NSPS), 40 CFR 60, Subpart KKKK applies to the combustion turbines.
- New Source Performance Standard (NSPS), 40 CFR 60, Subpart Dc applies to the auxiliary boiler and the fuel gas heater.
- New Source Performance Standard (NSPS), 40 CFR 60, Subpart IIII applies to the emergency generator and fire water pump.
- Maximum Achievable Control Technology (MACT), 40 CFR 63, Subpart ZZZZ applies to the emergency generator and fire water pump.
- Title IV Acid Rain Program.
- 9 VAC 5 Chapter 140, NO<sub>x</sub> Budget Trading Program, Clean Air Interstate Rule (CAIR) NO<sub>x</sub> Annual Trading Program, CAIR NO<sub>x</sub>

Ozone Season Trading Program, and CAIR SO<sub>2</sub> Annual Trading Program.

Rules that don't apply:

- The Combustion Turbine MACT, 40 CFR 63, Subpart YYYYY, applies to combustion sources located at major sources of HAP. GEP/S is an area source of HAPs and therefore is not an affected source under the Combustion Turbine MACT.
- The MACT for cooling towers, 40 CFR 63, Subpart Q, prohibits the use of chromium based water treatment chemicals in an industrial process using a cooling tower. This standard does not apply because the facility is not a major source of HAPs, and chromium-based cooling tower water treatment chemicals will not be used.

#### **D. Process/Equipment Description**

Green Energy Partners / Stonewall, LLC has proposed installation of the following combustion turbines and heat recovery steam generators:

- Two GE (Model GE.7FA) or two Siemens (SGT6-5000F5) natural gas-fired combustion turbine generators with inlet evaporative coolers (CCT1 and CCT2); each GE combustion turbine will produce 204.6 MW with the inlet evaporative coolers on and 193.3 MW with them off (at 92 °F). The maximum total gross power output is expected to be 230.9 MW at 18° F. For the Siemens option, each CT will produce 217.3 MW with the evaporative coolers on and 207.4 MW with them off (at 92 °F). The maximum total gross power output is expected to be 230.9 MW at 18° F.
- Two heat recovery steam generators (HRSG) with supplementary natural gas-fired duct burners (DB1, DB2), each rated at 650 MMBtu/hr heat input for the GE7FA.05 or 450 MMBtu/hr heat input for the SGT6-5000F5.

GEP/S has proposed the installation of the following ancillary equipment:

- One reheat, condensing steam turbine driven electric generator designed for variable pressure operation and capable of producing approximately 350 MW of electrical power;
- One natural gas-fired auxiliary boiler, rated at 75 MMBtu/hr heat input (AB1);

- One natural gas-fired fuel gas heater, rated at 20.0 MMBtu/hr heat input (FGH1);
- One diesel-fired Emergency Fire Water Pump, rated at 330 bhp (2.54 MMBtu/hr heat input) (EFP1);
- One diesel-fired Emergency Generator, rated at 2,088 bhp / 1,500 kW (15.04 MMBtu/hr heat input) (EG-1); and
- One 1,250-gallon fuel oil storage tank (EGT).
- One 400-gallon fuel oil storage tank (FWPT)
- One 12,000 gallon aqueous ammonia storage tank
- Ten cell mechanical draft cooling tower (MCT-1)

### **Combustion Turbine Generators (CT)**

Each gas turbine power block will include an advanced firing temperature combustion turbine air compressor section, gas combustion system (utilizing dry, low-NO<sub>x</sub> combustors), power turbine, and a generator.

The gas turbine is the main component of a combined-cycle power system. First, air is filtered, cooled by the evaporative cooler during warm weather, and compressed in a multiple stage axial flow compressor. Compressed air and fuel are mixed and combusted in the turbine combustion chamber. Lean pre-mix dry low-NO<sub>x</sub> combustors minimize NO<sub>x</sub> formation during natural gas combustion. Hot exhaust gases from the combustion chamber are expanded through a multi-stage power turbine that results in energy to drive both the air compressor and electric power generator.

In combined-cycle mode, the exhaust gas exiting the power turbine is ducted to a boiler commonly known as an HRSG where steam is produced to generate additional electricity in a steam turbine generator. Natural gas-fired duct burners located within the HRSGs are used for supplementary firing to increase steam output.

The combustion turbines are designed to operate in the dry low-NO<sub>x</sub> mode at loads from approximately 60 percent up to 100 percent rating and will normally be taken out of service for scheduled maintenance, or as dictated by economic or electrical demand conditions.

### **Turbine Inlet Evaporative Coolers**

Under certain meteorological conditions (e.g., hot, humid days), evaporative cooling will be used to cool the air entering the combustion turbine (CT) by evaporating water sprayed into the air intake, just behind the inlet filter. A mist eliminator will assure that no water droplets reach the turbine blades. The purpose of the cooling is to increase the density of the air entering the CT to increase its output capacity. The CT is a volumetric machine and thus produces more power with more pounds of air entering the machine. The evaporative cooler achieves this goal in the summer time by cooling the air when temperatures are high.

### **Heat Recovery Steam Generators (HRSG) with Duct Burners (DB)**

The proposed facility will use two HRSGs, one for each CT, which will use waste heat to produce additional electricity. Each HRSG will act as a heat exchanger to derive heat energy from the CT exhaust gas to produce steam that will be used to drive a steam turbine generator (ST). The HRSGs system will extract heat from the exhaust of each gas turbine. Exhaust gas entering the HRSG at approximately 1,100 °F will be cooled to 165 °F to 200 °F by the time it leaves the HRSG exhaust stack. Steam production in the HRSGs may be augmented using duct burners (DBs) that will be fired by natural gas, and will be limited by a permit condition to operate 1,400 hours a year (each) on a rolling 12-month basis. The proposed DBs will have a firing rate of 650 MMBtu/hr each for the GE 7FA.05 and 450 MMBtu/hr for the Siemens SGT6-5000F5. The heat recovered is used in the combined-cycle plant for additional steam generation and natural gas/feedwater heating. Each HRSG will include high-pressure superheaters, a high-pressure evaporator, high-pressure economizers, reheat sections (to reheat partially expanded steam), an intermediate-pressure superheater, an intermediate-pressure evaporator, an intermediate-pressure economizer, a low-pressure superheater, a low-pressure evaporator, and a low-pressure economizer. Control devices such as selective catalytic reduction (SCR) and oxidation catalysts will be installed to control NO<sub>x</sub> and CO, respectively.

The stack will be equipped with a Continuous Emissions Monitoring System (CEMS) for monitoring emissions of NO<sub>x</sub>, CO and concentration of oxygen.

### **Steam Turbine Generator (ST)**

The proposed project includes one reheat, condensing steam turbine designed for variable pressure operation. The high-pressure portion of the steam turbine receives high-pressure super-heated steam from the HRSGs,

and exhausts to the reheat section of the HRSGs. The steam from the reheat section for the HRSGs is supplied to the intermediate-pressure section of the turbine, which expands to the low-pressure section. The low-pressure turbine also receives excess low-pressure superheated steam from the HRSGs and exhausts to the condenser which is cooled with water from a cooling tower. The steam turbine set is designed to produce up to approximately 350 MW of electrical output at ISO conditions with duct firing.

#### **Ten Cell Mechanical Draft Cooling Tower (MCT1-MCT10)**

The proposed project will include a 10-cell, 187,400 gal/min mechanical draft cooling tower to service the condenser for the steam turbine. The tower will employ plume abatement to eliminate visible plumes except during extreme cold weather conditions. The cooling tower will also utilize highly efficient drift eliminators to reduce water losses during operation. The drift eliminators also serve the purpose of reducing particulate emissions from dissolved solids in the drift water.

#### **Auxiliary Boiler (AB1)**

The proposed facility will include an auxiliary boiler (AB1). The auxiliary boiler will provide sealing steam to the steam turbine generator at start-up and at cold starts to warm up the steam turbine generator rotor. The steam from the auxiliary boiler will not be used to augment the power generation of the CTGs or steam turbine. The proposed AB1 will be fired with natural gas, with a firing rate of 75 MMBtu/hr. GEP/S has requested the boiler to be permitted to operate without annual operating restrictions.

#### **Fuel Gas Heater (FGH1)**

The proposed facility will include a fuel gas heater (FGH1). The heater will be used to warm up the incoming natural gas fuel to prevent freezing of the gas regulating valves under certain gas system operating conditions. The proposed FGH1 will be fired with natural gas only and have a firing rate of 20 MMBtu/hr.

#### **Diesel-Fired Emergency Generator (EG1)**

The proposed facility will include a 2,088 bhp (15.04 MMBtu/hr and 1,500 kW/hr) diesel-fired emergency generator that will be operated up to 500 hours per year which includes the testing and maintenance hours. The emergency generator will provide power in emergency situations for turning gears, lube oil pumps, auxiliary cooling water pumps and water supply pumps. Testing and maintenance operation of the emergency

generator will be limited to 100 hours per year. The emergency diesel generator is not intended to provide sufficient power for a black start, peak shaving or non emergency power.

#### **Diesel-Fired Emergency Fire Water Pump (EFP-1)**

The proposed project will include a 330 bhp (2.54 MMBtu/hr) diesel-fired fire water pump operated as a fire water pump driver. The unit will be limited to 500 hours per year, including monthly testing and maintenance.

#### **Fuel Oil Storage Tanks**

The proposed project will include a 1,250-gallon fuel oil storage tank to provide fuel for the emergency generator, and a 400-gallon fuel oil tank to provide fuel for the fire water pump.

#### **Aqueous Ammonia Storage Tank**

The proposed project will include a 12,000-gallon aqueous ammonia storage tank to provide ammonia for the Selective Catalytic Reaction systems on the combined cycle combustion turbines.

### **E. Schedule of Project**

NRO received the modeling protocol for Green Energy Partners / Stonewall, LLC and Form 7 air permit application on July 24, 2012 (dated July 19, 2012). Application amendment information was submitted by GEP/S and received on August 16, 2012 and a revised application on October 4, 2012, and November 13, 2012. The proposed date for beginning actual construction is fall 2013. The target date for startup and electrical generation is 2014-2016.

## **III. Emissions Calculations**

### **A. Criteria Pollutants**

Proposed emissions are primarily products of combustion from the combustion turbines and duct burners. There are also emissions from the cooling towers, auxiliary boiler, fuel gas heater, emergency generator, and the emergency firewater pump.

Emissions from the combined-cycle units vary depending on ambient temperature, relative humidity, and percent of operating capacity ("load") of the unit. The CT manufacturer – GE or Siemens - provided criteria pollutant emissions for 6 operating scenarios (a.k.a. Operating Points) for

the GE 7FA.05, and four operating scenarios for the Siemens SGT6-5000F5 reflecting various temperature, humidity, and load conditions. Emissions for all operating loads (identified as Operating Point 1 through Operating Point 4, and one through 6) are shown in Table 5-2, and Table 5-3 of the application. SO<sub>2</sub> emissions are based on use of natural gas having a sulfur content of 0.1 grains per 100 standard cubic feet of gas, the maximum sulfur content allowed by the proposed permit.

Short-term emissions for the CTs and DBs have been based on the maximum hourly emission rates (“worst-case” from all operating scenarios) for each pollutant, as shown in Table 3a and Table 3b below.

*Table 3a. GE 7FA.05 operating scenarios having highest short-term emissions (each CT)*

Pollutant	Operating Point	% Load	Ambient Temp. (°F)	Relative Humidity (%)	Inlet Evaporative Coolers (On/Off)	Emissions (lbs/hr)
NO <sub>x</sub>	4	100	18	60	Off	21.00
CO	4	100	18	60	Off	12.70
SO <sub>2</sub>	4	100	18	60	Off	0.75
VOC	4	100	18	60	Off	7.29
PM-10	4	100	18	60	Off	16.2
PM-2.5	4	100	18	60	Off	16.2

Note: Operating point 4 shown above is with Duct Burner operation.

*Table 3b. Siemens SGT6-5000F5 operating scenarios having highest short-term emissions (each CT)*

Pollutant	Operating Point	% Load	Ambient Temp. (°F)	Relative Humidity (%)	Inlet Evaporative Coolers (On/Off)	Emissions (lbs/hr)
NO <sub>x</sub>	6	100	59	60	On	20.40
CO	6	100	59	60	On	12.50
SO <sub>2</sub>	6	100	59	60	On	0.696
VOC	6	100	59	60	On	5.68
PM-10	6	100	59	60	On	14.5
PM-2.5	6	100	59	60	On	14.5

Note: Operating point 6 shown above is with Duct Burner operation.

Annual emissions for the CTs were calculated based on the combinations of operating scenarios shown in Table 4a and Table 4b below. The combination, proposed by GEP/S in its application, yields a more realistic “worst-case” representation for annual emissions: it is assumed that the facility can operate 8,760 hours per year for each pollutant, but not at worst-case ambient conditions (such conditions would not occur for all 8,760 hours). As listed in Table 7 below, the worst case CT annual



emissions for CO and VOC are based on annual emissions that include the startup and shutdown scenarios shown in Tables 4a and 4b. The worst case CT annual emissions for all other pollutants are based on the combination of CT with duct burner firing at 1,400 hours per year and the CT only at 7,360 hours per year. (Please note that the draft permit requires GEP/S to include startup and shutdown emissions of all criteria pollutants in calculating emissions to show compliance with its annual emissions limits.) The maximum annual turbine emissions were calculated in GEP/S's application and are included in Attachment A.

Table 4a. GE 7FA.05 operating scenario structure used as basis for annual emissions (each CT)

Operating Mode	Hours	Case	% Load	Inlet Chilling (On/Off)	Ambient Temp. (° F)	Relative Humidity (%)	Emissions (lbs/hr)						
							NO <sub>x</sub>	CO	VOC	PM-10	PM-2.5	SO <sub>2</sub> <sup>1</sup>	H <sub>2</sub> SO <sub>4</sub>
W/O Duct Burner	7360	1	100	Off	0	60	16.0	9.9	2.8	9.6	9.6	0.58	0.31
W/Duct Burner	1400	4	100	Off	18	60	21.00	12.7	7.29	16.2	16.2	0.75	0.40
Start Up / Shut Down	2960	Offline	NA	NA	NA	NA	NA	0	0	NA	NA	NA	NA
	1400 W/ DB	4	100	Off	0	60	21.00	12.7	7.29	16.2	16.2	0.75	NA
	4182 W/O DB	1	100	Off	0	60	16.0	9.9	2.8	9.6	9.6	0.58	0.31
	25.7	Hot Start	NA	NA	NA	NA	72.9	771.4	25.7	NA	NA	NA	NA
	113.1	Warm Start	NA	NA	NA	NA	158.1	468.7	25.2	NA	NA	NA	NA
	33.3	Cold Start	NA	NA	NA	NA	90.4	631.6	89.8	NA	NA	NA	NA
	45.5	Shut down	NA	NA	NA	NA	72.9	745.7	38.6	NA	NA	NA	NA

<sup>1</sup> SO<sub>2</sub> emissions are based on conversion of all sulfur in fuel to SO<sub>2</sub>, so startup and shutdown do not affect SO<sub>2</sub> emissions appreciably.

Table 4b. Siemens SGT6-5000F5 operating scenario structure used as basis for annual emissions (each CT)

Operating Mode	Hours	Case	% Load	Inlet Chilling (On/Off)	Ambient Temp. (° F)	Relative Humidity (%)	Emissions (lbs/hr)					
							NO <sub>x</sub>	CO	VOC	PM-10/PM-2.5	SO <sub>2</sub>	H <sub>2</sub> SO <sub>4</sub>
W/O Duct Burner	7360	1	100	Off	0	60	17.1	10.4	3.0	14.5	0.696	0.31
W/Duct Burner	1400	4	100	Off	18	60	20.4	12.5	5.7	14.5	0.75	0.40
Start Up / Shut Down	2960	Offline	NA	NA	NA	NA	NA	0	0	NA	NA	NA
	1400 W/ DB	4	100	Off	0	60	20.4	12.5	5.7	14.5	0.75	0.40
	4244 W/O DB	1	100	Off	0	60	17.1	10.4	3.0	14.5	0.696	0.31
	58.7	Hot Start	NA	NA	NA	NA	106.9	405.0	161.3	NA	NA	NA
	47.5	Warm Start	NA	NA	NA	NA	112.1	413.7	165.8	NA	NA	NA
	10.7	Cold Start	NA	NA	NA	NA	106.9	444.4	130.3	NA	NA	NA
	39.0	Shut down	NA	NA	NA	NA	125.0	385.0	150.0	NA	NA	NA

NO<sub>x</sub>, CO, and SO<sub>2</sub> emissions from the auxiliary boiler and fuel gas heater were calculated based on the proposed BACT emission rates for natural gas-fired boilers and heaters provided in GEP/S's application. PM 10 and PM-2.5 emissions for the auxiliary boiler were calculated based on vendor data. The auxiliary boiler has a capacity of 75 MMBtu/hr and the fuel gas heater has a capacity of 20.0 MMBtu/hr and both will burn natural gas. Annual emissions for the boiler and heater are based on 8760 hours of operation per year. Hourly and annual emissions are shown in Table 5.

*Table 5. Emissions from auxiliary boiler (AB1) and fuel gas heater (FGH1)*

Pollutant	Auxiliary Boiler (AB1)		Fuel Gas Heater (FGH1)	
	lbs/hr	tons/yr	lbs/hr	tons/yr
NO <sub>x</sub> <sup>a</sup>	0.83	3.61	0.22	0.96
CO <sup>a</sup>	2.78	12.15	0.74	3.24
VOC <sup>b</sup>	0.15	0.66	0.04	0.18
PM-10 (Filterable and Condensable)	0.15	0.66	0.04	0.18
PM-2.5 (Filterable and Condensable)	0.15	0.66	0.04	0.18
SO <sub>2</sub>	0.02	0.087	0.005	0.002
GHG and CO <sub>2</sub> e	8,873	38,856	2,365	10,362

<sup>a</sup> Based on emission factors from the proposed BACT emission rates for natural gas-fired boilers and heaters.

<sup>b</sup> Based on emission factor from AP-42, Table 1.4-2 (Natural Gas Combustion).

<sup>c</sup> Based on vendor data (auxiliary boiler only).

Emissions from the emergency generator and the emergency fire water pump (EG1 and EFP1) were based on the NSPS Subpart IIII limits for Stationary Compression Ignition Internal Combustion Engines. The emergency units will use ultra-low sulfur distillate oil having a maximum sulfur content of 0.0015% by weight consistent with NSPS Subpart IIII requirements. Annual emissions from EG1 and EFP1 are based on 500 hours of operation each. Short-term and annual emissions are shown in Table 6.

*Table 6. Emissions from emergency equipment (EG1 and EFP1)*

Pollutant	Emergency Generator (EG1)		Fire Water Pump (EFP1)	
	lbs/hr	tons/yr	lbs/hr	tons/yr
NO <sub>x</sub> <sup>a</sup>	21.98	5.49	2.17	0.54
CO <sup>a</sup>	12.02	3.0	1.72	0.47
VOC <sup>a, c</sup>	21.98	5.49	2.17	0.54
PM-10 <sup>d</sup>	1.37	0.34	0.22	0.00543
PM-2.5	1.37	0.34	0.22	0.00543

SO <sub>2</sub> <sup>b</sup>	0.025	0.0006	0.00039	0.000097
GHG and CO <sub>2</sub> e	2,630	658	415	104

<sup>a</sup> Based on emission factors from NSPS Subpart IIII limits for Stationary Compression Ignition Internal Combustion Engines (reference 40CFR 89.112 Table 1). NO<sub>x</sub> emissions are assumed to be worst case as entire NMHC + NO<sub>x</sub> emission standard is used for NO<sub>x</sub> emission factor.

<sup>b</sup> lb/hr based on fuel sulfur.

<sup>c</sup> VOC = TOC.

<sup>d</sup> Since AP-42 does not provide an emission factor for PM-10, the PM emission rate was multiplied by a factor of 2 to conservatively estimate the contribution of condensable particulate matter (CPM).

A summary of estimated annual emissions from the proposed facility, showing the contribution from each emission unit type, is shown in Table 7.

Table 7. - Annual emissions of criteria pollutants from proposed facility (tons/yr)

Pollutant	Combined cycle units (CT-1+DB1, CT-2+DB2)	Auxiliary Boiler (AB1)	Fuel Gas Heater (FGH1)	Emergency Generator (EG1)	Emergency Firewater Pump (EFP1)	Mechanical Draft Cooling Tower (MCT1)	Total
NO <sub>x</sub>	148.2 <sup>a</sup> / 154.48 <sup>b</sup>	3.61	0.96	5.49	0.54		159 <sup>a</sup> / 164.9 <sup>b</sup>
CO	188.6 <sup>a</sup> /124.8 <sup>b</sup>	12.15	3.24	3.0	0.47		205.6 <sup>a</sup> / 143.6 <sup>b</sup>
VOC	30.96 <sup>a</sup> / 45.26 <sup>b</sup>	0.66	0.18	5.49	0.54		37.6 <sup>a</sup> / 51.9 <sup>b</sup>
PM-10 (Condensable and filterable)	93.66 <sup>a</sup> / 94.68 <sup>b</sup>	0.66	0.18	0.34	0.00543	10.27	105.2 <sup>a</sup> / 106.1 <sup>b</sup>
PM-2.5 (Condensable and filterable)	93.66 <sup>a</sup> / 94.68 <sup>b</sup>	0.66	0.18	0.34	0.00543	3.19	98.1 <sup>a</sup> / 99.1 <sup>b</sup>
SO <sub>2</sub>	5.2 <sup>a</sup> / 5.26 <sup>b</sup>	0.0857	0.02	0.0006	0.000097		5.44 <sup>a</sup> / 5.37 <sup>b</sup>
GHG / CO <sub>2</sub> e	2,418,272 <sup>a</sup> / 2,414,296 <sup>b</sup>	38,856	10,362	658	104		2,468,468 <sup>a</sup> / 2,464,490 <sup>b</sup>

a – Based on the GE F7A.05 emissions (includes both w/o duct burner and/ with duct burner operations)

b – Based on the Siemens SGT6-5000F5 emissions (includes both w/o duct burner and with duct burner operations)

Emission calculations and supporting documentation for criteria pollutants can be found in Appendix B of GEP/S's revised applications dated November 13, 2012.

## B. HAPs/Toxic Pollutants

Hazardous air pollutant (HAP) emissions were calculated to determine whether the proposed facility has the potential to be a major source of HAPs under Title III of the Clean Air Act Amendments of 1990. Based on worst case emission factors, HAP emissions are summarized in Tables 8 and 9 below for the GE and Siemens turbines, respectively; detailed emission calculations are provided in Table B-5 of Appendix B of GEP/S's revised permit applications dated October 4, 2012 and November 13, 2012.

Table 8. GE 7FA.05 - Potential HAP emissions

Pollutant	Potential emissions	
	lbs/hr	TPY
1,3 Butadiene	1.44E-03	5.90E-03
2-Methylnaphthalene	2.36E-05	2.48E-05
3-Methylchloranthrene	1.77E-06	1.86E-06
7,12-Dimethylbenz(a)anthracene	1.58E-05	1.65E-05
Acenaphthene	8.06E-05	2.16E-05
Acenaphthylene	1.63E-04	4.22E-05
Acetaldehyde	1.27E-01	5.48E-01
Acrolein	2.03E-02	8.76E-01
Anthracene	2.69E-05	8.61E-06
Arsenic	1.15E-03	2.60E-04
Benz(a)anthracene	1.60E-05	5.43E-06
Benzene	5.44E-02	1.70E-01
Benzo(a)pyrene	5.79E-06	2.39E-06
Benzo(b)fluoranthene	1.99E-05	6.38E-06
Benzo(g,h,i)perylene	1.01E-05	3.47E-06
Benzo(k)fluoranthene	5.67E-06	2.83E-06
Beryllium	6.89E-05	1.56E-05
Cadmium	6.31E-03	1.43E-03
Chromium	8.04E-03	1.82E-03
Chrysene	2.73E-05	8.23E-06
Cobalt	4.82E-04	1.09E-04
Dibenzo(a,h)anthracene	8.23E-06	3.00E-06
Dichlorobenzene	1.18E-03	1.24E-03
Ethylbenzene	9.99E-02	4.38E-01
Fluoranthene	8.71E-05	2.41E-05
Fluorene	2.83E-04	7.29E-05
Formaldehyde	7.65E-01	3.09E+00
Hexane	1.77E+00	1.86E+00
Indeno(1,2,3-cd)pyrene	9.38E-06	3.76E-06

Napthalene	6.97E-03	1.90E-02
PAHs	6.87E-03	3.01E-02
Phenanathrene	7.47E-04	2.00E-04
Propylene Oxide	9.05E-02	3.97E-01
Pyrene	7.67E-05	2.31E-05
Toluene	4.15E-01	1.78E+00
Xylene	2.04E-01	8.76E-01
Lead compounds	2.87E-03	6.50E-04
Manganese	2.18E-03	4.94E-04
Mercury	1.49E-03	3.38E-04
Nickel	1.21E-02	2.73E-03
Selenium	1.38E-04	3.12E-05
<b>Total HAPs</b>	<b>3.60*</b>	<b>10.10</b>
<b>Max Single HAP</b>	<b>-</b>	<b>3.09</b>

\* Federal major Hazardous Air Pollutant (HAP) source thresholds are annual (tons/yr); there are no short-term total HAP thresholds established.

*Table 9. Siemens SGT6-5000F5- Potential HAP emissions*

Pollutant	Potential emissions	
	lbs/hr	TPY
1,3 Butadiene	1.46E-03	5.98E-03
2-Methylnaphthalene	1.71E-05	2.02E-05
3-Methylchloranthrene	1.28E-06	1.51E-06
7,12-Dimethylbenz(a)anthracene	1.14E-05	1.34E-05
Acenaphthene	8.01E-05	2.12E-05
Acenaphthylene	1.63E-04	4.18E-05
Acetaldehyde	1.29E-01	5.55E-01
Acrolein	2.06E-02	8.88E-02
Anthracene	2.62E-05	8.15E-06
Arsenic	1.08E-03	2.05E-04
Benz(a)anthracene	1.55E-05	5.08E-06
Benzene	5.43E-02	1.72E-01
Benzo(a)pyrene	5.46E-06	2.16E-06
Benzo(b)fluoranthene	1.94E-05	6.04E-06
Benzo(g,h,l)perylene	9.79E-06	3.24E-06
Benzo(k)fluoranthene	5.18E-06	2.49E-06
Beryllium	6.49E-05	1.23E-05
Cadmium	5.95E-03	1.13E-03
Chromium	7.57E-03	1.44E-03
Chrysene	2.68E-05	7.89E-06
Cobalt	4.54E-04	8.61E-05
Dibenzo(a,h)anthracene	7.90E-06	2.77E-06
Dichlorobenzene	8.53E-04	1.01E-03
Ethylbenzene	1.01E-01	4.43E-01
Fluoranthene	8.63E-05	2.36E-05
Fluorene	2.82E-04	7.24E-05

Formaldehyde	7.54E-01	3.11E+00
Hexane	1.28E+00	1.51E+00
Indeno(1,2,3-cd)pyrene	8.89E-06	3.41E-06
Napthalene	6.85E-03	1.91E-02
PAHs	6.96E-03	3.05E-02
Phenanathrene	7.43E-04	1.97E-04
Propylene Oxide	9.18E-02	4.02E-01
Pyrene	7.53E-05	2.21E-05
Toluene	4.19E-01	1.81E+00
Xylene	2.06E-01	8.88E-01
Lead Compounds	2.70E-03	5.13E-04
Manganese	2.05E-03	3.90E-04
Mercury	1.41E-03	2.67E-04
Nickel	1.14E-02	2.15E-03
Selenium	1.30E-04	2.46E-05
<b>Total HAPs</b>	<b>3.11*</b>	<b>9.04</b>
<b>Max Single HAP</b>	<b>-</b>	<b>3.11</b>

Based on Tables 8 and 9, the maximum total HAPs from the proposed facility would be 10.10 tons per year; the single HAP emitted at the highest rate is formaldehyde at 3.11 tons per year. Major source thresholds for HAPs are 10 tons per year for an individual HAP or 25 tons per year total HAPs. Accordingly, GEP/S is not a major source of HAP and is not subject to requirements under 40 CFR Part 63 Subpart YYYY, the Combustion Turbine Maximum Achievable Control Technology (MACT) standard.

Since the combustion turbines are not subject to the Combustion Turbine MACT, the units are exempt to the state toxics standards in 9 VAC 5-60-300 *et seq.* Please see Section IV.B for further discussion of toxics emissions from the proposed facility.

## IV. Regulatory Review and Considerations

### A. Criteria Pollutants

The proposed facility meets the definition of major source under 9 VAC 5 Chapter 80 Article 8 (Prevention of Significant Deterioration (PSD)) because it is a fossil-fuel-fired steam electric plant of more than 250 MMBtu/hr heat input capacity and has the potential to emit (PTE) more than 100 tons per year of a regulated pollutant. When a new facility is subject to PSD, any regulated pollutant for which the area is in attainment having a PTE above the significance level is also subject to PSD. Additionally, based on 9VAC5 Chapter 85, GHGs from the proposed



project are subject to regulation based on PTE. The pollutants subject to PSD for the proposed project are CO, NO<sub>x</sub>, PM, PM10, and GHG.

The proposed facility will be locating in an area classified as an ozone and PM-2.5 nonattainment area as well as being part of the Ozone Transport Region (OTR) and meets the definition of a major source under 9 VAC 5 Chapter 80 (Permits for Major Stationary Sources and Modifications – Nonattainment Areas or Ozone Transport Region). Accordingly, the proposed facility is subject to major nonattainment NSR permitting for NO<sub>x</sub> emissions from the GE 7FA.05 CT and NO<sub>x</sub> and VOC emissions from the Siemens SGT6-5000F5-CT. Although the area is also nonattainment for PM-2.5, the proposed PM-2.5 emissions do not exceed the major source threshold and therefore Article 9 is not applicable.

Table 10 below compares the maximum proposed net emissions increases from GEP/S with PSD and NAA-MNSR significant increase levels.

*Table 10. Proposed emissions levels*

Pollutant	Maximum Allowable Emissions (tpy)	PSD Significant Threshold Levels (tpy)	Subject to PSD (Article 8) or Non Attainment MNSR (Article 9) <sup>c</sup>
NO <sub>x</sub>	159 <sup>a</sup> / 164 <sup>b</sup>	40	Article 8 & Article 9
CO	207 <sup>a</sup> / 143 <sup>b</sup>	100	Article 8
VOC	38 <sup>a</sup> / 52 <sup>b</sup>	NA <sup>d</sup>	Article 9 <sup>c</sup>
PM	105 <sup>a</sup> / 106 <sup>b</sup>	25	Article 8
PM-10	105 <sup>a</sup> / 106 <sup>b</sup>	15	Article 8
PM-2.5	98 <sup>a</sup> / 99 <sup>b</sup>	NA <sup>d</sup>	Article 9
SO <sub>2</sub>	5.44 <sup>a</sup> / 5.37 <sup>b</sup>	10	No
GHG (CO <sub>2</sub> e)	2,468,228 <sup>a</sup> / 2,464,25 <sup>b</sup>	100,000	Article 8
Sulfuric acid mist (H <sub>2</sub> SO <sub>4</sub> )	2.87 <sup>a</sup> / 2.81 <sup>b</sup>	7	No
Lead (Pb) <sup>1</sup>	0.02	0.6	No

a – Based on the GE F7A.05 emissions

b – Based on the Siemens SGT6-5000F5 emissions

c – Article 9 for the Siemens CT option only

d – Although there are PSD significance levels for VOC and PM-2.5, Loudoun County is non-attainment for ozone (VOC) and PM-2.5; Non Attainment MNSR requirements apply if VOC emissions are ≥ 50 tpy and also if PM-2.5 emissions are ≥ 100 tpy.

e – All pollutants were also reviewed for permitting applicability under Article 6 (mNSR).

The pollutants subject to nonattainment NSR are NO<sub>x</sub>, VOC (if using the Siemens model), and PM-2.5 and PSD review are NO<sub>x</sub>, PM, PM-10, and

CO. PSD regulations require modeling analysis to demonstrate compliance with the NAAQS and PSD increments (NO<sub>x</sub>, PM-10, and CO). It should be noted that although there is a designated significance level for PM, and VOC, there are no modeling requirements for these pollutants. The details of the modeling analysis are provided in Attachment C.

The facility is locating in a PM-2.5 nonattainment area but does not trigger MNSR. PM-2.5 was evaluated under Chapter 80, Article 6 and BACT was applied in accordance with 9VAC5-50-260.

## **B. HAPs/Toxic Pollutants**

The electric generating units proposed by GEP/S are subject to the toxic pollutant standards in 9 VAC 5-60-300. As a result, GEP/S conducted an evaluation of toxic pollutants in comparison to the emission standards in 9 VAC 5-60-300. This evaluation included a modeling analysis for five pollutants for which uncontrolled emissions were above the exemption levels in 9 VAC 5-60-300 (acrolein, formaldehyde, cadmium, chromium, and nickel). The modeling analysis indicates that the impacts of the five pollutants are well below their applicable Significant Ambient Air Concentrations (SAACs). Attachment B includes a table showing emissions of toxic pollutants from the proposed facility compared to the exemption thresholds. Attachment C contains the modeling results.

Table 11

Pollutant	tons/year <sup>a</sup>	tons/year <sup>b</sup>
Acrolein	8.76E-01	8.88E-02
Cadmium	2.25E-02	2.25E-02
Chromium	2.86E-02	2.86E-02
Formaldehyde	3.09E+00	3.11E+00
Nickel	4.29E-02	4.29E-02

a – Based on the GE F7A.05 emissions

b – Based on the Siemens SGT6-5000F5

40 CFR 63 Subpart YYYYY, National Emissions Standards for HAPs from Stationary Combustion Turbines, was promulgated March 5, 2004 and applies to CTs located at major HAP sources. According to GEP/S's application, the HAP emissions from the proposed GEP/S facility do not exceed major source thresholds for HAPs, i.e., 10 tons per year of a single HAP or 25 tons per year of all HAPs combined. Accordingly, the proposed facility is not subject to the MACT standard. It should be noted that the MACT stipulates oxidation catalyst as one way to comply with the MACT limits (oxidation catalysts not only reduce CO and VOC

emissions, they also reduce volatile HAP emissions such as formaldehyde, toluene, acetaldehyde and benzene). GEP/S has proposed oxidation catalyst to control CO and VOC from its facility.

### **C. Modeling Results**

The United States Forest Service (USFS), the United States Fish and Wildlife Service (FWS), and the National Park Service (NPS) each stated in an e-mail dated June 20, 2012, June 20, 2012, and July 3, 2012, respectively, that an AQRV analysis was not required since the project is not expected to show any significant additional impacts to AQRVs. Therefore, only a Class I area analysis to assess compliance with the Class I PSD increments was required.

The Class I and Class II air quality modeling analyses conform to 40 CFR Part 51, Appendix W - Guideline on Air Quality Models and were performed in accordance with their respective approved modeling methodology that were included in a protocol that was submitted in advance by the proposed facility.

The air quality modeling analyses results show compliance with all applicable NAAQS and PSD increments. The DEQ's air quality modeling analyses technical review memorandum is included as Attachment C.

### **D. Control Technology Analysis**

#### **1. BACT vs. LAER**

The permitting process involves two methods of control technology review: Best Available Control Technology (BACT) and Lowest Achievable Emission Rate (LAER). In geographic locations where ambient pollutant concentrations exceed the NAAQS, permit applicants are required to meet LAER. LAER is defined as the lowest emission rate achieved in practice on a similar design. Only technical and environmental factors are considered, without regard to cost. In areas where pollutant concentrations are within the NAAQS, the applicant must apply BACT. BACT represents the most stringent emission limit that is technically, environmentally, and economically feasible. EPA policy requires that LAER is the first consideration in the BACT analysis. Only when LAER is proven to be environmentally or economically infeasible may BACT be less stringent than LAER. However, in no case may BACT result in an emission rate less stringent than required by federal regulations such as NSPS or MACT requirements. Loudoun County is considered non

attainment for ozone and PM-2.5, and is attainment for CO, NO<sub>x</sub>, SO<sub>2</sub>, and PM-10. Therefore, a LAER analysis is required for emission controls for NO<sub>x</sub> and VOC, and BACT is considered for the remaining pollutants.

## 2. LAER Requirements

The proposed facility will be located in an ozone nonattainment area which is also part of the OTR. It will be major for NO<sub>x</sub> and VOC emissions for the Siemens CTGs configuration (and major for NO<sub>x</sub> for the GE CTGs configuration). Therefore, in accordance with 9VAC5-50-270, LAER must be applied for those pollutants. The NO<sub>x</sub> emissions are also subject to BACT as the region is attainment/unclassified for the NO<sub>x</sub> NAAQS. However because the region is nonattainment for the 1997 and 2008 8-hour ozone standards, LAER will be applied to the proposed CTGs for NO<sub>x</sub>. By applying LAER, which is the top level of control, the BACT requirement is presumed to be met.

Emission units addressed in the LAER determination submitted by GEP/S include the combined-cycle units, the auxiliary boiler, the fuel gas heater, the emergency generator, and the emergency firewater pump.

### **Combined-Cycle Combustion Turbine (CT)**

#### **NO<sub>x</sub> Control**

The combustion turbines and the HRSG duct burners are responsible for most of the emissions from the facility. The following control technologies were identified by GEP/S as applicable to NO<sub>x</sub> treatment for combined-cycle combustion turbines:

- Selective Catalytic Reduction (SCR)
- SCONOX™
- Selective Non-Catalytic Reduction (SNCR) and Non-Selective Catalytic Reduction (NSCR)
- Dry Low-NO<sub>x</sub> (DLN) Combustors
- Water or Steam Injection
- XONON™, LoTO<sub>x</sub>™, THERMALLONO<sub>x</sub>™, and Pahlmann™

Of the NO<sub>x</sub> control technologies that were reviewed for the GEP/S facility, SCR and Dry Low-NO<sub>x</sub> (DLN) combustors were the two most stringent techniques that have been applied to a combined

cycle turbine facility. A discussion of the control technologies is presented below.

**SCR** is a process that involves post combustion removal of  $\text{NO}_x$  from the flue gas with a catalytic reactor. In the SCR process, ammonia injected into the turbine exhaust gas reacts with nitrogen oxides and oxygen to form nitrogen and water. SCR converts nitrogen oxides to nitrogen and water through several possible reactions that take place on the surface of a catalyst. The function of the catalyst is to effectively lower the activation energy of the  $\text{NO}_x$  decomposition reaction. Technical factors related to this technology include increased turbine backpressure, exhaust temperature materials limitations, thermal shock/stress during rapid starts, catalyst masking/blinding, reported catalyst failure due to “crumbling”, design of the  $\text{NH}_3$  injection system, and high  $\text{NH}_3$  slip. SCR using ammonia as a reagent represents the state-of-the-art for back end gas turbine  $\text{NO}_x$  removal from base load, combined-cycle turbines.

**SCONOX™** is an emerging post-combustion technology that removes  $\text{NO}_x$  from the exhaust gas stream after formation in the combustion turbine. SCONOX™ employs a potassium carbonate bed that adsorbs  $\text{NO}_x$  where it reacts to form potassium nitrates. Periodically, a hydrogen gas stream is passed over the bed, resulting in the reaction of the potassium nitrates to re-form the potassium carbonate and the ejection of nitrogen gas and water.

SCONOX™ is reportedly capable of achieving  $\text{NO}_x$  emission reductions of 90% or more for combustion turbine application, and it is currently operating on several small natural gas-fired turbines. The most notable advantage of SCONOX™ over SCR is that it reduces  $\text{NO}_x$  without the use of ammonia. SCONOX™ thereby eliminates the possibility of “ammonia slip”, or emissions of excess (unreacted) ammonia, that is present with use of SCR for  $\text{NO}_x$  control. Similar to SCR, SCONOX™ only operates within a specific temperature range.

GEP/S’s application eliminated SCONOX™ as not technically feasible for application to this project since it is no longer being offered for large combustion turbines. SCONOX™ is considerably more complex than SCR, would consume significantly more water, and would require more frequent cleaning and other maintenance.

DEQ concurs with GEP/S's conclusion that at the present time, SCONOX™ cannot be considered a feasible control option for the proposed project.

**SNCR and NSCR** - Two other back-end catalytic reduction technologies, SNCR and NSCR, have been used to control emissions from certain other combustion process applications. However, both of these technologies have limitations that make them inappropriate for application to combustion turbines. SNCR requires a flue gas exit temperature in the range of 1,300 to 2,100 °F, with an optimum operating temperature zone between 1,600 and 1,900 °F. Simple-cycle combustion turbines have exhaust temperatures of approximately 1,100 °F, and combined-cycle turbines have exhaust temperatures much lower than simple-cycle turbines. Therefore, additional fuel combustion or a similar energy supply would be needed to create exhaust temperatures compatible with SNCR operation. This temperature restriction and related economic considerations make SNCR infeasible and inappropriate for the proposed combustion turbines. NSCR is only effective in controlling fuel-rich reciprocating engine emissions and requires the combustion gas to be nearly depleted of oxygen (<4% by volume) to operate properly. Since combustion turbines operate with high levels of excess oxygen (typically 14 to 16% O<sub>2</sub> in the exhaust), NSCR is infeasible and inappropriate for the proposed combustion turbines.

**Dry Low NO<sub>x</sub> Combustors** - Dry Low NO<sub>x</sub> (DLN) combustion control techniques reduce NO<sub>x</sub> emissions without injecting water or steam (hence "dry"). DLN combustors are designed to control peak combustion temperature, combustion zone residence time, and combustion zone free oxygen, thereby minimizing thermal NO<sub>x</sub> formation. This is accomplished by producing a lean, pre-mixed flame that burns at a lower flame temperature and excess oxygen levels than conventional combustors.

DLN combustors have been employed successfully for natural gas-fired combustion turbines for more than fifteen years.

**XONON™, LoTO<sub>x</sub>™, THERMALLONO<sub>x</sub>™, and Pahlmann™** A number of other combustion turbine NO<sub>x</sub> emissions control technologies for combustion turbines are being marketed including XONON™, LoTO<sub>x</sub>™, THERMALLONO<sub>x</sub>™, and Pahlmann™. None of these technologies has reached the commercial development stage for large combustion turbines that will be fired with natural gas, and thus none are considered to be technically

feasible for application to this project. DEQ concurs that these technologies are not yet commercially available technology suitable for controlling CTs of the size proposed at the GEP/S site.

LAER Determination:

GEP/S has proposed a combination of the remaining identified control options dry low-NO<sub>x</sub> combustors and selective catalytic reduction (SCR) as LAER. The proposed GE 7FA and Siemens SGT6-5000F5 model turbines use a two-stage premixed combustion design resulting in uncontrolled NO<sub>x</sub> emissions of 15 ppmvd at 15% O<sub>2</sub> when firing natural gas, the fuel proposed for use by GEP/S. The draft permit proposes use of dry low-NO<sub>x</sub> combustors and SCR to control NO<sub>x</sub> emissions from the CTs to the following level (at 15% O<sub>2</sub>):

2.0 ppmvd with and without the duct burner firing (16.0 lbs/hr for the GE 7FA and 17.1 lbs/hr for the Siemens SGT6-5000F5).

Compliance with the limits is to be based on a one-hour block average.

From 2007 to 20011, approximately fifteen projects were permitted at 2.0 ppmvd at 15% O<sub>2</sub> including two LAER determinations. Recent PSD permits at 2 ppmvd at 15% O<sub>2</sub> include a September 1, 2011 issued permit for the Thomas C Ferguson Power Plant in Texas and a December 17, 2010 issued permit for the Warren County Power Station in Virginia. There is one project that was permitted at a NO<sub>x</sub> emission rate of 1.5 ppmvd at 15% O<sub>2</sub> in the year 2000. However, this project has not been built and therefore, 1.5 ppmvd at 15% O<sub>2</sub> has not been demonstrated as achievable in practice. With that one exception, the proposed limits are as stringent as any listed in EPA's RACT/BACT/LAER Clearinghouse (RBLC) for electric generating facilities.

GEP/S's facility is expected to operate as a baseload plant, i.e., at close to 100% loading during most times. However, the proposed turbine units will serve the PJM electric grid capable of covering large swings in electric demand in short periods of time. As part of this process, the PJM system operator will take control of the units in order to meet the continuously changing demand. These load changes will necessitate ramping operation of the combustion turbines and, if necessary, the duct burners up and down to follow load demand. The permit does not restrict the facility from

operating at lower loads and the 2.0 ppmvd limit applies to the operation of the turbines at all load levels except during periods of startup and shutdown.

### **Auxiliary Boiler and Fuel Gas Heater**

GEP/S plans to install an auxiliary boiler and a fuel gas heater. Both units burn only pipeline quality natural gas and are relatively small emission sources when compared to the CTs.

#### **NO<sub>x</sub> control**

NO<sub>x</sub> emissions from the auxiliary boiler and fuel gas heater originate primarily as thermal NO<sub>x</sub>. The primary front-end combustion controls for boilers and heaters are low excess air, low-NO<sub>x</sub> burners, and ultra low-NO<sub>x</sub> burners. SCR can be used to remove NO<sub>x</sub> from the exhaust gas stream once NO<sub>x</sub> has been formed.

Both ultra low-NO<sub>x</sub> burners and SCR are capable of limiting NO<sub>x</sub> emissions to approximately 0.011 lb/MMBtu or 9 ppmvd at 3% O<sub>2</sub>. Data from EPA's RBLC show that recently permitted emission rates for natural gas-fired boilers and fuel gas heaters less than 250 MMBtu/hr are in the 0.035 lb/MMBtu to 0.060 lb/MMBtu range. However, several projects have been permitted in the 0.010 lb/MMBtu to 0.012 lb/MMBtu range including one boiler permitted at 0.012 lb/MMBtu as LAER and one fuel gas heater permitted at 0.021 lb/MMBtu as LAER.

The applicant proposes to burn only pipeline quality natural gas in the auxiliary boiler and fuel gas heater and to use ultra low-NO<sub>x</sub> burners to limit NO<sub>x</sub> emissions to 0.83 lb/hr, 0.011 lb/MMBtu (approximately 9 ppmvd at 3% O<sub>2</sub>). DEQ agrees that burning natural gas and using ultra low-NO<sub>x</sub> burners is LAER for NO<sub>x</sub> emissions from the auxiliary boiler and the fuel gas heater.

### **Emergency Generator and Fire Water Pump**

The facility will have a 1.5 MW emergency generator and a 330 bhp emergency firewater pump. Compliance with the New Source Performance Standard (40 CFR Part 60 Subpart IIII) is proposed as LAER for NO<sub>x</sub> and VOC.

#### **NO<sub>x</sub> control**



Because emergency engines must start quickly and change output rapidly to match fluctuating load demands, emergency units produce variations in exhaust temperature and flow rate as well as NO<sub>x</sub> concentration and are therefore not well-suited for a selective non-catalytic reduction (SNCR) or an SCR system. Additionally, because of the limited operating hours (a maximum of 500 per year as limited by the permit), control by SCR or SNCR would not be cost effective.

At 500 hours of operation, the maximum annual NO<sub>x</sub> emissions for the emergency generator would be 5.8 tons per year and for the fire water pump would be about 0.5 tons per year. The emission factors for NO<sub>x</sub> used as the basis for the emergency generator and fire water pump emissions limits are based on the NSPS Subpart III limits for Stationary Compression Ignition Internal Combustion Engines (40 CFR 60 Subpart III), the current federal standard for stationary engines.

Because of the low maximum emissions level at the limited allowed operating hours and the fact that the engines are required to meet the federal standards outlined in the NSPS, Subpart III, DEQ concurs that add-on control would not be appropriate for the emergency units and that the proposed emission levels meet LAER.

As also required by the NSPS, Subpart III, the permit requires GEP/S to use ultra-low sulfur fuel oil in its emergency units.

#### LAER VOC Control

The proposed facility will be located in an area designated nonattainment for the 1997 and 2008 8-hour ozone NAAQS and OTR and will be required to apply LAER for Volatile Organic Compounds (VOC) if the facility-wide VOC emissions are 50 tpy or more. The Siemens SGT6-5000F5 option results in VOC emissions greater than 50 tons per year resulting in a need to apply LAER, whereas the GE CTG option results in VOC emissions less than 50 tpy resulting in a need to apply BACT (under Article 6).

For VOCs, the Stonewall GE emissions are higher than Warren County. Although, there is no BACT or LAER requirement for VOC emissions for the Stonewall GE option, the VOC emission rates are consistent with LAER for the GE 7 FA combustion turbine. The available combustion turbine emission guarantees from GE are 1.4 ppmvd at 15% O<sub>2</sub> which is higher than the 1.0

ppmvd at 15% O<sub>2</sub> guarantees for the Siemens and MHI combustion turbines. For Stonewall, the 2.4 ppmvd emission rate with duct burning is attributable to the duct burner operating at a reduced load of 10%. With the duct burner at full load, the emissions will be 2.0 ppmvd at 15% O<sub>2</sub> which is higher than the Siemens or Warren County emissions due the higher GE combustion turbine emissions.

For VOCs, the Stonewall Siemens emissions are higher than Warren County without duct burning and slightly lower with duct burning. The available combustion turbine emission guarantees from Siemens and MHI are 1.0 ppmvd at 15% O<sub>2</sub>. The combustion turbine vendors indicate that they will not offer guarantees below 1.0 ppmvd at 15% O<sub>2</sub>. For the Warren County project, Dominion may have chosen to go beyond 1.0 ppmvd at 15% O<sub>2</sub> due the precedent set by older Warren County permits. Research into the Warren County project indicates that the original project was permitted and not built. The developer was expecting the oxidation catalysis to control excessive emissions; however, the project was never built to demonstrate the developer's claim. With duct burning the Stonewall Siemens emissions are slightly lower than the Warren County emissions.

The use of good combustion control and an oxidation catalyst represent LAER for VOC control for the proposed CTGs. Emissions depend upon the performance of each CTG, the use of duct burning, and the performance of the oxidation catalyst. Available performance guarantees are limited by the low VOC concentrations before control and uncertainties regarding the compounds that are actually emitted. The following VOC emission rates, based on VOC control by an oxidation catalyst, are proposed as LAER for the SGT6-5000F CTGs:

- 1.0 ppmvd @ 15% O<sub>2</sub> (DBs Off); and
- 1.5 ppmvd @ 15% O<sub>2</sub> (DBs On).

### 3. BACT Requirements

The EPA guidance document New Source Review Workshop Manual: Prevention of Significant Deterioration and Nonattainment Area Permitting prescribes that for PSD permitting, the most stringent BACT review, otherwise known as "top-down" review, be conducted. The "top-down" method provides that all available control technologies be ranked in descending order of control effectiveness. The applicant first examines the most stringent or "top" alternative. The top alternative is established as BACT unless the applicant

demonstrates that technical considerations or energy, environmental, or economic impacts justify that the most stringent technology is not feasible. If the most stringent is eliminated, the next most stringent is considered until BACT is established.

All pollutants subject to PSD review are subject to a “top-down” BACT analysis, as BACT is established on a pollutant basis. For the proposed GEP/S facility, the pollutants include NO<sub>x</sub>, CO, PM, PM<sub>10</sub>, and greenhouse gases. Emission units addressed in the BACT determination submitted by GEP/S include the combined-cycle units, the auxiliary boiler, the fuel gas heater, the emergency generator, and the emergency firewater pump.

A listing of BACT determinations included in the RACT/BACT/LAER Clearinghouse for similar facilities is included as Appendix C in GEP/S’s application.

### **Combined-Cycle Combustion Turbine (CT)**

#### *CO BACT*

Carbon monoxide emissions are formed in the exhaust of a combustion turbine as a result of incomplete combustion of the fuel. Similar to the generation of NO<sub>x</sub> emissions, the primary factors influencing the generation of CO emissions are temperature and residence time within the combustion zone. Variations in fuel carbon content have relatively little effect on overall CO emissions. Generally the effect of the combustion zone temperature and residence time on CO emissions generation is the exact opposite of their effect on NO<sub>x</sub> emissions generation. Higher combustion zone temperatures and residence times lead to more complete combustion and lower CO emissions, but higher NO<sub>x</sub> emissions. The applicant proposed good combustion control and an oxidation catalyst to control CO emissions (based on 85% CO control) to the following levels, all corresponding to 15% O<sub>2</sub> as a 1-hour rolling average:

- 2.0 ppmvd with and without duct burner firing

An oxidation catalyst is a post-combustion technology that removes CO from the exhaust gas stream after formation in the combustion turbine. In the presence of a catalyst, CO will react with oxygen present in the exhaust stream, converting it to carbon dioxide. No supplementary reactant is used in conjunction with an oxidation catalyst. The oxidation of CO to CO<sub>2</sub> utilizes the excess air present in the turbine exhaust; and the activation energy

required for the reaction to proceed is lowered in the presence of the catalyst. Technical factors relating to this technology include the catalyst reactor design, optimum operating temperature, back pressure loss to the system, catalyst life, and potential collateral increases in emissions of PM-10 and sulfuric acid mist emissions.

CO catalytic oxidation reactors operate in a relatively narrow temperature range. Optimum operating temperatures for these systems generally fall into the range of 700 °F to 1,100 °F. At lower temperatures, CO conversion efficiency falls off rapidly. Above 1,200 °F, catalyst sintering may occur, thus causing permanent damage to the catalyst. For this reason, the CO catalyst is strategically placed within the proper turbine exhaust lateral distribution (it is important to evenly distribute gas flow across the catalyst) and proper operating temperature at base load design conditions. Operation at part load, or during startup/shutdown will result in less than optimum temperatures and reduced control efficiency.

Typical pressure losses across an oxidation catalyst reactor (including pressure loss due to ammonium salt formation) are in the range of 0.7 to 1.0 inches of water. Pressure drops in this range correspond roughly to a 0.15 percent loss in power output and fuel efficiency or approximately 0.1 percent loss in power output for each 1.0 inch of water pressure loss.

Catalyst systems are subject to loss of activity over time. Since the catalyst itself is the most costly part of the installation, the cost of catalyst replacement should be considered on an annualized basis. Catalyst life may vary from the manufacturer's typical 3-year guarantee to a 5- to 6-year predicted life. Periodic testing of catalyst material is necessary to predict annual catalyst life for a given installation.

Oxidation catalysts have been employed successfully for two decades on natural gas combustion turbines. An oxidation catalyst is considered to be technically feasible for application to this project.

Good combustion practices consisting primarily of controlled fuel/air mixing and adequate temperature and gas residence time are used to minimize the formation of CO.

As shown in EPA's RBLC, only three projects have been permitted at CO emission rates below 2 ppmvd at 15% O<sub>2</sub>. For

CO, the Stonewall emissions are higher than Warren County without duct burning but lower with duct burning. Most of the projects in EPA's RACT/BACT/LAER Clearinghouse that are below 2 ppmvd @15% O<sub>2</sub> are various entries for Warren County from 2004 through 2010. Research into the Warren County project indicates that the original project was permitted and not built. The developer was expecting the oxidation catalysis to control excessive amounts of CO; however, the project was never built to demonstrate the developer's claim. The project was purchased by Dominion Energy and could not be built with the specified turbines listed in the permit because the turbines were no longer available. The current model offered had different emission characteristics, causing Dominion Energy to file for a new permit with a revised project configuration of 3 MHI turbines matched with one large steam turbine generator.

The Stonewall emissions are based on emission data provided by GE and Siemens and are consistent with similar projects listed in EPA's RACT/BACT/LAER Clearinghouse. This is further demonstrated by the most recent entries into the RACT/BACT/LAER Clearinghouse that followed the Warren County permitting.

The last 4 projects permitted in 2012 in Texas and Wyoming were permitted a 4.0 ppmvd CO at 15% O<sub>2</sub>. Two projects (Palmdale Hybrid Power Project (10/18/2011) and Avenal Energy Project (6/21/2011)), are listed at 1.5 ppmvd CO at 15% O<sub>2</sub>. The 1.5 ppmvd emission rate is a conditional rate that must be achieved during a demonstration period after the first 3 years of operation or a special condition will allow the permit limit to be adjusted up to 2.0 ppmvd CO at 15% O<sub>2</sub> for compliance.

Typically, CO emission rates of 2 ppmvd at 15% O<sub>2</sub> to 3.5 ppmvd at 15% O<sub>2</sub> are determined to be BACT and LAER. The higher CO emission rates generally account for the higher emissions associated with duct burning.

It should be noted that the lean pre-mix dry low-NO<sub>x</sub> combustion employed on the CTs also works to reduce CO emissions. DEQ concurs that the proposed oxidation catalyst control and good combustion practices constitute BACT for CO from the CTs.

#### VOC BACT

Only the GE CTG option is subject to PSD BACT for VOC emissions. Formation of VOC emissions in combustion turbines is attributable to the same factors as described for CO emissions above. VOC emissions are a result of incomplete combustion of carbonaceous fuels, and this is influenced primarily by the temperature and residence time within the combustion zone.

An oxidation catalyst is a post-combustion technology that removes VOC from the exhaust gas stream after formation in the combustion turbine. In the presence of a catalyst, VOC will react with oxygen present in the exhaust stream, converting it to carbon dioxide and water vapor. The performance of an oxidation catalyst is affected by the VOCs that are actually emitted. No supplementary reactant is used in conjunction with an oxidation catalyst. An oxidation catalyst is considered to be technically feasible for application to this project.

Good combustion practices consisting primarily of controlled fuel/air mixing and adequate temperature and gas residence time are used to minimize the formation of VOCs.

The two most recent BACT decisions are the Warren County, Virginia project with BACT emissions limits set for MHI 501 GAC CTGs at 1.5 ppmvd at 15% O<sub>2</sub> without duct burning and 2.4 ppmvd at 15% O<sub>2</sub> with duct burning, and the Kleen Energy project at 4.0 ppmvd at 15% O<sub>2</sub>. The available combustion turbine emission guarantees from GE are 1.4 ppmvd at 15% O<sub>2</sub> which is higher than the 1.0 ppmvd at 15% O<sub>2</sub> guarantees for the Siemens and Warren County's MHI combustion turbines. For Stonewall, the 2.4 ppmvd emission rate with duct burning is attributable to the duct burner operating at a reduced load of 10%. With the duct burner at full load, the emissions will be 2.0 ppmvd at 15% O<sub>2</sub> which is higher than the Siemens or Warren County emissions due the higher GE combustion turbine emissions.

The applicant has proposed to control VOC using good combustion practices in the CT and an oxidation catalyst. The oxidation catalyst is proposed for the dual purpose of controlling CO emissions and VOC emissions. The applicant proposed VOC limits, based on 30% control by an oxidation catalyst, as follows, all at 15% O<sub>2</sub> and as CH<sub>4</sub> (calculated as a three-hour average):

- 1.0 ppmvd without duct burner firing
- 1.5 ppmvd with duct burner firing

The VOC emissions are subject to the design of the turbine manufacturers who are balancing emissions while trying to achieve higher efficiency. The turbines will react differently, producing different emissions at different load conditions and with or without duct burner operations. Discussions with manufacturers indicate that 1.0 ppmvd at 15% O<sub>2</sub> for VOC is where they will guarantee their F class machines for this project. The Stonewall project has taken into account the various operating conditions that this project will face and has determined the BACT limit based on the worst case to set the not to exceed BACT limit for this project.

The use of good combustion control and an oxidation catalyst represent BACT for VOC control for the proposed combustion turbines.

#### PM/PM-10/PM-2.5 BACT

Particulate matter emissions from combustion turbines are a combination of filterable (front-half) and condensable (back-half) particulate. Filterable particulate matter is formed from impurities contained in the fuels and from incomplete combustion. Condensable particulate emissions, which contribute to PM-10 and PM-2.5, are attributable primarily to the formation of sulfates and possibly organic compounds.

The most stringent particulate control method demonstrated for gas turbines is the use of low ash and low sulfur fuel. No add-on control technologies are listed in EPA's RBLC. Proper combustion control and the firing of fuels with negligible or zero ash content and a low sulfur content for the combustion turbines is the only control method listed. Add-on controls, such as electrostatic precipitators (ESPs) or baghouses, have never been applied to commercial gas turbines. The use of ESPs and baghouses are considered technically infeasible, and do not represent an available control technology. The maximum PM-10 concentrations, including condensable PM-10, from combined cycle combustion units are approximately 0.002 gr/dscf which is lower than 0.01 gr/dscf, which is a typical baghouse performance specification.

Proper combustion control and the firing of fuels with negligible or zero ash content and a low sulfur content for the combustion turbines is considered to be technically feasible for application to this project.

The applicant proposed the use of good combustion practices and pipeline quality natural gas as BACT for PM, PM-10, and PM-2.5 control for the proposed combined-cycle turbines. The following PM/PM-10/PM-2.5 emission rates were proposed as BACT for the GE 7FA.05 and the Siemens SGT6-5000F5 combustion turbines in GEP/S's application:

GE 7FA.05

- 9.6 lb/hr without duct burner firing
- 16.2 lb/hr with duct burner firing
- $3.34 \times 10^{-3}$  lb/MMBtu at full load

Siemens SGT6-5000F5

- 10.1 lb/hr without duct burner firing
- 14.5 lb/hr with duct burner firing
- $3.74 \times 10^{-3}$  lb/MMBtu at full load

Unlike NO<sub>x</sub>, CO, or VOC, there are no demonstrated add-on technologies or design changes that are used for control of particulate matter. The specific combustion turbine models that GEP/S is considering for this project are more advanced than each manufacturer's comparable models currently in operation. The combustion turbine uses less fuel per kilowatt of power generated. The gain in generation efficiency allows the project to use comparatively less fuel to produce more power. While total fuel use will increase proportionately to the increased output capability of the new machines, the decrease in heat rate means that the gain in electric generation is a greater benefit. Fuel use is related to particulate matter generation because more fuel mass will equal more particulate mass out; however, use of the more efficient turbines will generate particulates at a lower rate (on an electrical output basis) than combustion turbines permitted ten years ago in California and other states. Combustion turbines (GE and Siemens turbine model versions) in California have been permitted at very low emission limits.

According to EPA's RBLC during the time period from 2005-2009, the PM emission limits on a lb/MMBtu basis for combined-cycle power plants ranged from 0.0055 to 0.0210 lb/MMBtu. Therefore, on a lb/MMBtu basis, the proposed CTs are comparable to those at other combined-cycle power plants. DEQ agrees that these emission rates along with limiting the fuel fired in the CTs to pipeline-quality natural gas having a maximum sulfur content of 0.0003 percent by weight (i.e., 0.1 grain or less of total sulfur per



100 standard cubic feet) and good combustion practices meets BACT for particulate matter emissions.

#### SO<sub>2</sub> and Sulfuric acid mist control

SO<sub>2</sub> and sulfuric acid mist emissions are not subject to PSD BACT or minor NSR review. Emissions of SO<sub>2</sub> from combustion turbines are a result of oxidation of fuel sulfur. Sulfuric acid mist emissions (SO<sub>3</sub>/H<sub>2</sub>SO<sub>4</sub>) result from oxidation of fuel sulfur as well as oxidation of SO<sub>2</sub> by the duct burners and catalysts used for NO<sub>x</sub>, CO, and VOC control.

The only technically feasible method for SO<sub>2</sub> and sulfuric acid mist emission control is the use of low sulfur fuels. The use of flue gas desulfurization is not technically feasible because the SO<sub>2</sub> emissions from the proposed combustion turbines are two orders of magnitude lower than emission rates achievable using flue gas desulfurization.

GEP/S proposed the following SO<sub>2</sub> and sulfuric acid mist emission rates based on a natural gas heating value of 1,020 Btu/scf for the GE 7FA.05 model and the Siemens CTG6-5000F5 model combustion turbines:

#### SO<sub>2</sub>

- 0.00026 lb/MMBtu with and without the duct burners firing

#### Sulfuric Acid Mist

- 0.00014 lb/MMBtu with and without the duct burner firing

The amount of SO<sub>2</sub> and sulfuric acid mist formation is directly proportional to the amount of sulfur present in the fuel. The applicant proposes to use only natural gas in the CTs to control SO<sub>2</sub> and sulfuric acid mist emissions.

#### Ammonia (NH<sub>3</sub>) control

Since ammonia is not a regulated pollutant, it is not subject to PSD or minor NSR BACT. However, as a precursor to PM-2.5, it can affect visibility. Ammonia emissions from combined-cycle gas turbine plants using SCR can be in the 5 to 10 ppmvd at 15% O<sub>2</sub> range. GEP/S proposed that ammonia emissions would be limited to 5 ppmvd at 15% O<sub>2</sub>.

CT & HRSG DB Greenhouse Gas (GHG) BACT

As fossil fuel-fired combustion sources, the combustion turbine and HRSG duct burners will emit three greenhouse gases: methane, nitrous oxide and carbon dioxide. Methane is emitted from combustion devices as a result of incomplete combustion. Methane emissions can be reduced by operating the combustion turbine generators at higher flame temperatures, increased residence time and higher excess oxygen; however this has the effect of increasing emissions of NO<sub>x</sub>. Nitrous oxide will be emitted in trace quantities from the combustion turbine generators as a result of partial oxidation of nitrogen from the excess oxygen used in combustion. Methane and nitrous oxide account for only 0.1% of all greenhouse gas emissions, with the remaining 99.9% of emissions being CO<sub>2</sub>. Carbon dioxide is a product of combustion of any carbon-containing fuel.

GHG emission controls that are currently available or under development are: 1) carbon capture and sequestration (CCS), 2) use of low carbon fuels, and 3) energy efficiency. The CTs will be fired on natural gas, which is considered a low carbon fuel as compared to coal.

Separating carbon dioxide from the gas streams of the combustion turbines was presented as challenging due to the dilute concentrations (3 to 4 volume percent for gas fired turbines), trace impurities in the flue gas can degrade sorbents and reduce the effectiveness of certain CO<sub>2</sub> capture processes. In addition to low concentration and impurities, compressing the captured CO<sub>2</sub> to pipeline pressure represented a large power consumption on the facility.

The facility would also be required to store the captured CO<sub>2</sub> in a geologic formation, and transport the CO<sub>2</sub> from the generation point to the storage location. The potential formations that could be used for storage are located in southwest Virginia.

The US DOE has estimated that CCS applied to a natural gas combined cycle power plant would more than double the total plant cost and increase the cost of electricity by 45%. The net result would be cost effectiveness in excess of \$100/ton of CO<sub>2</sub> controlled. In addition, CCS would consume 20% of the power plant energy output.

Based on the information presented, the DEQ agrees that carbon capture and sequestration is infeasible for this project.

On a ton/MMBtu basis, GHG from coal combustion are substantially higher than natural gas, as shown in the table below which lists several common fuels and their associated CO<sub>2</sub> emission factors. The use of low carbon fuels is technically feasible, and the proposed project will burn natural gas.

**Table 12 CO<sub>2</sub> Emission Factors**

<b>Fuel</b>	<b>kg CO<sub>2</sub>/MMBtu</b>
Bituminous Coal	93.40
Distillate Fuel Oil No. 2	73.96
Residual Fuel Oil	75.10
Coke	102.04
Wood & Wood Residuals	93.80
Natural Gas	53.02

Emission factors are from 40 CFR 98, Table C-1

Energy Efficiency: Since BACT is based on an emission limitation which reflects the maximum degree of reduction for a particular pollutant, then the best means of comparison is of emission limits rather than percent control efficiency. Since energy efficiency plays a role in emissions, one must compare efficiency limits based on output (Btu/kWh or lb/kWh) rather than mass limits based on heat input (lb/MMBtu). This is because, as a unit gets older and less efficient, it may still meet a lb/MMBtu limit while, at the same time, using more fuel to achieve its heat input need, therefore increasing emissions.

Stonewall is proposing to verify performance initially within 180 days of startup and once every Title V permit term (≈5 years) based on American Society of Mechanical Engineers Performance Test Code on Overall Plant Performance, ASME PTC 46-1996 or other method approved by DEQ. In order to establish a permit limit for these performance tests it is necessary to include margins to account for long term equipment performance.

To determine the heat rate limit for the permit, the following compliance margins were added to the base heat rate of 6,550 Btu (HHV)/gross kWh without duct burning and 6,940 MMBtu/hr with duct burning;

1. A 3.4% performance margin reflecting the efficiency losses due to permanent and recoverable combustion turbine degradation.
2. A 1.2% degradation margin reflecting operational variation and auxiliary power degradation. The operational variation assumes differences in operating techniques including but not limited to CT operation, degradation in catalyst life, HRSG tube leaks, excessive wear on equipment and design issues causing temporary derates, etc. Auxiliary power degradation includes efficiency losses over time of auxiliary (balance of plant) equipment including but not limited to pumps, motors, fans, etc.
3. A 7.1% degradation margin reflecting the efficiency losses over time of the steam turbine system including but not limited to CT gas performance (i.e., less mass flow), the HRSG, the cooling tower, etc.

Based on the above margins, Stonewall is proposing a 7,340 Btu (HHV)/gross kWh heat rate limit at full load, without duct burning, and a 7,780 Btu (HHV)/gross kWh heat rate limit at full load, with duct burning, adjusted to ISO conditions, which will be demonstrated once per Title V permit term.

Stonewall proposes to continuously monitor CO<sub>2</sub> emissions using 40 CFR Part 75 procedures. Emissions of CH<sub>4</sub> and N<sub>2</sub>O as CO<sub>2</sub>e will be based on emission factors and global warming potentials in EPA's Mandatory GHG reporting rule and fuel use. The resulting emission rate is 118.28 lb CO<sub>2</sub>e/MMBtu. The proposed GHG emission limit will be a lbCO<sub>2</sub>e/gross MWh limit as a 12-month rolling average, and will include startups, shutdowns, and low load operations. In order to establish a permit limit for continuous performance a 3% operational margin was added to the heat rate margins cited above. This operational margin accounts for dispatch variability and start-up and shut-down events. During startup and shutdown events, the combustion turbine power production efficiency is low and the steam turbine is not in operation until late in the event resulting in a much higher heat rate. The proposed annual average GHG emission rate is 903 lb CO<sub>2</sub>e/MWh (118.28 lb/MMBtu x 6,612 Btu/kWh x 1.034 x 1.012 x 1.071 x 1.03 x 1,000 kWh/MW x 1 MMBtu/1,000,000 Btu).

Only a handful of combined cycle combustion turbines have been permitted for GHG so a quick comparison can be made in the table below.

**Table 13 Comparison of GHG BACT Determinations**

Facility	Type	GHG BACT Limits	Basis
Green Energy Partners / Stonewall, Leesburg, VA	750 MW NGCC	7,340 Btu (HHV)/gross kWh w/o DB 7,780 Btu (HHV)/gross kWh w/ DB 903 lb/MWh Gross	Thermal Efficiency
Dominion VA – Brunswick, VA	1400 MW NGCC	7500 Btu/kWh (net HHV) and 920 lb/MWh	Thermal Efficiency
Cricket Valley Energy Ctr, NY	1000 MW NGCC	7605 Btu/kWh (net HHV) and 950 lb/MWh	Thermal Efficiency
Hess Newark Energy Center, NJ	655 MW NGCC	7522 Btu/kWh (net HHV) w/o DB and 887 lb/MWh (gross)	Thermal Efficiency
CPV Valley Energy, NY	630 MW NGCC	7605 Btu/kWh (LHV) w/o DB and 950 lb/MWh	Thermal Efficiency
PacifiCorp Lake Side, UT	629 MW NGCC	6918 Btu/kW (HHV) and 950 lb/MWh	Thermal Efficiency
Russell City Energy Ctr, CA	600 MW NGCC	7730 Btu/lWh and 242 tons/hr	Thermal Efficiency
LCRA Furguson Replacement, TX	590 MW NGCC	7720 Btu/kWh (net HHV) and 918 lb/MWh	Thermal Efficiency
Sevier Power Company, UT	580 MW NGCC	7515 Btu/kWh and 1,958,558 tons/yr	Thermal Efficiency
Palmdale Hybrid Power, CA	570 MW NGCC and 50 MW solar collectors	7319 Btu/kWh and 774 lb/MWh (source wide)	Thermal Efficiency
Pioneer Valley Energy, MA	431 MW CC (oil backup)	6840 Btu/kW and 895 lb/MWh	Thermal Efficiency
Deer Park (Calpine) Energy Ctr., TX	180 MW NGCC	7730 Btu/kWh (net) and 920 lb/MWh	Thermal Efficiency
Channel Energy Center, TX	180 MW NGCC	7730 Btu/kWh (net) and 920 lb/MWh	Thermal Efficiency
Kalama Energy Center, WA	346 MW NGCC (peaker)	858 lb/MWh	Thermal Efficiency

As can be seen in the table above, this project is similar in size and output to most of the other recently permitted or proposed NGCC projects. Keeping in mind that the thermal efficiency increases with larger turbines, and the net heat rate (Btu/kW) decreases, the BACT level proposed for the 750 MW Green Energy Partners / Stonewall Plant and the other permitted or proposed 180-1400 MW plants are comparable. When comparing a heat rate limit, it is important to know whether it is based on a HHV or LHV and whether it is for a gross power output or net power output, and duct fired or not duct fired operation. This is not always evident when researching other facilities. Also, some GHG BACT proposals include a “degradation factor” which takes into consideration the heat rate of a unit as it gets older and less efficient. More recently permitted plants have considered degradation, while earlier permitted plants may not have.

No information could be found on GHG BACT limits for a natural gas combined cycle power plant using CCS for comparison with a thermal efficiency approach, but estimates have shown it to be about 90 % effective in reducing GHG emissions.

Of the technologies discussed above, carbon capture and sequestration, use of low carbon fuels, and energy efficiency, CCS would be cost prohibitive. The remaining technologies, namely efficient power generation and the use of low carbon fuels are proposed for this facility and are accepted as BACT. Due to some

variability in size, manufacturer, configuration, cooling practice, elevation and the method used to determine the heat rate among the permitted plants, some variation in BACT determinations is expected, however, DEQ determined that the proposed emission level of CO<sub>2</sub>e and efficiency level are BACT for this facility. The plant will be required to operate at a higher heating value heat rate of no more than 7,780 Btu/kWh (gross) with duct burners on, and emit CO<sub>2</sub>e at an average annual rate not to exceed 903 lb CO<sub>2</sub>e/MWh (gross) (which reflects a 118.28 lb CO<sub>2</sub>e/MMBtu adjusted to account for emissions from start up and shut down and low load operation). This falls into the range of BACT for recently issued or drafted GHG PSD permits.

#### Circuit Breakers GHG BACT

The circuit breakers are electrical equipment insulated with sulfur hexafluoride (SF<sub>6</sub>), which is a greenhouse gas. SF<sub>6</sub> is a dielectric gas used in high voltage applications because of its ease of use and excellent insulation and arc-interruption properties.

The state of the art enclosed-pressure circuit breakers with leak detection equipment has been selected as BACT. The manufacturer guarantee is an annual leak rate of less than 1% for the proposed circuit breakers, and a low-pressure alarm will be installed to alert of fugitive leaks before a substantial quantity of SF<sub>6</sub> is released. Emissions will be monitored in accordance with the requirements of the Mandatory Greenhouse Gas Reporting rule for Electrical Transmission and Distribution Equipment Use (40 CFR 98, Subpart DD).

#### Auxiliary Boiler and Fuel Gas Heater

GEP/S plans to install an auxiliary boiler and a fuel gas heater. Both units burn only pipeline quality natural gas and are relatively small emission sources when compared to the CTs.

#### CO and VOC BACT

Available emission control techniques for CO are good combustion practices and oxidation catalysts. These controls are capable of limiting CO emissions to 0.037 lb/MMBtu, which is equivalent to 50 ppmvd at 3% O<sub>2</sub>. Data from EPA's RBLC show that recent emission rates for natural gas-fired boilers and fuel gas heaters less than 250 MMBtu/hr is in the range of 0.035 lb/MMBtu to 0.060 lb/MMBtu.

Oxidation catalysts may be technically feasible to achieve lower CO emissions than using good combustion practices alone. However, due to low emission potential of 12.15 tpy of CO emissions, oxidation catalyst is expected to be not economically feasible.

GEP/S proposes to implement good combustion practices as BACT in the auxiliary boiler and fuel gas heater to limit CO emissions to 0.037 lb/MMBtu. DEQ agrees that using good combustion practices is BACT for CO for the auxiliary boiler and the fuel gas heater.

Available emission control techniques for VOC are good combustion practices and oxidation catalysts. GEP/S proposes to burn only pipeline quality natural gas in the auxiliary boiler and the fuel gas heater and to use good combustion practices as BACT to limit emissions to 0.002 lb/MMBtu. Annual VOC emissions from the auxiliary boiler will be limited to 0.66 tons/yr while emissions from the fuel gas heater will be limited to 0.18 tons/yr. At this low emission potential of VOC emissions, oxidation catalyst is expected to be not economically feasible.

#### PM/PM-10/PM-2.5 BACT

Particulate matter emissions from the boiler and fuel gas heater are a combination of filterable and condensable particulate. Good combustion practices and limiting fuel use to only pipeline quality natural gas are proposed by the applicant as BACT for PM/PM-10/PM-2.5 emissions from the auxiliary boiler and fuel gas heater. DEQ agrees that this constitutes BACT for particulate emissions from the boiler and heater. Short-term PM-10/PM-2.5 emissions from the auxiliary boiler and the fuel gas heater will be limited to 0.15 lbs/hr and 0.04 lbs/hr, respectively. Annual PM-10/PM-2.5 emissions from the auxiliary boiler will be limited to 0.66 tons/yr while emissions from the fuel gas heater will be limited to 0.18 tons/yr.

#### SO<sub>2</sub> and Sulfuric Acid Mist control

SO<sub>2</sub> and sulfuric acid mist emissions are not subject to PSD BACT or minor NSR review. Emissions of SO<sub>2</sub> from the auxiliary boiler and fuel gas heater are a result of oxidation of fuel sulfur. Sulfuric acid mist emissions (SO<sub>3</sub>/H<sub>2</sub>SO<sub>4</sub>) are based on a 5% conversion of SO<sub>2</sub> to SO<sub>3</sub> by the boiler and heater.

The applicant has proposed the use of pipeline quality natural gas and good combustion practices to limit SO<sub>2</sub> and sulfuric acid mist emissions.

Greenhouse Gas (GHG) BACT

The use of low carbon fuels, oxidation catalyst and designs for high fuel to electricity efficiency are all considered technically feasible control technologies and are already being proposed as part of the Project. There are no technically feasible technologies for further reducing greenhouse gas emissions from the combustion turbine generators.

Emergency Diesel Generator and Diesel Fire Water Pump

The emergency generator will be operated only during interruptions in normal electrical power supply to the facility or for maintenance, testing, and operator training. The emergency fire water pump will be operated only in the event of a plant fire, maintenance, testing, and operator training. Each unit is limited to 500 hours of operation per year that includes 100 hours of operation per year for testing and maintenance.

CO BACT

Because of the limited hours of operation for the emergency units, resulting in low emissions, add-on controls for CO are not practical. The emission factors for CO used as the basis for the emergency generator and fire water pump emissions limits are based on the NSPS Subpart IIII limits for Stationary Compression Ignition Internal Combustion Engines, the current federal standard for stationary engines.

DEQ considers the federal standard from EPA's Tier II non road and stationary emergency engines of 3.5 g/brake horse power (bhp) to be acceptable as BACT. At 500 hours of operation, the maximum annual CO emissions for the generator would be 2.89 tons per year and for the firewater pump would be 0.47 tons per year. Given the limited allowable emissions, it is evident that add-on controls would not be cost effective.

PM/PM-10 /PM-2.5 BACT

Particulate matter emissions from oil-fired internal combustion engines may result from trace metals present in the fuel, unburned



carbon-containing materials and sulfate formation. The use of ultra-low sulfur fuel oil, good combustion practices, and a limitation on operating hours is considered BACT for PM/PM-10/PM-2.5 from the emergency units. The proposed emission rate for PM, based on NSPS Subpart IIII, is 0.002 lb/MMBtu for both the generator and the fire water pump. Annual PM/PM-10/PM-2.5 emissions from each unit are less than 0.5 ton per year, so DEQ finds the proposal acceptable as BACT for PM/PM-10/PM-2.5 from the generator and fire water pump.

It should be noted that the permit requirement to use ultra-low sulfur fuel per the federal motor vehicle diesel fuel standards (40 CFR 80.500 and 80.520) is expected to result in reduced PM/PM-10 emissions from the emergency equipment, as less sulfur will be available to form sulfates, a fine particulate.

#### VOC BACT

VOC emissions from internal combustion units are the result of incomplete combustion. Due to the limited operating hours for the emergency units, add-on controls, even if technically feasible, would not be economically feasible. The application proposes conservative VOC emission rates equal to the NSPS, Subpart IIII emission limits for non-methane hydrocarbons (NMHC) + NO<sub>x</sub> of 6.4 g/kW-hr for the generator and 4.0 g/kW-hr for the fire water pump as BACT. NO<sub>x</sub> and VOC are not segregated in the NSPS.

At 500 hours of operation, the maximum annual VOC emissions for the generator would be 5.29 tons per year and for the fire water pump would be 0.54 tons per year. DEQ concurs with the proposed limits as BACT.

#### SO<sub>2</sub> control

SO<sub>2</sub> emissions are not subject to PSD BACT or minor NSR review. GEP/S has proposed to use ultra-low sulfur fuel in the generators (distillate oil having no more than 0.0015% sulfur by weight).

#### Turbine Inlet Evaporative Coolers

Evaporative coolers are located in the gas turbine inlet duct. Water is sprayed over a media to cool the incoming air. This process increases the amount of air mass flowing through the turbine, increasing the power generated and the turbine's efficiency.

**Ten Cell Mechanical Draft Cooling Tower**

Green Energy Partners/Stonewall, LLC plans to install a 10-cell, 187,400 gal/min mechanical draft cooling tower to service the condenser for the steam turbine. The tower will employ plume abatement to eliminate visible plumes except during extreme weather conditions. A plume abated tower is essentially a hybrid or wet/dry cooling tower design. The tower contains the wet evaporative section to cool the circulating water and the dry section to abate or reduce the visible plume. A plume is the result of the wet evaporative process that generates heated saturated air. When the saturated air exits the cooling tower and comes into contact with the cooler ambient air temperatures, condensation will occur, which creates a visible plume. In order to abate or reduce the plume of a cooling tower, the saturated air that is created by the wet evaporative process is dried out before exiting the tower. This is achieved by extending the plenum height of the tower and installing a dry heat transfer section into the side of the plenum, where dry air can be drawn through louvers, heated and then introduced into the plenum area. Once the warm dry air has entered the plenum area, it comes into contact with features installed to facilitate the mixing of the saturated air and the warm dry air. The effect of mixing these two air masses essentially dries out the saturated air, so that when it exits the tower and comes into contact with the cooler ambient air temperatures, the result is either a substantially reduced plume or no visible plume. The plume abatement system will generally be less effective during periods of high relative humidity and cold weather conditions.

The cooling tower will also utilize highly efficient drift eliminators to reduce water losses during operation. The drift eliminators also serve the purpose of reducing particulate emissions from dissolved solids in the drift water. Table C-11 in Appendix C of the application summarizes the recent BACT determinations for cooling towers. All BACT determinations relate to controlling the drift from the cooling towers. As shown in Table C-11, the most stringent drift rate limit is 0.0005% of circulating water flow. Achieving a drift rate of 0.0005% is technically feasible. Consistent with recent BACT determinations, a drift rate of 0.0005% is proposed as BACT for the cooling tower for the Project. The maximum annual emissions from the operation of the ten cell mechanical draft cooling tower is 10.27 tons per year of PM-10 and 3.19 tons per year of PM-2.5.

The method of calculating the emissions are as follows:

Emission Rate (lb/hr) = Water Circulation Rate (gpm) x 60 min/hr  
x Drift (%) / 100 x 8.3453 (lb/gal) x TDS (ppmw, or lb  
PM/1,000,000 lb water) x Weight Percent of Particle Size (%) /  
100.

For PM10, this would be Emission Rate for Total Cooling Tower  
(lb/hr) = 187400 (gpm) x 60 (min/hr) x 0.0005 (%) / 100 x 8.3453  
(lb/gal) x 5000 (lb PM/1,000,000 lb water) x 100 (%) / 100 = 2.35  
lb/hr.

DEQ concurs with the proposed limits as BACT.

4. NESHAP (40 CFR Part 61)

National Emission Standards for Hazardous Air Pollutants (NESHAP), found at 40 CFR 61, regulate emissions of specific HAPs from a limited number of source categories. 40 CFR 61 standards are incorporated by reference into Virginia Regulations at 9 VAC 5 Chapter 60, Part II, Article 1 (Rule 6-1). None of these Part 61 regulations apply to natural gas-fired stationary combustion turbines or the other emissions units proposed for the GEP/S Stonewall Energy Project.

5. MACT (40 CFR Part 63)

Maximum Achievable Control Technology (MACT) standards, found at 40 CFR 63, designate emission standards for HAPs from specific source categories. 40 CFR 63 standards are incorporated by reference into Virginia Regulations at 9 VAC 5 Chapter 60, Part II, Article 2 (Rule 6-2).

40 CFR 63 Subpart YYYYY, National Emissions Standards for HAPs from Stationary Combustion Turbines, was promulgated March 5, 2004 and applies to CTs located at major HAP sources. The potential HAP emissions from the proposed GEP/S facility do not exceed major source thresholds for HAPs, i.e., 10 tons per year of a single HAP or 25 tons per year of all HAPs combined. Accordingly, the proposed facility is not subject to the MACT standard. It should be noted that the MACT stipulates oxidation catalyst as one way to comply with the MACT limits (oxidation catalysts not only reduce CO and VOC emissions, they also reduce HAP emissions such as formaldehyde, toluene, acetaldehyde and benzene). GEP/S has proposed oxidation catalyst to control CO and VOC from its facility.

40 CFR 63 Subpart ZZZZ, National Emissions Standards for HAPs for Stationary Reciprocating Internal Combustion Engines, was promulgated June 15, 2004 and applies to stationary reciprocating internal combustion (IC) engines located at major and area sources of HAP emissions. Per 40 CFR 63.6590(c), stationary IC engines subject to Regulations under 40 CFR Part 60 can meet the requirements of Subpart ZZZZ by meeting the requirements of 40 CFR 60 Subpart IIII for compression ignition engines. As mentioned below, 40 CFR 60 Subpart IIII applies to the proposed IC engines and the applicable requirements from Subpart IIII have been included in the permit. Therefore, no further requirements from Subpart ZZZZ apply to the engines.

6. NSPS (40 CFR Part 60)

New Source Performance Standards (NSPS), found at 40 CFR 60, designate emission standards for criteria pollutants (a few regulate HAPs as well) from new emissions units at specific source categories. 40 CFR 60 standards are incorporated into Virginia Regulations at 9 VAC 5 Chapter 50, Part II, Article 5 (Rule 5-5).

There are NSPS that apply to the CTs, the DBs, the auxiliary boiler, the fuel gas heater, the emergency generator, and the fire water pump at the proposed facility, as detailed below:

- *40 CFR 60 Subpart KKKK (Standards of Performance for Stationary Combustion Turbines)*

Subpart KKKK applies to gas turbines having a heat input at peak load equal to or greater than 10 MMBtu/hr, based on the higher heating value of the fuel fired. The subpart also applies to emissions from the associated duct burners. The rule imposes limits on NO<sub>x</sub> and SO<sub>2</sub> emissions and monitoring and testing requirements. Using the most conservative assumptions, the NO<sub>x</sub> limit in Subpart KKKK is 15 ppm at 15% O<sub>2</sub> and the SO<sub>2</sub> limit must be 0.060 lb SO<sub>2</sub>/MMBtu or lower.

The LAER determination codified in the permit are more stringent than the NSPS requirements. For example, the NO<sub>x</sub> permit limit is 2.0 ppmvd, the fuel sulfur content is limited to 0.0003 % by weight, and the SO<sub>2</sub> permit limit is 0.000261 lb/MMBtu. Testing and monitoring requirements mirror or exceed those in Subpart KKKK.

- *40 CFR 60 Subpart Da (Standards of Performance for Electric Utility Steam Generating Units for Which Construction is Commenced After September 18, 1978)*

Subpart Da applies to electric utility steam generating units capable of combusting more than 250 MMBtu/hr heat input of fossil fuel for which construction began after September 18, 1978. The DBs proposed by GEP/S meet the applicability criteria of the rule and are subject to its requirements.

However, duct burners regulated under NSPS, Subpart KKKK are exempted from the requirements of NSPS, Subparts Da, Db, and Dc.

- *40 CFR 60 Subpart Dc (Standards of Performance for Small Industrial-Commercial-Institutional Steam Generating Units)*

Subpart Dc applies to steam generating units with a maximum design heat input capacity in the range of 10 MMBtu/hr to 100 MMBtu/hr for which construction began after June 9, 1989.

The auxiliary boiler and the fuel gas heater meet the applicability criteria of the rule and are subject to its requirements. The applicable requirements for natural gas burning units have been incorporated into the permit.

- *40 CFR 60 Subpart IIII (Standards of Performance for Stationary Compression Ignition Internal Combustion Engines)*

Subpart IIII applies to stationary internal combustion (IC) engines with a displacement of less than 30 liters per cylinder where the model year is 2007 or later, for engines that are not fire pump engines. For fire pump engines, Subpart IIII applies beginning with the model years listed in Table 3 of the subpart. The rule imposes emission standards on NO<sub>x</sub>, CO, and PM emissions based on the engine model year and engine use (emergency, fire pump, etc.). The subpart also requires engine owners and operators to use ultra-low sulfur fuel in the generators (distillate oil having no more than 0.0015% sulfur by weight). The applicable requirements for the generator and fire pump engines have been incorporated into the permit.

Since the generator and fire pump engines will meet the requirements of Subpart IIII, the units do not have any further requirements under 40 CFR 63 Subpart ZZZZ (see above).

- 40 CFR 60 Subpart Kb (*Standards of Performance for Volatile Organic Liquid Storage Vessels*) is not applicable to the 1,250 and 400-gallon distillate oil storage tank proposed by the applicant. Subpart Kb applies only to storage vessels having a capacity of at least 10,566.88 gallons (40 m<sup>3</sup>).

## V. Offsets

Green Energy Partners / Stonewall, LLC is required to secure NO<sub>x</sub> emissions offsets at a 1.00:1.15 ratio in accordance with 9VAC5-80-2120 and 40 CFR Part 51, Appendix S. The permittee shall secure NO<sub>x</sub> emission offsets of no less than 159 tons x 1.15 = 182.85 tons for the GE 7FA.05 combustion turbines, and 164.9 tons x 1.15 = 189.64 tons for the Siemens SGT6-5000F5 combustion turbines. If GEP/S chooses the Siemens combustion turbines, a VOC emission offset in the ratio of 1.15:1.00 will also be required. The permittee shall secure VOC emission offsets of no less than 51.9 tons x 1.15 = 59.69 tons for the Siemens SGT6-5000F5 combustion turbines. GEP/S had not yet identified the source of the offsets but is required to assure they are state and federally enforceable prior to beginning operation.

## VI. Compliance Determination

### A. Stack testing requirements

The permit requires initial compliance testing for NO<sub>x</sub>, SO<sub>2</sub>, CO, PM-10, PM-2.5, and VOC from the combined-cycle units. The need for periodic performance testing will be evaluated during processing of the Title V permit for the facility based on the results of the initial testing and operating data. A condition allowing DEQ to require additional testing has been included in the permit.

### B. Fuel testing requirements

The permit allows the permittee to use the fuel quality characteristics in a current, valid purchase contract, tariff sheet or transportation contract for the fuel to verify that the sulfur content of the natural gas is 0.1 grain or less of total sulfur per 100 standard cubic feet. Alternatively, per 40 CFR 60.4370, the permit allows GEP/S to determine the sulfur content of the natural gas by testing using two custom monitoring schedules or an EPA-approved schedule. The permit also requires the permittee to obtain fuel supplier certification for each shipment of distillate oil used in the emergency units.

### C. Visible emissions evaluations

A visible emissions evaluation (VEE), concurrent with the initial CT stack test, is required by the permit. Periodic CT stack visible emission inspections, which trigger a VEE according to EPA Method 9 if visible emissions are observed, have been included in the permit.

#### **D. Continuous emissions monitoring systems (CEMS)**

The permit requires that the CT stacks be equipped with CEMS meeting the requirements of 40 CFR Part 75 (Acid Rain program) for NO<sub>x</sub> and SO<sub>2</sub> (unless an alternative method of determining SO<sub>2</sub> emissions has been approved for that purpose). In addition to providing a means to demonstrate compliance with the permit NO<sub>x</sub> limits, the CEMS will satisfy the NSPS Subpart KKKK requirement to monitor NO<sub>x</sub> emissions using a CEMS. The permit also requires that the CT stacks be equipped with CEMS meeting the monitoring requirements in 40 CFR 60.13 for CO.

In addition to the CEMS, the draft permit requires GEP/S to conduct extensive, continuous monitoring of key operational parameters on the control devices to assure proper operation and performance (see Conditions 5 through 9).

Compliance with NO<sub>x</sub> and CO emission limits for the CCCTs will be determined using Continuous Emission Monitoring Systems (CEMS).

#### **E. Recordkeeping requirements**

- Compliance with SO<sub>2</sub> emission limits will be determined through fuel sulfur monitoring and records of fuel usage.
- VOC, CO, PM-10, and PM-2.5 emission factors (lb/MMBtu) will be verified during initial compliance testing. Since annual emission limits for these pollutants are based 8760 hours of operation with each unit operating at worst case conditions, compliance with annual emission limits can be demonstrated with fuel throughput records and operational limits. Accordingly, monthly record keeping of “rolling” 12-month totals is required for natural gas throughput to each turbine and to each duct burner.

Additionally, the permit requires that the following records be kept:

- Time, date, and duration of each CT startup, shutdown, and malfunction period;
- Annual number of startup and shutdown occurrences for each CT calculated monthly;

- Continuous records of heat input and power output for each CT;
- Emissions calculations sufficient to verify compliance with the annual emission limits in Conditions 35, 37, 38, 40, 41, 42, and 43 (calculated monthly as the sum of each consecutive 12-month period), and records sufficient to allow calculation of actual annual emissions from the remainder of the facility. Calculation methods are to be approved by the DEQ;
- CEMS data, calibrations and calibration checks, percent operating time, and excess emissions;
- Annual operating hours of the emergency generator and the fire water pump for emergency purposes and maintenance/testing, calculated monthly as the sum of each consecutive 12-month period;
- Time, date, and duration of operation of emergency generator and fire water pump for maintenance and testing and the operational status of each CT during that time;
- Fuel supplier certifications for distillate oil;
- Records of engine manufacturer data;
- Operation and monitoring records for each SCR system and each oxidation catalyst;
- Records of steady-state vs. non-steady-state operation of each CT unit, the ammonia slip monitoring plan, and ammonia slip monitoring results;
- Scheduled and unscheduled maintenance and operator training;
- Results of all stack tests, VEEs, visible emissions inspections, and performance evaluations;
- Monthly and annual fuel throughput to the auxiliary boiler and fuel gas heater;
- Records of good combustion practices for the auxiliary boiler and fuel gas heater;
- Records for emission offsets; and
- Records of CEMS quality control program.

The records must be available for DEQ inspection and maintained for five years.

## **VII. Public Participation**

### **A. Applicant Informational Briefing**

In accordance with Section 9 VAC 5-80-1775 C of the Regulations, the applicant held an informational briefing at 6:30 p.m. on September 24, 2012 at the Rust Library in Leesburg, Virginia. As required, the briefing was advertised in the Loudoun Times Mirror at least 30 days in advance (on August 24, 2012).



**B. Public Briefing**

9 VAC 5-80-1775 J specifies that a briefing be scheduled prior to the public comment period if appropriate. NRO has scheduled a public briefing at 6:00 p.m. to 7:00 p.m. on April 3, 2013 at Stone Bridge High School located at 43100 Hay Road in Ashburn, Virginia 20147. The briefing requires a 30-day (at minimum) notification period. A legal advertisement for the briefing was placed in the Washington Post and Loudoun Times Mirror on February 27, 2013.

**C. Public Hearing**

In accordance with 9 VAC 5-80-1775 E, NRO will hold a public hearing to accept comments on the air quality impact of the proposed source, alternatives to the source, the control technology required, and other appropriate considerations tentatively scheduled for April 3, 2013 at the Stone Bridge High School located at 43100 Hay Road in Ashburn, Virginia 20147 from 7:00 p.m. to 9:00 p.m. A legal advertisement for the hearing has been published in the Washington Post and Loudoun Times Mirror on February 27, 2013.

**D. Documents Concerning Public Comment Period**

Copies of the documents used in development of the draft permit were available for review at NRO. Additionally, a copy of Green Energy Partners / Stonewall, LLC permit application, modeling information and correspondence was placed online at the DEQ website. Upon completion of the application analysis and prior to the public briefing, the permit application, draft permit, and draft engineering analysis and all items contained in the attached Document List were accessible from DEQ's website at:

<http://www.deq.virginia.gov/Programs/Air/PermittingCompliance/Permitting/PowerPlants/GreenEnergyPartners.aspx>.

**E. Public Comment**

The public comment period which runs for at least 45 days and includes 15 days after the public hearing begins on February 28, 2013 and ends on April 19, 2013. All comments received will be recorded, reviewed and a Response to Comments document will be written.

**VIII. Notification of Other Government Agencies**

**A. Local Zoning**

Because the proposed facility constitutes a new stationary source subject to air permitting regulations, a local governing body certification form is required in accordance with Department policy and § 10.1-1321.1 of the Code of Virginia. On May 13, 2010 Tim Hemstreet, the County Administrator for Loudoun County certified that the proposed facility is fully consistent with local ordinances.

**B. Environmental Protection Agency (EPA)**

In accordance with 9 VAC 5-80-2070, there are specific notification requirements to advise EPA of sources impacting nonattainment areas. Accordingly, a copy of the permit application, including supplemental addenda, and DEQ's initial letter of determination were provided to EPA Region III. EPA will be provided with a copy of the draft permit and will be notified of the public comment period and the final determination on permit issuance.

**C. Federal Land Managers**

Because of GEP's distance to SNP (see Table 1), DEQ has worked with the Federal Land Managers (FLMs) whose responsibility it is to oversee such areas. In accordance with the Memorandum of Understanding dated March 31, 1993, between DEQ and Shenandoah National Park (SNP) and the Jefferson National Forest, both the National Park Service (NPS) and U.S. Forest Service (USFS) were provided copies of GEP's permit application and supplemental addenda, most notably the Class I and Class II modeling analyses.

Upon completion of DEQ's application analysis, DEQ provided the FLMs copies of correspondence generated in reaching its permit determination. On August 7, 2012, DEQ sent both NPS and USFS copies of the preliminary permit determination and provided notification that the application was considered complete and that the FLM 60-day review period had begun. Two updated applications were submitted to the DEQ, and on January 14, 2013 the FLM was notified. According to 9 VAC 5-80-1765 B, that notification must be provided at least 60 days before the scheduled public hearing on the application. In emails dated June 20, 21, and July 3, 2012, the USFS, FWS, and NPS responded to the DEQ notification letter by stating that they did not plan to issue any finding of adverse impact on visibility and other applicable AQRVs as a result of emissions from the proposed GEP/S facility. Copies of the draft permit and engineering analysis were sent to the FLMs prior to the beginning of the public comment period.

## **IX. Pollution Prevention**

The natural gas-fired combined-cycle turbine configuration may itself be considered a pollution prevention alternative in that it produces power much more cleanly (in pounds of pollutant emitted per kilowatt hour of power produced) than conventional coal or oil-fired power plants. The HRSGs are an important factor in clean power generation because they recover heat that would otherwise be lost to the atmosphere and use it to produce additional electrical power.

Site-specific pollution prevention measures have been included as requirements in the permit, such as the following:

- Use of clean fuels (natural gas containing no more than 0.0003 % sulfur by weight in the CTs, auxiliary boiler, and fuel gas heater;
- Use of clean firing technology (lean premix low-NO<sub>x</sub> burners);
- In the emergency generator and firewater pump, use of ultra low-sulfur (no more than 0.0015% sulfur by weight) distillate oil. Use of such fuels reduces emissions of not only sulfur dioxide and sulfuric acid mist but also of PM/PM-10/PM-2.5 (a component of which is sulfates) and is expected to reduce NO<sub>x</sub> emissions as well.

The permit also includes requirements related to emissions of ammonia from the SCR. Ammonia is injected in the SCR system to induce the catalytic reduction of NO<sub>x</sub>, and, to ensure maximum conversion of NO<sub>x</sub>, ammonia in excess of its stoichiometric requirement (the minimum amount required to react with a given amount of NO<sub>x</sub>) is used. Any unreacted ammonia remaining is released to the atmosphere and is referred to as “ammonia slip”. Although ammonia is not a regulated pollutant, ammonia emissions can nonetheless contribute to condensable particulate, regional haze, and nitrogen deposition. Furthermore, excessive ammonia emissions can indicate poor SCR system performance. Accordingly, the permit includes an ammonia emission limit of 5 ppmvd during operating conditions (as a one-hour average) for at least 95 % of the time that the SCR is operating and a requirement to submit a plan for monitoring ammonia slip.

## **X. Title V Operating Permit (9 VAC 5 Chapter 80, Article 1)**

GEP/S is required by Virginia regulations to obtain a federal operating permit under Title V of the Clean Air Act. The Regulations require that GEP/S submit a Title V permit application no later than one year after startup of the facility.

## **XI. Acid Rain Operating Permit (9 VAC 5 Chapter 80, Article 3)**

GEP/S is required by Virginia Regulations to obtain a permit under the federal Acid Rain program. Federal regulations require that a complete Acid Rain Program permit application be submitted at least 24 months prior to commencement of operation.

## **XII. NO<sub>x</sub> and SO<sub>2</sub> Trading Programs (9 VAC 5 Chapter 140)**

Virginia has established several emissions trading programs to meet the requirements of Section 110(a)(2)(D) of the Clean Air Act regarding transport of emissions from upwind states to downwind nonattainment areas. Electric generation units that have capacities above 25 MW and certain industrial boilers are generally subject to the restrictions of the trading programs, which EPA created to satisfy the mandates within Section 110(a)(2)(D) of the Clean Air Act to minimize impacts on downwind air quality. Accordingly, GEP/S will be required to comply with 9 VAC 5 Chapter 140 upon commencement of operation (first day any of the combustion turbines burn fuel).

The emission trading programs rely on regional cap and trade mechanisms that provide an economic incentive to minimize emissions from applicable units. These programs include provisions for construction of new facilities by allowing new units to access limited amounts of pollution allocations, called new source set asides. New units also may purchase allocations on the cap and trade market to cover emissions.

The NO<sub>x</sub> Budget Trading Program (9 VAC 5 Chapter 140 Part I “Regulations for Emissions Trading – NO<sub>x</sub> Budget Trading Program”) became effective in 2002. This program required that applicable units participate in a regional NO<sub>x</sub> ozone season cap and trade program. This regulation was later superseded by the more stringent Clean Air Interstate Rule (CAIR), which not only regulated NO<sub>x</sub> emissions during the ozone season but also regulated SO<sub>2</sub> and NO<sub>x</sub> emissions on an annual basis. The CAIR rules, as adopted into Virginia’s SIP, may be found at 9 VAC 5 Chapter 140 Part II, “Regulation for Emissions Trading – NO<sub>x</sub> Annual Trading Program”; Part III, “Regulations for Emissions Trading – NO<sub>x</sub> Ozone Season Trading Program”; and Part IV, “Regulations for Emissions Trading – SO<sub>2</sub> Annual Trading Program.” Similar to the NO<sub>x</sub> Budget Trading Program, the CAIR rules required that applicable units participate in regional NO<sub>x</sub> ozone season and annual trading programs as well as a regional SO<sub>2</sub> annual trading program.

A December 2008 court decision remanded CAIR to EPA but kept the requirements of CAIR in place temporarily until a new rule could be issued. The new rule, called the Cross-State Air Pollution Rule (CSAPR) was finalized on July 6, 2011. CSAPR was subsequently remanded back to EPA due to an August 21, 2012, ruling by the D. C. Circuit Court of Appeals. EPA has filed a petition seeking rehearing of this ruling. However, at this time, CAIR is in effect, and the

turbines and duct burners at GEP/S will be subject to the CAIR trading programs for annual and ozone season NO<sub>x</sub> emissions as well as for annual SO<sub>2</sub> emissions.

The fact that GEP/S is subject to CAIR will provide an incentive for the facility to minimize the number of times it starts up its CTs. During CT startup, NO<sub>x</sub> emissions from the unit are higher than they are during normal operation. If the facility has too many startups during a given period, it may exceed its NO<sub>x</sub> emission allotment. Such an exceedance in the trading program will cost the facility in that it may be required to purchase allowances to cover the additional emissions.

### **XIII. Document List**

A list of documents used in preparing the application analysis is included as Attachment E.

### **XIV. Recommendation**

Approval to proceed with public comment period is recommended.

### **Attachments**

Attachment A: Maximum Annual Turbine Emissions with Startups and Shutdowns

Attachment B: Public Hearing Notice

Attachment C: DEQ Air Quality Modeling Analysis Memorandum

Attachment D: Local Governing Body Form

**ATTACHMENT A:**

**Maximum Annual Turbine Emissions  
with Startups and Shutdowns**



**ATTACHMENT B:**  
**Public Hearing Notice**



## Public Notice – Environmental Permit

**PURPOSE OF NOTICE:** To seek public comment and announce a public hearing and an information briefing on a draft permit from the Department of Environmental Quality to limit air pollution from a facility proposed to be located in Loudoun County, Virginia.

**PUBLIC COMMENT PERIOD:** February 27, 2013 to April 19, 2013

**PUBLIC HEARING:** Cafeteria, Stone Bridge High School, 43100 Hay Road, Ashburn, Virginia 20147 on April 3, 2013 from 7:00 p.m. to 9:00 p.m.

**INFORMATION BRIEFING:** Same date and location as Public Hearing from 6:00 p.m. to 7:00 p.m.

**PERMIT NAME:** Prevention of Significant Deterioration (PSD) & Non-Attainment New Source Review Permit issued by DEQ, under the authority of the Air Pollution Control Board

**APPLICANT NAME AND REGISTRATION NUMBER:** Green Energy Partners / Stonewall, LLC; 73826

**FACILITY NAME AND PROPOSED LOCATION:** Stonewall Combined Cycle Project; 20077 Gant Lane, Leesburg, Virginia 20175 (approximately 4 miles south/south east of Leesburg & north of Dulles Toll Road (SR267), 39.058° N Latitude, 77.545° W Longitude).

**PROJECT DESCRIPTION:** Green Energy Partners / Stonewall, LLC has applied for a permit to construct and operate the Stonewall Combined-Cycle Project, a natural gas fired combined-cycle combustion turbine electric power generating facility having an electrical output capacity of approximately 750 MW. This area is in non-attainment for both the 1997 & 2008 8-hour ozone National Ambient Air Quality Standard (NAAQS) and the 1997 annual PM<sub>2.5</sub> NAAQS. The area is in attainment for all other NAAQS. The maximum annual emissions of air pollutants from the facility are expected to be: 164.9 tons per year (tpy) of nitrogen oxides (as NO<sub>2</sub>), 205.6 tpy of carbon monoxide (CO), 51.9 tpy of volatile organic compounds (VOCs), 106.1 tpy of total filterable & condensable particulate matter (PM) with a diameter less than or equal to 10 microns (PM<sub>10</sub>), 99.1 tpy of PM<sub>2.5</sub> (PM with a diameter less than or equal to 2.5 microns), 5.44 tpy of sulfur dioxide (SO<sub>2</sub>), 2,468,468 tpy of green house gasses (carbon dioxide equivalent) and 10.1 tpy of total hazardous air pollutants. The applicant proposes to use 40.9 billion cubic feet per year of natural gas for fuel, and 9,370 gallons of diesel fuel per year. The proposed Stonewall Combined-Cycle Project does not cause or significantly contribute to a predicted violation of any applicable NAAQS or Class II area PSD increment. Emission offsets are required for nitrogen oxides and potentially for VOCs.

**HOW TO COMMENT AND/OR REQUEST BOARD CONSIDERATION:** DEQ accepts comments and requests for Board consideration by hand-delivery, e-mail, fax or postal mail. All comments and requests must be in writing and be received by DEQ during the comment period. Submittals must include the names, mailing addresses and telephone numbers of the commenter/requester and of all persons represented by the commenter/requester. A request for Board consideration must also include: 1) The reason why Board consideration is requested. 2) A brief, informal statement regarding the nature and extent of the interest of the requester or of those represented by the requestor, including how and to what extent such interest would be directly and adversely affected by the permit. 3) Specific references, where possible, to terms and conditions of the permit with suggested revisions. Board consideration may be granted if public response is significant, based on individual requests for Board consideration, and there are substantial, disputed issues relevant to the permit.

**CONTACT FOR PUBLIC COMMENTS, DOCUMENT REQUESTS AND ADDITIONAL INFORMATION:**

Thomas Valentour; Northern Regional Office, 13901 Crown Court, Woodbridge, VA 22193; Phone: (703) 583-3931; E-mail: [Thomas.valentour@deq.virginia.gov](mailto:Thomas.valentour@deq.virginia.gov); Fax: (703)583-3821. The public may review the draft permit and application at the DEQ office named above by appointment or may request copies of the documents from the contact person listed above.

**ATTACHMENT C:**

**DEQ Air Quality Modeling Analysis Memorandum**



## MEMORANDUM

### DEPARTMENT OF ENVIRONMENTAL QUALITY *Office of Air Quality Assessments*

629 East Main Street, Richmond, VA 23219  
8<sup>th</sup> Floor

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To: James LaFratta, Air Permit Manager (NRO)

From: Mike Kiss, Director - Office of Air Quality Assessments (AQA)

Date: December 14, 2012

Subject: Virginia Department of Environmental Quality (DEQ) Technical Review of the Air Quality Analyses in Support of the Permit Application for the Proposed Green Energy Partners/Stonewall, LLC Natural Gas-Fired Electric Generating Facility in Loudoun County, Virginia (Stonewall Combined-Cycle Project)

Copies: Bobby Lute

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#### **I. Project Background**

Green Energy Partners/Stonewall, LLC has proposed to construct and operate a 750 megawatt (MW) natural gas-fired electric generating facility on an approximately 101-acre parcel located south-southeast of the Town of Leesburg Airport and north of the Dulles Greenway in Loudoun County, Virginia. The proposed new facility, called the Stonewall Combined-Cycle Project, will consist of two identical natural gas-fired only combined-cycle turbines, each with its own duct-fired heat recovery steam generator (HRSG), one steam turbine generator, a 10-cell mechanical draft cooling tower, a natural gas-fired only auxiliary boiler, a natural gas-fired only fuel heater, a diesel-fired emergency generator and fire water pump, two distillate fuel oil storage tanks, and circuit breakers. Green Energy Partners/Stonewall, LLC has proposed the installation of either General Electric (7FA.05) or Siemens (SGT6-5000F5) turbines.

The proposed facility meets the definition of major source under 9 VAC 5 Chapter 80, Article 8 (Prevention of Significant Deterioration (PSD)) of the Commonwealth of Virginia Regulations for the Control and Abatement of Air Pollution because it is a fossil-fuel-fired steam electric plant of more than 250 MMBtu/hr heat input capacity and has the potential to emit 100 tons per year or more of a regulated pollutant. Also, the proposed facility has the potential to emit

greenhouse gas (GHG) emissions equal to or greater than 75,000 TPY carbon dioxide equivalent (CO<sub>2</sub>e). The pollutants subject to PSD review are nitrogen oxides (NO<sub>x</sub>), particulate matter having an aerodynamic diameter equal to or less than 10 microns (PM<sub>10</sub>), carbon monoxide (CO), and greenhouse gases (GHGs). As a result, PSD regulations require an air quality analysis be performed that demonstrates that the projected air emissions from the proposed facility will neither cause or significantly contribute to a violation of any applicable National Ambient Air Quality Standard (NAAQS) or PSD increment. In addition, PSD regulations require that an additional impact analysis consisting of a vegetation and soil analysis, a growth analysis, and a visibility impairment analysis be conducted.

Loudoun County is included in the Washington, D.C. Metropolitan Statistical Area (MSA) which is currently designated as a “marginal” nonattainment area for the 2008 ozone NAAQS. NO<sub>x</sub> and volatile organic compound (VOC) emissions, precursors to ozone formation, are subject to the nonattainment permitting provisions.

The Washington, D.C. MSA, including Loudoun County, is also currently designated as nonattainment for the 1997 annual particulate matter having an aerodynamic diameter of 2.5 microns or less (PM<sub>2.5</sub>). However, the current air quality in the region is significantly below the 1997 annual PM<sub>2.5</sub> NAAQS. Virginia intends to formally submit a request to EPA in 2013 to redesignate the area to attainment.

The proposed facility’s permit application addressed the following two possible PM<sub>2.5</sub> scenarios because the area will not be formally reclassified during the review of the permit application:

- The area is nonattainment for PM<sub>2.5</sub>.
- The area is eventually redesignated as attainment for PM<sub>2.5</sub>.

As a result, an air quality analysis was also performed for PM<sub>2.5</sub> to demonstrate that the projected PM<sub>2.5</sub> emissions from the proposed facility will not cause or significantly contribute to a violation of any applicable PM<sub>2.5</sub> NAAQS or PSD increment.

An analysis of the project’s impact on air quality and air quality related values (AQRVs) in any affected Class I area may also be required, contingent upon input from the Federal Land Managers (FLMs). The United States Forest Service (USFS), the United States Fish and Wildlife Service (FWS), and the National Park Service (NPS) each stated in an e-mail dated June 20, 2012, June 20, 2012, and July 3, 2012, respectively, that an AQRV analysis was not required since the project is not expected to show any significant additional impacts to AQRVs. Therefore, only a Class I area analysis to assess compliance with the Class I PSD increments was required.

The following is a summary of the AQA’s review of the required air quality analyses for the Stonewall Combined-Cycle Project for both Class I and Class II PSD areas. The worst-case impacts from all operating loads, including startup and shutdown operations, are presented in this memorandum.

## **II. Modeling Methodology**

The Class I and Class II air quality modeling analyses conform to 40 CFR Part 51, Appendix W - Guideline on Air Quality Models and were performed in accordance with their respective approved modeling methodology. The air quality model used for the Class I area analysis was the EPA-approved regulatory version of the CALPUFF modeling system (Version 5.8, Level 070623). The CALPUFF modeling system is the preferred model for long-range transport applications and is contained in Appendix W of 40 CFR Part 51. The air quality model used for the Class II area analysis was the most recent version of the AERMOD modeling system (Version 12060). The AERMOD modeling system is the preferred EPA-approved regulatory model for near-field applications and is also contained in Appendix W of 40 CFR Part 51.

Additional details on the modeling methodology can be found in the applicable sections of Green Energy Partners/Stonewall, LLC's revised air permit application submittal dated November 2012.

## **III. Modeling Results**

### **A. Class II Area - Preliminary Modeling Analysis**

A preliminary modeling analysis for criteria pollutants was conducted in accordance with PSD regulations to predict the maximum ambient air impacts. The preliminary analysis modeled emissions from the proposed facility only to determine whether or not the impacts were above the applicable significant impact levels (SILs). For those pollutants for which maximum predicted impacts were less than the SIL, no further analyses was required (i.e., predicted maximum impacts less than SILs are considered insignificant and of no further concern). For impacts predicted to be equal to or greater than the SIL, a more refined air quality modeling analysis (i.e., full impact or cumulative impact analysis) is required to assess compliance with the NAAQS and PSD increment.

The emissions associated with six (6) representative operating loads for the General Electric turbine option and four (4) representative operating loads for the Siemens turbine option were modeled, as well as their startup/shutdown emissions. Tables 1 and 2 below show the maximum predicted ambient air concentrations for the General Electric and Siemens turbine options, respectively.

Table 1  
Class II Preliminary Modeling Analysis Results vs. Significant Impact Levels  
General Electric Turbines

Pollutant	Averaging Period	Maximum Predicted Concentration From Proposed Facility ( $\mu\text{g}/\text{m}^3$ )	Class II Significant Impact Level ( $\mu\text{g}/\text{m}^3$ )
NO <sub>2</sub>	1-hour	75.43	7.5
	Annual	0.97	1
PM <sub>10</sub>	24-hour	2.89	5
	Annual	0.37	1
PM <sub>2.5</sub>	24-hour	1.70	1.2
	Annual	0.20	0.3
CO	1-hour	2,381.37	2,000
	8-hour	113.58	500

Table 2  
Class II Preliminary Modeling Analysis Results vs. Significant Impact Levels  
Siemens Turbines

Pollutant	Averaging Period	Maximum Predicted Concentration From Proposed Facility ( $\mu\text{g}/\text{m}^3$ )	Class II Significant Impact Level ( $\mu\text{g}/\text{m}^3$ )
NO <sub>2</sub>	1-hour	70.69	7.5
	Annual	0.96	1
PM <sub>10</sub>	24-hour	2.72	5
	Annual	0.37	1
PM <sub>2.5</sub>	24-hour	1.35	1.2
	Annual	0.19	0.3
CO	1-hour	633.91	2,000
	8-hour	48.21	500

The modeling results for NO<sub>2</sub> (annual averaging period), PM<sub>10</sub> (24-hour and annual averaging periods), PM<sub>2.5</sub> (annual averaging period), and CO (8-hour averaging period) were less than the applicable SILs for both turbine options. Also, the modeling results for CO (1-hour averaging period) for the Siemens turbine option only were less than the applicable SIL. Therefore, a full impact analysis for these pollutants and averaging periods was not required. Furthermore, the additional pollution from this facility would not cause or

contribute to a violation of any applicable NAAQS or PSD increment for all pollutants and averaging periods with impacts below the applicable SILs.

A full impact analysis for CO (1-hour averaging period, General Electric turbine option only), NO<sub>2</sub> (1-hour averaging period), and PM<sub>2.5</sub> (24-hour averaging period) was conducted because the preliminary modeling analysis results exceeded the applicable SILs. Additionally, a full impact analysis was conducted for PM<sub>2.5</sub> (annual averaging period) at the request of DEQ even though the facility's predicted impact was below the SIL. This was done to provide additional assurance of NAAQS compliance in the Washington, D.C. MSA.

The AQA has adopted the NO<sub>2</sub> (1-hour) SIL in Table s1 and 2 based on a review of the following documentation:

*Guidance Concerning the Implementation of the 1-hour NO<sub>2</sub> NAAQS for the Prevention of Significant Deterioration Program, Stephen D. Page, EPA, June 29, 2010.*

The staff concurs with the EPA recommendations in this memorandum that it is appropriate to derive an interim 1-hour NO<sub>2</sub> SIL by using an impact equal to 4% of the 1-hour NO<sub>2</sub> NAAQS (4 ppb is equivalent to 7.5 µg/m<sup>3</sup>). The AQA believes that it is reasonable to adopt this value based on consideration of the impact level relative to the NAAQS and past EPA rationale for existing short-term averaging period SILs. The use of 4% of the NAAQS as a threshold is also consistent with previous EPA rulemaking and supporting documentation as described in the June 29, 2010 EPA memorandum.

## **B. Class II Area – Cumulative Impact Modeling Analysis**

The cumulative impact analysis described below consisted of separate analyses to assess compliance with the NAAQS for CO (General Electric turbine option only), NO<sub>2</sub>, and PM<sub>2.5</sub> and the PSD increment for PM<sub>2.5</sub> for the indicated averaging periods. It is important to note that the cumulative impact modeling results (both NAAQS and PSD increment) can sometimes be less than the “source only” modeling results in Tables 1 and 2 of this memorandum. This is due to the fact that source only modeling uses the maximum concentration to determine significance, whereas the cumulative modeling results reflect the form of the air quality standard. For example, the following criteria must be met to attain the NAAQS:

- CO (1-hour) - Not to be exceeded more than once per year.
- NO<sub>2</sub> (1-hour) - To attain this standard, the 3-year average of the 98<sup>th</sup> percentile of the daily maximum 1-hour average at each monitor within an area must not exceed the standard.
- PM<sub>2.5</sub> (24-hour) - To attain this standard, the 3-year average of the 98<sup>th</sup> percentile of 24-hour concentrations at each population-oriented monitor within an area must not exceed the standard.

- PM<sub>2.5</sub> (annual) - To attain this standard, the 3-year average of the weighted annual mean PM<sub>2.5</sub> concentrations from single or multiple community-oriented monitors must not exceed the standard.

#### *NAAQS Analysis*

The NAAQS analysis included emissions from the proposed source, emissions from existing sources from Virginia, West Virginia, and Maryland, and representative ambient background concentrations of NO<sub>2</sub>, PM<sub>2.5</sub>, and CO. The results of the analysis are presented in Tables 3 and 4 for the General Electric and Siemens turbine options, respectively, and demonstrate compliance with the applicable NAAQS.

Table 3  
NAAQS Modeling - Cumulative Impact Results  
General Electric Turbines

Pollutant	Averaging Period	Total Modeled Concentration (µg/m <sup>3</sup> )	Ambient Background Concentration (µg/m <sup>3</sup> )	Total Concentration (µg/m <sup>3</sup> )	NAAQS (µg/m <sup>3</sup> )
NO <sub>2</sub>	1-hour	117.72	47	164.72	188
CO	1-hour	2,273.97	2,530	4,803.97	40,000
PM <sub>2.5</sub>	24-hour	2.21	20	22.21	35
	Annual	0.42	9.5	9.92	15

Table 4  
NAAQS Modeling - Cumulative Impact Results  
Siemens Turbines

Pollutant	Averaging Period	Total Modeled Concentration (µg/m <sup>3</sup> )	Ambient Background Concentration (µg/m <sup>3</sup> )	Total Concentration (µg/m <sup>3</sup> )	NAAQS (µg/m <sup>3</sup> )
NO <sub>2</sub>	1-hour	117.72	47	164.72	188
PM <sub>2.5</sub>	24-hour	1.76	20	21.76	35
	Annual	0.37	9.5	9.87	15

#### *PSD Increment Analysis*

The 24-hour and annual PM<sub>2.5</sub> PSD increment analysis included emissions from the proposed source. Tables 5 and 6 below present the results of the analysis for the General Electric and Siemens turbine options, respectively, and show that the 24-hour and annual PM<sub>2.5</sub> concentrations were below their applicable PSD increment.



Table 5  
PSD Increment Modeling - Cumulative Impact Results  
General Electric Turbines

Pollutant	Averaging Period	Modeled Concentration ( $\mu\text{g}/\text{m}^3$ )	Class II PSD Increment ( $\mu\text{g}/\text{m}^3$ )
PM <sub>2.5</sub>	24-hour	2.13	9
	Annual	0.25	4

Table 6  
PSD Increment Modeling - Cumulative Impact Results  
Siemens Turbines

Pollutant	Averaging Period	Modeled Concentration ( $\mu\text{g}/\text{m}^3$ )	Class II PSD Increment ( $\mu\text{g}/\text{m}^3$ )
PM <sub>2.5</sub>	24-hour	1.62	9
	Annual	0.20	4

#### *NAAQS and PSD Increment Analyses Conclusions*

Based on AQA's review of the NAAQS and PSD increment analyses, assuming DEQ's regional office processing the permit application approved all of the emission estimates and associated stack parameters for the modeled scenarios, the proposed Stonewall Combined-Cycle Project does not cause or significantly contribute to a predicted violation of any applicable NAAQS or Class II area PSD increment.

#### *Toxics Analysis*

The source is subject to the state toxics regulations at 9 VAC 5-60-300 et al. An analysis was conducted in accordance with the regulations and the predicted concentrations for each toxic pollutant were below their respective Significant Ambient Air Concentrations (SAAC). Tables 7 and 8 summarize the toxic pollutant modeling analysis results for the General Electric and Siemens turbine options, respectively.

Table 7  
Toxics Analysis Maximum Predicted Concentrations  
General Electric Turbines

Toxic Pollutant	Averaging Period	Maximum Modeled Concentration From Project ( $\mu\text{g}/\text{m}^3$ )	SAAC ( $\mu\text{g}/\text{m}^3$ )
Acrolein	Annual	1.00E-04	0.46
Formaldehyde	1-hour	3.21E-01	62.5
	Annual	4.58E-03	2.4
Cadmium	1-hour	2.64E-03	2.5
	Annual	5.00E-05	0.1
Chromium	1-hour	3.36E-03	2.5
	Annual	7.00E-05	0.1
Nickel	1-hour	5.04E-03	5
	Annual	1.00E-04	0.2

Table 8  
Toxics Analysis Maximum Predicted Concentrations  
Siemens Turbines

Toxic Pollutant	Averaging Period	Maximum Modeled Concentration From Project ( $\mu\text{g}/\text{m}^3$ )	SAAC ( $\mu\text{g}/\text{m}^3$ )
Acrolein	Annual	8.00E-05	0.46
Formaldehyde	1-hour	3.18E-01	62.5
	Annual	4.35E-03	2.4
Cadmium	1-hour	2.44E-03	2.5
	Annual	5.00E-05	0.1
Chromium	1-hour	3.11E-03	2.5
	Annual	7.00E-05	0.1
Nickel	1-hour	4.66E-03	5
	Annual	1.00E-04	0.2

#### Additional Impact Analysis

In accordance with the PSD regulations, additional impact analyses were performed to assess the impacts from the proposed facility on visibility, vegetation and soils, and the potential for and impact of secondary growth. These analyses are discussed below.

### *Visibility*

A screening modeling analysis using the VISCREEN model was conducted to assess the potential for visual plume impacts in Class II areas within 50 kilometers (km) of the project site. A review of National Parks and other potential areas of interest near the project site was conducted. It was determined that Manassas National Battlefield Park is the closest area of potential interest. Manassas National Battlefield Park is approximately 23 km southeast of the project site.

The visibility screening modeling approach followed guidance provided in EPA's *Workbook for Plume Visual Impact Screening and Analysis (Revised)* (October 1992; EPA-454/R-92-023). The two visibility metrics that were evaluated in the VISCREEN modeling analysis are:

- **Plume contrast ( $|C|$ ):** Contrast can be defined at any wavelength as the relative difference in the intensity (called spectral radiance) between the viewed object (e.g., plume) and its background (e.g., sky). Plume contrast results from an increase or decrease in light transmitted from the viewing background through the plume to the observer.
- **Plume perceptibility ( $\Delta E$ ):** A parameter used to characterize the perceptibility of a plume on the basis of the color difference between the plume and a viewing background such as the sky, a cloud, or a terrain feature.

The VISCREEN results were developed for the worst-case normal operating scenario. All results were below the significance criteria in the nearest Class II area. Therefore, the plume is expected to be imperceptible against the background sky and the terrain in the Manassas National Battlefield Park.

The visibility in the area near the proposed facility will be protected by operational requirements, such as air pollution controls and clean burning fuels, and stringent limits on visible emissions that are incorporated into the draft permit.

### *Vegetation and Soils*

An analysis on sensitive vegetation types with significant commercial or recreational value was conducted. The analysis compared maximum predicted concentrations from the proposed facility against a range of injury thresholds found in various peer-reviewed research articles as well as criteria contained in the EPA document *A Screening Procedure for the Impacts of Air Pollution Sources on Plants, Soils, and Animals* (EPA, 1980). Tables 9 and 10 show the maximum modeled concentrations for NO<sub>2</sub>, PM<sub>10</sub>, and CO for the General Electric and Siemens turbine options, respectively, were all below the respective thresholds (i.e., the minimum reported levels at which damage or growth effects to vegetation may occur). As a result, no adverse impacts on vegetation are expected.

Table 9  
Comparison of Vegetation Sensitivity Thresholds to Maximum Modeled  
Concentrations from the Stonewall Combined-Cycle Project  
General Electric Turbines

Pollutant	Averaging Period	Maximum Modeled Concentration From Proposed Facility ( $\mu\text{g}/\text{m}^3$ )	Sensitive Vegetation Threshold ( $\mu\text{g}/\text{m}^3$ )
NO <sub>2</sub>	1-hour	14.71	280
	4-hour	99.98	3,760
	1-month	1.34	564
	Annual	0.97	94
PM <sub>10</sub>	24-hour	2.89	150
CO	1-week	23.24	1,800,000

Table 10  
Comparison of Vegetation Sensitivity Thresholds to Maximum Modeled  
Concentrations from the Stonewall Combined-Cycle Project  
Siemens Turbines

Pollutant	Averaging Period	Maximum Modeled Concentration From Proposed Facility ( $\mu\text{g}/\text{m}^3$ )	Sensitive Vegetation Threshold ( $\mu\text{g}/\text{m}^3$ )
NO <sub>2</sub>	1-hour	14.68	280
	4-hour	99.98	3,760
	1-month	1.34	564
	Annual	0.96	94
PM <sub>10</sub>	24-hour	2.72	150
CO	1-week	23.24	1,800,000

The impact of the emissions on soils in the vicinity of the proposed project was evaluated. The soil type was determined from data collected from the United States Department of Agriculture's Natural Resources Conservation Service (NRCS) Soil Survey Geographic (SSGUGO) database and the NRCS Web Soil Survey tool. The soil types within Loudoun County, Virginia and Montgomery County, Maryland were examined.

The predominant soil types for Loudon County are a variety of silt loams. In Montgomery County, the predominate soil types are also a variety of silt loams, with some small areas of sandy loams.

The soil types in these counties are generally considered to have a moderate to high buffering capacity and have adequate capacity to absorb acidic deposition without changing the soil pH. Based on the soil types and quantity of emissions from the proposed project, no adverse impact on local soils is anticipated.

### *Growth*

The work force for the proposed facility is expected to range from 600 to 700 jobs during various phases of the construction. It is expected that a significant regional construction force is already available to build the proposed facility. Therefore, it is anticipated that no new housing, commercial, or industrial construction will be necessary to support the Stonewall Combined-Cycle Project during the two-year construction schedule. The proposed facility will also require approximately 25 to 30 permanent positions. It is assumed that individuals that already live in the region will perform a number of these jobs. No new housing requirements are expected for any new personnel moving to the area. In addition, due to the small number of new individuals expected to move into the area to support the Stonewall Combined-Cycle Project and the existence of some commercial activity in the area, new commercial construction would not be necessary to support the permanent work force. Additionally, no significant level of industrial related support will be necessary for the Stonewall Combined-Cycle Project. Therefore, industrial growth is not expected.

Based on the growth expectations discussed above, no new significant emissions from secondary growth during the construction and operation phases of the Stonewall Combined-Cycle Project are anticipated.

## **C. Class I Area Modeling Analysis**

The FLMs are provided reviewing authority of Class I areas that may be affected by emissions from a proposed source by the PSD regulations and are specifically charged with protecting the Air Quality Related Values (AQRVs) within the Class I areas. The closest Class I area to the proposed facility is Shenandoah National Park (SNP). It is approximately 57 km from the proposed facility. The other Class I areas within 300 km of the proposed facility, but located at a distance greater than 57 km, are Dolly Sods Wilderness Area, Otter Creek Wilderness Area, James River Face Wilderness Area, and Brigantine National Wildlife Refuge.

Modeling guidance contained in the *Federal Land Managers' Air Quality Related Values Work Group (FLAG) Phase I Report – Revised (2010)* provides screening criteria for determining whether a source may be excluded from performing a Class I area AQRV

modeling analysis. The FLMs may consider excluding a source from modeling if its total SO<sub>2</sub>, NO<sub>x</sub>, PM<sub>10</sub>, and H<sub>2</sub>SO<sub>4</sub> annual emissions (in tons per year, based on 24-hour maximum allowable emissions) divided by the distance (in km) from the Class I area is less than or equal to 10. The sum of the emissions for the proposed project is not expected to exceed approximately 317.2 tons per year (tpy). Therefore, the FLAG 2010 screening criteria for SNP is 5.6 (317.2 tpy/57 km). The screening criteria for all other Class I areas is less than 5.6 because these areas are located at a distance greater than 57 km. As a result, the USFS, the FWS, and the NPS each stated in an e-mail dated June 20, 2012, June 20, 2012, and July 3, 2012, respectively, that an AQRV analysis was not required since the project is not expected to show any significant additional impacts to AQRVs.

However, even though an AQRV analysis was not required to be conducted, an analysis to assess compliance with the Class I PSD increments for PM<sub>10</sub>, PM<sub>2.5</sub>, and NO<sub>2</sub> was required. A preliminary modeling analysis for PM<sub>10</sub>, PM<sub>2.5</sub>, and NO<sub>2</sub> was conducted to determine whether or not the predicted maximum ambient air impacts in the closest Class I area (i.e., SNP) were above the Class I SILs. This analysis was limited to only SNP because the impacts will be higher relative to the other Class I areas since its proximity to the proposed facility is nearly 100 km closer than the other Class I areas. The emissions used in the Class I area modeling were the same as those used for the Class II area modeling. A more refined air quality modeling analysis (i.e., cumulative impact analysis) would be required to assess compliance with the Class I PSD increments for impacts predicted to be equal to or above the Class I SIL. No additional air quality analysis would be required for pollutants when the proposed project's impacts were less than the SIL.

Tables 11 and 12 below present the proposed facility's maximum predicted ambient air concentrations for PM<sub>10</sub>, PM<sub>2.5</sub>, and NO<sub>2</sub> for the General Electric and Siemens turbine options, respectively, in Shenandoah National Park.

Table 11  
Summary of Maximum Predicted Concentrations from the Proposed  
Facility in Shenandoah National Park  
General Electric Turbines

Pollutant	Averaging Period	Maximum Predicted Concentration From Proposed Facility (µg/m <sup>3</sup> )	Class I Significant Impact Level (µg/m <sup>3</sup> )
PM <sub>10</sub>	24-hour	0.1117	0.3
	Annual	0.0029	0.2
PM <sub>2.5</sub>	24-hour	0.1117	0.07
	Annual	0.0029	0.06
NO <sub>2</sub>	Annual	0.0025	0.1

Table 12  
Summary of Maximum Predicted Concentrations from the Proposed  
Facility in Shenandoah National Park  
Siemens Turbines

Pollutant	Averaging Period	Maximum Predicted Concentration From Proposed Facility ( $\mu\text{g}/\text{m}^3$ )	Class I Significant Impact Level ( $\mu\text{g}/\text{m}^3$ )
PM <sub>10</sub>	24-hour	0.1002	0.3
	Annual	0.0026	0.2
PM <sub>2.5</sub>	24-hour	0.1002	0.07
	Annual	0.0026	0.06
NO <sub>2</sub>	Annual	0.0024	0.1

The modeling results for NO<sub>2</sub> (annual averaging period), PM<sub>10</sub> (24-hour and annual averaging periods), and PM<sub>2.5</sub> (annual averaging period) were less than the applicable SILs for both turbine options. Therefore, a cumulative impact analysis to assess compliance with the Class I PSD increments was not required for these pollutants and their averaging periods. However, a cumulative impact analysis for PM<sub>2.5</sub> (24-hour averaging period) for both turbine options was conducted because the preliminary modeling analysis results exceeded the applicable SIL.

#### *PSD Increment Analysis*

The 24-hour PM<sub>2.5</sub> PSD increment analysis included emissions from the proposed source and nearby PM<sub>2.5</sub> increment consuming sources. Table 12 presents the results of the analysis for both the General Electric and Siemens turbine options and shows that the 24-hour PM<sub>2.5</sub> concentrations for both turbine options were below the applicable PSD increment.

Table 12  
PSD Increment Modeling - Cumulative Impact Results for Shenandoah National Park

Turbine Option	Pollutant	Averaging Period	Modeled Concentration ( $\mu\text{g}/\text{m}^3$ )	Class I PSD Increment ( $\mu\text{g}/\text{m}^3$ )
General Electric	PM <sub>2.5</sub>	24-hour	1.250	2
Siemens			1.249	

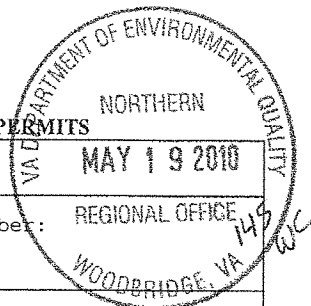
*Summary of Class I Area Analysis*

Based on AQA's review of the Class I area modeling analyses, the proposed Stonewall Combined-Cycle Project does not cause or significantly contribute to a predicted violation of any applicable Class I area PSD increment.



**ATTACHMENT D:**  
**Local Government Body Form**

**VIRGINIA DEPARTMENT OF ENVIRONMENTAL QUALITY - AIR PERMITS**



LOCAL GOVERNING BODY CERTIFICATION FORM	
Facility Name: <b>Green Energy Partners/Stonewall Energy Plant</b>	Registration Number: <b>New Source</b>
Applicant's Name: <b>Green Energy Partners/Stonewall, LLC</b>	Name of Contact Person at the site: <b>Jordan Dimoff, Project Manager</b>
Applicant's Mailing address: <b>Andrews Community Investment Corp. P.O. Box 660, Hamilton, Virginia 20159</b>	Contact Person Telephone Number: <b>540-338-9040</b>
Facility location (also attach map): <b>Four miles south, southeast of Leesburg, north of the Dulles Toll Road, adjacent Gant Lane and Cochran Mill Road</b>	
Facility type, and list of activities to be conducted: <b>Power Generation</b>	
<p>The applicant is in the process of completing an application for an air pollution control permit from the Virginia Department of Environmental Quality. In accordance with § 10.1-1321.1, Title 10.1, Code of Virginia (1950), as amended, before such a permit application can be considered complete, the applicant must obtain a certification from the governing body of the county, city or town in which the facility is to be located that the location and operation of the facility are consistent with all applicable ordinances adopted pursuant to Chapter 22 (§§ 15.2-2200 et seq.) of Title 15.2. The undersigned requests that an authorized representative of the local governing body sign the certification below.</p>	
Applicant's signature: <i>[Signature]</i>	Date: <b>5-3-2010</b>
<p>The undersigned local government representative certifies to the consistency of the proposed location and operation of the facility described above with all applicable local ordinances adopted pursuant to Chapter 22 (§§ 15.2-2200 et seq.) of Title 15.2, of the Code of Virginia (1950) as amended, as follows:</p> <p>(Check one block)</p> <p><input checked="" type="checkbox"/> The proposed facility is fully consistent with all applicable local ordinances.</p> <p><input type="checkbox"/> The proposed facility is inconsistent with applicable local ordinances; see attached information.</p>	
Signature of authorized local government representative: <i>[Signature]</i>	Date: <b>5/13/10</b>
Type or print name: <b>Tim Hemstreet</b>	Title: <b>County Administrator</b>
County, city or town: <b>Loudoun County</b>	

[THE LOCAL GOVERNMENT REPRESENTATIVE SHOULD FORWARD THE SIGNED CERTIFICATION TO THE APPROPRIATE DEQ REGIONAL OFFICE AND SEND A COPY TO THE APPLICANT.]

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
WASHINGTON, D.C. 20460

JAN 25 1995

OFFICE OF  
ENFORCEMENT AND  
COMPLIANCE ASSURANCE

SUBJECT: Guidance an Enforceability Requirements for  
Limiting Potential to Emit through SIP and §112 Rules  
and General Permits

FROM: Kathie A. Stein, Director  
Air Enforcement. Division

TO: Director, Air and, Pesticides and Toxics  
Management Division, Regions I and IV  
Director, Air and Waste Management Division,  
Region II  
Director, Air, Radiation and Toxics Division,  
Region III  
Director, Air and Radiation Division,  
Region V  
Director, Air, Pesticides and Toxics Division,  
Region VI  
Director, Air and Toxics Division,  
Regions VII, VIII, IX, and X

Attached is a guidance document developed over the past year by the former Stationary Source compliance Division in coordination with the Air Enforcement Division, Office of Air Quality Planning and Standards, OAR's Office of Policy Analysis and Review, and the Office of General Counsel, as well-as with significant input from several Regions.

A number of permitting authorities have begun discussions with or have submitted programs for review by EPA that would provide alternative mechanisms for limiting potential to emit. Several authorities have submitted SIP rules and at least one State has been developing a state general permit approach.; We believe that this guidance is important to assist the EPA Regions as well as States in approving and developing such approaches.

For additional information regarding this guidance, please contact me or Clara Poffenberger of my staff at (202) 564-8709.

cc: John Rasnic, Director  
Manufacturing, Energy, and Transportation Division Office of Compliance

Air Branch Chiefs, Regions I -X

## Enforceability Requirements for Limiting potential to Emit Through SIP and §112 Rules and General Permits

### Introduction

As several EPA guidance describe, there are several mechanisms available for sources to limit potential to emit. EPA guidance have also describe the importance of practical enforceability or the means used to limit the Potential to Emit. This guidance is intended to provide additional guidance on practical enforceability for such limits. We provide references for guidance an practical enforceability for permits and rules in general and provide guidance in this document for application of the same principles to "limitations established by rule or general permit," as described in the guidance document issued January 25, 1995, entitled "Options for Limiting Potential to Emit (PTE) of a Stationary Source under section 112 and Title V of the Clean Air Act (Act)." The description is as follows:

Limitations established by rules. For less complex plant sites, and for source categories involving relatively few operations that are similar in nature, case-by-case permitting may not be the most administratively efficient approach to establishing federally enforceable restrictions. One approach that has been used is to establish a general rule which creates federally enforceable restrictions at one time for many sources (these rules have been referred to as "prohibitory" or "exclusionary" rules). The concept of exclusionary rules is described in detail in the November 3, 1993 memorandum ["Approaches to Creating Federally Enforceable Emissions Limits," from John S. Seitz]. A specific suggested approach for VOC limits by rule was described in EPA's memorandum dated October 15, 1993 entitled "Guidance for State Rules for Optional Federally Enforceable Emissions Limits Base Upon Volatile Organic Compound (VOC) Use." An example of such an exclusionary rule is a model rule developed for use in California. (The California model rule is attached, along with a discussion of its applicability to other situations - see Attachment 2). Exclusionary rules are included in a State's SIP or 112 program and generally become effective upon approval by the EPA.

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The EPA prefers the term "exclusionary rule" in that this phrase is a less ambiguous description of the overall purpose of these rules.

General permits -A concept similar to the exclusionary rule is the establishment Of a general permit for a given source type. A general permit is a single permit that establishes terms and conditions that must be complied with by all sources subject to that permit. The establishment of a general permit could provide for emission limitations in a one-time permitting process, and thus avoid the need to issue separate permits for each source. Although this concept is generally thought of as an element of Title V permit programs there is no reason that a state or local agency could not submit a general permit program as a SIP submittal Aimed at creating synthetic minor sources. Additionally FESOP [Federally Enforceable State Operating Permit usually referring to Title I State Operating Permit Programs approved under- the criteria established by EPA in the June 28, 1989 Federal Register notice, 54 FR 27274] programs can include general permits as an element of the FESOP program being approved into the SIP. The advantage of a SIP general permit, when compared to an exclusionary rule, is that upon approval by the EPA of the state's general permit program, a general permit could be written for an additional source type without triggering the need for the formal SIP revision process. (January 25, 1995 Seitz and Van Heuvelen memorandum, page 4.)

#### SIP or §112 Rules

Source-category standards 'approved in the. SIP. or under 112,if enforceable as a. practical matter, can be used as federally enforceable limits on potential to emit. Such provisions require public participation and EPA review. Once a specific source qualifies under the applicability requirements of the source category rule, additional public participation is not required to make the limits federally enforceable as a matter of legal sufficiency since the rule itself underwent public participation and EPA review. The rule must still be enforceable as practical matter in order to be considered federally enforceable. A source that violates this type of rule limiting potential to emit below major a source thresholds or is later determined not to qualify for coverage under the rule, could be subject to enforcement action for violation of the rule and for constructing or operating without a proper permit (a. part 70, a New Source Review permit, or operating without meeting §112 requirements, or any combination thereof).

#### General Permits

The title V regulations set out provisions for general permits covering numerous similar sources. The primary purpose of general permits is to provide a permitting alternative where

the normal permitting process would be overly burdensome, such as for area sources under section 112. General permits may be issued to cover any category of numerous similar sources, including major sources, provided that such sources meet certain criteria laid out in 40 CFR part 70. Sources may be issued general permits strictly for the purpose of avoiding classification as major source. In other words, general permits may be used to limit the potential to emit for numerous similar sources. However, general permits must also meet both legal and practical federal enforceable requirements.

With respect to legal sufficiency, the operating permit regulations provide that once the general permit has been issued, after opportunity for public participation and, EPA and affected State review, the permitting authority may grant or deny a sources request to be covered by a general permit without further public participation or EPA or affected State review. The action of granting or denying the source's request is not subject to judicial review. A general permit does not carry a permit shield. A source may be subject to enforcement action for operating without a part 70 permit if the source is later determined not to qualify for coverage under the general permit. Sources covered by general permits must comply with all part 70 requirements.

#### State SIP or 112(1) General Permits

Another mechanism available to limit potential to emit is a general permit program approved into the SIP or under section 112(1), the hazardous air pollutant program authority. This mechanism allows permitting authorities to issue and revise general permits consistent with SIP or 112(1) program requirements without going through the SIP or 112(1) approval process for each general permit or revision of a general permit. The program is also separate from title V, like Title I state operating permits, and issuance and revisions of the permits are to comply with title V procedures.

Once a program is approved, issuing and revising general permits should be significantly less burdensome and time-consuming for State legislative and rulemaking authorities. The EPA review should also be less burdensome and time-consuming. After a program is approved, permitting authorities have the flexibility to submit and issue general permits as needed rather than submitting them all at once as part of a SIP submittal. Given the reduced procedural burden, permitting authorities should be able to issue general permits to small groups or categories or sources rather than attempt to cover broad categories with a generic rule. We anticipate that specific permit requirements or general permits may be readily developed with the assistance of interested industry groups.

The state general permit approach may allow sources to meet the federal the federal enforceability requirements more easily than other approaches. However, to use this approach, states must have a federally enforceable program that provides the state the authority, to issue such permits; to accomplish this, EPA must approve the program into the SIP or pursuant to section. 112(1) of the Clean Air Act.

### Enforceability Principles

In 1989, in response to challenges from the Chemical Manufacturers Association and other industry groups, EPA reiterated its position that controls and limitations used to limit a source's Potential to emit must be federally enforceable. See 54 FR 27274 (June 28, 1989). Federally enforceable limits can be established by Clean Air Act programs such as NSPS, NESHAPs, MACTs, and SIP requirements. However, source-specific limits are generally set forth in permits. Generally, to be considered federally enforceable, the permitting program must be approved by EPA into the SIP and include provisions for public participation. "In addition, permit terms and conditions must be practicably enforceable to be considered federally enforceable. EPA provided specific guidance on federally enforceable permit conditions in a June 13, 1989 policy memo "Limiting Potential to Emit in New Source Permitting" from John Seitz and in the June 28, 1989 Federal Register notice (54 FR 27274) Additional guidance Can also be found in United states v. Louisiana Pacific, 682 F. Supp 1122 (D. Colo. 1987) 682 F. Supp 1141 (D. Colo.1988), which led to these guidance statements and a number of other memoranda covering practicable enforceability as it relates to rolling averages, short-term averages, and emission caps. See "Use of Long Term Rolling Averages to Limit Potential to Emit," form John. B. Rasnic to David Kee, February 24, 1992; "Limiting Potential to Emit;" from Mamie Miller to George Czerniak, August, 1992; "Policy Determination an Limiting Potential to Emit for Koch Refining Company's Clean Fuels Project", from John B. Rasnic to David Kee, March 13, 1992; and "3M Tape Manufacturing Division Plant, St. Paul, Minnesota" from. John B. Rasnic to David Kee, July 14, 1992.

In 1987, EPA laid out enforceability criteria that SIP rules must meet. see "Review of State Implementation Plans and Revisions for Enforceability and Legal Sufficiency," from Michael Alushin, Alan Eckert, and John Seitz, September 3, 1987 (1997 SIP memo). The criteria include clear statements as to applicability, specificity as to the standard that must be met, explicit statements of the compliance time frames (e.g. hourly, daily, monthly, or 12-month averages, etc.), that the time frame and method of compliance employed must be sufficient to protect the standard involved, record keeping requirements must be specified, and equivalency provisions must meet certain requirements.

Based on these precedents this guidance describes six enforceability criteria which a rule or a general permit must meet to make limits enforceable as a practical matter. In general, practical enforceability for a source-specific permit term means that the provision must specify (1) a technically accurate limitation and the portions of the source subject to the limitation; (2) the time period for the limitation (hourly, daily, monthly, annually); and (3) the method to determine compliance including appropriate monitoring, record keeping and reporting. For rules and general permits that apply to categories of sources, practical enforceability additionally requires that the provision (4) identify the categories of sources that are covered by the rule; (5) where coverage is optional, provide for notice to the permitting authority of the source's election to be covered by the rule; and (6) recognize the enforcement consequences relevant to the rule.

This guidance will address requirements (4) "and (5) first as they are concepts that are unique to rules and general' permits.

#### A. Specific Applicability

Rules and general permits designed to limit potential to emit must be specific as to the emission units or sources covered by the rule or permit. In other words, the rule or permit must clearly identify the category(ies) of the sources that qualify for the rule's coverage. The rule must apply to categories of sources that are defined specifically or narrowly enough so that specific limits and compliance monitoring can be identified and achieved by all sources in the categories defined.

A rule or general permit that covers, a homogeneous group of sources should allow standards to be set that limit potential to emit and provide the specific monitoring requirements. (Monitoring is more fully addressed in section D.) The State can allow for generic control efficiencies where technically sound and appropriate, depending on the extent of the application and ability to monitor compliance with resultant emission limits. Similarly, specific and narrow applicability may allow generic material usage or limits on hours of operation to be sufficient. For example, a rule or general permit that applies to fossil fuel fired boilers of a certain size may allow for limits on material usage, such as fuel-type and quantity. A rule or general permit that applies, only to standby diesel generators or emergency generators may allow restrictions on hours of operation to limit potential to emit. The necessary compliance terms (i.e., monitoring or record keeping) associated with any of these limits, such as with hours of operation, can readily be specified in the rule or the general permit itself.

General permits under Title V are assumed to include this



enforceability principle because the Part 70 regulations set out specific criteria that states should consider in developing their general permit provisions (See 57 FR 32278). These factors include requirements that

"categories of sources covered by general permits should be generally homogenous in terms of operations, processes, and emissions. All sources in the category should have essentially similar operations or processes and emit pollutants with similar characteristics."

Another factor stated is "sources should be subject to the same or substantially similar requirements governing operation, emissions, monitoring, reporting, or record keeping." Examples of source categories appropriate for general permits include: degreasers, dry cleaners, small heating systems, sheet fed printers, and VOC storage tanks (see 57 FR 32278).

B. Reporting or Notice to Permitting Authority

The rule or general permit should provide specific reporting requirements as part of the compliance method. Although the compliance method for all sources must include record keeping requirements, the permitting authority may make a determination that reporting requirements for small sources would provide minimal additional compliance assurance. Where ongoing reporting requirements are determined not to be reasonable for a category of sources, the rule or general permit should still provide that the source notify the permitting authority of its coverage by the rule or the permit. In the limited situation where all the sources described in a source category are required to comply with the all of the provisions of a rule or general permit, notice is not needed. However, where there are no reporting requirement's and no opt-in provisions, the permitting authority must provide the public with the names and locations of sources subject to the rule or permit.

For Title V general permits, Part 70 requires sources to submit an application for a general permit which must be approved or disapproved by the permitting authority. For SIP or §112 rules and SIP or §112 general permits, in response to receiving the notice or application, the permitting authority may issue an individual permit, or alternatively, a letter or certification. The permitting authority may also determine initially whether it will issue a response for each individual application or notice, and may initially specify a reasonable time period after which a source that has submitted an application or notice will be deemed to be authorized, to operate under the general permit or SIP or §112 rule.

### C. Specific Technically Accurate Limits

The rule or general permit issued pursuant to the SIP or §112 must specify technically accurate limits on the potential to emit. The rule or general permit must clearly specify the limits that apply, and include the specific associated compliance monitoring. (The compliance monitoring requirements are discussed further in the next section.) The standards or limits must be technically specific and accurate to limit potential to emit, identifying any allowed deviations.

The 1987 policy on SIP enforceability states that limitations "must be sufficiently specific so that a source is fairly on notice as to the standard it must meet." For example, "alternative equivalent technique" provisions should not be approved without clarification concerning the time period over which equivalency is measured as well as whether the equivalency applies on a per source or per line basis or is facility-wide.

Further, for potential to emit limitations, the standards set must be technically sufficient to provide assurance to EPA and the public that they actually represent a limitation on the potential to emit for the category of sources identified. Any presumption for control efficiency must be technically accurate and the rule must provide the specific parameters as enforceable limits to assure that the control efficiency will be met. For example, rules setting presumptive efficiencies for incineration controls applied to a specific or broad category must state the operating temperature limits or range, the air flow, or any other parameters that may affect the efficiency on which the presumptive efficiency is based. Similarly, material usage limits such as fuel limits, as stated above, require specifying the type of fuel and may require specifying other operating parameters.

A rule that allows sources to submit the specific parameters and associated limits to be monitored may not be enforceable because the rule itself does not set specific technical limits. The submission of these voluntarily accepted limits on parameters or monitoring requirements would need to be federally enforceable. Absent a source-specific permit and appropriate review and public participation of the limits, such a rule is not consistent with the EPA's enforceability principles.

### D. Specific compliance Monitoring

The rule must specify the methods to determine compliance. Specifically, the rule must state the monitoring requirements, record keeping requirements, reporting requirements, and test methods as appropriate for each potential to emit limitation; and clarity which methods are used for making a direct determination of compliance with the potential to emit limitations.

"Monitoring" refers to many different types of data collection, including continuous emission or opacity monitoring, and measurements of various of Parameters of process or control devices (e.g. temperature, pressure drop, fuel usage) and record keeping of parameters that been limited ,such as hours of operation, production levels, or raw material usage. Without a verifiable plantwide, verifiable emission limits must assigned to each unit or group of units subject to the subject to he rule or general permit. Where monitoring cannot be used to determine emissions directly, limits on appropriate operating parameters must be established for the units or source, and must the monitoring must be sufficient to yield data form the relevant time period that is representative of the source's compliance with the standard or limit. Continuous emissions monitoring, especially in the case of smaller sources, is not required.

E. Practicably Enforceable Averaging Times

The averaging time for all limits must be practicably enforceable. In other words, the averaging time period must readily allow for determination of compliance. EPA policy expresses a preference toward short term limits, generally daily but not to exceed one month. However, EPA policy allows for rolling limits not to exceed 12 months or 365 days where the permitting authority finds that the limit provides an assurance that compliance can be readily determined and verified. See June 13, 1989 "Guidance on Limiting Potential to Emit," February 24, 1992 memorandum "Use of Long Term Rolling Averages to Limit Potential to Emit" from John Rasnic to David Kee and March 13 1992 "Policy Determination on Limiting Potential to Emit for Koch Refining Company Clean Fuels Project" from John B. Rasnic to David Kee, stating that determinations to allow an annual rolling average versus a shorter term limit must be made on a case by case basis. Various, factors weigh in favor of allowing a long term rolling average, such as historically unpredictable emissions. Other factors may weigh in favor of shorter term limit, such as the inability to set interim limits during the first year. The permitting agency must make a determination as to what monitoring and averaging period is warranted for the particular source-category in light of how close the allowable emissions would be to the applicability threshold.

F. Clearly Recognized Enforcement

Violations of limits imposed by the rule or general permit that limit potential to emit constitute violations of major source requirements. In other words the source would be violating a "synthetic minor" requirement which may result in the source being treated as a major source under Titles I and V. The 1989 Federal Register Notice provides for separate enforcement

and permitting treatment depending on whether the source subsequently chooses to become a major or remain minor. Thus violations of the rule or general permit or violation of the specific conditions of the rule or general permit subjects the source to potential enforcement under the Clean Air Act and state law. The operating permit rule states that notwithstanding the shield provisions of part 70, the source subject to a general permit may be subject to enforcement action for operating without a part 70 permit if the source is later determined not to qualify or the conditions and terms of the general permit. Moreover, violation of any of the conditions of the rule or general permit may result in a different determination of the source's potential to emit and thus may subject the source to major requirements and to enforcement action for failure to comply with major source requirements from the initial determination.

G. Rule Requirements for State General Permit Programs

As discussed above, general permit programs must be submitted to EPA for approval under SIP authority or under section 112(1), or both, depending on its particular pollutant application. SIP and §112(1) approval and rulemaking procedures must be met, including public notice and comment. The specific application of the enforceability principles for establishing State SIP or §112(1) general permit programs require that the rule establishing the program set out these principles as rule requirements. In other words, these principles must be specific rule requirements to be met by each general permit.

The rule establishing the program must require that (1) general permits apply to a specific and narrow category of sources; (2) sources electing coverage under general permits where coverage is not mandatory, provide notice or reporting to the permitting authority; (3) general permits provide specific and technically accurate (verifiable) limits that restrict the potential to emit; (4) general permits contain specific compliance requirements; (5) Limits in general permits are established based on practicably enforceable averaging times; and (6) violations of the permit are considered violations of the state and federal requirements and result in the source being subject to major source requirements.

In addition, since the rule establishing the program does not provide the specific standards to be met by the source, each general permit, but not each application under each general permit, must be issued pursuant to public and EPA notice and comment. The 1989 Federal Register notice covering enforceability of operating permits requires that SIP operating permit programs issue permits pursuant to public and EPA notice and comment. Title V requires that permits, including general permits, be issued subject to EPA objection.

Finally, sources remain liable for compliance with major source requirements if the specific application of a general permit to the source does not limit the source's potential to emit below major source or major modification thresholds. (The limits provided in these mechanisms may actually limit the potential to emit of sources but may not limit the potential to emit for some sources to below the threshold necessary to avoid major source requirements. For example, a general permit for industrial boilers may in fact provide limits that are sufficient to bring a source with only two or three boilers to below the subject thresholds but a source with more than three boilers may have a limited PTE but not limited below the major source threshold.) Also, where the source is required to use another mechanism to limit potential to emit, i.e., a construction permit, the general permit may not be relied upon by the source or the State, to limit potential to emit.

Permits issued pursuant to the approved program, meeting the above requirements, are adequate to provide federally enforceable limits on potential to emit for New Source Review, title V, and §112 programs as long as they are approved pursuant to SIP (section 110) and section 112(1) authorities.

September 15, 1998

MEMORANDUM

SUBJECT: Periodic Monitoring Guidance for Title V Operating  
Permits Programs

FROM: Eric V. Schaeffer, Director /s/  
Office of Regulatory Enforcement (2241-A)

John S. Seitz, Director /s/  
Office of Air Quality Planning and Standards (MD-10)

TO: Addressees

Attached is the Periodic Monitoring Guidance for the Clean Air Act's title V operating permits programs. Our offices, acting in concert with Region VII, as lead Regional Office, and the Office of General Counsel, developed this guidance to address questions and concerns raised by State and local permitting authorities. The clarifications provided in this guidance should speed permit application development, as well as draft and proposed permit review.

Please share this guidance with permitting authorities and applicants in your jurisdiction. As mentioned in the guidance, specific questions should be directed to Regional title V permitting personnel. This guidance is also available on EPA's TTN web site at [www.epa.gov/ttn/oarpg/tvmain.html](http://www.epa.gov/ttn/oarpg/tvmain.html).

Finally, we want to thank Region VII for its leadership in coordinating Regional views on this topic.

Attachment

Addressees:

Director, Office of Environmental Stewardship, Region I  
Director, Office of Ecosystem Protection, Region I  
Director, Division of Enforcement and Compliance Assurance,  
Region II  
Director, Division of Environmental Planning and Protection,  
Region II  
Director, Air Protection Division, Region III  
Director, Air, Pesticides, and Toxics Management Division,  
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Compliance and Environmental Justice, Region VIII  
Assistant Regional Administrator, Office of Pollution  
Prevention, State, and Tribal Assistance, Region VIII  
Director, Air Division, Region IX  
Director, Office of Air Quality, Region X

cc: T. Curran  
L. Wegman

## **Periodic Monitoring Guidance**



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## I. Introduction

Many State and local permitting authorities have begun issuing title V operating permits. One of the most challenging aspects of this process has been the "periodic monitoring" requirement of the Environmental Protection Agency's (EPA's or Agency's) rules implementing title V, codified at title 40 of the Code of Federal Regulations (40 CFR), part 70. The issues raised have sometimes revealed significantly different interpretations of this requirement among permitting authorities, EPA, and permitted sources. On several occasions, EPA Regions have objected to permits because the periodic monitoring provisions were lacking or inadequate. It is likely that understanding of the technical aspects of implementing periodic monitoring will continue to evolve over time. However, EPA believes this is an appropriate time for issuance of guidance that addresses certain basic principles, necessary for adequate periodic monitoring.

The purpose of this guidance is to clarify certain principles to be applied when implementing the periodic monitoring requirements contained in 40 CFR, sections 70.6(a)(3) and 71.6(a)(3). Section I provides background on why and when periodic monitoring is necessary. Section II offers a description of the periodic monitoring evaluation process and clarifies important concepts like "relevant time period." Sections III and IV describe how periodic monitoring can be made enforceable through the title V permit and what level of documentation should accompany the permit record. Sections V and VI explain EPA's role in the periodic monitoring evaluation process and where the applicant, the permitting authority, or public may find more information about the process. Section VII describes the effect of this guidance.

### A. Periodic Monitoring is Required by the Act and its Implementing Regulations

All title V permits must contain sufficient monitoring, including periodic monitoring, to assure compliance with the applicable requirements in the permit. Section 504 of the Clean Air Act (Act) makes it clear that each title V permit must include "conditions as are necessary to assure compliance with applicable requirements of [the Act], including the requirements of the applicable implementation plan" and "inspection, entry, monitoring, compliance certification, and reporting requirements to assure compliance with the permit terms and conditions." In addition, section 114(a) of the Act requires "enhanced monitoring" at major stationary sources, and authorizes EPA to establish periodic monitoring, record keeping, and reporting requirements at such sources. The regulations at 40 CFR,

sections 70.6(a)(3) and 71.6(a)(3), specifically note that each permit shall contain periodic monitoring sufficient to yield reliable data from the relevant time period that are representative of the source's compliance with the permit where the applicable requirement does not require periodic testing or instrumental or noninstrumental monitoring (which may consist of record keeping designed to serve as monitoring).

It has been and continues to be the Agency's view that sources are under an obligation to comply with permit limits, State implementation plan (SIP) limits, national emissions standards for hazardous air pollutants (NESHAP), and new source performance standards (NSPS) requirements at all times. Consistent with this view of "compliance" and with our stated approach in the compliance assurance monitoring (CAM) rule (40 CFR part 64), we believe that periodic monitoring requirements in title V permits must provide a reasonable assurance of compliance over all anticipated operating conditions.<sup>1</sup>

One of the purposes of the periodic monitoring requirement is to collect and record information that can be used by the source, in conjunction with any other relevant information, to assess that emission point's compliance with applicable requirements. Thus, periodic monitoring requires the actual recording and retention of information related to emissions, not just the displaying of that information at the time it is being generated.

#### B. Why Periodic Monitoring Is Required

The Act, through the title V program and section 114(a), places the responsibility on source owners and operators to have sufficient knowledge of their source operations to certify whether their emission units are in compliance with all

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<sup>1</sup>This guidance interprets sections 70.6(a)(3)'s and 71.6(a)(3)'s requirement that periodic monitoring be sufficient to yield reliable data that are "representative of the source's compliance with the permit" to require the same level of compliance assurance as part 64's requirement that monitoring and monitoring data provide "reasonable assurance of compliance with emission limitations or standards for the anticipated range of operations at a pollutant-specific emissions unit." Both part 70's "representative of compliance" standard and part 64's "reasonable assurance of compliance" standard are reasonable interpretations of the Act, section 504's mandate to include monitoring to "assure compliance" with title V permit terms and conditions. In light of this, this guidance will use the terms "representative of compliance," "reasonable assurance of compliance," and "assure compliance" interchangeably. Moreover, when these terms are used, compliance shall mean continuous compliance.

applicable air pollution control requirements. Periodic monitoring can be used by source operators to quickly identify unusual periods of operation and to take the necessary corrective action. Further, data from periodic monitoring--in conjunction with other required monitoring data and other available information--provide a basis on which a responsible official for a source may certify its compliance status. Data from periodic monitoring are also important to permitting authorities and citizens for the purpose of assessing sources' compliance with applicable requirements.

### C. Where Periodic Monitoring is Required

Periodic monitoring is required for each emission point at a source subject to title V of the Act that is subject to an applicable requirement, such as a Federal regulation or a SIP emission limitation. No emission units at a title V source subject to an applicable requirement, including those subject only to generic applicable requirements, are categorically exempt from the requirement that the permit contain monitoring, compliance certification, and reporting provisions to assure compliance with the permit terms and conditions.

For many emission points at most sources, monitoring already exists in current Federal or State regulations that satisfies the part 70 periodic monitoring requirement. First, all new standards proposed under the authority of section 111 NSPS and section 112 NESHAP after November 15, 1990 are presumed to have adequate monitoring to meet the periodic monitoring requirement for those standards. Second, for emission units at major sources that are subject to Federal or SIP emission limitations, or standards for which the Federal standard specifies a continuous compliance determination method,<sup>2</sup> the existing monitoring used to determine continuous compliance is sufficient to meet the title V monitoring requirements [see 62 FR 54899, 40 CFR section 64.1, and 40 CFR section 64.2(b)(1)(vi)]. Third, for emission units subject to the acid rain requirements pursuant to sections 404, 405, 406, 407(a), 407(b), or 410 of the Act, EPA has determined that these regulations contain sufficient monitoring for the acid rain requirements. Therefore, permits incorporating monitoring in the Federal regulations for units subject to any of the above

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<sup>2</sup>A continuous compliance determination method means a method specified by the applicable standard which: (1) is used to determine compliance with an emission limitation or standard on a continuous basis, consistent with the averaging period established for the emission limitation or standard; and (2) provides data either in units of the standard or correlated directly with the compliance limit.

identified applicable requirements will not need any additional monitoring for these standards.

In addition, on October 22, 1997, EPA promulgated the CAM rule, 40 CFR part 64, which addresses monitoring for certain emission units at major sources. The CAM rule, which applies only to emission units with active control devices whose potential pre-control device emissions are at or above the major source thresholds, requires the title V permit for these sources to contain monitoring sufficient to give a "reasonable assurance of compliance" with applicable standards for the units subject to CAM. Thus, emission units with an approved CAM plan will have sufficient monitoring to satisfy the periodic monitoring requirement under title V and part 70. In other words, although units subject to part 64 are also subject to part 70's periodic monitoring requirement, an adequate CAM plan will also satisfy the periodic monitoring requirements of part 70 for those emission units covered by the CAM plan.

The CAM rule generally will not require implementation of its requirements for most units subject to CAM until the first round of title V permit renewals, which will generally be 5 years after initial permit issuance. Therefore, until emission units become subject to the requirements of part 64, the initial title V permit for major sources with units subject to Federal or SIP regulations will need to include periodic monitoring for these CAM units. The most obvious periodic monitoring for these units in this interim period before permit renewal would be to begin to establish monitoring based on CAM principles as the units' method of complying with part 70's monitoring requirements. These units, however, may also use periodic monitoring that is not based on CAM principles as periodic monitoring, but only until 40 CFR part 64 becomes applicable to the unit and only to the extent that the monitoring reasonably assures compliance.

If an emission unit does not fall within one of the general categories identified in the previous three paragraphs, periodic monitoring is required when the applicable requirement does not require periodic testing or instrumental or noninstrumental monitoring sufficient to yield reliable data from the relevant time period that are representative of the source's compliance with the permit. Clearly, when an applicable requirement imposes a one-time testing requirement, periodic monitoring is not satisfied, and so additional monitoring must be required consistent with sections 70.6(a)(3) or 71.6(a)(3). In addition, additional periodic monitoring may be necessary in cases where some monitoring exists in an applicable requirement, but such monitoring does not provide the necessary assurance of compliance. Further, if an applicable requirement lacks

monitoring or testing, periodic monitoring is not satisfied unless the unit is an insignificant emissions unit (IEU) for which no additional monitoring may be necessary, as discussed in section II.F below.

In light of the general categories above for which periodic monitoring requirements are already satisfied, emission units subject to pre-1990 NSPS and NESHAP regulations and emissions units subject to specific SIP standards or permit terms created under SIP-approved programs should be examined for determining whether the applicable requirement's existing monitoring is sufficient to assure compliance or whether additional monitoring is necessary to satisfy part 70's periodic monitoring requirement.

## II. The Periodic Monitoring Evaluation Process

Periodic monitoring must be adequate to provide a reasonable assurance of compliance with requirements applicable to the source and with all permit terms and conditions over the anticipated range of operation. As described above, periodic monitoring must be evaluated and established as appropriate for each applicable requirement for which the present monitoring is nonexistent or otherwise inadequate. In many cases, this will require a case-by-case, unit-by-unit, pollutant-by-pollutant analysis to devise an adequate monitoring scheme. However, in other cases, it may be appropriate to simply evaluate periodic monitoring for a "like" class of emission units and applicable requirements. Monitoring for "like" situations is described further in section II.F below.

The periodic monitoring process should begin by evaluating whether monitoring, including record keeping, reporting, or periodic testing, applies to the emissions unit in question under existing applicable requirements for that unit. If the already-required monitoring is sufficient to yield reliable data from the relevant time period and is representative of the source's compliance with a particular applicable requirement, then no further monitoring—for that applicable requirement at that emission unit—is required in the permit. If additional monitoring is required, then the permitting authority should consider all of the relevant factors listed below, as well as other factors that may apply on a case-by-case basis, in order to arrive at the appropriate periodic monitoring methodology.

Those factors include:

- The likelihood of violating the applicable requirement (i.e., margin of compliance with the applicable requirement);
- Whether add-on controls are necessary for the unit to meet the emission limit;
- The variability of emissions from the unit over time;
- The type of monitoring, process, maintenance, or control equipment data already available for the emission unit;
- The technical and economic considerations associated with the range of possible monitoring methods; and
- The kind of monitoring found on similar emission units.

While EPA does not plan to specify any particular protocol in implementing periodic monitoring, the preceding factors provide an outline of how to analyze what is appropriate periodic monitoring for an emission unit with a particular applicable standard. The process is informed at each step by the underlying purpose of periodic monitoring, to provide a reasonable assurance of compliance with the applicable requirement for the anticipated range of operations.

In all cases, the rationale for the selected periodic monitoring method must be clear and documented in the permit record. In many cases, the effectiveness of the periodic monitoring technique will be obvious--as in the case of continuous emissions monitoring--and will require little additional documentation in the administrative record. At other times, a technical justification may be necessary in the permit record. Overall, it is important for permitting authorities to properly document the permit record for reference in future title V permitting actions.

Examples of how these and other factors should be considered in the periodic monitoring selection process are described throughout the remainder of the guidance. In particular, Sections II.B through II.F discuss many of the different types of activities that can constitute periodic monitoring for different applicable requirements. The discussion of these different monitoring options should not suggest, however, that there is a hierarchy to deciding what periodic monitoring is appropriate.

A. The Relevant Time Period for Periodic Monitoring

For the purposes of this guidance, "relevant time period" from 40 CFR section 70.6(a)(3) and 40 CFR section 71.6(a)(3) is clarified to mean *"the averaging period of the applicable requirement."* The "relevant time period" is not to be confused with the semi-annual reporting and annual compliance certification cycles also found in parts 70 and 71. For example, the relevant time period for many opacity requirements is 6 minutes. If an applicable requirement measures compliance with an SO<sub>2</sub> emission limit pursuant to a rolling 30-day average, then the relevant time period is a rolling 30-day period. In some cases, the applicable requirement may not expressly state an averaging time. For example, 40 CFR part 60, subpart O limits particulate matter to 0.65 g/kg of dry sludge. However, the standard specifies that Method 5 shall be used and specifies the sampling time and volume for each run. In this example, the relevant time period would be the cumulative sampling time needed to perform the Method 5 test (e.g., 3 hours representing the cumulative sampling time of three 1-hour runs). In some cases the relevant time period is instantaneous. For example, if a work practice standard requires a lid to be free of holes or cracks, a violation exists if the lid has a hole or crack for any amount of time.

However, it is important to note that the duration of periodic monitoring, in many instances, will not match the relevant time period of the applicable requirement. Instead, the duration of the monitoring simply needs to allow the results of the monitoring to relate to, that is, to provide an assurance of compliance during, the relevant time period. In this way, the requirement that periodic monitoring data be from the "relevant time period" is closely related to the requirement that the data be "representative of compliance." Data are "representative of compliance" if they allow for a reasonably supportable conclusion regarding the compliance status during each relevant time period.

For example, suppose that a boiler is subject to an SO<sub>2</sub> limit with a 1-hour averaging time and the source is using a low sulfur oil that would assure compliance with the limit. The periodic monitoring might consist of testing the oil purchased by the source. In this example, although the "relevant time period" is one-hour, it is obvious that neither the sampling nor analysis of the oil must occur for the full hour. Instead, it is clear that the results of an analysis of the sulfur content of a representative oil sample relate to the 1-hour averaging period of the limit for that fuel shipment, provided that the sulfur content is consistent.



Furthermore, periodic monitoring does not require that every "relevant time period" be monitored. Instead, the frequency of the monitoring would be determined during the periodic monitoring evaluation process. Take the example of a flare that is subject to the requirements of 40 CFR section 60.18. The design requirements at section 60.18(c)(1) require that the flare be designed for and operated with no visible emissions except for periods not to exceed a total of 5 minutes during any 2 consecutive hours. Compliance is determined by using Reference Method 22 with an observation period of 2 hours. Performing a Method 22 for every 2-hour period is neither practical nor necessary.

#### B. Use of Existing Continuous Emissions Monitors

Several Federal rules, including certain NSPS and NESHAP subparts and Acid Deposition Control, already require source operators to install, maintain, operate, and quality assure continuous monitoring devices to directly measure emissions. Similarly, many SIPs and construction permits require such devices. Where the source has already installed a continuous emission monitoring system (CEMS), a predictive emission monitoring system (PEMS), or a continuous opacity monitoring system (COMS), such systems will be the periodic monitoring method except in highly unusual circumstances.

For example, most coal fired utility boilers are required to install, operate, maintain, and quality assure SO<sub>2</sub>, NO<sub>x</sub>, and CO<sub>2</sub> flow, and opacity monitoring equipment under the acid rain program. These monitoring systems are to be operated during all periods of operation, including periods of startup, shutdown, and malfunction, and during times when alternative fuels may be combusted. In these cases, the existing monitoring systems are to be specified as the periodic monitoring method for applicable requirements under the SIP and other requirements such as the NSPS. In nearly all cases, data from these monitoring systems provide the fundamental building blocks for determining compliance with different emissions limits and averaging times, at little or no additional cost. Further, since the acid rain program requires these monitoring systems to be operated at all times, including periods of time when the unit is combusting alternative fuels, the monitoring systems provide useful information that the source may use to verify compliance with the standards.

While it may be technically possible to craft different monitoring scenarios for each different operating condition, the permitting authority should strive to minimize confusion where possible. For example, even though opacity and SO<sub>2</sub> emissions

will likely never exceed the corresponding emission limitations when a coal-fired utility unit fires natural gas during periods of startup, shutdown, malfunction, or coal curtailment, data on opacity and SO<sub>2</sub> emissions should still be supplied during those periods using the COMS and SO<sub>2</sub> CEMS. The use of a single, standardized monitoring methodology allows the source, State and local agencies, EPA, and the general public to evaluate one set of compliance data.

C. When Existing Testing or Monitoring is Inadequate

Part 70 requires an evaluation of a permit's applicable requirements to determine whether monitoring in these requirements meets the periodic monitoring criteria and is, therefore, adequate to provide a reasonable assurance of compliance with the applicable requirement over the anticipated range of operations. Whether existing monitoring is adequate, therefore, must be judged according to the periodic monitoring criteria, namely whether the monitoring yields reliable data from the relevant time period that are representative of the source's compliance with the applicable requirement. A different interpretation would lead to the anomalous and unacceptable result that an applicable requirement that lacked monitoring altogether would be supplemented to a greater degree in the title V permit than an applicable requirement with monitoring that is minimal and inadequate.

In general, existing testing or monitoring is inadequate if the data are not reliable, if the data collection frequency is not specified, or if the data collected are not representative of the emission unit's compliance performance. Where the applicable requirement does not contain adequate monitoring, reporting, or record keeping to provide a reasonable assurance of compliance for the anticipated range of operations, periodic monitoring must be added to fulfill the requirements of 40 CFR sections 70.6 and 71.6.

While reference method tests and emission factors all play an important role in the air pollution control program, none of these methods constitutes periodic monitoring unless it provides reliable information at a frequency sufficient to provide a reasonable assurance of compliance with the applicable requirement. For example, a once-a-year stack test is not sufficient to assure compliance with a 3-hour emission limitation unless the source can provide additional parametric data to provide a reasonable assurance of compliance with the standard. Likewise, while AP-42 or other emission factors are helpful for estimating emission levels, they are generally not appropriate for determining compliance with an applicable requirement unless

the factor has either been developed directly from the emission unit in question or substitutes for a proven mass-balance relationship. Further, monthly fuel sampling and analysis also may not be adequate for short-term emission limits where the fuel composition varies. In the event the permitting authority determines that shorter-term monitoring is technically infeasible or cost prohibitive, a less frequent sampling frequency may be established as long as the period is sufficiently representative of the source's compliance with the emission limitations. Otherwise, additional monitoring must be used to show compliance between stack tests.

D. CEMS, PEMS, or COMS Should be Considered When Developing Periodic Monitoring

The permitting authority should give consideration to requiring installation, operation, maintenance, and quality assurance of CEMS, PEMS, or COMS for vents or stacks which carry a major portion of the plant's emissions and have an applicable requirement that the emission unit is likely to exceed. In addition, any other equipment for which an NSPS establishes a CEMS, PEMS, or COMS requirement--whether or not that equipment is subject to the NSPS--should be considered candidates for emission monitors.<sup>3</sup> Note that even where CEMS, PEMS, or COMS are technically and economically feasible, other periodic monitoring may be selected consistent with the relevant factors in section II of this guidance.

E. Use of Parametric Monitoring

Parametric monitoring that provides a reasonable assurance of compliance should be considered for periodic monitoring. The CAM rule should be consulted for guidance on the type of parametric monitoring that might satisfy periodic monitoring.

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<sup>3</sup>For example, through its NSPS program, EPA has already determined that COMS are both technically and economically feasible for a large number of emission units, including industrial, institutional, commercial, and utility steam boilers firing other than natural gas or "clean" fuel oil; fluidized catalytic cracking units; portland cement kilns and clinker coolers; primary metal smelters; ferroalloy and steel arc furnaces; pulp mill recovery furnaces; glass melting furnaces; rotary lime kilns; and phosphate rock and other mineral dryers, calciners, and grinders. Similarly, the NSPS establish SO<sub>2</sub>, NO<sub>x</sub>, H<sub>2</sub>S, and other continuous monitoring requirements for a variety of emission units. The above list is not meant to limit the source types for which monitors may be appropriate, but instead provides examples of the source types for which monitors are known to be both technically and economically feasible.

Information on parameter data that the source is already collecting and that could be used to indicate compliance should be considered.

When using parametric data to satisfy the periodic monitoring requirement, the permit should specify a range which will provide a reasonable assurance that the source is in compliance with the underlying requirement. Wherever possible, the proposed range should be supported by documentation indicating a site-specific developed relationship between parameter indicator ranges and compliance with the emission limit, although it is not required that the range be set such that an excursion from the range will prove noncompliance with the associated limit. Operational data collected during performance testing is a key element in establishing indicator ranges; however, other relevant information in establishing indicator ranges would be engineering assessments, historical data, and vendor data. The permit should also include some means of periodically verifying the continuing validity of the parameter ranges.<sup>4</sup>

For example, the permit may require periodic stack testing to verify direct compliance with the applicable requirement. At the same time, the test data and other engineering information could be used to set the parameter ranges that will be used to determine compliance between tests. The permit should also specify what happens when a parameter exceeds the established range. For example, the permit should specify whether excursion from the established range is considered a violation or whether it will instead trigger corrective action and/or additional monitoring or testing requirements to determine the compliance status of the source. Where documentation of a site-specific developed relationship between parametric monitoring and compliance with the emission limit is not possible because data are lacking and because generation of such data are not feasible prior to issuance of the permit, it may be necessary to include in the permit milestones, including source testing, for

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<sup>4</sup>The discussion of parametric monitoring for compliance purposes in this document is necessarily brief. More complete discussions, including examples and illustrations, of compliance assurance monitoring principles, parametric monitoring designs, and appropriate justifications are available in the CAM rule (40 CFR part 64) and the CAM Technical Guidance Document. Both of these documents as well as other related materials are available electronically through the Emission Measurement Center site on EPA's Technology Transfer Network ([www.epa.gov/ttn/emc](http://www.epa.gov/ttn/emc)). Responses to specific questions about the CAM rule and related material are available through the emission testing information hotline, The Source, at (919) 541-0200.

establishing such relationship. The EPA expects this will only rarely be the case.

F. Other Forms of Periodic Monitoring, Including Record Keeping and Permit Limitations

The Agency recognizes that periodic monitoring may take many forms other than the direct measurement of emissions or parametric monitoring, including record keeping and permit limitations. As stated earlier in this guidance, the conclusion about what is appropriate periodic monitoring should be reached by analyzing all relevant factors in section II of this guidance for each emission unit and each applicable requirement.

The maintenance of records, whether emission calculations, fuel content information, or some other relevant information, may be sufficient periodic monitoring for certain emission units, and applicable requirements. For example, record keeping of required work practices, pollutant content of fuel or raw material, and inspections of design or equipment specifications may satisfy periodic monitoring depending on the applicable requirements and the type of emission units.

As an example, many state rules establish particulate matter limitations based on a process-weight-rate table or formula. In cases where these limits can be met with minimal or no controls, it may be acceptable for the permitting authority to specify record keeping as adequate periodic monitoring because the likelihood that the source will exceed the emission limitation, even while operating at full load, is extremely low. In this case, retaining information on the material inputs to the process would constitute adequate periodic monitoring. Of course, if some level of control is necessary to comply with the standard, then the permit must either specify frequent measurement of particulate matter and/or collection of control equipment parameters to assure proper operation and maintenance of the control device.

Similarly, an enforceable permit limitation may constitute adequate periodic monitoring in the proper circumstances. For example, a permitting authority may conclude that the likelihood of violating an SO<sub>2</sub>, particulate matter, or opacity emission standard for gas combustion units firing pipeline grade natural gas is virtually impossible as long as the unit is properly maintained and burns pipeline grade natural gas. Thus, appropriate periodic monitoring for this situation might consist of maintaining adequate records of fuel type and making the fuel type and the proper maintenance of the unit enforceable conditions of the permit. The EPA believes that there are many

other combinations of requirements, emission units, raw materials and fuels, in addition to the two examples above, where record keeping and/or permit restrictions would satisfy the periodic monitoring requirement.

In situations where a particular class of "like" applicable requirements associated with "like" emission units would all require the identical periodic monitoring (e.g., all natural gas fired boilers needing record keeping to provide a reasonable assurance of compliance with a 20 percent opacity standard), a permitting authority may, after adequate justification, determine the periodic monitoring for that class of units. Of course, if a particular source is found to differ from such a class due to a history of inconsistent operating conditions or difficulties in providing a reasonable assurance of compliance, for example, then class treatment may not be appropriate. Permitting authorities may opt to create a policy or other guidance document explaining the class treatment and rationale for use in all subsequent permitting actions. Any such policy should be made readily available to the public and other interested parties, including EPA.<sup>5</sup>

Although periodic monitoring may consist of record keeping and/or a permit limitation such as a fuel restriction, in no case will EPA accept a periodic monitoring determination based solely on the size, hours of operation, or the past compliance history of the emission unit. Operational and process flexibility, changes in ownership, fuel flexibility, age of unit, and many other factors can adversely influence a source's future compliance status, despite its past good performance. Of course, information on past compliance history is relevant to the likelihood of violating the applicable standard (one of the six factors discussed previously in this guidance) and will help inform the source and permitting agency on the appropriate monitoring to provide a reasonable assurance of compliance.

The EPA also acknowledges that there may be a small class of IEU's for which no additional monitoring may be necessary. While discussing IEU's subject to generally applicable requirements, White Paper Number 2 for Implementation of The Part 70 Operating Permits Program states that where the establishment of a regular program of monitoring would not significantly enhance the ability of the permit to assure compliance with the general applicable requirement, the permitting authority can provide that the status

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<sup>5</sup>Although any such policy will undergo formal review by EPA only when presented in the context of a particular title V permit, advanced coordination with and review by EPA is encouraged.

quo (e.g., no monitoring) will meet the requirements of section 70.6(a)(3)(i). This is based on the belief that IEU's typically are associated with inconsequential environmental impacts and present little potential for violations of generically applicable requirements.

Of course, where a potential for violation of the applicable requirement exists, the permitting authority shall consider adding monitoring requirements. For example, a small coal and natural gas-fired boiler (an IEU in some programs) may need monitoring for opacity while the unit is burning coal to provide a reasonable assurance of compliance with the SIP's opacity limit, while a large turbine that is major for NO<sub>x</sub> and that can only burn pipeline natural gas, may not need monitoring for the SIP's opacity or SO<sub>2</sub> limit. It should be emphasized that whether a reasonable assurance of compliance is achieved without additional monitoring must be judged in the context of a particular emission unit, or as discussed above, a class thereof. That a unit was approved as an "insignificant activity" by EPA relates to the level of detail necessary to be included in a title V permit application and not whether compliance with any applicable requirement is assured without further monitoring. The fact that a unit is an IEU is not, by itself, a justification for no monitoring.

### III. Enforceability of Periodic Monitoring Provisions

Vague or unenforceable monitoring requirements in permits are not sufficient to address the requirement for periodic monitoring. For example, statements in the permit that the source shall prepare a monitoring plan, that testing shall be performed at the request of the permitting authority, or that the permitting authority's inspectors will conduct the periodic monitoring for the source are not sufficient. Responsibility for compliance with the title V permit rests upon the source. Therefore, permit conditions that rely on a permitting agency to conduct periodic monitoring are not enforceable. While permitting authorities may conduct frequent inspections or compliance tests for certain sources as part of the permitting authorities' general compliance program, the source cannot guarantee that this practice will continue in the future, or that it will provide adequate data to assure compliance with all applicable requirements. Additionally, the source is in a better position to detect and correct changes in normal operations before they become violations.

Monitoring methods approved by the permitting authority must result in information that is enforceable as a practical matter. For example, if monitoring and recording the usage of fuel is the

method chosen by the permitting authority for determining compliance with an emission limit, the data must be collected at a frequency so as to allow a presumption of compliance on the part of the source. Permitting authorities can assure such practical enforceability by confirming that the following elements are identified in the title V permit for each monitoring approach where appropriate: the frequency of monitoring, the data averaging period used, the procedures used to check data validity, the minimum period that data must be available, the requirements for record keeping, and the requirements to provide prompt deviation and summary reports.

#### IV. Periodic Monitoring and the Permit Public Record

The periodic monitoring in each permit must be supported by the permit record. Discussion of the decisions the permitting authority makes related to monitoring may appear in the statement that sets forth the legal and factual basis for the draft permit required by section 70.7(a)(5) or may be documented elsewhere in the permit record, including the permit application if the permitting authority finds the periodic monitoring methodologies proposed by the source are adequate. The rationale for periodic monitoring decisions that require substantial explanation should be put in documents other than the formal title V permit. This approach allows inspectors, sources, and other interested readers to focus on the actual requirements of the permit rather than having to evaluate background materials.

#### V. EPA's Role

The EPA in general, and Regional Offices in particular, will continue to provide technical assistance to permitting authorities to assure that adequate monitoring exists in permits. Further, the Regions will continue to evaluate whether the public records for periodic monitoring decisions are complete and technically sound. While EPA respects the role of the permitting authority as the primary implementer of the title V permit program, the Agency has a responsibility to maintain oversight to help ensure consistency in implementing the requirements and to fulfill EPA's role in assuring compliance with applicable requirements of the Act. The Regions should work with permitting authorities to resolve any periodic monitoring deficiencies expeditiously and at an early stage. However, the Regional Offices may object to a permit that is lacking adequate periodic monitoring if no other resolution can be reached prior to the end of EPA's 45-day review period.

While periodic monitoring by nature may be very source specific, the Regional Offices have a responsibility to ensure a



level of broad consistency in how different permitting authorities implement periodic monitoring. Therefore, the Regions will continue to coordinate reviews of periodic monitoring. The EPA expects that understanding of the technical aspects of periodic monitoring will evolve. Accordingly, EPA views consistency as a goal that must be achieved over time.

The EPA's limited resources do not allow it to review all permits or all proposals for periodic monitoring. Given the Agency's constraints in reviewing all proposed permits, EPA will concentrate its efforts on periodic monitoring associated with those emission units that have uncontrolled or pre-control potential emissions equivalent to or in excess of the major source threshold for the pollutant of interest. In addition, EPA will focus on non-major units that utilize control devices, non-major emission units that involve environmental justice concerns, those units that are located in a particular area where non-major emission units significantly impact air quality or have toxic emissions that could impose significant risks to public health, those units for which the public raised significant concern during the comment period, and those units for which the proposed title V permit contains no monitoring.

#### VI. For More Information

Source representatives with specific questions about periodic monitoring should first contact their local or state permitting authority. If appropriate, the permitting authority may then wish to involve the Regional Office in discussions on periodic monitoring. On the whole, permitting authorities should feel free to discuss any periodic monitoring issues with their EPA Regional Office.

Those interested in periodic monitoring developments may also want to periodically visit the various EPA Headquarters and Regional Office web sites for specific details on periodic monitoring. Many regions have been working with their state and local permitting authorities to improve the process and are making objection letters and other guidance and policy documents available to the public through the Internet.

## VII. Effect of This Guidance

While offering specific recommendations, this guidance is not intended to prescribe or prohibit periodic monitoring for specific applicable requirements or emissions sources. The policies set forth in this paper are intended solely as guidance, do not represent final Agency action, and cannot be relied upon to create any rights enforceable by any party. The Agency may choose to issue more detailed, technical guidance in the future. Further, this guidance does not address and in no way affects use of periodic monitoring data under the Credible Evidence Revisions (see 62 FR 8314). Finally, nothing in this guidance is intended to limit EPA's authority and ability to object to periodic monitoring that the Agency determines to be inadequate or otherwise not in compliance with part 70.

BEFORE THE ADMINISTRATOR  
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

IN THE MATTER OF	)	
	)	
	)	PETITION NUMBERS VI-2010-05, VI-
	)	2011-06 AND VI-2012-07
CONSOLIDATED ENVIRONMENTAL	)	
MANAGEMENT, INC. – NUCOR STEEL	)	ORDER RESPONDING TO THE
ST. JAMES PARISH, LOUISIANA	)	JUNE 25, 2010 REQUEST FOR
	)	OBJECTION TO THE ISSUANCE
PIG IRON AND DRI MANUFACTURING	)	OF A TITLE V OPERATING
PERMIT NUMBERS: 2560-00281-V0;	)	PERMIT AND PARTIAL ORDER
2560-00281-V1; AND 3086-V0	)	RESPONDING TO MAY 3, 2011
	)	AND OCTOBER 3, 2012
ISSUED BY THE LOUISIANA DEPARTMENT	)	REQUEST FOR OBJECTION TO THE
OF ENVIRONMENTAL QUALITY	)	ISSUANCE OF TITLE V OPERATING
	)	PERMITS
	)	
	)	
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**ORDER GRANTING IN PART AND DENYING IN PART THREE PETITIONS FOR  
OBJECTION TO PERMITS**

This Order responds to issues that were raised in three related Petitions received by the U.S. Environmental Protection Agency (EPA) from the Louisiana Environmental Action Network (LEAN) and Sierra Club (collectively referred to as “Petitioners”) on June 25, 2010 (the 2010 Petition), May 3, 2011 (the 2011 Petition), and October 3, 2012 (the 2012 Petition) pursuant to Section 505(b)(2) of the Clean Air Act (“CAA” or “Act”), 42 United States Code (U.S.C.) § 7661d(b)(2). The Petitions regard one or more of the following three operating permits issued by the Louisiana Department of Environmental Quality (LDEQ) to Consolidated Environmental Management, Inc. - Nucor Steel Louisiana (Nucor): an operating permit for the pig iron manufacturing process (pig iron process) (permit number 2560-00281-V0), a modification to the operating permit for the pig iron process (permit number 2560-00281-V1), and an operating permit for the direct reduced iron manufacturing process (“DRI process” or “DRI”) (permit number 3086-V0). The Petitions request that the EPA object to each of these permits for a number of reasons outlined below. The source is located in Convent (St. James Parish), Louisiana. These permits are operating permits issued pursuant to title V of the CAA, CAA §§ 501-507, 42 U.S.C. §§ 7661-7661f, the Louisiana Administrative Code (L.A.C.) at 33:III.507, and the EPA’s implementing regulations at 40 Code of Federal Regulations (C.F.R.) Part 70. These operating permits, also referred to as title V permits or part 70 permits, also include provisions from Nucor’s preconstruction review permits, including two Prevention of Significant Deterioration (PSD) permits, and many of the issues raised in the Petitions regard the preconstruction review conditions associated with the facility.

## I. INTRODUCTION

The Petitioners timely filed the June 25, 2010 Petition. The 2010 Petition requested that the Administrator object to the operating permit issued by LDEQ on May 24, 2010, for Nucor's pig iron process (permit number 2560-00281-V0) on the basis that: (1) the permit fails to apply the appropriate maximum achievable control technology (MACT) standards, 2010 Petition at 4-8; (2) the modeling submitted by Nucor to support its PSD analysis is flawed, 2010 Petition at 8-45 and (3) the title V permit fails to incorporate conditions sufficient to assure compliance with PSD, 2010 Petition at 45-67.

The Petitioners timely filed the May 3, 2011, Petition. The 2011 Petition requested that the Administrator object to two operating permits issued by LDEQ on January 27, 2011: a modified operating permit for the pig iron process (the modified pig iron title V permit) (permit number 2560-00281-V1); and a new operating permit for the DRI process (DRI title V permit) (permit number 3086-V0).<sup>1</sup> The Petition identifies the following bases on which the EPA should object: (1) LDEQ failed to aggregate PSD permitting for emissions from the entire facility, 2011 Petition at 5-6; (2) the modified pig iron permit fails to apply MACT standards for the topgas boilers, 2011 Petition at 7-8; (3) LDEQ failed to include emission limits for particulate matter less than 2.5 microns (PM<sub>2.5</sub>), 2011 Petition at 8-10; (4) the limit for natural gas consumption is not the best available control technology (BACT) for greenhouse gas (GHG) emissions from the DRI process, 2011 Petition at 10-17 and (5) the DRI permits must specify procedures estimating GHG emissions, 2011 Petition at 17-19.

The Petitioners timely filed the October 3, 2012, Petition. The 2012 Petition requested in relevant part that the Administrator object to Nucor's pig iron title V permit, the modified pig iron title V permit, and the DRI title V permit for the reasons expressed in the 2010 and 2011 Petitions, which were incorporated by reference and attached as Attachments B and C to the 2012 Petition. 2012 Petition at 1. The 2012 Petition did not provide any additional information, analysis, or argument in support of the claims it re-raised from the 2010 and 2011 Petitions. *See id.* Thus, the EPA's responses to those claims in this order also respond to and resolve those claims as they were re-raised in the 2012 Petition. Accordingly, the responses in today's order address claims in the 2010 and 2011 Petitions, which were re-raised in the 2012 Petition, as well as addressing the corresponding claims in the 2012 Petition, and the EPA's responses below should be understood in this light. Because the 2012 Petition re-raised issues from the earlier petitions by attaching those petitions, the claim numbers and page numbers are the same in the 2012 Petition as in the earlier petitions. For ease of reference and clarity, we refer to the claim numbers and page numbers as originally raised in the 2010 and 2011 Petitions.

Pursuant to a settlement agreement entered into by the EPA and the Petitioners, the EPA agreed to sign an order or orders granting or denying the 2010 Petition and 2011 Petition (except "Specific Objection I"). "Specific Objection I" is the claim that LDEQ failed to aggregate pig

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<sup>1</sup> For the sake of clarity, we adopt the following naming convention for the various title V and PSD permits that have been issued for Nucor and that are discussed in this Order: the "pig iron title V permit" for Permit # 2560-00281-V0; the "pig iron PSD permit" for Permit # PSD-LA-740; the "modified pig iron title V permit" for Permit # 2560-00281-V1; the "DRI title V permit" for Permit # 3086-V0; and the "DRI PSD permit" for Permit # PSD-LA-751.

iron and DRI processes under a single PSD permit, which was the first claim in the 2011 Petition as described above, and which was excepted because, as recognized in the settlement agreement, on June 19, 2013, the EPA issued a partial order denying “Specific Objection I” of the 2011 Petition and as re-raised in the 2012 Petition. Today’s order addresses all the issues originally raised in the 2010 Petition and all the remaining issues originally raised in the 2011 Petition, which were all re-raised in the 2012 Petition. This order does not address the issues originally raised in “Specific Objection I” (which were also re-raised in the 2012 Petition) because those were addressed in the previously issued June 19, 2013 Order. Thus, with the June 19, 2013 Order and today’s order together, the EPA has responded to all of the issues originally raised in the 2010 and the 2011 Petitions, which were re-raised in the 2012 Petition. Similarly, the EPA has now also responded to the 2012 Petition, as that Petition re-raises issues in the 2010 and 2011 Petitions.

Thus, as relevant to this order, the Petitioners asked the EPA to object to the title V permits because they assert that they do not comply with the CAA, the EPA regulations, and the Louisiana state implementation plan (SIP) for six reasons from the above-referenced Petitions:

- (1) the permit fails to apply the appropriate MACT standards, 2010 Petition at 4–8; 2011 Petition at 7–8, 2012 Petition, Att. B at 4-8 and Att. C at 7-8;
- (2) the modeling submitted by Nucor to support its PSD analysis is flawed, 2010 Petition at 8–45, 2012 Petition, Att. B at 8-45;
- (3) the title V permit fails to incorporate conditions sufficient to ensure compliance with PSD, 2010 Petition at 45–67, 2012 Petition, Att. B at 45-67;
- (4) LDEQ failed to include emission limits for PM<sub>2.5</sub>, 2011 Petition at 8–10, 2012 Petition, Att. C at 8-10;
- (5) the limit for natural gas consumption is not BACT for GHG emissions from the DRI process, 2011 Petition at 10–17, 2012 Petition, Att. C at 10-17; and
- (6) the DRI permits must specify procedures estimating GHG emissions, 2011 Petition at 17–19, 2012 Petition, Att. C at 17-19.

Based on a review of the Petitions, and other relevant materials, including the Nucor permits and permit records, and relevant statutory and regulatory authorities, and as explained more fully below, I grant in part and deny in part the Petitions requesting that the EPA object to the Nucor permits. Specifically, I grant or grant in part on issues (1), (3), and (4).

## **II. STATUTORY AND REGULATORY FRAMEWORK**

CAA § 502(d)(1), 42 U.S.C. § 7661a(d)(1), requires each state to develop and submit to the EPA an operating permit program to meet the requirements of title V of the CAA. The State of Louisiana originally submitted its title V program governing the issuance of operating permits in 1993, and the EPA granted full approval on September 12, 1995. 60 *Fed. Reg.* 47296; 40 C.F.R. Part 70, Appendix A. This program, which became effective on October 12, 1995, is codified in L.A.C. Title 33, Part III, Chapter 5.<sup>2</sup>

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<sup>2</sup> Date of signature by the Secretary is November 9, 1993; promulgated in the *Louisiana Register*, Volume 19, Number 11, 1420-1421, Nov. 20, 1993.

All major stationary sources of air pollution and certain other sources are required to apply for title V operating permits that include emission limitations and other conditions as necessary to assure compliance with applicable requirements of the CAA, including the requirements of the applicable SIP. CAA §§ 502(a) and 504(a), 42 U.S.C. §§ 7661a(a) and 7661c(a). The title V operating permit program generally does not impose new substantive air quality control requirements (referred to as “applicable requirements”), but does require permits to contain adequate monitoring, recordkeeping, reporting and other requirements to assure sources’ compliance with applicable requirements. 57 *Fed. Reg.* 32250, 32251 (July 21, 1992). One purpose of the title V program is to “enable the source, States, the EPA, and the public to understand better the requirements to which the source is subject, and whether the source is meeting those requirements.” *Id.* Thus, the title V operating permit program is a vehicle for ensuring that air quality control requirements are appropriately applied to facility emission units and for assuring compliance with such requirements.

Applicable requirements for a new major stationary source or for a major modification to a major stationary source include the requirement to obtain a preconstruction permit that complies with applicable new source review (NSR) requirements. The NSR program is comprised of two core types of preconstruction permit programs for major sources. Part C of Title I of the CAA establishes the PSD program, which applies to areas of the country, such as St. James Parish, Louisiana, that are designated as attainment or unclassifiable for the national ambient air quality standards (NAAQS). CAA §§ 160-169, 42 U.S.C. §§ 7470-7479. Part D of Title I of the Act establishes the nonattainment NSR program, which applies to areas that are designated as nonattainment with the NAAQS. At issue in this order is the PSD part of the NSR program, which requires a major stationary source in an attainment area to obtain a PSD permit before beginning construction of a new facility or undertaking certain modifications. CAA § 165(a)(1), 42 U.S.C. § 7475(a)(1). The analysis under the PSD program must address two primary and fundamental elements (among other requirements) before the permitting authority may issue a permit: (1) an evaluation of the impact of the proposed new or modified major stationary source on ambient air quality in the area, and (2) an analysis ensuring that the proposed facility is subject to BACT for each pollutant subject to regulation under the Act. CAA §§ 165(a)(3), (4), 42 U.S.C. §§ 7475(a)(3), (4); *see also* L.A.C. 33:III.509.

The EPA has two largely identical sets of regulations implementing the PSD program, one set, found at 40 C.F.R. § 51.166, contains the requirements that state PSD programs must meet to be approved as part of a SIP. The other set of regulations, found at 40 C.F.R. § 52.21, contains the EPA’s federal PSD program, which applies in areas without a SIP-approved PSD program. The EPA has approved LDEQ’s PSD SIP. *See* 61 *Fed. Reg.* 53639 (October 15, 1996) and 40 C.F.R. § 52.970(c) (discussing approval of PSD provisions in L.A.C. 33:III.509); *see also* 40 C.F.R. § 52.999(c) and 52.986. As LDEQ administers a SIP-approved PSD program, the applicable requirements of the Act for new major sources or major modifications include the requirement to comply with PSD requirements under the Louisiana SIP. *See, e.g.*, 40 C.F.R. § 70.2.<sup>3</sup> In this

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<sup>3</sup> Under 40 C.F.R. § 70.1(b), “[a]ll sources subject to [the title V regulations] shall have a permit to operate that assures compliance by the source with all applicable requirements.” “Applicable requirements” are defined in 40 C.F.R. § 70.2 to include “(1) [a]ny standard or other requirement provided for in the applicable implementation plan approved or promulgated by EPA through rulemaking under title I of the [Clean Air] Act that implements the relevant requirements of the Act, including any revisions to that plan promulgated in [40 C.F.R.] part 52; (2) [a]ny

case, the “applicable requirements” include Louisiana’s PSD provisions contained in L.A.C. 33:III.509, as approved by the EPA into Louisiana’s SIP.

### **A. Raising PSD Issues in a Petition**

Where a petitioner’s request that the Administrator object to the issuance of a title V permit is based in whole, or in part, on a permitting authority’s alleged failure to comply with the requirements of its approved PSD program (as with other allegations of inconsistency with the Act), the burden is on the petitioners to demonstrate to the Administrator that the permitting decision was not in compliance with the requirements of the Act, including the requirements of the SIP. Such requirements, as the EPA has explained in describing its authority to oversee the implementation of the PSD program in states with approved programs, include the requirements that the permitting authority (1) follow the required procedures in the SIP; (2) make PSD determinations on reasonable grounds properly supported on the record; and (3) describe the determinations in enforceable terms. *See, e.g., In the Matter of Wisconsin Power and Light, Columbia Generating Station*, Order on Petition No. V-2008-01 (October 8, 2009) (*Columbia Generating Order*) at 8.<sup>4</sup>

As the permitting authority for Louisiana’s SIP-approved PSD program, LDEQ has substantial discretion in issuing PSD permits. Given this discretion, in reviewing a PSD permitting decision, the EPA will not substitute its own judgment for that of Louisiana. Rather, consistent with the decision in *Alaska Dep’t of Env’tl Conservation v. EPA*, 540 U.S. 461 (2004), in reviewing a petition to object to a title V permit raising concerns regarding a state’s PSD permitting decision, the EPA generally will look to see whether the petitioner has shown that the state did not comply with its SIP-approved regulations governing PSD permitting or whether the state’s exercise of discretion under such regulations was unreasonable or arbitrary. *See, e.g., In re Louisville Gas and Electric Company*, Order on Petition No. IV-2008-3 (Aug. 12, 2009)(hereafter “*LG&E Order*”); *In re East Kentucky Power Cooperative, Inc. Hugh L. Spurlock Generating Station*, Order on Petition No. IV-2006-4 (Aug. 30, 2007)(hereafter “*Spurlock Order*”); *In re Pacific Coast Building Products, Inc.* (Order on Petition) (Dec. 10, 1999); *In re Roosevelt Regional Landfill Regional Disposal Company* (Order on Petition) (May 4, 1999).

### **B. Review of Issues in a Petition**

State and local permitting authorities issue title V permits pursuant to the EPA-approved title V programs. Under CAA § 505(a), 42 U.S.C. § 766ld(a), and the relevant implementing

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term or condition of any preconstruction permits issued pursuant to regulations approved or promulgated through rulemaking under title I, including parts C or D, of the Act.”

<sup>4</sup> As the EPA has previously explained, in reviewing PSD permit determinations in the context of a petition to object to a title V permit, the standard of review applied by the Environmental Appeals Board (EAB) in reviewing the appeals of federal PSD permits provides a useful analogy. *In the Matter of Louisville Gas and Electric Company*, Order on Petition No. IV-2008-3 (Aug. 12, 2009) at 5 n.6; *see also In the Matter of East Kentucky Power Cooperative, Inc., Hugh L. Spurlock Generating Station*, Order on Petition No. IV-2006-4 (Aug. 30, 2007) at 5. The standard of review applied by the EAB in its review of federal PSD permits is discussed in numerous EAB orders as the “clearly erroneous” standard. *See, e.g., In re Prairie State Generation Company*, 13 E.A.D. 1, 10 (EAB, Aug. 24, 2006)(*Prairie State*); *In re Kawaihae Cogeneration*, 7 E.A.D. 107, 114 (EAB, April 28, 1997). In short, in such appeals, the EAB has explained that the burden is on a petitioner to demonstrate that review is warranted.

regulations found at 40 C.F.R. § 70.8(a), states are required to submit each proposed title V operating permit to the EPA for review. Upon receipt of a proposed permit, the EPA has 45 days to object to final issuance of the permit if the EPA determines that the permit is not in compliance with applicable requirements of the Act. CAA §§ 505(b)(1), 42 U.S.C. § 7661d(b)(1); *see also* 40 C.F.R. § 70.8(c) (providing that the EPA will object if the EPA determines that a permit is not in compliance with applicable requirements or requirements under 40 C.F.R. Part 70). If the EPA does not object to a permit on its own initiative, § 505(b)(2) of the Act and 40 C.F.R. § 70.8(d), provide that any person may petition the Administrator, within 60 days of the expiration of the EPA's 45-day review period, to object to the permit. The petition shall be based only on objections to the permit that were raised with reasonable specificity during the public comment period provided by the permitting agency (unless the petitioner demonstrates in the petition to the Administrator that it was impracticable to raise such objections within such period or unless the grounds for such objection arose after such period). CAA § 505(b)(2), 42 U.S.C. § 7661d(b)(2); 40 C.F.R. § 70.8(d). In response to such a petition, the Act requires the Administrator to issue an objection if a petitioner demonstrates to the Administrator that a permit is not in compliance with the requirements of the Act. CAA § 505(b)(2), 42 U.S.C. § 7661d(b)(2); 40 C.F.R. § 70.8(c)(1); *see also New York Public Interest Research Group, Inc. (NYPIRG) v. Whitman*, 321 F.3d 316, 333 n.11 (2nd Cir. 2003). Under § 505(b)(2) of the Act, the burden is on the petitioner to make the required demonstration to the EPA. *MacClarence v. EPA*, 596 F.3d 1123, 1130-33 (9th Cir. 2010); *Sierra Club v. Johnson*, 541 F.3d 1257, 1266-1267 (11th Cir. 2008); *Citizens Against Ruining the Environment v. EPA*, 535 F.3d 670, 677-78 (7th Cir. 2008); *WildEarth Guardians v. EPA*, 728 F.3d 1075, 1081-1082 (10th Cir. 2013); *Sierra Club v. EPA*, 557 F.3d 401, 406 (6th Cir. 2009) (discussing the burden of proof in title V petitions); *see also NYPIRG*, 321 F.3d at 333 n.11. In evaluating a petitioner's claims, the EPA considers, as appropriate, the adequacy of the permitting authority's rationale in the permitting record, including the response to comments (RTC).

The petitioner's demonstration burden is a critical component of CAA § 505(b)(2). As courts have recognized, CAA § 505(b)(2) contains both a "discretionary component," to determine whether a petition demonstrates to the Administrator that a permit is not in compliance with the requirements of the Act, and a nondiscretionary duty to object where such a demonstration is made. *NYPIRG*, 321 F.3d at 333; *Sierra Club v. Johnson*, 541 F.3d at 1265-66 ("it is undeniable [CAA § 505(b)(2)] also contains a discretionary component: it requires the Administrator to make a judgment of whether a petition demonstrates a permit does not comply with clean air requirements"). Courts have also made clear that the Administrator is only obligated to grant a petition to object under CAA § 505(b)(2) if the Administrator determines that the petitioners have demonstrated that the permit is not in compliance with requirements of the Act. *See, e.g., Citizens Against Ruining the Environment*, 535 F.3d at 667 (§ 505(b)(2) "clearly obligates the Administrator to (1) determine whether the petition demonstrates noncompliance and (2) object if such a demonstration is made") (emphasis added); *NYPIRG*, 321 F.3d at 334 ("§ 505(b)[2] of the CAA provides a step-by-step procedure by which objections to draft permits may be raised and directs the EPA to grant or deny them, *depending on* whether non-compliance has been demonstrated.") (emphasis added); *Sierra Club v. Johnson*, 541 F.3d at 1265 ("Congress's use of the word 'shall' ... plainly mandates an objection *whenever* a petitioner demonstrates noncompliance") (emphasis added). When courts review the EPA's interpretation of the



ambiguous term “demonstrates” and its determination as to whether the demonstration has been made, they have applied a deferential standard of review. *See, e.g., Sierra Club v. Johnson*, 541 F.3d at 1265-66; *Citizens Against Ruining the Environment*, 535 F.3d at 678; *MacClarence*, 596 F.3d at 1130-31. We discuss certain aspects of the petitioner demonstration burden below; however, a fuller discussion can be found in *In the Matter of Consolidated Environmental Management, Inc. – Nucor Steel Louisiana*, Order on Petition Numbers VI-2011-06 and VI-2012-07 (June 19, 2013) (*Nucor II Order*) at 4-7.

The EPA has looked at a number of criteria in determining whether the petitioner has demonstrated noncompliance with the Act. *See generally Nucor II Order* at 7. For example, one such criterion is whether the petitioner has addressed the state or local permitting authority’s decision and reasoning. The EPA expects the petitioner to address the permitting authority’s final decision, and the permitting authority’s final reasoning (including the RTC). *See MacClarence*, 596 F.3d at 1132-33; *see also, e.g., In the Matter of Noranda Alumina, LLC*, Order on Petition No. VI-2011-04 (December 14, 2012) (*Noranda Order*) at 20 (denying title V petition issue where petitioners did not respond to state’s explanation in response to comments or explain why the state erred or the permit was deficient); *In the Matter of Kentucky Syngas, LLC*, Order on Petition No. IV-2010-9 (June 22, 2012) at 41 (*2012 Kentucky Syngas Order*) (denying title V petition issue where petitioners did not acknowledge or reply to state’s response to comments or provide a particularized rationale for why the state erred or the permit was deficient). Another factor the EPA has examined is whether the petitioner has provided the relevant analyses and citations to support its claims. If the petitioner does not, the EPA is left to work out the basis for petitioner’s objection, contrary to Congress’ express allocation of the burden of demonstration to the petitioner in CAA § 505(b)(2). *See MacClarence*, 596 F.3d at 1131 (“the Administrator’s requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”); *In the Matter of Murphy Oil USA, Inc.*, Order on Petition No. VI-2011-02 (Sept. 21, 2011)(hereafter “*Murphy Oil Order*”) at 12 (denying a title V petition claim, where petitioners did not cite any specific applicable requirement that lacked required monitoring). Relatedly, the EPA has pointed out in numerous orders that, in particular cases, general assertions or allegations did not meet the demonstration standard. *See, e.g., In the Matter of Luminant Generation Co. – Sandow 5 Generating Plant*, Order on Petition Number VI-2011-05 (Jan. 15, 2013) at 9; *In the Matter of BP Exploration (Alaska) Inc., Gathering Center #1*, Order on Petition Number VII-2004-02 (Apr. 20, 2007) at 8; *In the Matter of Chevron Products Co., Richmond, Calif. Facility*, Order on Petition No. IX-2004-10 (Mar. 15, 2005) (hereafter “*Chevron Order*”) at 12, 24. Also, if the petitioner did not address a key element of a particular issue, the petition should be denied. *See, e.g., In the Matter of Public Service Company of Colorado, dba Xcel Energy, Pawnee Station*, Order on Petition Number: VIII-2010-XX (June 30, 2011) at 7–10; *See, e.g., In the Matter of Georgia Pacific Consumer Products LP Plant*, Order on Petition No. V-2011-1 at 6-7, 10-11 (July 23, 2012) at 10–11, 13–14.

### **III. BACKGROUND**

#### **A. The Facility**

The Nucor facility is located on an approximately 4,000-acre site on the Mississippi River, in St. James Parish, near Convent, Louisiana, outside of the Baton Rouge Ozone Nonattainment Area.

The facility, as permitted, is composed of two primary manufacturing processes: a pig iron process and a DRI process, both of which produce feedstock for steelmaking. The pig iron process is designed to produce pig iron, while the DRI process is designed to produce sponge iron. The pig iron process was originally permitted (as reflected in the pig iron title V permit) with two blast furnaces (including hot blast stoves and top gas boilers), two coke oven batteries of 140 ovens each (with associated coke charging, pushing and quenching operations), iron ore sintering, furnace slag handling, storage piles, and material handling and transfer operations and haul roads. The capacity of the pig iron process was reduced by approximately half through removal of one blast furnace and associated units, in a subsequent permitting action (the modified pig iron title V permit). As described in the DRI title V permit, issued on the same day as the modified pig iron title V permit, the DRI process consisted of two production lines, each consisting of a natural gas reformer<sup>5</sup> (where reducing gases are produced), a reduction furnace (where reducing gases are passed through the iron ore), package boilers (which produce steam used in emission control systems), and material handling and transfer operations and haul roads. The DRI process differs from the pig iron process in that it does not use blast furnaces, coke ovens, or slag handling operations because the iron ore is reduced in solid form.

## **B. Nucor Permitting History**

Underlying the 2010 and 2011 Petitions are two sets of permits that LDEQ issued to Nucor for the two processes: one set for the pig iron process and the other set for the DRI process. On May 24, 2010, LDEQ separately but concurrently issued the pig iron title V permit and a related pig iron PSD permit. On August 20, 2010, Nucor submitted an application for the new construction of a DRI process to be built on the same site as the pig iron process. On October 13, 2010, Nucor submitted a permit application asking for modification of the May 24, 2010, pig iron title V permit for several reasons. Specifically, Nucor requested that the production capacity be reduced, that certain material handling and haul road activities be transferred over to the DRI process (under development by LDEQ at that time) “in order to allow for construction and operation of the DRI facility to proceed independently of the [pig iron] permit,”<sup>6</sup> and proposed the addition of selective catalytic reduction (SCR) emission controls at several pig iron emission units. On October 28, 2010, Nucor submitted an addendum to the October 13<sup>th</sup> application asking for removal of the coke battery heat recovery steam generator (HRSG) bypass vents that had been permitted for the pig iron process.

On January 27, 2011, the second set of permits was issued by LDEQ, including the modified pig iron title V permit. At the time of permit issuance on January 27, 2011, LDEQ also placed an administrative stay on the modified pig iron title V permit, which stated that it “shall affect the

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<sup>5</sup> Nucor has subsequently modified its title V and PSD permits for the DRI process to replace one of the reformer-based DRI units with a reformer-less DRI unit. See a modified title V permit for the DRI process, Permit No. 3086-V1, issued on March 8, 2012, a second modified title V permit for the DRI process, Permit No. 3086-V2, issued on November 26, 2013, and a modified PSD permit for the DRI process, Permit No. PSD-LA-751(M-1), issued on November 16, 2012.

<sup>6</sup> See LDEQ Electronic Data Management System (EDMS) Document ID 769711 at page 10. This document may be accessed through the EDMS, the LDEQ's electronic repository of official records, available at <http://edms.deq.louisiana.gov/app/doc/querydef.aspx> (use Agency Interest ID “157847” to find the Nucor permitting record generally). Such records may be searched using a variety of search terms including document date, but most directly by using the EDMS assigned document ID number (EDMS Doc. ID).

permit as modified and precludes the commencement of construction as authorized by the permit.” *Stay of Effectiveness of Permit No. 2560-00281-VI*, at 1 (Jan. 27, 2011), EDMS Doc. ID 7806741. The modified pig iron title V permit reduces production capacity and removes the material handling and haul road units that Nucor had requested to be transferred to the DRI process. The modified pig iron permit also requires operation of SCR and removal of HRSG bypass vents at the pig iron process, as Nucor requested in its October 13, 2010, and October 28, 2010, applications. The record for the permit modification stated that LDEQ was not revising the pig iron PSD permit.

The second set of permits consists of title V and PSD permits for the DRI process, which were issued separately but concurrently on January 27, 2011. These permits also include the material handling operations and haul roads that Nucor requested to be transferred from the pig iron process to the DRI process in its permit application of October 13, 2010. Because this permit was issued after GHGs became a regulated pollutant for purposes for PSD, LDEQ included a BACT determination intended to address GHGs in the DRI PSD permit.

The EPA additionally notes that Nucor’s pig iron and DRI permits have subsequently been modified. With respect to the pig iron process, LDEQ issued a modified PSD permit on July 9, 2013 (Permit No. PSD-LA-740(M-1)). With respect to the DRI process, LDEQ issued a modified title V permit on March 8, 2012 (Permit No. 3086-V1) and a subsequent modification on November 26, 2013 (Permit No. 3086-V2), as well as a modified PSD permit on November 16, 2012 (Permit No. PSD-LA-751(M-1)). The Petitions themselves regard three particular title V permits issued for the pig iron and DRI processes (permit numbers 2560-00281-V0, 2560-00281-V1, and 3086-V0).

### **C. Relevant Prior Petition History**

On March 23, 2012, the EPA issued an order granting two other petitions on the Nucor permits from a different petitioner, Zen-Noh Grain Corp (Zen-Noh). *In the Matter of Consolidated Environmental Management, Inc. – Nucor Steel Louisiana*, Order on Petition Nos. VI-2010-02 and VI-2011-03 (Permit Numbers 2560-00281-V0, 3086-V0, and 2560-00281-V1) (Mar. 23, 2012) (*Zen-Noh Order*). One of Zen-Noh’s claims raised in its 2011 petition was that LDEQ’s determination that the PSD air quality analysis need not be conducted on the aggregate emissions from the DRI and pig iron processes was not based on reasonable grounds or properly supported in the record. *See Zen-Noh’s 2011 Petition* at 18. As part of the first ground for granting Zen-Noh’s petitions, the EPA determined that the permit record did not provide an adequate basis to allow the EPA to determine whether the PSD requirement to conduct an ambient air quality impact analysis for the source had been satisfied. *Zen-Noh Order* at 13.<sup>7</sup> The EPA granted the

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<sup>7</sup> The *Zen-Noh Order* has resulted in two separate lawsuits. In one, Zen-Noh brought a lawsuit arguing that the EPA had a nondiscretionary duty to deny the Nucor title V permits. The judge in that case granted the EPA’s motion to dismiss the case on jurisdictional grounds. *See Zen-Noh Grain Corp. v. Jackson*, Order, Doc. No. 35, Civ. Action No. 12-2535 (E.D. La. April 30, 2013). In the other, LDEQ sought judicial review of the *Zen-Noh Order* in the U.S. Court of Appeals for the 5<sup>th</sup> Circuit, and Nucor intervened; the EPA defended the *Zen-Noh Order* in that case and argued that CAA § 505(c) precluded the court from exercising jurisdiction. *See LDEQ v. EPA*, 730 F.3d 446 (5<sup>th</sup> Cir. 2013). On September 13, 2013, the Court issued a decision in the EPA’s favor, dismissing LDEQ’s petition for review for lack of subject matter jurisdiction. *Id.* at 448-50. LDEQ and Nucor are seeking rehearing of that decision and that request remains pending.

Zen-Noh petitions on the basis that “[t]he respective permit records for the pig iron and DRI title V permits, including the responses to comments, fail to provide an adequate basis and rationale for the EPA to determine that these permits assure compliance with applicable requirements and are in compliance with the Act.” *Zen-Noh Order* at 10. In sum, after considering Zen-Noh’s petitions under the standard in CAA § 505(b)(2), the EPA explained that “the decision to grant these petitions is based on two threshold issues”: “(1) LDEQ has not adequately justified its decision to permit the DRI and pig iron processes as two separate projects for purposes of PSD analysis; and (2) LDEQ has not provided permit records from which the full scope of applicable requirements for the pig iron and DRI title V permits can be determined and, in particular, has not adequately explained the basis for its transfer of emissions units between the pig iron and DRI processes via the title V permits, and its incorporation by reference of permit requirements established in a title V permit into a PSD permit.” *Zen-Noh Order* at 10.

On June 21, 2012, LDEQ submitted a response, which it also described as a supplement to the permit record, to the EPA’s *Zen-Noh Order* granting an objection to Nucor’s title V permits. LDEQ’s Response disagreed with the *Zen-Noh Order* on multiple grounds and defended the Nucor permits, arguing that LDEQ satisfied SIP and title V requirements. In its Response, LDEQ also provided some clarification of how it viewed both the permitting approach and the interaction between the title V and PSD permits. *See, e.g.*, Memorandum from Sam L. Phillips, Assistant Secretary, LDEQ, to Jeffrey Robinson, U.S. EPA Region 6, *Re: Order Responding to Petition VI-2010-02 & VI-2010-03 Consolidated Environmental Management, Inc. – Nucor Steel Louisiana*, ( June 21, 2012), EDMS Document ID 8437945 (hereafter “2012 LDEQ Response”) at 6–7, 16–21. For example, LDEQ stated that “LDEQ agrees that the pig iron and DRI manufacturing facilities constitute a single ‘major stationary source.’” *Id.* at 6. LDEQ also explained its view that “the pig iron and DRI project do not have to be addressed in a single PSD permit (i.e. a single physical document).” *Id.* at 7. In support, LDEQ explained that in a situation where “a single site includes more than one process,” LDEQ interprets its regulations to mean that “a single permit may be issued to include all processes at the site” or that “multiple permits may be issued each of which addresses one or more processes at the site.” *Id.* at 7 n. 43. LDEQ’s Response also committed to make certain PSD permit revisions to address the second threshold issue. *See* 2012 LDEQ Response at 18, 20.

In the *Zen-Noh Order*, the EPA explained that it would entertain future petitions from Zen-Noh, LEAN or Sierra Club raising any of the issues in their 2010 and 2011 petitions that they still wished to raise after LDEQ’s Response to that objection, as well as any new claims based on any new proposed permit. *Zen-Noh Order* at 16–17 and n. 9. On September 26, 2012, counsel for the EPA contacted counsel for Zen-Noh to emphasize that the EPA viewed LDEQ’s June 21, 2012 Response to the *Zen Noh Order* as a new proposed title V permit for Nucor, and that the proper course to raise any issues from the 2010 or 2011 Petitions that the Petitioners still wished to raise, or any new claims based on the new proposed permit, would be to submit a title V petition, by October 3, 2012. On the same day, the EPA also contacted counsel for LEAN and Sierra Club to emphasize the EPA’s view on this issue. 2012 Petition, Att. A. On October 3, 2012, as described above, LEAN and Sierra Club filed a new petition, which, among other things, requested that the EPA object to the DRI and pig iron title V permits for the reasons stated in the 2010 and 2011 Petitions.<sup>8</sup>

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<sup>8</sup> The 2012 Petition also disagreed with the EPA’s interpretation that LDEQ’s Response was a new proposed permit

Issues originally raised in the 2010 Petition are discussed in section IV below; issues originally raised in the 2011 Petition are discussed in section V.

#### **IV. EPA DETERMINATIONS ON ISSUES ORIGINALLY RAISED BY THE PETITIONERS ON THE PIG IRON PERMIT IN THE 2010 PETITION**

##### **A. Petitioners' Contention that the Permit Fails to Apply Appropriate MACT Standards**

The Petitioners raise three issues regarding the CAA § 112 MACT standards in the revised pig iron permit: (a) the permit fails to apply a CAA § 112(j) case-by-case MACT standard for the topgas boiler and construction without a CAA § 112(g) case-by-case MACT determination would be illegal, 2010 Petition at 4–7 and 2011 Petition at 7<sup>9</sup>; (b) the revised permit fails to comply with 40 C.F.R Part 63, Subpart L requirements for coal charging operations at coke oven batteries, 2010 Petition at 7; and (c) the permit fails to apply a CAA § 112(j) case-by-case MACT standard for the heat recovery coke ovens and construction without a CAA § 112(g) case-by-case MACT determination would be illegal. *Id.* at 7–8. These claims are discussed in more detail below.

##### **1. The Permit Fails to Include a Case-by-Case MACT Determination for the Topgas Boiler**

*Petitioners' Claims.* The Petitioners claim that Nucor's pig iron title V permits violate CAA § 112 because it does not contain case-by-case MACT standards under CAA § 112(j) for hazardous air pollutants (HAPs) emitted from the facility's topgas boiler. 2010 Petition at 4-5 and 2011 Petition at 7-8. Petitioners also claim that construction without a case-by case MACT determination under CAA § 112(g) would be illegal. *Id.* Petitioners contend that because Nucor is a major source under CAA § 112 and because the EPA's Industrial Boiler MACT Rule was vacated, CAA § 112(g) and CAA § 112(j) requirements apply and further contend that the EPA must object to the pig iron title V permits because LDEQ failed to assure compliance with them.<sup>10</sup> *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 4-5, Att. C at 7-8.

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that had created another title V petition opportunity. *See Nucor II Order* at 14-15 (discussing the EPA's views on this issue).

<sup>9</sup> This issue is raised in a similar fashion in the 2010 and 2011 Petitions, and those issues were re-raised in the 2012 Petition. For the sake of clarity, we address the similar MACT issue from the 2011 Petition in this section, rather than in Section V.

<sup>10</sup> CAA § 112(j) provides generally that major sources in a listed category or subcategory for which the EPA fails to promulgate standards by CAA deadlines must submit permit applications for case-by-case emission limits and that federal or state permit writers must then determine on a case-by-case basis emission limits equivalent to the limitation that would apply if an emission standard had been issued in a timely manner under CAA §§ 112(d) or (h) of the Act. *See* CAA § 112(j)(5), 40 C.F.R. § 63.55(a). Under CAA § 112(g), no person may begin actual construction or reconstruction of a major source of HAP unless the permitting authority determines that new source MACT requirements will be met and such determination shall be made on a case-by-case basis where there is no applicable federal MACT standard in place.

*EPA's Response.* For the reasons provided below, the EPA denies these CAA § 112(j) and 112(g) claims.<sup>11</sup>

The relevant CAA § 112(d) emissions standard for the boilers at issue is now promulgated and in effect. 76 *Fed. Reg.* 15554 (March 21, 2011). Even if Petitioners' claims on these issues were correct, they are now moot. The requested relief would no longer be appropriate. The EPA does not believe it would be appropriate to now require the pig iron permit be revised to reflect requirements or standards under CAA § 112(j) which is no longer applicable. *See Noranda Order* at 22; *Cf. In the Matter of CF&I Steel LP dba EVRAZ Rocky Mountain Steel*, Order on Petition No. VIII-2011-1 (May 31, 2012) at 23 (denying title V petition issue where provisions claimed to be applicable requirements no longer existed). Moreover, the EPA's regulations implementing CAA § 112(j) at 40 C.F.R. § 63.50(c) provide that no further action to develop a case-by-case limit are required after a federal standard has been promulgated. In addition, with a CAA § 112(d) standard in place, there is no requirement to obtain a § 112(g) case-by-case determination prior to construction of the top-gas boiler.<sup>12</sup>

Further, the Petitioners failed to acknowledge or address the LDEQ's RTC which set forth LDEQ's view that the CAA §§ 112(g) and 112(j) do not apply. LDEQ responded to comments raising CAA §§ 112(g) and 112(j) issues in LDEQ's 2010 response to comments document (*Public Comments Response Summary, Part 70 Operating Permit 2560-00281-VO and Prevention of Significant Deterioration Permit PSD-LA-740, May 24, 2010*, EDMS Document ID 2947527 (hereafter the "2010 RTC") at 230-233. In the 2010 RTC, LDEQ stated that the vacatur of the boiler rule raises the issue of whether CAA § 112(j) has been triggered and then summarized two arguments that "proffered" that CAA § 112(j) has not been triggered. Petitioners do not acknowledge or address these arguments in their 2010 petition and Petitioners have failed to demonstrate why LDEQ's rationale is deficient. *See Kentucky Syngas Order* at 41 (denying title V petition issue where Petitioners failed to acknowledge or reply to state's response to comments or provide a particularized rationale for why the state erred or the permit was deficient).<sup>13</sup>

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<sup>11</sup> The EPA notes that the modified pig iron title V permit issued January 27, 2011 did not modify any requirements applicable to the topgas boilers and thus Claim II in the 2011 Petition concerning applicability of CAA §§ 112(g) and 112(j) to the topgas boilers is outside the scope of issues that may be raised in a petition to object to the modified title V permit. *See In the Matter of Wisconsin Public Service Corporation – Weston Generating Station*, Order on Petition Number V-2006-4 (Dec. 19, 2007) (*Weston Order*) at 11-17 ("[I]n evaluating a petition objecting to a significant modification permit, the EPA will determine based on the facts whether the issues raised by the petitioner are directly related to the permit modification action."). This provides a basis to deny the claims as originally raised in the 2011 Petition. *Id.* (denying petition claim that was "not directly related to the permit modification action.") However, we are addressing the substantive CAA §§ 112(g) and 112(j) issues as they were originally raised in the 2010 Petition. This analysis would also apply to the issues raised in the 2011 Petition, although the issues were not properly raised in light of the scope of the permit action.

<sup>12</sup> The EPA has been advised (via electronic mail from Herman Robinson, LDEQ Counsel to Suzanne Murray, Regional Counsel, EPA Region 6, on March 7, 2011, re: clarifying scope of stay) that construction of the pig iron plant, where the topgas boilers would be located, has not commenced. *See also Stay of Effectiveness of Permit No. 2560-00281-V1*, at 1 (Jan. 27, 2011). The EPA believes this stay on such construction is still in effect. *See a* subsequently modified pig iron PSD permit, Permit No. PSD-LA-740 (M-1) (July 9, 2013) ("The stay of effectiveness, which remains in effect, 'precludes the commencement of construction as authorized by the permit.'")

<sup>13</sup> Although the EPA's view is that the CAA §§ 112(g) and (j) claims in the 2011 Petition are outside the scope of issues that can be raised in a Petition on the modified pig iron title V permit issued January 27, 2011, the EPA notes

Notwithstanding the denial of these claims on this issue, we note that following promulgation of the CAA § 112(d) standard, permitting authorities may be required under 40 C.F.R. § 70.7(f)(1)(i) to reopen title V permits to ensure they incorporate newly applicable requirements under CAA § 112(d) of the CAA, 42 U.S.C. § 7412(d). Thus, the appropriate course in this situation is for a permitting authority to consider whether a title V permit must be reopened under title V to incorporate the newly applicable requirements under CAA § 112(d).

For these reasons, the EPA denies these CAA § 112(j) and 112(g) claims.

## **2. Permit Fails to Comply with Charging Requirements for Coke Oven Batteries**

*Petitioners' Claims.* The Petitioners contend that Nucor's Pig Iron Title V Permit fails to comply with the coke oven charging requirements set forth in 40 C.F.R. § 63.303(b)(2).<sup>14</sup> The Petition states that Nucor requested no controls based on its plan to compact the coal and LDEQ issued a permit with a permit shield excusing Nucor from complying with the National Emissions Standard for Hazardous Air Pollutants (NESHAP) for coal charging. 2010 Petition at 7. The Petitioners further urge the EPA to object based on LDEQ's failure to comply with 40 C.F.R. Part 63, Subpart L for the reasons discussed in certain public comments. *Id.*; *id.*, n.16.

These claims were re-raised in the 2012 Petition, Att. B at 7.

In order to clarify the nature of the issue and give context for LDEQ's response, which is summarized below, a summary of comment numbers 154-157 may be helpful. These comments allege that 40 C.F.R. § 63.303(b)(2) requires the facility have some type of emission control for charging operations. 2010 RTC at 120-122. The comments note that "charge" or "charging period" as defined in 40 C.F.R. § 63.301 means "the period of time that commences when coal begins to flow into an oven and ends when the push side door is replaced." The comments note that Nucor relies on two proposed conditions to fulfill the charging requirement of the MACT: (i) baghouse control on the coal brick preparation and (ii) negative pressure on the oven system. The comment asserts that the use of a negative pressure coke oven and compacted coal charging does not replace the need for emission controls during charging. *Id.* at 121. The comments request that Nucor demonstrate how their process will meet either the NESHAP standard for charging operations or some alternative emission standard established in accordance with 40 C.F.R. § 63.6(g). *Id.* at 122.

LDEQ responded to these comments, reasoning that use by the regulation of the word "flow" in the definition of "charge" or "charging period" clearly indicates that the regulators intend the

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that the 2011 Petition does not acknowledge or reply to additional arguments raised in LDEQ's 2011 RTC document and failure to do so would provide an additional basis for the EPA's denial of the claims in the 2011 Petition. For instance, the 2011 Petition does not acknowledge or reply to LDEQ's argument that § 112(j) does not require the permit to contain a CAA § 112(j) limit because 40 C.F.R. § 63.52(b) provides that a CAA § 112(j) limit for this source would not be required until 30 days after startup of the source.

<sup>14</sup> Subpart L, the NESHAP for Coke Oven Batteries at 40 C.F.R. § 63.303(b)(2) provides that "[f]or charging operations, the owner or operator shall install, operate, and maintain an emission control system for the capture and collection of emissions in a manner consistent with good air pollution control practices for minimizing emissions from the charging operation."

regulation to apply to loose coal and that “Nucor’s use of a coal/brick preparation process and the use of the brick on plate transfer system to a negative pressure oven system is itself controlled in accordance with 40 C.F.R. § 63.303(b)(2).” *Id.* at 121. LDEQ further stated that this matter was addressed by means of a permit shield and that 40 C.F.R. § 70.6(f) provides that a permitting authority may expressly include in a part 70 permit a provision stating that compliance with the conditions of the permit shall be deemed compliance with any applicable requirements as of the date of permit issuance. *Id.* LDEQ explained that it determined that the combination of negative pressure ovens and compacted coal charging will satisfy the requirements of 40 C.F.R. § 63.303(b)(2). *Id.* LDEQ added that Nucor will be required to develop a site specific stack test plan to demonstrate compliance. Nucor must demonstrate this equivalency by complying with the particulate matter limitation of 0.0081 pounds per ton of dry coal charged imposed by 40 C.F.R. § 63.303(d)(2). *Id.*

*EPA’s Response.* For the reasons provided below, the EPA grants on these claims.<sup>15</sup>

The permit fails to incorporate the applicable requirement of 40 C.F.R. § 63.303(b)(2) that “[f]or charging operations, the owner or operator shall install, operate, and maintain an emission control system for the capture and collection of emissions in a manner consistent with good air pollution control practices for minimizing emissions from the charging operation.” The lack of a control device is contrary to 40 C.F.R. § 63.303(b)(2) and 40 C.F.R. § 63.313(d)(1) does not allow a state to create an alternative standard through use of a permit shield or otherwise.<sup>16</sup> The EPA does not agree with LDEQ that the term “flow” indicates that 40 C.F.R. § 63.303(b)(2) only applies to loose coal. The use of the term “flow” was not intended to narrow the definition of charging to the filling of a coke oven with loose coal.<sup>17</sup> In addition, compliance with the

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<sup>15</sup> As noted above, the 2010 Petition further urges the EPA to object based on LDEQ’s failure to comply with 40 C.F.R. Part 63, Subpart L for the reasons presented in comments 154–158 in LDEQ’s 2010 RTC and also references comments 169, 198 and 264.G in LDEQ’s 2010 RTC on this point. 2010 Petition at 7 and n.16. The EPA notes that these comments raise issues relating to both MACT and BACT requirements. However, it does not appear that the Petitioners are raising an objection related to the relevant BACT determinations in this claim, since the claim specifically cites these comments with regard to “LDEQ’s failure to comply with 40 C.F.R. Part 63, Subpart L.” *Id.* In addition, the EPA notes that such general references to comments, without any attempt to explain how those comments are relevant to the argument in the Petition and without addressing the state’s final permit decision, including the state’s response to comments, is not sufficient to meet the Petitioners’ demonstration burden. *See MacClarence*, 596 F.3d at 1132-33; *see generally Nucor II Order* at 7 (discussing demonstration burden). LDEQ provided a response to these comments, including responses regarding BACT. 2010 RTC at 120-123; *see also* 2010 RTC at 130, 154-155, 305-306. The Petition does not acknowledge or address LDEQ’s response to these comments, or point to any flaw in LDEQ’s explanation. *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Noranda Order* at 20. Thus, the EPA is not reaching the points contained in those comments relating to the BACT determinations as part of this grant.

<sup>16</sup> 40 C.F.R. § 63.313(d)(1) provides that the authority for approval of alternatives to the requirements in §§ 63.300 and 63.302 through 63.308 (except the authorities in § 63.306(a)(2) and (d)) “cannot be delegated to State, local, or Tribal agencies....” *See also* 40 C.F.R. § 63.91(g)(2)(i) & (ii)(A) (providing that state may not seek delegation of authority to approve § 63.6(g) alternative non-opacity standards), CAA § 112(l)(1) (a delegation shall not include authority to set standards less stringent than those promulgated by the Administrator) and 75 *Fed. Reg.* 19252, 19254-55 (April 14, 2010) (the EPA’s action approving the delegation of the Subpart L NESHAPs to LDEQ was based on a finding that the state program is no less stringent than the federal program and the EPA’s oversight of the delegation includes ensuring that LDEQ does not make decisions that decrease the stringency of the delegated standards.). The EPA notes that Nucor could seek an alternative emission limit for the charging operation pursuant to the procedures in 40 C.F.R. § 63.6(g).

<sup>17</sup> *See 57 Fed. Reg.* 57534, 57542 (Dec. 4, 1992) (in describing the charging emission point, the EPA indicated that



performance tests and procedures set forth at 40 C.F.R. § 63.309(k) contemplate a capture and control device and it is not clear how compliance (stack) tests could be performed without such a device. Further, LDEQ's reliance on its authority to establish a permit shield is not valid. The permit shield provision in CAA § 505(f) and the title V regulations at 40 C.F.R. § 70.6(f) only authorize a permit shield for applicable requirements if the permit "includes the applicable requirements" or the requirements are determined to be "not applicable." As described above, we have determined that the requirements of 40 C.F.R. § 63.303(b)(2) are applicable and the permit does not include them. Furthermore, the permit shield in the pig iron permit specifically refers to terms and conditions that purport to comply with 40 C.F.R. § 63.303(b)(2). Since the EPA has determined that those terms and conditions do not satisfy the requirements of 40 C.F.R. § 63.303(b)(2), the permit shield itself is also invalid and needs to be revised. Thus, the permit must be revised to include the relevant applicable requirement consistent with 40 C.F.R. § 63.303(b)(2) and the permit shield should likewise be revised with the new terms and conditions associated with 40 C.F.R. § 63.303(b)(2).

The EPA additionally notes that in Claim III.B.1 as originally raised in the 2010 Petition, certain assertions are made about the monitoring required to assure compliance for the controls addressed in this claim with respect to certain emission units. 2010 Petition at 52-53. LDEQ explained that the BACT limits for these units was established at 0.0081 pound per ton of dry coal charged, the limit associated with 40 CFR 63, Subpart L – National Emission Standards for Coke Oven Batteries. 2010 RTC, Comment 258.C.1, at 214. In addressing this grant on Claim I.B, LDEQ should also consider whether any revisions are required for these BACT limits and ensure that the permit includes monitoring sufficient to assure compliance with any revised permit terms. *See, supra*, section C.2.

For the reasons provided above, the EPA grants on these claims.

### **3. The Permit Fails to Apply A Case-by-Case MACT Determination for the Heat Recovery Process**

*Petitioners' Claims.* The Petitioners state that there are two types of coke ovens, byproduct and heat recovery, and that although there are national emission standards for byproduct coke ovens (at 40 C.F.R part 63, Subparts L and CCCCC) there are no national emission standards for heat recovery coke ovens. The Petitioners claim that therefore, Nucor, as a major source, is required "to obtain a case-by-case MACT determination from LDEQ" under CAA § 112(j) and that construction of the facility would be illegal under CAA § 112(g). 2010 Petition at 8. The Petitioners assert that Nucor's pig iron permit violates CAA § 112 because it fails to include a case-by-case MACT determination. *Id.* at 7. The Petitioners also incorporate by reference two comments by Sierra Club and Zen-Noh, known as comment 3 and comment 265 in LDEQ's 2010 RTC. *Id.* at 8.<sup>18</sup>

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it considered charging more generally as "when the hot oven is being filled with coal.").

<sup>18</sup> While the Petition does not include any further description or analysis of these comments, for context, the EPA notes that these comments discuss the need to address various toxic pollutants, such as mercury, dioxin and furan, and hydrochloric acid and sulfuric acid. Comment 3 asserts that LDEQ failed to consider the adverse affects of Nucor's mercury emissions and failed to require mercury controls. 2010 RTC at 12-13 (citing *inter alia* 40 C.F.R. 52.21(o) and mercury controls required by Ohio EPA for a coke plant). The EPA notes that the claim discussed in

These claims were re-raised in the 2012 Petition, Att. B at 7-8.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

The Petitioners have not demonstrated that the permit is not in compliance with the requirements of the Act. Petitioners' claim that there is no national emission standard for heat recovery coke ovens is incorrect. Heat recovery coke ovens<sup>19</sup> are a type of "non-recovery coke oven" as defined in 40 C.F.R. § 63.302 and are regulated under Part 63, subparts L<sup>20</sup> (NESHAP for coke oven batteries) and CCCCC<sup>21</sup> (NESHAP for coke ovens). The term "non-recovery" refers to lack of recovery of chemical by-products and a coke oven that recovers heat (but not by-product) is a "non-recovery coke oven." *See* 66 *Fed. Reg.* 35326, 35328 (July 3, 2001) (explaining that non-recovery coke oven process allows for recovery of heat rather than by-products). Because there is a national emission standard in place for heat recovery coke ovens, CAA §§ 112(j) and 112(g) are not applicable.

In addition, the Petitioners' mere incorporation by reference of Comment 3 and Comment 265 into this argument without any attempt to explain how these comments relate to the argument in the Petition and without confronting LDEQ's reasoning supporting the final permit is not sufficient to satisfy the Petitioners' demonstration burden. *See generally Nucor II Order* at 7 (discussing demonstration burden). In particular, LDEQ provided a response to these comments. *See* 2010 RTC at 13-15; 2010 RTC at 309, 311-312, 316. The Petition does not acknowledge or address LDEQ's response to these comments, or point to any flaw in LDEQ's explanation. *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Noranda Order* at 20.

For these reasons, the EPA denies these claims.

## **B. The Modeling Submitted by Nucor to Support its PSD Analysis is Flawed**

The Petitioners raise six issues in regards to the modeling submitted by Nucor for its PSD analysis: (a) Nucor cannot use Significant Impact Levels (SILs) to justify modeled class I PSD increment violations; (b) Nucor's air modeling uses Baton Rouge Airport wind data, which excludes low wind speeds necessary for verifying compliance with the NAAQS and Class II PSD increments; (c) LDEQ improperly exempted Nucor from PSD monitoring requirements; (d) Nucor's finding that class I area particulate matter less than 10 microns (PM<sub>10</sub>) impacts are

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the body of the 2010 Petition does not discuss or cite 40 C.F.R. § 52.21(o), which relates to PSD requirements, but instead raises issues related to requirements under CAA § 112. Thus, the requirements of 40 C.F.R. § 52.21(o) appear to be outside of the scope of the claim specifically raised and discussed by the Petition. Comment 265 generally claims that "Nucor should be required to quantify and implement MACT for all HAP emissions," including hydrogen chloride, mercury, dioxins, and furans, from the non-recovery coke ovens and the HRSG bypass vents. *Id.* at 307-14. The comment also asserts that LDEQ cannot rely on "NESHAP Subpart CCCCC" for the HAP emissions MACT standard because Subpart CCCCC applies to byproduct coke ovens and not to the coke ovens used by Nucor. *Id.* at 308, 310.

<sup>19</sup> A heat recovery coke oven operates under negative pressure to combust coal to produce heat and electricity used in the plant.

<sup>20</sup> 40 C.F.R. Part 63 Subpart L - NESHAP for Coke Oven Batteries.

<sup>21</sup> 40 C.F.R. Part 63 Subpart CCCCC - NESHAP for Coke Ovens: Pushing, Quenching, and Battery Stacks.

insignificant neglects contributions from secondary particulate formation; (e) emissions calculations that Nucor submitted to LDEQ to support Nucor's PSD analysis are unverifiable; and (f) LDEQ could not provide the modeling input files necessary to evaluate Nucor's class I area impact modeling. 2010 Petition at 8–45. These allegations are discussed in more detail immediately below in Sections IV.B.1–IV.B.6 of this Order (Petition Claims II.A–II.F as originally raised in the 2010 Petition).

## **1. Nucor Cannot Use SILs to Justify Modeled Class I PSD Increment Violations**

*Petitioners' Claims.* The Petitioners generally contend that it is inappropriate to use SILs to determine whether emissions from the proposed pig iron process would cause or contribute to a predicted violation of the PSD increments. 2010 Petition at 8-17. The Petitioners state that Nucor's cumulative source modeling identified violations of the Class I increments for the 24-hour and 3-hour sulfur dioxide (SO<sub>2</sub>) standards but that Nucor argued that its impacts on the day and location that it modeled the PSD increments were below the applicable SILs. *Id.* at 8–9.

The Petitioners state that the use of SILs in this manner is inconsistent with the Wyoming Supreme Court decision in *Powder River Basin Resource Council, et al. v. Wyoming Dept. of Env'tl. Quality*, 2010 WY 25 (Wyo. 2010). The Petitioners state that the *Powder River Basin* decision held that SILs can be used only to determine if a cumulative increment analysis is necessary, not to determine whether an air concentration that exceeds increment levels contributes to a violation in the cumulative analysis. 2010 Petition at 9-10. The Petitioners disagree with LDEQ's statement that the Wyoming law at issue in the *Powder River Basin* decision is distinguishable from the Louisiana statute because the Wyoming law requires a demonstration that the “predicted impact” will not cause or contribute to a violation. *Id.* at 11-12. The Petitioners contend that the Louisiana statute also requires a demonstration based on modeled values. *Id.* at 12.

The Petitioners further state that LDEQ's issuance of the permit was inconsistent with the statutory requirements of CAA § 165(a)(3). 2010 Petition at 10-11. The Petitioners interpret § 165(a)(3) to prohibit the construction of a facility that makes “any contribution” to an exceedance of a NAAQS or increment. *Id.* at 11 (“The statute does not state that the contribution must be significant in order for the construction to be prohibited.”). The Petitioners contend that the “EPA has made clear in other circumstances that such a use of SILs is improper.” *Id.* The Petitioners note that in 2002, the EPA commented on a proposed North Dakota SIP revision, stating that it is not appropriate to establish Class I SILs when an increment violation exists and that any impact caused by the facility in such an area would be considered to contribute to that violation. *Id.* (citing Letter from Richard R. Long, EPA, to Terry L. O'Clair, North Dakota Department of Health, Apr. 12, 2002). Therefore, the Petitioners contend that LDEQ cannot label some contributions “not significant.” *Id.* at 11.

The Petitioners contend that the EPA's regulations establishing SILs at 40 C.F.R. § 51.165(b)(2) apply only to NAAQS and not increments. *Id.* at pp. 12-13. The Petitioners state that the regulations also do not allow for the exemption of modeled violations when Nucor's contribution is below the significance threshold at the time and location of each predicted violation. The Petitioners contend that “locality” in § 51.165(b)(2) refers to contributions in a “broader region,

such as the zone of impact or even the air quality control region, not a specific modeled receptor.” *Id.* at 13. The Petitioners therefore contend that while the EPA guidance asserts that project impacts are insignificant only if they exceed the SIL at the same time and location as the identified increment violation, this interpretation is beyond the intent of SILs set forth in the regulation and interferes with the intentions of the CAA. *Id.* at 14-15. The Petitioners explain that air dispersion models are not designed to pinpoint project impacts at specific locations and time periods, and LDEQ’s application of the SILs based on such modeling implies a false level of model accuracy, citing the EPA guideline on Air Quality Models to support the assertion that models cannot be reliably used in this manner. *Id.* at 15 (citing 40 C.F.R. Part 51 Appendix W, *Guidelines on Air Quality Models* (hereafter “Appendix W”), at § 9.2.1). The Petitioners conclude that Nucor could only capture specific time and location violations by using “infinitely more receptors to identify all possible source-to-receptor combinations.” *Id.* at 15-16.

Although conceding that LDEQ did not reference the 1990 Draft New Source Review Workshop Manual (U.S. EPA, New Source Review Workshop Manual, Prevention of Significant Deterioration and Nonattainment Area Permitting (Draft, October 1990) (hereafter “NSR Manual”)) to support the use of SILs in the cumulative increment analysis, the Petitioners state that reliance on the document as a supporting reference is inconsistent with the EPA regulations. *Id.* at 13. The manual may be helpful when it explains how to implement the statute and regulations, but it cannot supersede statutory requirements. *Id.* at 13-14. The Petitioners note that the EPA indicated the manual is not intended to be a final agency action or an official statement of policy, and in the case of a conflict with regulations and policy, those regulations and policies govern. *Id.* (citing NSR Manual at Preface). Moreover, the Petitioners state that the manual is outdated and has not been updated to reflect regulatory changes that have occurred in the last 20 years, such as the development of the PM<sub>10</sub> increments in 1993. *Id.* at 14.

Finally, the Petitioners contend that the NAAQS SILs provide inadequate protection of the increments, which are much smaller values than the respective NAAQS. *Id.* at 16. In particular, the Petitioners state that the use of the NAAQS SILs for the PM<sub>10</sub> increment analysis is unlawful because the 24-hour PM<sub>10</sub> NAAQS is five times the allowable increment, yet LDEQ applied the same SIL value to both. *Id.* While the Petitioners contend that the use of SILs for NAAQS “makes sense,” PSD increments are not protected with regional ambient air monitoring networks and other SIP-planning requirements in the same way as the NAAQS, such that without a full modeling analysis increment violations are never detected or prevented. *Id.* at 17.

These claims were re-raised in the 2012 Petition, Att. B at 8-17.

*EPA’s Response.* For the reasons provided below, the EPA denies these claims.

The EPA does not agree with the Petitioners that the language of either the statute or the regulations prohibit the use of significant impact levels in the increment analysis to determine whether a source contributes to an existing increment violation. As explained in various authorities, the EPA has long interpreted the Act to permit the use of SILs to determine whether a proposed new or modified major source will cause or contribute to a violation of the NAAQS or increment. The Petitioners have failed to demonstrate that the LDEQ acted inconsistent with the statute or regulations in applying this interpretation to the permit.

Section 165(a)(3) of the CAA requires the owner or operator of a major emitting facility, as a condition of obtaining a construction permit, to demonstrate that the facility will not “cause, or contribute to, air pollution in excess of any (A) maximum allowable increase or maximum allowable concentration for any pollutant in any area to which this part applies more than one time per year, (B) national ambient air quality standard in any air quality control region, or (C) any other applicable emission standard or standard of performance under this chapter.” The statute does not define the phrase “cause, or contribute to,” or specify how a facility is to “demonstrate” that it does not cause or contribute to a violation of the NAAQS or increments. Therefore, the statute is ambiguous with respect to the precise questions at issue here. The EPA recently defended this interpretation of the statute in the U.S. Court of Appeals for the D.C. Circuit in *Sierra Club v. EPA*, 705 F.3d 458 (D.C. Cir. 2013). The court declined to rule on the precise question, but in the briefing, the EPA explained that it has long interpreted the phrase “cause, or contribute” to refer to significant or non-*de minimis* emission contributions. See Brief of Respondents at 26-32, 37-44.

In particular, the EPA has long interpreted and continues to interpret this ambiguity in the statute to permit the use of SILs to determine if the impact from a source contributes to an existing violation. For example, in 1980, Richard Rhoads, Director of the EPA’s Control Programs Development Division, issued a memo explaining, “If the proposed source or modification has no significant contribution to the nonattainment problem, then the proposed project does not contribute to this violation.” Memo from R. Rhoads, Director, Control Programs Development Division, to A. Smith, Director, Air & Hazardous Materials Division, Region X, re: Interpretation of “Significant Contribution” (Dec. 16, 1980), at 1.

In 1988, Gerald Emison of the EPA’s Office of Air Quality and Planning Standards (OAQPS) issued a memo to resolve inconsistent practices among the Regions in applying the significance standard to the air quality analysis. Memo from G. Emison, Director, OAQPS, to T. Maslany, Director, Air Management Division, re: *Air Quality Analysis for Prevention of Significant Deterioration (PSD)* (July 5, 1988) (hereafter “Emison Memo”). The memo notes that “[h]istorically, the Environmental Protection Agency’s (EPA’s) position has been that a PSD source will not be considered to cause or contribute to a predicted NAAQS or increment violation if the source’s estimated air quality impact is insignificant (i.e., at or below defined *de minimis* levels).” *Id.* at 1. The Emison Memo notes that one approach used by some Regions was “where a proposed source would automatically be considered to cause or contribute to any modeled violation that would occur within its impact area. . . . The permit would be denied, even if the source’s impact was not significant at the predicted site of the violation during the violation period.” *Id.* The second approach included an “additional step [which] determines whether the emissions from the proposed source will have a significant ambient impact at the point of the modeled NAAQS or increment violation when the violation is predicted to occur. If it can be demonstrated that the proposed source’s impact is not ‘significant’ in a spatial and temporal sense, then the source may receive a PSD permit.” *Id.* at 2. The Emison Memo concludes that the second approach is the most appropriate. *Id.*

The NSR Manual similarly concludes that a source’s impact on a NAAQS or increment violation only contributes to an existing violation where it is significant:

When a violation of any NAAQS or increment is predicted at one or more receptor in the impact area, the applicant can determine whether the net emissions increase from the proposed source will result in a significant ambient impact at the point (receptor) of each predicted violation, and at the time the violation is predicted to occur. The source will not be considered to cause or contribute to the violation if its own impact is not significant at any violating receptor at the time of each predicted violation. In such case, the permitting agency, upon verification of the demonstration, may approve the permit.

*Id.* at C.52.<sup>22</sup>

The EAB issued an order relying on these authorities to uphold this use of SILs in the cumulative NAAQS or increment analysis in *Prairie State*. The EAB concluded that the “cause, or contribute to” language “must mean that some non-zero emission of a NAAQS parameter is permissible, otherwise such a demonstration [that emissions from a proposed facility will not ‘cause, or contribute to’ air pollution in excess of a NAAQS standard] could not be made. Courts have long recognized that the EPA has discretion under the Clean Air Act to exempt from review ‘some emission increases on grounds of de minimis or administrative necessity.’” *Id.* at 104-05 (quoting *Alabama Power Co. v. Costle*, 636 F.2d 323, 400 (D.C. Cir. 1979)).<sup>23</sup>

The Petitioners attempt to distinguish these precedents by citing one isolated document, a 2002 letter sent from the Director of the Air and Radiation Program in Region 8 to the North Dakota Department of Health. In that letter, the Region provided comments on a proposed SIP revision, including a comment expressing the position that SILs should not be used when an increment violation already exists in a Class I area because any impact on a receptor that shows a violation of the “increment would be considered to contribute to that violation.” Letter from R. Long, EPA Region 8, to T. O’Clair, North Dakota Department of Health (April 12, 2002), Attachment at 5-6. It does not appear that this particular document was cited or analyzed in the Petitioners’ underlying comments during the period for public comment in the permitting action. Rather, the Petitioners’ are raising the points in response to statements LDEQ made in the RTC. Thus, LDEQ has not had an opportunity to consider and respond to the Petitioners’ points. Nevertheless, although the 2002 letter indicates that the Region “consulted with our Headquarters offices,” the letter does not necessarily represent the EPA’s interpretation of the Clean Air Act or federal PSD regulations. The Petitioners have not cited to and the EPA is not

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<sup>22</sup> Although conceding that LDEQ did not reference the NSR Manual in its response to comments, the Petitioners contend that the manual cannot be used to justify the use of SILs in the cumulative air quality analysis. As discussed further in this order, the EPA does not agree that the guidance described in the manual conflicts with the requirements of the statute or the regulations. Rather, it is consistent with previous and subsequent guidance provided by the agency as to this issue. Moreover, the fact that the manual does not include increments for PM<sub>10</sub> is irrelevant to the Petitioners’ claim that LDEQ should not have used SILs for the SO<sub>2</sub> increment analysis.

<sup>23</sup> The *Prairie State* order and LDEQ also rely on Appendix W, which states with respect to SO<sub>2</sub> emissions for sources located in areas designated as attainment or unclassifiable that “the demonstration as to whether the source will cause or contribute to an air quality violation should be based on sufficient data to show whether” among other things, “the source *contributes significantly*, in a temporal and spatial sense, to any modeled violation.” Appendix W, § 10.2.3.2.a (emphasis added). Although this language applies specifically to the NAAQS analysis, nothing in Appendix W suggests this same standard could not also be applied to the increment analysis, consistent with existing EPA guidance.

aware that this position has been reiterated in any official guidance or rulemaking in the decade since that letter was drafted.

On the contrary, in 1996, the EPA proposed SILs for SO<sub>2</sub>, PM, and NOs, including Class I SILs to be used to determine whether a source would cause or contribute to a NAAQS or increment violation. 61 *Fed. Reg.* 38250, 38292 (July 23, 1996). Although those values were not finalized at that time, the EPA did finalize Class I increment SILs for PM<sub>2.5</sub> in 2010 for purposes of determining whether a source would cause or contribute to a NAAQS violation. 75 *Fed. Reg.* 64864, 64890-95 (Oct. 20, 2010). In proposing the PM<sub>2.5</sub> SILs, the EPA noted that it was aware that many states had been using the SILs proposed in the 1996 proposal as screening tools. 72 *Fed. Reg.* 54112, 54140 (Sept. 21, 2007). *See also* Memo from J. Calcagni, Director, AQMD, to T. Maslany, Director, ARTD (Sept. 10, 1991) (“EPA does not have a national policy defining air quality significant impact levels for Class I increments. I see no reason, however, why the concept of a significant impact should not also be applied to Class I increments, provided the significant impact levels are determined in a reasonable manner.”). At no point in those rulemakings did the EPA indicate or suggest that the Class I SILs could not be used or approved into a state’s SIP where there was an existing increment violation.<sup>24</sup> Thus, the 2002 letter from Region 8 would appear to be an isolated communication that is inconsistent from both prior and subsequent EPA statements generally supporting the application of SILs in Class I areas.

The Petitioners’ reliance on the language of the EPA’s regulations at § 51.165(b)(2) also does not compel the EPA to object to the permit. The regulations in that section provide:

A major source or major modification will be considered to cause or contribute to a violation of a national ambient air quality standard when such source or modification would, at a minimum, exceed the following significance levels at any locality that does not or would not meet the applicable national standard:  
[Table of values].

40 C.F.R. § 51.165(b)(2). Although the regulation explicitly applies the use of SILs to evaluating

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<sup>24</sup> In the *Sierra Club* litigation mentioned above and in the preamble to the final rule at issue in that case, the EPA acknowledged that there may be circumstances in which an impact from an individual source could cause a NAAQS or increment violation even if the impact falls below the applicable SILs. *See* Brief of Respondents at pp. 32-33; 75 *Fed. Reg.* at 64894 (“[W]e have historically cautioned states that the use of a SIL may not be appropriate when a substantial portion of any NAAQS or increment is known to be consumed.”). The EPA concluded that “notwithstanding the existence of a SIL, permitting authorities should determine when it may be appropriate to conclude that even a *de minimis* impact will ‘cause or contribute’ to an air quality problem and to seek remedial action from the proposed new source or modification.” 75 *Fed. Reg.* at 64892. The Petitioners cite no evidence in the record indicating that the source in this case is *causing* an increment violation despite the fact that its impact is not significant. Rather the question at issue is whether the source is considered to “contribute” to the modeled violation. Subsequent to the court decision in *Sierra Club*, the EPA issued guidance regarding the continued use of the SILs. *See* EPA Office of Air Quality Planning and Standards, Circuit Court Decision on PM<sub>2.5</sub> Significant Impact Levels and, Significant Monitoring Concentration, Questions and Answers (March 4, 2013). The EPA explained that “[a]s part of a cumulative analysis, the applicant may continue to show that the proposed source does not contribute to an existing violation of the PM<sub>2.5</sub> NAAQS by demonstrating that the proposed source’s PM<sub>2.5</sub> impact does not significantly contribute to an existing violation of the PM<sub>2.5</sub> NAAQS.” *Id.* at 3. The permit at issue in this claim was issued well before the *Sierra Club* court decision, but even so, the permit record does not contradict any holdings of that decision or EPA’s subsequent guidance.

a contribution to a NAAQS violation, nothing in the language of the regulation precludes the application of a similar concept to the increment analysis.

The EPA also does not agree that the term “locality” in this regulation precludes the application of the SILs at the time and location of the violation. Nothing in 40 C.F.R. § 51.165(b)(2) or any other regulation precludes states from considering both a geographical and temporal element when using SILs in increment analysis. Such an approach is consistent with the EPA guidance. *See* 1980 Rhoads memo at 1-2 (“if the proposed PSD source can demonstrate that its new emissions would not have a significant impact at the point of the violation when that violation is actually occurring, then the proposed source would meet the requirements of 40 C.F.R. §52.21(k)(1) provided that it would not cause any new violations of the NAAQS”); 1988 Emison Memo at 2 (“If it can be demonstrated that the proposed source's impact is not ‘significant’ in a spatial and temporal sense, then the source may receive a PSD permit.”); NSR Manual at C.52 (“When a violation of any NAAQS or increment is predicted at one or more receptors in the impact area, the applicant can determine whether the net emissions increase from the proposed source will result in a significant ambient impact at the point (receptor) of each predicted violation, and at the time the violation is predicted to occur. The source will not be considered to cause or contribute to the violation if its own impact is not significant at any violating receptor at the time of each predicted violation.”); *Prairie State* at 105 (“With respect to SO<sub>2</sub> emissions, Appendix W states that, for sources located in attainment or unclassifiable areas, ‘the demonstration as to whether the source will cause or contribute to an air quality violation should be based on,’ among other things, ‘the *significance* of the spatial and temporal contribution to any modeled violation.’”) (citing Appendix W § 11.2.3.2(a) (1995)); Appendix W § 10.2.3.2.a (“the demonstration as to whether the source will cause or contribute to an air quality violation should be based on,” among other things, whether “the source contributes significantly, in a temporal and spatial sense, to any modeled violation”). The Petitioners do not clearly explain why these authorities contradict the statute or the regulations. Rather, the Petitioners make conclusory statements that this policy “clearly” contradicts the intentions of both the statute and the regulations. As discussed above, the EPA does not agree that the statute or the regulations so limit the application of SILs in the increment analysis.

Also, the claim about the number of receptors modeled by Nucor does not discuss any of the relevant statutes, regulations or EPA guidance on the number of receptors or geographical extent of receptors required to be used in refined modeling, and thus, is not sufficient to demonstrate an error in the ambient impact assessment performed for Nucor. *See, e.g., MacClarence*, 596 F.3d at 1131 (“the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA section 505(b)(2)). Moreover, the Petitioners’ contention that the number of receptors modeled by Nucor is insufficient does not discuss any of the relevant statutes, regulations or EPA guidance that have a bearing on the number of receptors or geographical extent of receptors required for refined modeling, and thus, is not sufficient to demonstrate an error in the ambient impact assessment performed for Nucor. The claim does not demonstrate an error by LDEQ in the selection of receptors used for the refined PSD modeling performed for Nucor. In particular, Appendix W § 7.2.2 Critical Receptor Sites, provides that “[r]eceptor sites for refined modeling



should be utilized in sufficient detail to estimate the highest concentrations and possible violations of a NAAQS or a PSD increment. In designing a receptor network, the emphasis should be placed on receptor resolution and location, not total number of receptors. The selection of receptor sites should be a case-by-case determination taking into consideration the topography, the climatology, monitor sites, and the results of the initial screening procedure.” The Petitioners did not address any of the source-specific factors identified in Appendix W with respect to the selection of receptors for modeling at the Nucor facility. Thus, LDEQ’s selection of receptors was not demonstrated to be unreasonable for the Nucor facility and the Petitioners have not identified any error in the modeling or the application of the SILs on this basis.

The Petitioners’ reliance on the Wyoming Supreme Court decision in *Powder River Basin* is also misplaced. In that case, the court was evaluating whether Wyoming law permitted the Wyoming Department of Environmental Quality (WDEQ) to use the SILs in the cumulative analysis to determine whether a source (the Dry Fork Station) caused or contributed to modeled exceedances on a nearby Indian reservation classified as a Class I area. 2010 WY 25, P7-39. A state court decision interpreting a state regulation is not binding on either the EPA’s interpretation of its own regulations or another state’s interpretation of its regulations. However, even if the opinion is substantively evaluated, it does not preclude or undermine LDEQ’s use of the SILs in the present permitting action.

The Wyoming court (and the Petitioners here) agreed that SILs may be used in the initial impact analysis to determine whether a cumulative analysis was required, but expressed doubt about whether the SILs could be relied upon in the cumulative analysis to determine that a source’s impact on a modeled exceedance would not cause or contribute to an increment violation. *Id.* at 17-22. The court evaluated the EPA authorities on this issue and acknowledged that many demonstrated the EPA’s support for the use of SILs in the cumulative phase of the air quality analysis. *Id.* at 23-24 (citing, e.g., NSR Manual; *Prairie State*). However, the Court’s holding ultimately turned on the plain language of the Wyoming regulation, which reads: “A permit to construct . . . shall be issued only . . . if the predicted impact . . . is less than the maximum allowable increment . . . .” *Id.* at 27-28. (quoting 6 WAQSR § 4(b)(i)(A)(I)). The court held that the state regulatory language requiring the “predicted impact” be lower than the increment “does not provide authority for the DEQ to treat small exceedances as de minimis and issue the permit anyway.” *Id.* at 29. The court continued:

Given the language of the regulation, we see no room for the DEQ to waive application of the increment through the use of [SILs], and no authority for the DEQ to invoke [SILs] to issue a permit despite modeled exceedances of the increment, no matter how small those exceedances might be or how small the proposed source’s contribution may be. We therefore conclude that the DEQ’s reliance solely on [SILs] is not consistent with the language of the regulations.

*Id.* Thus, the Wyoming court decision did not turn on either the language of the CAA, the federal regulations or EPA guidance. Rather, as LDEQ correctly stated in its response to comment, the *Prairie River Basin* decision turned on a peculiarity in Wyoming law. *See* 2010 RTC at 242.<sup>25</sup>

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<sup>25</sup> Modeling using the maximum allowable emissions indicated that the Dry Fork Station would contribute between 0.0002 and 0.0009 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) to 29 modeled increment exceedances in the Class I area. *Id.*

The Petitioners have not identified a similar peculiarity in Louisiana law or in the federal regulations that prohibits the use of the SILs in the cumulative analysis. In fact, the Louisiana law mimics the federal regulations as to this point, reading:

The owner or operator of the proposed source or modification shall demonstrate that allowable emission increases from the proposed source or modification, in conjunction with all other applicable emission increases or reductions, including secondary emissions, would not cause or contribute to air pollution in violation of: 1. any national ambient air quality standard in any air quality control region; or 2. any applicable maximum allowable increase over the baseline concentration in any area.

*Compare* LAC 33:III.509.K, *with* 40 C.F.R. § 51.166(k)(1). The Petitioners contend that the requirement for LDEQ to “demonstrate” that source emissions will not violate a NAAQS or increment means that the analysis must be based on modeled values and the Louisiana regulation is therefore comparable to the Wyoming regulation. The EPA disagrees that the language of the Louisiana regulation, like the Wyoming regulation, explicitly prohibits the issuance of a permit where the modeled impacts exceed the increment in all cases. Rather, as explained above, the term “contribute” is ambiguous and allows the permitting authority (in this case, LDEQ) and the EPA to determine that certain impacts simply do not contribute to an existing violation. The Petitioners did not cite any other requirement or decision that suggests that LDEQ did not follow the proper procedures under its own SIP-approved regulations, nor did they show that its exercise of discretion under such regulations was unreasonable or arbitrary. *See, e.g., infra* at 5.

Finally, the Petitioners have not demonstrated that LDEQ’s use of the SILs in this case does not adequately protect the Class I increments. The Petitioners generally contend that the SILs are not protective of increments in the same manner as they protect the NAAQS, but they do not address the SILs used by LDEQ in the final increment analysis in this permitting action. In fact, LDEQ did not use the SIL values found at 40 C.F.R. § 51.165(b)(2) for the Class I increment. Rather, according to the final permitting record, LDEQ compared the impact of the Nucor facility to a SIL of 1.0 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) for the 3-hour  $\text{SO}_2$  standard and 0.2  $\mu\text{g}/\text{m}^3$  for the 24-hour standard. *See* pig iron PSD permit at 113. These SIL values represent 4 percent of the respective increments of 25  $\mu\text{g}/\text{m}^3$  and 5  $\mu\text{g}/\text{m}^3$ .

The Petitioners use the example of the  $\text{PM}_{10}$  SILs to support their contention that the SILs generally are not adequately protective of the increments, arguing that the  $\text{PM}_{10}$  SIL represents only 3.3 percent of the 24-hour NAAQS standard as compared to 16.7 percent of the 24-hour increment. This example is irrelevant for several reasons. First, the Petitioners do not appear to be challenging the application of the SILs in the  $\text{PM}_{10}$  increment analysis in this petition. In fact, the final permitting record demonstrates that there were no modeled Class I increment violations

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at 15-16. Using maximum actual emissions, no exceedances were predicted. *Id.* at 14. The Wyoming court ultimately concluded that, while the state could not rely on the SILs in the cumulative air quality analysis, it was entitled to technical deference to determine whether the source would actually have any predicted impact on the modeled exceedances. *Id.* at 34-35. Because the state determined that the predicted exceedances occurred only when modeling the maximum allowable emissions and were exceedingly small, the court held that the state was within its discretion to rely more heavily on the modeling of the maximum actual emissions as providing a better prediction of source impacts. *Id.* at 36.

for the 24-hour PM<sub>10</sub> NAAQS. *See* the pig iron PSD permit, Permit No. See pig iron PSD permit at 113. Second, the fact that the PM<sub>10</sub> SIL may represent a more significant fraction of the PM<sub>10</sub> increment has no bearing on whether the entirely different SIL values used by LDEQ to evaluate the Class I SO<sub>2</sub> increments are sufficiently protective of the increment. As the SIL values used by LDEQ represent 4 percent of the applicable increment, they are more comparable to the PM<sub>10</sub> SILs as compared to the PM<sub>10</sub> NAAQS, where the former represents 3.3 percent of the latter. The Petitioners do not address whether the use of a SIL that represents 4 percent of the increment would be adequately protective of the increments in their view.

The Petitioners contend that increment violations cannot be detected without a full modeling analysis and that the use of SILs in the increment analysis would preclude this preventative measure. The use of SILs in an increment analysis does not necessarily preclude a finding that a source has caused or contributed to a predicted violation of an increment and does not preclude a state from taking appropriate measures to remediate existing violations at any source. However, earlier in the petition, the Petitioners only objected to the use of SILs to determine whether a source's impact contributes to an existing violation identified in the full or cumulative modeling analysis. The Petitioners did not object to the use of the SILs in the preliminary modeling analysis to determine whether a cumulative modeling analysis was required in the first place. In this case, LDEQ and Nucor actually conducted a cumulative modeling analysis. Thus, LDEQ has been made aware of the existing increment violations and can take appropriate measures to remediate the emissions contributing to those violations. The EPA guidance specifically instructs states to address known increment violations in such circumstances. *See* Emison memo at 2 (“[T]he State must also take the appropriate steps to substantiate the NAAQS or increment violation and begin to correct it through the SIP. The EPA Regional Offices’ role in this process should be to establish with the State agency a timetable for further analysis and/or corrective action leading to a SIP revision, where necessary. Additionally, the Regional Office should seriously consider a notice of SIP deficiency, especially if the State does not provide a schedule in a timely manner.”); NSR Manual at C.52 (“However, the agency must also take remedial action through applicable provisions of the state implementation plan to address the predicted violation(s).”).

For these reasons, the EPA denies these claims.

## **2. Nucor’s Air Modeling Uses Baton Rouge Airport Wind Data, Which Excludes Low Wind Speeds Necessary for Verifying Compliance with the NAAQS and Class II PSD Increments.**

*Petitioners’ Claims.* The Petitioners contend that Nucor’s use of five years of wind data from the Baton Rouge Airport in the modeling analysis was inconsistent with the EPA’s *Meteorological Monitoring Guidance for Regulatory Modeling Applications*. 2010 Petition at 17 (citing EPA-454/R-99-005, *Meteorological Monitoring Guidance for Regulatory Modeling Applications* (2000)). The Petitioners assert that the main problem with use of airport wind data is that any wind speed below three knots, or about 1.5 meters per second, is regarded as calm, which means that the effects of wind speeds lower than 1.5 meters per second will be excluded from the modeling analysis. *Id.* at 18. The Petitioners claim that while the airport data label any winds below 1.5 meters per second as calm, the EPA guidance states that wind speed measuring devices should have a starting threshold of 0.5 meters per second or less. *Id.* (citing

*Meteorological Monitoring Guidance for Regulatory Modeling Applications* at 1-1). The Petitioners explain that the worst case conditions for pollution modeling occur at low wind speeds (1.0 meters per second) because modeled impacts are inversely proportional to wind speed, and conclude that using airport data results in severely underestimated modeled impacts. *Id.* The Petitioners note that this concern is relevant for Nucor because its 24-hour PM<sub>10</sub> air modeling results are over 93 percent of the available PSD increment. *Id.* at 19. Furthermore, the Petitioners claim that these low wind speeds occurred during 23 percent of the hours collected from the airport wind data. *Id.* The Petitioners note that between the low wind speeds and another 8 percent of missing hours, the airport wind data only reflect 69 percent of the possible hours for the AERMOD modeling. *Id.* The Petitioners conclude that since the data were missing the “worse-case dispersion conditions,” the airport wind data were inadequate to provide the AERMOD model with the low wind speed hours that must be included for realistically verifying compliance with the NAAQS and PSD increments. *Id.* at 20.

These claims were re-raised in the 2012 Petition, Att. B at 17-20.

*EPA’s Response.* For the reasons provided below, the EPA denies these claims.

In its response to Petitioners’ comment, the LDEQ stated that the EPA-approved AERMOD model was used, following approved LDEQ modeling guidelines, and using 5 years of surface data from LDEQ recommended meteorological stations. LDEQ cited Appendix W § 8.3.4.1.a, to state that

treatment of calm or light and variable wind poses a special problem in model applications since steady-state Gaussian plume models assume that concentration is inversely proportional to wind speed. Furthermore, concentrations may become unrealistically large when wind speeds less than 1m/s are input to the model. Procedures have been developed to prevent the occurrence of overly conservative concentration estimates during periods of calms. These procedures acknowledge that a steady-state Gaussian plume model does not apply during calm conditions, and that our knowledge of wind patterns and plume behavior during these conditions does not, at present, permit the development of a better technique. Therefore, the procedures disregard hours which are identified as calm. The hour is treated as missing and a convention for handling missing hours is recommended.

40 C.F.R. Part 51, Appendix W § 8.3.4.1.a. LDEQ also stated that the use of airport data was based on studies and Louisiana modeling guidance updated and made public in 2006. *See* 2010 RTC at 249-250.

The Petitioners’ analysis in this claim does not address or consider LDEQ’s relevant analysis in the RTC. *See MacClarence*, 596 F.3d at 1132-33; *see also, e.g., Kentucky Syngas Order* at 41 (denying title V petition issue where petitioners did not acknowledge or reply to state’s response to comments or provide a particularized rationale for why the state erred or the permit was deficient). In addition, the Petitioners rely on the EPA’s *Meteorological Monitoring Guidance for Regulatory Modeling Applications Guidance* to support their claim, which generally describes the collection of meteorological data for regulatory modeling applications, but it does not address the use of the meteorological data in AERMOD modeling in particular. The most

appropriate guidance on how to use meteorological data to conduct PSD modeling is found in Appendix W. In particular, the Petitioners did not address or consider Appendix W § 8.3, Meteorological Input Data, which provides “that model input data are normally obtained either from the NWS or as part of a site-specific measurement program” (see *id.* § 8.3.b), and did not cite any provision of Appendix W that invalidates meteorological data used for modeling based on the percentage of wind data that is deemed calm. See, e.g., Appendix W § 8.3.4, Treatment of Near Calms and Calms. While it may be true that using winds less than 1 m/s (calm winds) in AERMOD could hypothetically increase modeled impacts greater than 93 percent of the increment, at the time the analysis was performed (2009) and at the time the PSD permit was issued (May 24, 2010) Appendix W did not require impacts to be calculated using calm wind speeds; thus, the use of this wind data for modeling was consistent with Appendix W. Also, the Petitioners did not show that the wind data were inconsistent with any statutory or regulatory provision, or the EPA-approved modeling protocol, *Dispersion Modeling Protocol, PSD Permit Application for the Proposed Pig Iron Plant, St. James Parish, Louisiana* (Nucor Corp., March 12, 2009), EDMS Document ID 6322690 (hereafter “Modeling Protocol”), or that LDEQ’s approach was unreasonable. See, e.g., *MacClarence*, 596 F.3d at 1131 (“the Administrator’s requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA section 505(b)(2)). Thus, the Petitioners did not demonstrate that LDEQ failed to comply with its SIP-approved regulations governing PSD permitting or that it acted unreasonably in using the Baton Rouge wind data at the time of permitting. See, e.g., *In the Matter of Cash Creek Generation, LLC*, Order on Petition No. IV-2010-4 (June 22, 2012) (hereafter “2012 *Cash Creek Order*”) at 4-5.

For these reasons and for the reasons described in the discussion that follows, the EPA denies these claims.

**a. LDEQ’s Understanding of How AERMOD Treats Calm Hours is Incorrect**

*Petitioners’ Claims.* The Petitioners dispute LDEQ’s claim in response to Comment 129 that AERMOD can model calculated pollutant concentrations during calm wind hours. *Id.* at 20 (quoting what appears to be an earlier version of LDEQ’s response to Comment 129). The Petitioners assert that AERMOD treats calm and missing hours by setting “the concentration values to zero for that hour, and calculat[ing] the short term averages according to the EPA’s calm policy, as set forth in the Guideline.”” *Id.* at 21 (quoting EPA-454/B-03-001, *User’s Guide for the AMS/EPA Regulatory Air Model: AERMOD*, at 3-3 to 3-4 (2004)). The Petitioners then quote the EPA recommendations in § 8.3.4.2 of Appendix W, including: “For annual averages, the sum of all valid hourly concentrations is divided by the number of non-calm hours during the year. AERMOD has been coded to implement these instructions.”” *Id.* (quoting § 8.3.4.2 from the EPA’s Final Rule, *Revision to the Guideline on Air Quality Models: Adoption of a Preferred General Purpose (Flat and Complex Terrain) Dispersion Model and Other Revisions*, 70 Fed. Reg. 68218, 68246 (Nov. 9, 2005)). The Petitioners then state that LDEQ’s misunderstanding of AERMOD has “serious permitting ramifications” since the Baton Rouge Airport data includes “many calm hours.” *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 20-21.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

In its response to Petitioners' comment, the LDEQ stated that the LDEQ response to Comment 129 that the Petition references and quotes was a preliminary draft that was inadvertently made public, that it was an internal draft that had not been finalized or reviewed, and that it was not the final and official response. *See* LDEQ response to comment No. 260.C in the 2010 RTC at 250. Thus, LDEQ asserts that the response of the preliminary draft should not be relied on. *Id.* In addition, in the final response to this comment in the 2010 RTC, LDEQ provides a different explanation for the treatment of calm winds in Gaussian models, such as AERMOD, citing Appendix W. *See* 2010 RTC at 109 (referring to LDEQ response to comment No. 260.C at 249).

The Petitioners do not acknowledge or address that fact that LDEQ removed this statement from the final response to comments; nor do the Petitioners confront the explanation in LDEQ's response to comment that the quoted statement did not represent the final and official response. *See MacClarence*, 596 F.3d at 1132-33; *see also, e.g., Kentucky Syngas Order* at 41 (denying title V petition issue where petitioners did not acknowledge or reply to state's response to comments or provide a particularized rationale for why the state erred or the permit was deficient). In addition, the Petitioners have not demonstrated that the draft, un-reviewed response had any bearing on, or led to any errors in, the modeling analysis performed for the pig iron PSD permit. The Petitioners merely said that LDEQ's misunderstanding has "serious permitting ramifications," but did not identify a specific error or flaw that occurred with respect to the permit or the modeling analyses. This general and unsupported assertion does not meet the Petitioners' demonstration burden. *See, e.g., Kentucky Syngas Order* at 41; *see also Nucor II Order* at 7.

For these reasons, the EPA denies these claims.

**b. Replacing Standard (ASOS) [Automated Surface Observing Stations] Data with True Hourly-Average Winds Will Increase Modeled Impacts**

*Petitioners' Claims.* The Petitioners note that the AERMOD Implementation Workgroup had previously stated that using AERMOD with airport data would likely underestimate modeled impacts because of the high number of calm and missing hours in standard ASOS data, such as Nucor used. *Id.* at 22 (citing AERMOD Implementation Workgroup, *ASOS and Met Data Processing Subgroup, EPA R/S/L Modelers Workshop*, at 3-4 (2009)). The Petitioners state that these concerns are heightened for Nucor, since the 24-hour PM<sub>10</sub> modeled impacts were "very close to the allowable PSD increment." *Id.* at 23. The Petitioners additionally describe a method that the AERMOD Implementation Group has been developing to calculate hourly-averaged winds based on one-minute average ASOS data, which can then be used in AERMOD. *Id.* at 22-23. The Petitioners assert that using such hourly averaged wind data "invariably results in higher modeled concentrations than standard ASOS data" because they contain low wind speeds that are "most culpable for peak impacts." *Id.* at 23.

These claims were re-raised in the 2012 Petition, Att. B at 21-23.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

As noted above, in the 2010 RTC, LDEQ explained that the EPA-approved AERMOD model was used, following an approved LDEQ modeling guidelines and using data from LDEQ recommended meteorological stations, consistent with Appendix W. LDEQ also stated that the use of airport meteorological data was based on studies and Louisiana modeling guidance, updated and made public in 2006. *See* 2010 RTC at 249-250. Despite the Petitioners' claims, the Petition notes that the method to which it refers "is not currently available to the public." 2010 Petition at 23. Thus, it is not clear how such an approach could reasonably be expected to be used for Nucor's analysis. Furthermore, when the pig iron PSD permit was issued, AERMOD was not capable of handling one-minute data to replace low wind speed hours in the standard ASOS meteorological dataset. The tool to handle low wind speeds, a preprocessor to AERMET, called AERMINUTE, that can read 2-minute average ASOS winds (reported every minute) in the National Climatic Data Center (NCDC) DSI-6405 dataset (NCDC, 2006), and calculate hourly average wind speeds and directions, was developed in February 2011. *See* Memorandum from Tyler Fox, Group Leader, Air Quality Modeling Group, U.S. EPA, to Regional Modeling Contacts; *Subject: Use of ASOS meteorological data in AERMOD dispersion Modeling* (March 8, 2013).<sup>26</sup> The tool to handle low wind is currently available, but was under development at the time Nucor submitted its modeling; thus, it would have been unreasonable to expect Nucor to utilize this approach. The modeling was performed using meteorological data consistent with Appendix W and that had been approved for modeling use. Accordingly, the Petitioners did not demonstrate that LDEQ failed to comply with its SIP-approved regulations governing PSD permitting or that it acted unreasonably in not requiring the use of such data at the time of permitting. *See, e.g., 2012 Cash Creek Order* at 4-5.

In addition, the Petitioners did not demonstrate that the meteorological data they claim would have yielded more accurate results for AERMOD modeling was required by any regulatory or statutory provision, or relevant guidance, such as Appendix W, or the EPA-approved Modeling Protocol at the time the analysis was performed or the PSD permit was issued, or that LDEQ's decision to use different data lacked a reasoned basis at that time. *See, e.g., MacClarence*, 596 F.3d at 1131 ("the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive"); *Nucor II Order* at 7 (explaining that EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA section 505(b)(2)).

Finally, the Petitioners do not acknowledge or respond to any of the discussion or analysis in LDEQ's response to comments, and thus have not met their demonstration burden in this claim. *See MacClarence*, 596 F.3d at 1132-33; *see also, e.g., Kentucky Syngas Order* at 41 (denying title V petition issue where petitioners did not acknowledge or reply to state's response to comments or provide a particularized rationale for why the state erred or the permit was deficient).

For these reasons, the EPA denies these claims.

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<sup>26</sup> Available at [http://www.epa.gov/scram001/guidance/clarification/20130308\\_Met\\_Data\\_Clarification.pdf](http://www.epa.gov/scram001/guidance/clarification/20130308_Met_Data_Clarification.pdf).

**c. Replacing Standard ASOS Data with LDEQ's Measured Hourly-Average Winds Will Increase Modeled Impacts**

*Petitioners' Claims.* The Petitioners assert that LDEQ has air monitoring data which can be used in AERMOD and that includes low wind speeds, and they identify an air monitoring site that is designated as a surrogate surface station for modeling impacts in the Nucor region, but assert that Nucor did not use any surface data from this location. 2010 Petition at 24. The Petitioners then state that they performed a 24-hour PM<sub>10</sub> model for the Nucor facility, remodeling Nucor's permit application PM<sub>10</sub> emissions using revised meteorological data based on LDEQ's wind and temperature measures from a combination of sites. *Id.* at 25–26. The Petitioners present their modeled impacts and claim that their results show that “modeling Nucor's emissions with LDEQ surface winds will result in modeled impacts from about 1.77 to 2.96 times higher than the impacts modeled with Baton Rouge Airport wind data.” *Id.* at 29. Based on this modeling, the Petitioners conclude, “[w]ithout exception, [the] AERMOD analyses using LDEQ surface meteorological data show that Nucor's proposed project will substantially violate the 24-hour PM<sub>10</sub> PSD increment of 30 µg/m<sup>3</sup>,” in contrast to Nucor's analyses, which “do not identify any violations of the increments or standards.” *Id.* at 29. The Petitioners assert that LDEQ's permit approval is unacceptable because it used meteorological data that are unsuitable for determining compliance with the NAAQS and PSD increments. *Id.* at 30.

The Petitioners further assert that, in the 2010 RTC, LDEQ relied on the wrong section of Appendix W, § 8.3.4.1.a, and should instead have relied on § 8.3.4.1.b, which explains how AERMOD does not use wind speeds labeled as calm in the model. *Id.* at 30. They also contend that LDEQ's response is against the weight of the evidence, since the AERMOD Implementation Workgroup and the Petitioners' modeling analysis both show that AERMOD will under-predict air impacts when run with airport data. *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 24-31.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

The Petitioners failed to demonstrate that the standard ASOS meteorological data used in Nucor's modeling was inconsistent with any specific statutory or regulatory requirement, such as Appendix W, relevant guidance, or the EPA-approved Modeling Protocol for Nucor. Further, the Petitioners did not identify a requirement for Nucor to use the data that the Petitioners stated should be used in the modeling. *See, e.g., MacClarence*, 596 F.3d at 1131 (“the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”). Instead, Petitioners suggest an alternative modeling methodology, or alternative consideration of certain data, but do not identify the federal requirement making such an interpretation mandatory. *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA section 505(b)(2)).

Furthermore, the issues raised by the Petitioners do not demonstrate that the modeling conducted by Nucor and LDEQ was inconsistent with the modeling tools and guidance available at the time the modeling analysis was submitted or the PSD permit was issued. The fact that Petitioners



contend that different results could be achieved from use of different data sets does not necessarily mean that the approach that Nucor took with respect to the meteorological data, which LDEQ approved, was technically unreasonable at the time the modeling was performed or when the pig iron PSD permit was issued. In fact, the EPA and LDEQ approved the Baton Rouge NWS (ASOS) meteorological data, as part of the Modeling Protocol approval process, as appropriate and representative. *See* Modeling Protocol at 8. The Modeling Protocol was approved on March 20, 2009, by the EPA and March 23, 2009, by LDEQ.<sup>27</sup> Further, Nucor and LDEQ used the Appendix W methodology associated with such data, as described in 40 C.F.R. Part 51, Appendix W § 8.3.4.1. While Petitioners suggest an alternative, they do not show that LDEQ's analysis, or quoting of App. W, § 8.3.4.1.a, in the 2010 RTC, led to a substantive error in the permits or modeling analysis, or that App. W, § 8.3.4.1.b, would have required different data to be used. Thus, the Petitioners did not demonstrate that LDEQ failed to comply with its SIP-approved regulations governing PSD permitting or that it acted unreasonably, under the circumstances at the time, in accepting Nucor's use of the Baton Rouge data. *See, e.g., 2012 Cash Creek Order* at 4-5.

For these reasons, the EPA denies these claims.

### **3. LDEQ Improperly Exempted Nucor from PSD Monitoring Requirements**

*Petitioners' Claims.* The Petitioners claim that LDEQ failed to require Nucor to perform required pre- and post-construction monitoring. 2010 Petition at 31, 37. First, they claim that Nucor failed to meet the requirements of the CAA and the Louisiana PSD regulations because it failed to "gather any pre-construction air monitoring data prior to their permit application." *Id.* at 31. In particular, the Petitioners contend that the CAA requires applicants to gather pre-construction monitoring data in the one year period prior to the date of a permit application, *id.* (quoting 42 U.S.C. § 7475(e)(2)), and contend that although Louisiana's PSD regulations can allow for collection of preconstruction monitoring data for a period shorter than a year, they never allow for a period less than four months. *Id.* at 34 (quoting LAC 33:III.509.M.1.d). The Petitioners further claim that Louisiana's PSD regulations at LAC 33:III.509.M require Nucor to collect preconstruction monitoring data for PM<sub>10</sub> and SO<sub>2</sub> because the permit application indicated that Nucor would exceed the PSD monitoring significance levels set forth in LAC 33:III.509.I for those pollutants. *Id.* at 32-37. The Petitioners claim that because Nucor failed to do such preconstruction monitoring, the PSD application was incomplete and LDEQ's approval of it was improper. *Id.* at 34.

The Petitioners further assert that the availability of existing air quality monitoring data does not exempt Nucor from the pre- and post-construction monitoring requirements in LAC 33:III.509.M. 2010 Petition at 34. The Petitioners also take issue with statements in LDEQ's response to public comments that cited provisions from Appendix W § 8.2, contending that Appendix W does not exempt Nucor from the monitoring requirements. *Id.* at 36-37.

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<sup>27</sup> EDMS Document ID 6332480 is a letter dated March 23, 2009 from LDEQ approving the protocol. The EPA electronic mail conditionally approving the protocol and response from the company are in EDMS Document ID 6439208 at 1-4.

Furthermore, the Petitioners claim that a statement in LDEQ's response to comments that ambient air quality data can be required if representative air quality data are not available misstates the law. *Id.* at 37 (citing 2010 RTC at 106, 250). The Petitioners note that Nucor submitted 2001-2005 LDEQ PM<sub>10</sub> and SO<sub>2</sub> data from East Baton Rouge with the permit application. *Id.* at 34, 37. The Petitioners claim this data falls short of the requirement for ambient air quality monitoring because they contend that LAC 33:III.509.M.1.d is clear that the data must be gathered within the year preceding the permit application, and that the permit application was submitted no earlier than 2008 and because they contend that LDEQ did not have authority to waive that requirement for Nucor. *Id.* at 37.

The Petitioners claim that "[p]ost-construction monitoring for PM<sub>10</sub> and SO<sub>2</sub> is essential" because LDEQ has already permitted numerous sources that are violating the NAAQS and PSD increments, as indicated by existing violations that were identified in Nucor's modeling. *Id.* at 37 (citing LAC 33:III.509.M.2).

Finally, the Petitioners claim that the required pre-application monitoring data was "not available at the April 15, 2010 public hearing for the Nucor project" in violation of the CAA. *Id.* at 31 (quoting 42 U.S.C. § 7475(e)(2)). The Petitioners further claim that the permit included with LDEQ's April 15, 2010 hearing notice was incomplete because it did not address the preconstruction monitoring requirements for PM<sub>10</sub> and SO<sub>2</sub>. *Id.* at 35.

These claims were re-raised in the 2012 Petition, Att. B at 31-38.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

To begin, the Petitioners have not demonstrated that Nucor's use of existing air quality monitoring data in lieu of collecting site-specific preconstruction data violated the applicable PSD requirements. Section 165(e)(2) of the CAA, 42 U.S.C. § 7475(e)(2), provides that the analysis of the ambient air quality at the proposed site and in areas which may be affected by emissions from the facility applying for a PSD permit "shall include continuous air quality monitoring data gathered for purposes of determining whether emissions from such facility will exceed the maximum allowable increases or the maximum allowable concentration permitted under this part" and further provides that "[s]uch data shall be gathered over a period of one calendar year preceding the date of application for a permit under this part unless the State, in accordance with regulations promulgated by the Administrator, determines that a complete and adequate analysis for such purposes may be accomplished in a shorter period." Consistent with the EPA's regulations at 40 C.F.R. § 51.166(m) implementing this statutory provision, Louisiana's SIP-approved PSD regulations provide in relevant part that "the continuous air quality monitoring data that is required shall have been gathered over a period of at least one year and shall represent at least the year preceding receipt of the application, except that if the administrative authority determines that a complete and adequate analysis can be accomplished with monitoring data gathered over a period shorter than one year (but not to be less than four months), the data that is required shall have been gathered over at least that shorter period." LAC 33:III.509.M.1.d. This language mirrors the corresponding provision in the EPA's regulations in the material respects. *See* 40 C.F.R. § 51.166(m)(1)(iv); *cf.* 40 C.F.R. § 52.21(m)(1)(vi).

None of these provisions, however, require every PSD permit applicant to independently collect its own pre-construction monitoring data before submitting the permit application. The EPA has not interpreted CAA § 165(e)(2) or the EPA's implementing regulations to mandate collecting new site-specific preconstruction monitoring data for each permit application, but rather has explained that, where the circumstances warrant, existing representative data from off-site locations or from times other than the year immediately preceding the permit application may be compiled by the permit applicant to satisfy the requirement for monitoring data. *In re Northern Michigan University Ripley Heating Unit*, PSD Appeal No. 08-02, slip op. at 56 (EAB Feb. 18, 2009) ("hereafter "*Northern Michigan Univ.*") at 62; *In re Hawaii Elec. Light Co.*, 8 E.A.D. 66, 97-98 (EAB 1998). The EPA has further explained that this understanding is supported by statements of congressional intent. *Northern Michigan Univ.* at 61-62 ("preconstruction, onsite air quality monitoring may be for less than a year if the basic necessary information can be provided in less time, or it may be waived entirely if the necessary data [are] already available" (quoting H.R. Rep. No. 95-294, at 171 (1977); "one-year monitoring requirement 'may be waived by the [s]tate'" (quoting H.R. Rep. No. 95-564, at 152 (1977) (Conf. Rep.)).

PM<sub>10</sub> and SO<sub>2</sub> are the only specific pollutants for which the Petitioners claim that Nucor was required to collect preconstruction monitoring data. LDEQ's response to comments indicated that representative air quality data may be used where available. *See* 2010 RTC at 106 ("Pre-construction ambient air quality monitoring can be required if the proposed source exceeds the monitoring de minimis concentrations or if representative ambient air quality data are not available.") (cited by 2010 RTC at 254, responding to the Petitioners' comment on this issue). Further, in response to a public comment that LDEQ must require Nucor to perform both pre- and post-construction air monitoring for PM<sub>10</sub> and SO<sub>2</sub>, LDEQ specifically explained that ambient monitoring data was available from monitors located in the area of the facility, that data from these monitors was "representative of air quality conditions at the facility's location," and that "[p]reconstruction monitoring requirements for PM<sub>10</sub> and SO<sub>2</sub> have been met by these monitors." 2010 RTC at 348-349. Thus, LDEQ concluded that it was "unnecessary to require new monitors at the site for these pollutants." *Id.*

The Petitioners do not address information provided in the Modeling Protocol (EDMS Document ID 6333698) or LDEQ's response in the 2010 RTC, explaining that the data Nucor used was representative. Further, Petitioners do not demonstrate that LDEQ's explanation or conclusion lacked a reasoned basis. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments and citing *MacClarence*, 596 F.3d at 1132-34). The Petition quotes a statement made by a commenter during the public comment period asserting that LDEQ had not made a determination that the data used in the air quality modeling was representative of the air quality at the proposed site and that such a determination would have been improper because the 2001-2005 Baton Rouge data was gathered 40 miles from the proposed site and is not representative of the proposed site. 2010 Petition at 35 (quoting Comment 124 and LDEQ's response). However, the Petition's critique of LDEQ's response to this comment does not focus on the representativeness of the data but rather asserts that LDEQ's response "fails to understand how background concentrations relate to the pre- and post-construction monitoring requirements," *id.* at 36, and that LDEQ's interpretations of certain provisions of § 8.2 of Appendix W are mistaken, concluding that "LDEQ cannot interpret Appendix W as exempting Nucor" from the monitoring requirements in LAC

33:III.509.M. *Id.* at 37. The Petition does not provide any rationale for why the data used was not representative, nor does the Petition address other parts of the response to comments and permit record (such as the Modeling Protocol) in which specific information was provided explaining what data was used as well as information supporting the use of such data.

The Petitioners claimed that LDEQ misstated the law in the 2010 RTC by explaining that ambient air quality data can be required if representative air quality data are not available. The Petitioners also challenged the 2001-2005 ambient monitoring data Nucor used by asserting that it does not comply with LAC 33:III.509.M.1.d because it was not gathered in the year prior to the permit application, which they claim was received no earlier than 2008. *See* 2010 Petition at 37; *cf.* pig iron PSD permit at (listing initial pig iron application date as May 12, 2008). The Petitioners have not supported their assertion because the relevant provision requires that the data “*shall represent at least the year preceding receipt of the application,*” LAC 33:III.509.M.1.d (quoted by 2010 Petition at 34) (emphasis added), but does not state that the data must be *gathered* during the year before the permit application. The Petitioners have not shown that the monitoring background data did not represent the year preceding the permit application. In addition, they have not demonstrated that the data failed to comply with the requirements of LAC 33:III.509.M.1.d, nor have they shown that LDEQ’s use of such data was unreasonable. In addition, the EPA notes that use of 2001-2005 data was consistent with the EPA-approved Modeling Protocol for the pig iron PSD permit. *See* Modeling Protocol at 10, 13.<sup>28</sup> The EPA notes that use of representative monitored data gathered over a number of years is in many cases reasonable and appropriate because data gathered only in a single year may be influenced by unusual events or emissions (e.g., exceptional events). Thus, a single year of site specific data are not necessarily more representative of the air quality that would be expected at the proposed site or more protective of air quality than representative data collected from another site or during a different time period. The EPA also notes that for similar reasons, the EPA recommends modeling with 5 years of meteorological data for a representative set of meteorological conditions, if the data are available. *See, e.g.,* Appendix W § 8.3.1.2 (2005). Notably, compliance with many of the NAAQS and PSD increment are based on a multi-year analysis approach in Appendix W (PM<sub>10</sub> increment, 1-hour NO<sub>2</sub>, 1-hour SO<sub>2</sub>), which further supports that evaluation of monitoring data using more than one year of data is within the normal demonstration of monitored compliance and therefore just as informative for background monitoring data.

Accordingly, the Petitioners did not demonstrate that Nucor’s use of existing ambient air quality monitoring data violated the applicable PSD requirements, and did not demonstrate that LDEQ’s acceptance of such data lacked a reasoned basis or failed to assure compliance with applicable requirements of the CAA. *See, e.g., 2012 Cash Creek Order* at 4-5.

The Petitioners have also not demonstrated that Nucor or LDEQ failed to provide information that was required at the time of the public hearing under CAA § 165(e)(2). Section 165(e)(2) states that the “results of such analysis shall be available at the time of the public hearing on the application” for a PSD permit, referring to the analysis of ambient air quality required by CAA § 165(e). The Petition does not assert that any results of such analysis were not available during the public comment period; nor does it assert that data used by Nucor was not available during

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<sup>28</sup> Available at EDMS, Doc. ID 6333698.

the relevant public comment period. 2010 Petition at 31. In fact, the Petition states that the 2001-2005 PM<sub>10</sub> and SO<sub>2</sub> monitoring data was submitted with Nucor's 2009 permit application and provides citations to portions of that application. *Id.* at 34, n.44. That application preceded the 2010 public comment period on Nucor's pig iron permits, which was public noticed on March 10, 2010. *See* pig iron PSD permit at 9; 2010 RTC at 1, n.1. Instead, the Petitioners' claim appears to be that failure to provide new, site-specific pre-construction monitoring data at the April 15, 2010 public hearing violated CAA § 165(e)(2). Because, as explained above, the Petitioners have not demonstrated that Nucor was required to collect new pre-construction monitoring data, they also have not demonstrated that Nucor or LDEQ were required to provide any such data during the public comment period or at the public hearing, nor that failure to do so failed to assure compliance an applicable requirement of the Act.

In the Petition (at 35), Petitioners contend that the April 15, 2010 hearing notice "remains silent on the PSD monitoring requirements for PM<sub>10</sub> and SO<sub>2</sub>. Further, Petitioners state that the permit fails to address the pollutants that did exceed the preconstruction monitoring level. Petition at 35. This information is not completely accurate. The Basis for Decision for the pig iron title V permit provides a lengthy explanation of the modeling analysis surrounding the PSD preconstruction requirements for the relevant pollutants. *See Basis for Decision, Part 70 Operating Permit No.2560-00281-VO and Prevention of Significant Deterioration Permit No. PSD-LA-740*, EDMS Document ID 2947527 (May 24, 2010)(hereafter "2010 Basis for Decision") at 17-22. The Petitioners do not explain how information in the permit is inconsistent with the outcome of the analysis described in the 2010 Basis for Decision. Additionally, the Petitioners do not identify or analyze any regulatory or statutory provision, or any guidance document, to support their contention that the permit must "address" certain pollutants that exceeded preconstruction monitoring levels (Petition at 35). *See, e.g., MacClarence*, 596 F.3d at 1131 ("the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive"); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA § 505(b)(2)). Nor do Petitioners explain why the lengthy analysis provided in the 2010 Basis for Decision is flawed or otherwise inconsistent with applicable requirements. Therefore, it is not clear whether this discussion is intended to raise additional deficiencies in the permit or public process (and if so, what the basis for those alleged deficiencies would be), or whether this discussion is simply intended as an extension of the Petitioners' points discussed above. As indicated above, preconstruction monitoring requirements were addressed during the permitting process, including in the EPA-approved Modeling Protocol, the 2010 Basis for Decision, the public comments, and in LDEQ's responses to those comments. *See* Modeling Protocol at 13; 2010 RTC at 348-349, 253-254. The Petitioners have not explained why the permit would have needed to include additional discussion of preconstruction requirements in order to be complete. Nor have the Petitioners provided any explanation of how the failure to include this information in that permit deprived them of a meaningful opportunity for participation in the permit proceedings, including how the alleged flaw resulted in, or may have resulted in, a deficiency in the contents of the permit. *See Noranda Order* at 11; *2012 Cash Creek Order* at 9. Further, the fact that commenters, including the Petitioners, were able to offer comments on LDEQ's approach to preconstruction monitoring, including for PM<sub>10</sub> and SO<sub>2</sub>, suggests that relevant information was available during the public comment period.

Finally, we consider the Petitioners' assertion that post-construction monitoring under LAC 33:III.509.M.2 is essential for PM<sub>10</sub> and SO<sub>2</sub> because of existing violations of the NAAQS and PSD increments. With respect to post-construction monitoring, the cited provision of Louisiana's PSD rules states that: "The owner or operator of a major stationary source or major modification shall, after construction of the stationary source or modification, conduct such ambient monitoring *as the administrative authority determines is necessary* to determine the effect emissions from the stationary source or modification may have, or are having, on air quality in any area." LAC 33:III.509.M.2 (emphasis added). *See also* 40 C.F.R. § 51.166(m)(2). This language provides LDEQ substantial discretion concerning whether to require post-construction monitoring for a particular source, including the discretion to require no post-construction ambient air monitoring at all. *See In the Matter of Pacific Coast Bldg. Prod. Inc.* (Order on Petition) (Dec. 10, 1999) at 9-10 (denying a title V petition claim based on similar language in an approved SIP and explaining that such language gave the permitting authority "full authority to require no ambient air monitoring as long as that determination was made in a manner that was not arbitrary, capricious or otherwise unlawful."). The Petitioners have neither asserted nor demonstrated that LDEQ acted arbitrarily, capriciously, or otherwise not in accordance with law in not requiring Nucor to conduct post-construction monitoring for PM<sub>10</sub> and SO<sub>2</sub> emissions. *See id.* The Petitioners have not explained why it was necessary for LDEQ to exercise its discretion to require post-construction monitoring for the pig iron process after Nucor had otherwise demonstrated that it would not significantly contribute to existing exceedances around other facilities.

For these reasons, the EPA hereby denies these claims.

#### **4. Nucor's Finding that Class I Area PM<sub>10</sub> Impacts are Insignificant Neglects Contributions from Secondary Particulate Formation.**

*Petitioners' Claims.* The Petitioners claim that Nucor was required to account for nitrate and sulfate formation in their PM<sub>2.5</sub> and PM<sub>10</sub> Class I area impacts modeling in the Breton National Wildlife Refuge (Breton NWR). 2010 Petition at 38. In particular, the Petitioners state that Nucor's Class I area PM<sub>10</sub> modeling found impacts below the proposed significance levels but that the modeling fails to address formation of sulfates and nitrates that it also must consider as PM<sub>10</sub>. *Id.* The Petitioners additionally assert that Nucor's emissions of the precursors SO<sub>x</sub> and nitrogen oxides (NO<sub>x</sub>) far outweigh its emissions of particulate matter (PM), and state that its emissions of PM<sub>10</sub> are much smaller than its emissions of sulfate and nitrate precursors (SO<sub>2</sub> and NO<sub>x</sub>). *Id.* at 38-39. Therefore, the Petitioners assert that Nucor has "failed to include what are likely the greatest contributors to its project's PM<sub>10</sub> and PM<sub>2.5</sub> Class I area impacts" and that "Nucor must reanalyze these project impacts, including the effects of sulfate and nitrate at Breton." *Id.* at 38. The Petitioners further state that Nucor ran CAMx for its ozone analysis and could have used the same model to calculate PM<sub>2.5</sub> and PM<sub>10</sub> impacts at Breton NWR. *Id.* at 39.

These claims were re-raised in the 2012 Petition, Att. B at 38-39.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

With respect to PM<sub>2.5</sub>, as LDEQ explained in its response to comments, the EPA's final rule for

implementation of NSR requirements for PM<sub>2.5</sub> did not require states with SIP-approved PSD programs, like Louisiana, to regulate SO<sub>2</sub> and NO<sub>x</sub> as precursors to PM<sub>2.5</sub> under PSD before May 16, 2011, when the period provided for states with SIP-approved programs to revise their regulations and incorporate the necessary requirements ended. 2010 RTC at 40, 354;

*Implementation of the New Source Review (NSR) Program for Particulate Matter Less Than 2.5 Micrometers (PM<sub>2.5</sub>)*, 73 Fed. Reg. 28321, 28343 (May 16, 2008)(hereafter “the PM<sub>2.5</sub> NSR Implementation Rule”). In addition, referring to the PM<sub>10</sub> Surrogacy Policy, which is discussed in more detail below, LDEQ stated that its “current obligation” at the time of issuing the pig iron permits was only to demonstrate that PM<sub>10</sub> is a reasonable surrogate for PM<sub>2.5</sub> and further stated that a Class I PM<sub>2.5</sub> analysis was not required, but that sulfates and nitrates were calculated by CALPUFF (an air quality dispersion model) as part of the Air Quality Related Values analysis of visibility impacts. 2010 RTC at 354. Additionally, in discussing a PM<sub>2.5</sub> air dispersion modeling analysis submitted by Nucor on January 27, 2010 to address air quality impacts from proposed PM<sub>2.5</sub> emissions, LDEQ explained that monitor selection was then the only available method to account for secondary formation in the absence of the EPA guidance on how to address secondary formation of PM<sub>2.5</sub> and further stated that the Bayou Plaquemine monitor had been selected to account for secondary formation and transport of PM<sub>2.5</sub>. 2010 RTC at 190.

With respect to PM<sub>10</sub>, LDEQ explained in its response to comments that at that time, CALPUFF was deemed incapable of properly representing secondary particle formation for the purpose of estimating PM<sub>10</sub> and PM<sub>2.5</sub> impacts, so the U.S. Fish and Wildlife Service, the Federal Land Manager (FLM),<sup>29</sup> for the Breton NWR, accepted a CALPUFF analysis in which the PM<sub>10</sub> impacts are limited to the effect of emitted particulate matter and which does not include secondary particle formation. 2010 RTC at 354.

The only response that the Petitioners make to the discussion in LDEQ’s response to comments is to explain that the Petitioners did not say that CALPUFF is necessarily the best method to be used for assessing PM<sub>2.5</sub> impacts at Breton NWR, and that their point was that sulfate and nitrate impacts were occurring from Nucor’s emissions and “were being ignored for all NAAQS and PSD increment compliance ... analyses.” 2010 Petition at 39. The Petitioners additionally observed that Nucor could have used CAMx to calculate PM<sub>2.5</sub> and PM<sub>10</sub> impacts, which would include sulfate and nitrate impacts. *Id.* This discussion, however, fails to address salient points in LDEQ’s response to comments concerning the obligation to address secondary particulate matter (PM) impacts in PSD permitting. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state’s final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). LDEQ correctly pointed out that the EPA did not require states to address secondary formation of PM<sub>2.5</sub> for purposes of PSD permitting before May 16, 2011. *See* PM<sub>2.5</sub> NSR Implementation Rule, at 28321, 28343. Nucor’s PSD permit for the pig iron process was issued before that date, on May 24, 2010, and the Petitioners have not identified or analyzed any provision of the approved SIP that required PSD permits in Louisiana

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<sup>29</sup> Federal Land Managers or FLMs are federal officials charged with responsibility for management of Class I areas, such as National Parks and Wildlife Refuges. 42 U.S.C. § 7475(d); 40 C.F.R. § 51.166(p). FLMs have an affirmative responsibility to protect air quality related values (including visibility) in Class I areas such as Breton NWR. Whenever construction of a new or modified source requiring a PSD permit may affect a class I area, the FLM must be notified and provided with information sufficient to evaluate whether the proposed construction will have an adverse impact on air quality related values in the Class I area. 40 C.F.R. §§ 51.166(p), 51.307.

to address secondary formation of PM<sub>2.5</sub> at the time the permit was issued.<sup>30</sup> In addition, the Petitioners have not addressed LDEQ's statement in the response to comments that a Class I analysis was not required for PM<sub>2.5</sub>, and have not provided any citations or analysis to support their assertion that Nucor's Class I analysis should have addressed PM<sub>2.5</sub>. *See, e.g., MacClarence*, 596 F.3d at 1131 ("the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive"); *Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's response to comments, citing *MacClarence*, 596 F.3d at 1132-34, and has looked to whether they have provided the relevant citations and analyses to support the claim).

The Petitioners also assert that nitrates and sulfates must be considered as PM<sub>10</sub>, but have not identified or analyzed any statutory or regulatory provision to support this statement. *See, e.g., MacClarence*, 596 F.3d at 1131 ("the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive"); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support the claim in determining whether it has a duty to object under CAA § 505(b)(2)). In contrast to PM<sub>2.5</sub>, the EPA has not required SIPs to regulate secondary formation of PM<sub>10</sub> in PSD permitting. While the EPA's PSD regulations governing SIP-approved PSD permitting programs do identify precursors for PM<sub>2.5</sub>, they do not identify any such precursors for PM<sub>10</sub>. *See* 40 C.F.R. § 51.166(b)(49)(i)(b)-(c). Moreover, the Petitioners have not identified any provision of the CAA or the approved SIP that they claim requires consideration of formation of sulfates and nitrates as PM<sub>10</sub>. The Petitioners have also not addressed LDEQ's response that the FLM accepted a CALPUFF analysis for PM<sub>10</sub> that does not include secondary particle formation. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's response to comments, citing *MacClarence*, 596 F.3d at 1132-34). In addition, the Petition does not provide any factual information or analysis to show that Nucor's sulfate and nitrate emissions are impacting compliance with any NAAQS or PSD increment, including at Breton NWR.

For these reasons, the EPA hereby denies these claims.

## **5 Emissions Calculations That Nucor Submitted to LDEQ to Support Nucor's PSD Analysis are Unverifiable.**

*Petitioners' Claims.* The Petitioners claim that it was impossible to verify the numerous calculations for Nucor's emission inventory because its permit application included emission calculations and emission reporting tables in a PDF file, rather than as an unlocked Excel spreadsheet showing the equations and assumptions made by Nucor when preparing the application. 2010 Petition at 39. The Petitioners list four reasons why a PDF file without

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<sup>30</sup> The Petitioners cite a draft guidance document for demonstrating attainment of air quality goals for PM<sub>2.5</sub> and regional haze, but do not provide any analysis or explanation of how this document supports this claim. 2010 Petition at 38, n.50 (citing VISTAS, Draft Guidance for Demonstrating Attainment of Air Quality Goals for PM<sub>2.5</sub> and Regional Haze, Jan. 2, 2001, pp. 14-15). The referenced pages of this document provide guidance for states on using modeled results to determine whether an air quality goal is met, particularly on the recommended modeled tests for attainment of the two PM<sub>2.5</sub> NAAQS. These pages do not specifically discuss requirements for PSD permitting, and the Petitioners have provided no explanation of why they believe the recommendations in this guidance document are relevant to PSD permitting.



emission calculations is insufficient: (1) the public cannot verify or review the actual emission calculations applied in the permit application without checking the equations by hand, which could involve many thousands of individual calculations; (2) without “having the native spreadsheets, LDEQ could not itself have reviewed the facility emission calculations in any meaningful fashion” and emission calculation errors could adversely impact permit issuance for the Class I modeling impacts and the 24-hour PM<sub>10</sub> modeled air concentrations, which were over 93 percent of the allowable increment; (3) the name of the spreadsheet and worksheet, which appears in the footer of the PDF file, includes the extension “.XLS”, indicating that the calculations were performed with Excel; Nucor could have provided the Excel files to LDEQ and requesting reviewers but “has never made these Excel files available”; and (4) the “printout of the emission calculation spreadsheets provided by LDEQ are frequently difficult to read” and it is sometimes impossible to determine essential numbers. *Id.* at 39–40.

The Petitioners also claim that meaningful public review requires full transparency of an applicant’s modeling work. *Id.* at 40. The Petitioners contend that without the electronic spreadsheets used to perform the emission calculations, there is no meaningful opportunity for public comment. *Id.* The Petitioners point out that their review is limited by comment deadlines and state that the lack of an electronic version of the calculations forced them to spend an inordinate time attempting to read and recreate the calculations, which was a “roadblock to the public having the ability to understand and comment on the Nucor permit.” *Id.* at 41. Finally, the Petitioners point to a communication between Nucor’s consultant, ERM, and LDEQ in which the consultant noted an error in a calculation because the “formula was drawing from the incorrect cell. Fortunately, the result is an emissions decrease, if only slightly....” *Id.* (quoting “LDEQ Resp. \_Zen-Noh\_Doc.Request.pdf, p. 14/24”). According to the Petitioners, it is “impossible to find that error without having the actual Excel spreadsheet used.” *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 39-41.

*EPA’s Response.* For the reasons provided below, the EPA denies these claims.

LDEQ’s response to comments on this point explained that LDEQ does not require or typically request electronic copies of the permit application, but rather scans hard copies of permit applications and other correspondence to make such information electronically available to the public through its EDMS system. 2010 RTC at 235. LDEQ further stated that it is “not impossible to verify the accuracy of the emissions calculations,” explaining that Nucor submitted “the emissions calculations, the origin or basis for the calculations, and all assumptions and/or variables which serve as inputs necessary to calculate potential emissions.” *Id.* at 236. LDEQ also said that its staff reviewed the calculations in detail and asked many questions directly referencing the calculations. *Id.* Further, LDEQ stated that the scanned version of the permit application is legible and that it is not impossible to determine essential numbers, and suggested that any person who could not decipher the electronic files and did not wish to refer to the hard copy available for public review, could have contacted LDEQ for assistance. *Id.* In regard to a quotation (“the source code needs to be open for public access and scrutiny to enable meaningful opportunity for public comment on new source permits, PSD increment consumption and SIPs”) which the comment attributed to the *Guideline on Air Quality Models*, LDEQ explains that the citation is incorrect, that the quoted passage is actually from a rule preamble, and that the citation

relates to the availability of the source code for dispersion models, not emissions calculations. *Id.* (citing 68 *Fed. Reg.* 18440 (Apr. 15, 2003)).

The Petitioners do not respond to LDEQ's explanation of this quotation or to the corrected citation. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). The Petitioners respond to LDEQ's RTC by stating that the public "must have the available electronic emission calculations to have any chance of reviewing the calculations in the allotted time frame" and that LDEQ's response that it is "not impossible" to figure out the emissions calculations is a "poor excuse" for not providing the electronic calculations that Nucor could have easily emailed to LDEQ and reviewers. 2010 Petition at 41.

The EPA has recognized the importance of the legibility of the information provided in a title V permit application necessary to determine applicable requirements. *See In the Matter of the Huntley Generating Station*, Order on Petition No. II-2002-I (July 31, 2003) at 17-18 (noting that the EPA was unable to determine whether a final permit was in compliance with all applicable requirements where emission limits in the permits appended to the title V permit application were illegible and requiring the state upon reopening to provide the EPA and the public a legible draft of the underlying permits). The Petitioners, however, do not respond to LDEQ's statements in the response to comments that the scanned version of the application is legible and that anyone could have referred to the hard copy or contacted LDEQ for help deciphering the file if needed. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). Moreover, the Petitioners have not pointed to any specific value or page that was illegible, and the EPA additionally notes that the version of the calculations in the permit application publicly available in EDMS generally appears legible, *see* EDMS Doc. ID 642067. Nor have the Petitioners identified any particular applicable requirement that could not be determined because of alleged difficulties in reviewing the PDF documents.

With respect to the Petitioners' claims that without the electronic emission calculations, a meaningful opportunity for public participation was not possible, it is not clear whether the Petitioners are referring to the opportunity for public participation provided for under title V or under PSD. For purposes of a title V petition, in order to show an error in the public process on a PSD permit incorporated into a title V permit, a petitioner must demonstrate that an alleged error in the public process deprived the public of an opportunity to meaningfully comment on the applicable PSD requirements. *See Noranda Order* at 11 (describing the standard for showing an error in the public notice for a PSD permit). Analogously, when a title V petition seeks an objection based on the unavailability of information during the public comment period in violation of title V's public participation requirements, the EPA has stated that the petitioner must demonstrate that the unavailability of the information deprived the public of the opportunity to meaningfully participate during the permitting process and that the EPA would generally look to whether the petitioner had demonstrated that the alleged flaws resulted in or may have resulted in a deficiency in the permit's content. *See, e.g., 2012 Cash Creek Order* at 9. The EPA further noted that where a permitting authority has explained its decision not to make information available during the public comment period, the petitioner bears the burden of demonstrating that the explanation is unreasonable. *Id.*

The Petitioners have not shown that they were deprived of a meaningful opportunity for participation under either standard. LDEQ explained that it does not require or typically request electronic submissions for permit applications, and it explained that Nucor submitted “the emissions calculations, the origin or basis for the calculations, and all assumptions and/or variables which serve as inputs necessary to calculate potential emissions.” 2010 RTC at 236; *see also* EDMS Document ID 42013758 (previously known as Document ID 6462061) (June 29, 2009 pig iron permit application, specifically Appendices B and C). The Petitioners do not show that LDEQ’s explanation is unreasonable or incorrect. *See 2012 Cash Creek Order* at 9. Further, the Petitioners have not demonstrated that the lack of the electronic emissions calculations in the materials submitted as part of Nucor’s application resulted in or may have resulted in a deficiency in permit content. *Id.* The Petition quotes one communication from Nucor’s consultant, which mentions an error in the calculation annual average emission rate from one emissions unit. 2010 Petition at 41 (quoting “LDEQ\_Resp.\_Zen Noh\_Doc.Request.pdf, p. 14/24”). However, the quotation from that communication in the Petition indicates that correcting the error led to a slight emissions decrease, and the Petitioners have not explained how this decrease resulted or may have resulted in a flaw in the permit.

Moreover, the Petition does not assert that the difficulties in reviewing the emissions calculations led to any specific difficulty in evaluating PSD requirements, and the Petitioners’ comments during the public comment period extensively addressed PSD issues, including Nucor’s air quality analysis. *See, e.g., Comments submitted on behalf of LEAN, Sierra Club, and O’Neill Couvillion on the Proposed Part 70 Operating Permit and Prevention of Significant Deterioration Permit, for Consolidated Environmental Management, Inc., Nucor Steel Facility*, (April 19, 2010)(EDMS Document ID 6756728) at 7-19, 21-38; *see also Addendum to Comments submitted on behalf of LEAN, Sierra Club, and O’Neill Couvillion on April 19 Regarding the Proposed Part 70 Operating Permit and Prevention of Significant Deterioration Permit, for Consolidated Environmental Management, Inc., Nucor Steel Facility* (May 3, 2010)(EDMS Document 4889042) at 1-5 (providing additional comments on Nucor’s air quality modeling and Class I area analysis). The Petitioners have not argued that they would have made any other comments had the Excel spreadsheets been made available during the public comment period. *See Noranda Order* at 13.

The crux of the Petitioners’ claim appears to be that it would be difficult to verify the volume of emissions calculations provided in the time allotted for public review without access to the Excel spreadsheets and that it would have been easy for Nucor to provide the data. *See 2010 Petition* at 39 (noting that Nucor provided 329 pages of calculations, with potentially hundreds individual calculations per page); *id.* at 41 (noting that it is not realistic review the calculations in the allotted time frame without the electronic emissions calculations). However, the Petition does not indicate whether the Petitioners sought additional time to review the calculations. The response to comments indicates that LDEQ extended the public comment period until May 3, 2010, 2010 RTC at 1, n.1, and it is possible that the Petitioners might have been able to obtain additional time for their review. Moreover, the Petitioners’ assertions that it would have been easy for Nucor to provide the electronic data does not establish that LDEQ was required to request or Nucor was required to submit data in electronic form during this permitting process or that either LDEQ or Nucor was required to provide information that was available in paper form to public commenters in electronic form. *See, e.g., MacClarence*, 596 F.3d at 1131 (“the Administrator’s

requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA § 505(b)(2)). Accordingly, the Petitioners have not demonstrated that the information that was available in the record was insufficient to allow for a meaningful opportunity to comment for title V or PSD purposes.

For these reasons, the EPA denies these claims.

## **6. LDEQ Could Not Provide Modeling Input Files Necessary for Evaluating Nucor’s Class I Area Impact Modeling.**

*Petitioners’ Claims.* The Petitioners claim that LDEQ did not provide the Petitioners with certain modeling files that were used in Nucor’s modeling to assess air and air-quality related impacts on the Breton NWR Class I Area and that they requested from LDEQ. In particular, the Petitioners assert that LDEQ failed to provide requested files containing meteorological data and ozone data used by Nucor, specifically the “CALMET monthly VISTAS Domain 1 meteorological data files and the yearly VISTAS Domain 1 ozone data files used by Nucor.” 2010 Petition at 41–43, 44. The Petitioners claim that they sent a hard drive to LDEQ and received the data back from LDEQ on April 19, 2010, but that it contained CALMET meteorological data files that were not part of Nucor’s modeling analysis and did not contain any of the ozone data used by Nucor. *Id.* at 43. The Petitioners explain that they were able to obtain the appropriate VISTAS Domain 1 meteorological from the National Park Service (NPS), but not the three yearly ozone files. *Id.* at 44. The Petitioners further state that they requested the data files because they “need[ed] the exact data files modeled by Nucor in order to replicate their analyses.” *Id.* The Petitioners also claim that LDEQ did not appear to have ever obtained these files from Nucor, and that without them “LDEQ could not have replicated or independently evaluated the Class I modeling performed by Nucor.” *Id.* Additionally, the Petitioners claim that they had not yet finished their Class I modeling review because of the protracted effort to obtain the referenced data. In addition, the Petitioners note that a recent, undated LDEQ document indicates that the Fish and Wildlife Service (FWS) also “had difficulties reviewing Nucor’s Class I modeling.” *Id.* at 44–45 (citing “LDEQ\_Resp.\_Zen-Noh\_Doc.Request.pdf, p.23-24/24” and quoting communications from Jill Webster, FWS, to Bryan Johnston).

These claims were re-raised in the 2012 Petition, Att. B at 41-45.

*EPA’s Response.* For the reasons provided below, the EPA denies these claims.

LDEQ’s response to comments on this point explained that the most recent Class I modeling has been available for public review since early 2009 and that the referenced data files are large files, so that the requestor must send LDEQ a hard drive to download the information. 2010 RTC at 352. LDEQ states that it received the Petitioners’ hard drive on April 13, 2010, that it was ready on April 15, 2010, and that the requested ozone files were included. *Id.* LDEQ states that there was no further response from the Petitioners, so it had no reason to believe that there were any deficiencies in the information. *Id.* With respect to the VISTAS meteorological data, LDEQ explained that because the files are large and that the data has been previously approved by the

FLM and the EPA, LDEQ did not require the data to be submitted. *Id.* LDEQ additionally states that the data was available from the FLM and that the Petitioners were aware of that. *Id.*

The Petition does not identify or analyze any specific regulatory, statutory, or other legal requirement that it alleges LDEQ failed to meet with respect to the Class I modeling data or with respect to the public review process. *See, e.g., MacClarence*, 596 F.3d at 1131 (“the Administrator’s requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive”); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA § 505(b)(2)).

In addition, the Petitioners’ reply to LDEQ’s response to comments does not show that LDEQ’s approach failed to comply with its SIP-approved regulations governing PSD permitting or that it acted unreasonably. *See, e.g., 2012 Cash Creek Order* at 4-5. The Petitioners state that the response to comments reflects that LDEQ was aware that they did not have the proper data. 2010 Petition at 44. However, this assertion is contrary to LDEQ’s statement that “it had no reason to believe that there were any deficiencies or problems with the information provided.” 2010 RTC at 352. The Petition further states that the ozone files LDEQ provided were for the wrong analysis and were not the ozone files used by Nucor. 2010 Petition at 44. But there is no indication that LDEQ knew or should have known that the Petitioners believed the data provided was not the data they had requested. In addition, the Petition states that the Petitioners were concerned that the NPS data might be for different time blocks than Nucor modeled, which “may or may not” affect their efforts to replicate Nucor’s modeling. 2010 Petition at 44. As the Petition earlier states that the NPS sent the Petitioners “the appropriate VISTAS Domain 1 meteorological data files,” *id.*, it appears that the Petitioners obtained the meteorological data they sought.

The Petition also contends that the FWS also had difficulty reviewing Nucor’s Class I modeling. *Id.* at 44-45. (citing “LDEQ\_Resp.\_Zen-Noh\_Doc.Request.pdf, p.23-24/24” and quoting statements from Jill Webster from the FWS). The Petition does not contend that the Fish and Wildlife Service ultimately found the Class I area review inadequate or the Class I impacts unacceptable. To the contrary, a February 13, 2009 email from Jill Webster, which contains the same statements quoted in the petition, also indicates that Nucor’s impacts are acceptable. *See* Email from Jill Webster, FWS, to Brian Johnston, Subject: Breton Class I Impacts-Nucor, LA (Feb. 13, 2009), available at EDMS Doc. No. 2628621. LDEQ’s response to comments cites this email and its indication that Nucor’s impacts are acceptable and states that LDEQ “also repeated [the] public participation process, including notice to EPA, such that all interested parties could have an opportunity comment on Nucor’s Class I analysis.” 2010 RTC, No. 312, at 409. The Petition does not acknowledge or address these points in the state’s response. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state’s final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34).

For these reasons, the EPA denies these claims.

### C. The Permit Lacks Conditions Sufficient to Ensure Compliance with PSD Requirements

As originally presented in the 2010 Petition, the Petitioners raise five main claims about which the Petitions contend that the pig iron title V permit fails assure compliance with PSD requirements (Claims III.B – F), and these claims contain multiple subclaims. 2010 Petition at 45-67. These main claims will be addressed in order below. The initial part of Claim III begins with “III.A. Legal Background.” 2010 Petition at 45-48. The Petitioners state that a title V permit must identify all emission limits for the source, including enforceable emission limitations and standards and requirements to assure compliance with the permit terms and conditions. *Id.* at 45 (citing *Sierra Club v. EPA*, 536 F.3d 673, 674 (D.C. Cir. 2008), 42 U.S.C. § 7661(c)(a) & (c)). This section also includes a discussion of BACT (including top-down BACT, which the Petitioners contend Nucor and LDEQ used), and it contends that the PSD permitting process requires establishing federally enforceable limits to ensure that BACT determinations are implemented. *Id.* at 45. Accordingly, the Petitioners contend that BACT limits must be met ““on a continual basis at all levels of operations”” and must be ““enforceable as a practical matter.”” *Id.* at 46 (quoting the NSR manual at B.56). This section does not include any contentions that the permit is deficient and does not contain a separate request that the EPA object to the permit. As a result, the EPA does not interpret the statements in this section as separate petition claims that require a response but rather as background for the claims that follow and therefore is not responding to the statements made in this section.

The next section, Claim III.B, is entitled, “The Monitoring Frequency for Numerous Emission Units is not Adequate to Ensure Enforceability.” This section raises numerous contentions about inadequate monitoring for BACT limitations,<sup>31</sup> however, it appears that specific units to which

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<sup>31</sup> In Claim III.B, the Petitioners make the following contentions – many of which appear to be repeated in III.B.1-5 with additional specificity. Thus, the EPA is responding to Claim III.B and III.B.1-5, together.

*The Petitioners’ Claims in III.B may be summarized as follows:*

Petitioners initially contend generally that the PSD and title V permits do not meet the requirements under the CAA and Louisiana law to contain permit terms and conditions to ensure compliance with applicable limits. 2010 Petition at 48. They contend that the PSD permit for the pig iron process contains “no testing, monitoring, or record-keeping provisions” and therefore, “is fundamentally flawed.” *Id.* However, Petitioners do not state that this asserted flaw is a separate ground for an objection to the title V permit. Petitioners go on to state that monitoring and record keeping are found only in the “draft” title V permit and none of the “title V” monitoring is specifically directed at BACT limitations. *Id.* Petitioners explain that the BACT determinations are expressed as concentrations not emission rates, and as a result, the “draft permits” in effect contain no monitoring and record keeping for the BACT limitations. *Id.* Petitioners state that examples of this include control efficiencies for scrubbers and baghouses and TDS concentrations for cooling and quench towers. *Id.* For support, Petitioners cite to § 504(c) of the CAA, 42 U.S.C. § 7661(c) and *Sierra Club v. Whitman*, 536 F.3d 673 (D.C. Cir. 2008). Petitioners note that the testing provisions for all sources except for a select few are not enforceable because “compliance testing is either not required at all or is infrequent,” such as one stack test over the life of the facility. 2010 Petition at 49. The Petitioners argue that the absence of testing or infrequent (and ambiguous) testing renders BACT limits unenforceable as a practical matter and violates Section 504(c) of the CAA, 42 U.S.C. § 7661(c). Citing to the NSR Manual, Petitioners discuss their views on the “hierarchy” for monitoring in a permit and state that the monitoring in the “draft permits” do not comport with EPA guidance. *Id.* Petitioners further contend that except for the limited use of continuous emissions monitoring systems (CEMSs), all testing is by periodic stack tests which Petitioners contend measure “about 0.007%” of emissions from the facility over its lifetime. *Id.* Petitioners provide additional information regarding their views that stack tests do not reflect full emissions from the facility, including startup, shutdown and malfunction emissions. 2010 Petition at 50. The Petitioners explain that the compliance provisions must assure that BACT conditions are met on a “continual” basis and that the proposed testing is not “adequate to assure compliance with

Petitioners' contentions apply are detailed in Claim III.B.1-5. Since the EPA is responding directly to the specific claims on the specific units in III.B.1-5, the EPA is responding to Petition sections III.B and III.B.1-5 together, below.

These points were re-raised in the 2012 Petition, Att. B at 45-67.

**1. The Title V Permit Requires no Testing for Many Point Sources Subject to BACT Limits.**

*Petitioners' Claims.* The Petitioners contend that the draft title V permit does not require "any testing" for BACT limits at 20 listed units. 2010 Petition at 51. The Petition includes PM<sub>10</sub> limits in tons per year (tpy) for each unit in parentheses. The Petition states that the title V permit must be modified to require at least an initial stack test for sources that vent to a baghouse and that larger sources of PM<sub>10</sub>, such as the Coke Battery 2 Quench Tower and the Sinter Plant Main Dedusting Baghouse Vent should be tested at least annually. 2010 Petition at 52. The Petition also states that surrogate monitoring should be required for vent sources with emissions under one ton per year. *Id.* The Petitioners state testing on one quench tower should not exempt the other quench tower from testing. *Id.* Petitioners then address parts of LDEQ's response to comment by stating that LDEQ's explanation "violates the NESHAPS." 2010 Petition at 53. For support, Petitioners state that LDEQ cannot rely on the use of compacted coal to satisfy the NESHAPS (and that also cannot be used to exempt Nucor from monitoring to determine compliance) and further state that the regulations cited for compliance do not require any testing. *Id.* Petitioners further state that the monitoring for opacity and visible emissions (citing conditions 78 and 79) cannot assure compliance with limits expressed as pounds per hour or tons per year. *Id.* In response to some changes made by LDEQ to the Coke Battery 2 Coke Quench Tower provisions, Petitioners contend that compliance with MACT testing requirements does not assure compliance with BACT limits. Petitioners conclude by stating that certain visible emissions conditions (Conditions 662, 655, 666, and 685) are ambiguous and cannot assure continuous compliance. *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 51-54.

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any emission rate, especially the BACT determinations." *Id.* Specifically, Petitioners state that it is feasible to directly and continuously monitor filterable PM<sub>10</sub> CO, VOC, SO<sub>2</sub>, and NO<sub>x</sub> emissions from sources and to periodically monitor both PM<sub>10</sub> and PM<sub>2.5</sub>, as well as conduct more frequent stack testing. *Id.* Noting LDEQ's response to Petitioners' comments on the permits, that all emission limits, monitoring, recordkeeping, and reporting requirements of the title V permit for PM/PM<sub>10</sub>/PM<sub>2.5</sub>, SO<sub>2</sub>, NO<sub>x</sub>, carbon monoxide (CO), and volatile organic compounds (VOC) are incorporated into the PSD permit, Petitioners simply state that LDEQ's response did not address their concerns. 2010 Petition at 51. The Petitioners state that many BACT limits are expressed in terms other than pounds per hour and tons per year and no monitoring or recordkeeping is required to assure these limits are met. *Id.* Petitioners also state that the title V permit's specific conditions are deficient because they lack any "responsive monitoring or recordkeeping," so the incorporation does not address their concerns. *Id.* However, the Petitioners do not contend that the incorporation of the terms of the title V permit into the PSD permit is inconsistent with the requirements of the SIP or title V or that the record contains insufficient justification to provide the basis for that incorporation. Thus, the Petitioners are not raising the same issue that was discussed in the objection granted in the *Zen-Noh Order* and LDEQ's response to that objection. *See Zen-Noh Order* at 10, 14-16; LDEQ's Response at 19-20.

*EPA's Response.* For the reasons provided below, the EPA grants in part and denies in part on these claims.

As a preliminary matter, the issue raised here regarding compacted coal and coal charging (2010 Petition at bottom of 52 - top of 53) is addressed in the EPA's response to Claim I.B as originally raised in the 2010 Petition, *supra*. Additionally, the EPA notes that the Petition discusses visible emissions monitoring conditions contained in the permit, and states that "Petitioners object" to those conditions. 2010 Petition at 54. To the extent the Petitioners are responding to LDEQ's mention of visible emissions monitoring in its RTC by asserting that LDEQ requires only visible emissions monitoring and the conditions are ambiguous and cannot assure continuous compliance, we note that below we have addressed the monitoring claims for each unit raised in the petition and thus are not separately discussing those issues here. However, to the extent that Petitioners intended to raise a separate claim based on these conditions, the EPA denies this issue on procedural grounds because it does not appear to have been raised in public comments to LDEQ. *See* 2010 RTC at 120, 214.<sup>32</sup> This procedural deny is supplemented by the discussion below.

#### *Rationale for Partial Grant*

For the reasons provided below, the EPA grants this claim as it regards the following units and the issues summarized above: COK-101, COK-201, COK-102, COK-202, COK-203, SIN-102, COK-104, COK-204, COK-112, COK-212, COK-113, COK-213, SIN-105, SIN-106, SLG-405, SLG-407, SLG-408, SLG-402, STC-101, and STC-210. During the public comment period, Petitioners provided comments to LDEQ raising concerns with the enforceability of certain emission limits associated with the above-identified units. Specifically, the comments raised with reasonable specificity the same issues described in the Petitions. 2010 RTC at 214.

The EPA has previously addressed monitoring claims raised in the title V petition context, and has recently provided the following legal framework regarding such claims. *See, e.g., In the Matter of United States Steel Corporation, Granite City Works*, Order on Petition No. V-2011-2 (December 3, 2012) (hereafter "*US Steel Order*") at 10-11.

As explained in the *US Steel Order*, section 504(c) of the CAA requires all title V permits to contain monitoring requirements to assure compliance with permit terms and conditions. 42 U.S.C. § 7661c(c). The EPA's Part 70 monitoring rules (40 C.F.R. § 70.6(a)(3)(i)(A) and (B) and 70.6(c)(1)) must be interpreted to carry out § 504(c) of the Act's directive. *Sierra Club v. EPA*, 536 F.3d 673 (D.C. Cir. 2008). As a general matter, permitting authorities must take three steps to satisfy the monitoring requirements in the EPA's Part 70 regulations. First, under 40 C.F.R. § 70.6(a)(3)(i)(A), permitting authorities must ensure that monitoring requirements contained in applicable requirements are properly incorporated into the title V permit. Second, if the

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<sup>32</sup> Pursuant to CAA § 505(b)(2), a petition "shall be based only on objections to the permit that were raised with reasonable specificity during the public comment period provided by the permitting agency (unless the petitioner demonstrates in the petition to the Administrator that it was impracticable to raise such objections within such period or unless the grounds for such objection arose after such period)." 42 U.S.C. § 7661d(b)(2). None of the comments submitted by any commenter raise the issue that Conditions 662, 655, 666, and 685 are ambiguous and cannot assure continuous compliance. Nor do Petitioners demonstrate that it was impracticable to raise this issue in comments, or that it arose after the close of the comment period.



applicable requirement contains no periodic monitoring, permitting authorities must add “periodic monitoring sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.” 40 C.F.R. § 70.6(a)(3)(i)(B). Third, if there is some periodic monitoring in the applicable requirement, but that monitoring is not sufficient to assure compliance with permit terms and conditions, permitting authorities must supplement monitoring to assure such compliance. 40 C.F.R. § 70.6(c)(1). *In the Matter of CITGO Refining & Chemicals Co.*, Order on Petition No. VI-2007-01 (May 28, 2009) (hereafter “*CITGO Order*”) at 6-7. As the EPA has explained, “[b]oth of these monitoring rules (40 C.F.R. §§ 70.6(a)(3)(i)(A) and (B) and 70.6(c)(1)) are designed to address the statutory requirement that ‘[e]ach permit issued under [title V] shall set forth . . . monitoring . . . requirements to assure compliance with the permit terms and conditions.’ CAA § 504(c), 42 U.S.C. § 7661c(c). Thus, in evaluating whether the permit contains monitoring sufficient to assure compliance under 40 CFR 70.6(c)(1), EPA believes it is appropriate to consider whether such monitoring is ‘sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.’” *In the Matter of United States Steel Corporation, Granite City Works*, Order on Petition No. V-2009-03 (January 31, 2011) at 6.

Further, as explained in the *US Steel Order*, the rationale for the monitoring requirements selected by a permitting authority must be clear and documented in the permit record (*e.g.*, in the Statement of Basis). *See* 40 C.F.R. § 70.7(a)(5); *see also CITGO Order* at 7. Furthermore, permitting authorities do not have the discretion to issue a permit without specifying the monitoring methodology needed to assure compliance with applicable requirements in the title V permit. *In the Matter of Wheelabrator Baltimore, L.P.*, (Order on Petition) at 10 (April 14, 2010) (hereafter “*Wheelabrator Order*”). In the *Wheelabrator Order*, the permit condition in question required the source to develop a way to convert data in order to demonstrate compliance with PSD emission limits. *Id.* at 11. Both the establishment and approval by the permitting authority of this conversion method were to occur “outside of the title V permitting process.” *Id.* The EPA found this methodology “inconsistent with the requirements of § 504(c) of the Act to include – *in the title V permit* – monitoring to assure compliance with applicable requirements,” and instructed the permitting authority to revise the permit to explicitly include the conversion method that would assure compliance with the emission limits. *Id.* (emphasis in original.)

While it is not clear from the permit record that the numeric PM limits listed in parentheses in the 2010 Petition (*e.g.*, at 51-52) and taken from the Emission Rates for Criteria Pollutant Table (pig iron title V permit, “Emission Rates for Criteria Pollutants”) are “BACT” limits, LDEQ appears to refer to these limits as PSD-related limits in the 2010 RTC. 2010 RTC at 214-216. At a minimum, LDEQ appears to treat these limits as if they are federally enforceable limits, and if they are, they would need adequate periodic monitoring under title V. The following analysis is based on the position that these emission limits are federally enforceable limits. As a result, the analysis below anticipates that the limits identified by the Petitioners apply at all times.

*Units COK-101 and COK-201 Coke Battery 1 and 2 Coal Charging.* In addition to the EPA’s response to Claim I.B. in the 2010 Petition, *supra*, the EPA provides the following additional response. Petitioners assert that the “Title V permit does not require any testing to determine if BACT limits for these sources are met.” 2010 Petition at 51. In its response to similar comments during the state public comment period, LDEQ explained that BACT limits for Coke Battery 1

and 2 Coal Charging have been set at 0.0081 pounds (lb)/ton of dry coal charged, and that 40 C.F.R. § 63.309(k), which requires a performance test, will not be used to determine compliance with this emission limitation because it requires a ventilation stack, which will not exist at Nucor. 2010 RTC at 214-215. “Instead, compliance shall be determined with other applicable procedures described in 40 C.F.R. § 63.309(a)-(m) and 40 C.F.R. § 63.7300(a).” 2010 RTC at 214-215. The regulations at 40 C.F.R. § 63.309(a)-(m), excluding (k), require, among other elements, daily opacity observations and 40 C.F.R. § 63.7300(a) requires good air pollution control practices and an operation and maintenance plan. The permit itself references the Part 63 citations but provides no additional information regarding how that monitoring assures compliance with either numeric PM limit in the permit. *See, e.g.*, pig iron title V permit, unit EQT 0001-22 (Specific Requirements at 3). LDEQ’s response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is “sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.” 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). As a result, the EPA grants this claim in the Petition and directs LDEQ to provide an explanation as to how the monitoring identified in the permit assures compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units, considering that the limits apply at all times. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Units COK-102 and COK-202 Coke Pushing.* LDEQ’s response to issues raised during the public comments was that the BACT limit set for these units of 0.04 lb of filterable PM<sub>10</sub> per ton of coke pushed was the limit associated with the NESHAP for Coke Ovens: Pushing, Quenching, and Battery Stacks (40 C.F.R. Part 63, Subpart CCCCC). 2010 RTC at 215. Further, LDEQ explained that the performance testing and monitoring provisions “ensure compliance with the BACT limit.” *Id.* LDEQ’s response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is “sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.” 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). Further, LDEQ has not explained how the cited MACT monitoring is sufficient to assure compliance with the BACT limit, consistent with title V. Thus, the EPA grants this issue and directs LDEQ to explain how the monitoring requirements in the permit are sufficient to assure compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Units COK-104 and COK-204 Coke Battery 1 and 2 Coke Handling.* LDEQ’s response to issues raised during the public comments was that the units are “controlled via baghouses and are subject to daily monitoring provisions.” 2010 RTC at 215. However, LDEQ’s response in the 2010 RTC does not explain how these monitoring requirements are related to the numeric PM limits identified in the Petition. Although there are monitoring requirements for both units COK-104 and 204 (pig iron title V permit, units EQT-0004 and 0010 and PCS-002), LDEQ’s response does not explain how compliance with the numeric PM limit will be calculated. LDEQ’s

response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is “sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.” 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). The EPA therefore grants this issue and directs LDEQ to explain how these monitoring requirements assure compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Units COK-112, COK-113, COK-212, COK-213 Coke Battery 1 and 2 FGD Lime Silo Unloading and Coke Battery 1 and 2 FGD Waste Loading.* LDEQ’s response to issues raised during public comments was that the units are controlled via baghouses and are subject to daily monitoring provisions and that testing of these sources is not warranted because potential particulate emissions from these sources are 0.015 tpy (for COK-112 and 212) and 0.09 tpy (for COK-113 and 213). 2010 RTC at 215. As noted by LDEQ, the Permit does contain numerous testing requirements and some compliance demonstration information; however, neither the permit nor LDEQ’s response explain how the testing provided for in the permit assures compliance with the numeric PM emission limits identified by the Petitioners for the above-referenced units. *See, e.g.,* pig iron title V permit, units EQT-0005 and 0006. LDEQ’s response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is “sufficient to yield reliable data from the relevant time period that are representative of the source’s compliance with the permit.” 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). Thus, the EPA grants this issue and directs LDEQ to explain how the monitoring requirements in the permit assure compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Units SIN-102 Sinter Plant Main Dedusting Baghouse Vent.* LDEQ’s response to issues raised during public comments was that 40 C.F.R. Part 63, Subpart FFFFF, applies and that compliance with the 0.005 grains per dry standard cubic feet concentration and the 0.0482 lbs/ton finished sinter limit will be demonstrated per the performance testing and monitoring provisions of Subpart FFFFF “and additional stack testing requirements.” 2010 RTC at 215. In response, the Petition contends that MACT testing requirements do not assure compliance with BACT limits, which must be met on a continuous basis, among other points. 2010 Petition at 53. LDEQ’s simply points to Subpart FFFFF in the 2010 RTC but does not explain how testing and monitoring in that Subpart are sufficient to assure compliance with the numeric PM limit identified by Petitioners. For unit SIN-102, the permit itself includes numerous testing and monitoring provisions. *See* pig iron title V permit, unit EQT-0032 (Specific Requirements at 37). In the permit provisions associated with the emission limits identified by the Petitioners, for units EQT-0032-401 and 403, there does not appear to be any specific monitoring or compliance information. LDEQ’s response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is “sufficient to yield reliable data from the relevant time period that

are representative of the source's compliance with the permit." 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). Thus, the EPA grants this issue and directs LDEQ to explain how the monitoring requirements assure compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Units SIN-105, SIN-106, SLG-405, SLG-407, SLG-408, SLG-402, STC-101, and STC-210.* LDEQ's response to issues raised during public comments was that these sources are controlled via baghouses and are subject to daily monitoring provisions, and that due to low emissions, testing of these sources is not warranted. 2010 RTC at 215-216. LDEQ also references applicability of Subpart FFFFF. *Id.* As LDEQ's response does not explain how the monitoring in the permit assures compliance with the numeric PM limits identified by the Petitioners. Specifically, LDEQ did not explain how the monitoring is "sufficient to yield reliable data from the relevant time period that are representative of the source's compliance with the permit." 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). The permit itself references various testing and monitoring requirements, including references to Subpart FFFFF, but does not clearly correlate these conditions with compliance with the numeric PM limits at these units. As a result, the EPA grants this issue and directs LDEQ to explain the specific permit monitoring requirements that will result in information to assure compliance with the numeric PM limits identified by Petitioners. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

*Unit COK-103 and COK-203 Coke Battery 2 Coke Quench Tower.* With regard to the quench towers, the Petition raises two separate claims. In the first claim, the Petitioners repeat statements made during the public comment period regarding testing for the two similar quench towers. 2010 Petition at 52. Petitioners explain that just because the two quench towers are similar does not mean that both towers do not need to be tested. In the second claim, Petitioners discussed LDEQ's response to their comments on the testing for the quench towers where LDEQ pointed to monitoring, recordkeeping and reporting requirements in 40 C.F.R. Part 63, Subpart CCCCC. For the reasons described here, and consistent with the other issues discussed in this section, the EPA denies the Petition as to the first claim and grants the petition as to the second claim.

With regard to the first claim, LDEQ's response to issues raised during public comments was that "a testing requirement identical to that associated with Coke Battery 1 Coke Quench Tower has been added to the permit" and annual testing is not required because Subpart CCCCC has additional monitoring, recordkeeping and reporting requirements. 2010 RTC at 215. Thus, it appears that testing for units COK-103 and COK-203 no longer differ in the ways that resulted in Petitioners' claim in the Petition. *Id.* Further, Petitioners appear to acknowledge this change (Petition at 53) but do not raise any further issues regarding that specific change to the permit. The Petition reiterates LDEQ's response and then provides no further additional information demonstrating that the permit is not in compliance with the CAA. 2010 Petition at 53. LDEQ appears to have made changes to the permit in light of Petitioners' comments, but Petitioners did not consider the responsive changes or demonstrate how such changes were inadequate to ensure

that the permit was in compliance with the Act. *See, e.g., MacClarence*. For these reasons, the Petition is denied as to this claim.

With regard to the second claim, The permit terms, and LDEQ's RTC points to 40 C.F.R. Part 63, Subpart CCCCC (and specific provisions therein), but does not explain how those provisions are sufficient to assure compliance with the numeric PM emission limit. Specifically, LDEQ did not explain how the monitoring is "sufficient to yield reliable data from the relevant time period that are representative of the source's compliance with the permit." 40 C.F.R. § 70.6(a)(3)(i)(B); *see also* 40 C.F.R. § 70.6(c)(1). Thus, the EPA grants this issue and directs LDEQ to explain how the monitoring requirements in the permit assure compliance with the numeric PM emission limit identified by the Petitioners for the above-referenced units. To the extent that LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10.

#### *Rationale for Partial Deny*

*General Statements Regarding Monitoring.* Throughout this section of the Petition (2010 Petition at 45-54), Petitioners appear to be raising "general" issues not necessarily specific to the Nucor permits – but general assertions regarding monitoring. For example, the Petition makes blanket statements regarding stack testing (Petition at 50). To the extent that this portion of the Petition makes general assertions and does not identify an issue upon which the EPA could object to the Nucor permit, the EPA is not obligated to respond to such general assertions. Pursuant to CAA § 505(b)(2), the Administrator "shall issue an objection...if the petitioner demonstrates to the Administrator that the permit is not in compliance with the requirements of" the CAA. 42 U.S.C. § 7661d(b)(2). To the extent that the Petitioners are simply stating their positions on legal, policy, or technical points without specifically demonstrating that the Nucor permit fails to assure compliance with the Act, such issues do not warrant a response by the EPA. *See also MacClarence v. EPA*, 596 F.3d 1123 (9<sup>th</sup> Cir. 2010) (upholding the EPA's finding that an unsupported general assertion failed to demonstrate that the permit is not in compliance with the Act and stating "(the Administrator's requirement that [a title V petitioner] support his allegations with legal reasoning, evidence, and references is reasonable and persuasive)"; *see also Nucor II Order* at 7. In addition, we note that many of these general statements directly repeat what was said in the corresponding public comments, and to the extent that the Petitioners have failed to address the response that LDEQ provided in the 2010 RTC, these assertions would not satisfy the Petitioners' demonstration burden. *See Nucor II Order* at 7 (explaining that the EPA expects title V Petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). The EPA has provided a unit-by-unit substantive response above, and granted on numerous monitoring related issues.

In addition, the Petitioners assert that an initial stack test is required to confirm emission calculation assumptions for sources that vent to a baghouse, that annual stack testing and surrogate monitoring must be conducted for the large sources of PM<sub>10</sub>, and that surrogate monitoring must be performed for the vent sources below 1 ton per year. 2010 Petition at 52. However, the Petitioners do not cite or analyze any statutory or regulatory provisions that would require those specific forms of testing or monitoring for those units. *See, e.g., MacClarence*, 596 F.3d at 1131 ("the Administrator's requirement that [a title V petitioner] support his allegations

with legal reasoning, evidence, and references is reasonable and persuasive”); *Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA section 505(b)(2)). Accordingly, the aspect of the claim is denied. While we are granting on numerous monitoring-related issues, as explained above, we are not determining that the forms of monitoring cited on page 52 of the Petition are necessarily required for the emissions units identified by the Petitioners.

For these reasons, the EPA grants in part and denies in part these claims.

## **2. The Title V Permit Requires No Testing for Many Non-Point Sources Subject to BACT Limits**

*Petitioners’ Claims.* In the petition on the pig iron process, the Petitioners identify claims raised in context of pig iron process units that they contend include non-point source emissions that are not subject to emission limits, testing or record keeping requirements in the title V permit. 2010 Petition at 54-55. Petitioners state that the permit is unlawful without such conditions. 2010 Petition at 55. With regard to units FUG-101 and FUG-102, Petitioners contend that LDEQ failed to respond to their comments regarding the enforceability of certain conditions that apply to those sources. 2010 Petition at 55. Petitioners then discuss “storage piles” generally (no unit citations provided) and contend that LDEQ’s response to issues raised in the comment period was “unsupported and incorrect.” 2010 Petition at 56. Petitioners cite to rules and practices of the South Coast Air Quality Management District (SQAMD) in California for support that LDEQ’s responses were incorrect. Petitioners take particular issue with LDEQ’s statement that Nucor’s storage piles cannot feasibly be covered and Petitioners offer several reasons as to why they believe such a statement is incorrect. 2010 Petition at 56-57. First, Petitioners suggest that LDEQ should have developed a plot plan to enclose the piles. 2010 Petition at 56. Second, Petitioners suggest that the California rules reached a different conclusion. 2010 Petition at 56-57. Third, Petitioners suggest that emissions from roadways and storage piles are underestimated and that control efficiencies are unenforceable. *Id.* With regard to the Dust Management Plan (DMP), Petitioners contend that it is not adequate to ensure that the dust control efficiencies used to estimate emissions, model ambient impacts and satisfy BACT are met and that monitoring is “not a substitute for assuring that emissions do not exceed the assumed levels.” *Id.* Petitioners provide three bases for this contention involving weekly monitoring being inadequate, and triggers for responses being very high, and again, citing to a SQAMD report. *Id.* at 57-58. Finally, in response to LDEQ’s statement that direct measurement of emissions from paved and unpaved roads is not technically feasible, the Petitioners state that such a demonstration must be “on the record.” *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 54-58.

*EPA’s Response.* For the reasons provided below, the EPA denies these claims.

In response to Petitioners’ comments regarding non-point (fugitive) source monitoring, LDEQ provided unit-by-unit information responsive to Petitioners’ comments and highlighted existing monitoring in the permit that Petitioners did not reference. 2011 RTC at 160-163. Specifically,

LDEQ explained, “Nucor’s Dust Management Plan requires actual monitoring of dust during both the construction and operation of the facility with deposition gauges, portable monitors, and visual inspections. This plan also includes quantifiable action levels and prescribes corrective actions. LDEQ has determined these work practice standards meet BACT for fugitive particulate emissions. See also LDEQ Response to Comment No. VII.45 for our response.” *Id.* at 160-163 (response to comment VII.46). The permit itself and the Dust Management Plan<sup>33</sup> (compliance with which is a condition of the permit - *see* pig iron title V permit, condition 937, at 91 of 93) include numerous conditions to monitor emissions from each of the units identified in the Petition at 54.

The Petition generally asserts that specific emission limits, testing and recordkeeping of emission inputs must be added to the permit. Although section III.A. of the petition (beginning on page 45) includes a broad legal background, the Petitioners do not apply that legal background to this section such that it is apparent that there is a legal basis for the specific objections requested by the Petitioners to the permits. The Petition simply lays out a general legal overview, and then specific facts, without any analysis connecting the two. *See, infra*, Section II “Statutory and Regulatory Framework.”

As noted earlier in this Order, the EPA has looked at a number of criteria in determining whether the petitioner has demonstrated noncompliance with the Act. *See Nucor II Order* at 7. With regard to these issues in the Petitions, the EPA concludes that the Petitioners have not met their burden of demonstrating that the permit is not in compliance with the Act, or that LDEQ lacked a reasoned basis for the work practices standards and related compliance conditions related to fugitive dust sources in the permit. Notably, although Petitioners cite to 15 separate units in the Petition (at page 54), the remainder of the discussion is focused on only a few of those units (except for the enclosure discussion). LDEQ provided a unit-specific response in the 2010 RTC, the vast majority of which is not referenced or discussed by Petitioners at all. 2010 RTC at 216-218, and 339. Further, instead of identifying applicable requirements that apply for this permit, Petitioners instead rely on a SCAQMD rule and staff report, neither of which are applicable requirements for Nucor or LDEQ. 2010 Petition at 56. Petitioners do not explain why they should apply, or why it would be informative in this circumstance, despite these items clearly not being applicable requirements in Louisiana, as well as the significant air quality and climatological differences between Southern California and Louisiana. Petitioners focus in the Petition on LDEQ’s statement that enclosing the piles would be infeasible; however, the Petitioners do not demonstrate that LDEQ’s statement in the 2010 RTC was unreasonable in light of requirements applicable to Nucor and Louisiana. While it is clear that the Petitioners might prefer the fugitive dust management approach of the SCAQMD rule and report for the Nucor facility, Petitioners do not explain why LDEQ’s analysis is unreasonable under the applicable requirements that actually apply to the Nucor facility.

In addition, some of Petitioners’ contentions appear simply inaccurate. For example, Petitioners contend that the DMP is unenforceable, but the document is specifically referenced in the pig iron title V permit, (Condition 937 at 91), and includes specific, enforceable requirements. *See, e.g.*, Section 9.0 - 12.0 of the DMP (EDMS Document ID 6462271, page 269-300)<sup>34</sup>. As to the

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<sup>33</sup> See EDMS Document ID 6462271 (June 29, 2009), at 269.

<sup>34</sup> The EPA notes that the DMP is attached to the DRI title V permit at 61-92 and the DRI PSD permit at 84-115.

claim that the DMP is not adequate to ensure that 90-95 percent control efficiencies are met, Petitioners do not respond to statements made by LDEQ regarding that issue – which addressed technical information regarding AP-42<sup>35</sup> and provided a reasoned basis for LDEQ’s conclusions. 2010 RTC at 163-166. Finally, it does not appear that the specific concerns in the petition about the DMP (e.g., that monitoring only occurs weekly with a handheld monitor and that the thresholds are very high) were raised with reasonable specificity during the comment period. *See, e.g.*, 2010 RTC at 102. Nor is there any demonstration in the Petitions that it was impracticable to do so or that the grounds arose after the comment period. Thus, to the extent that the Petitioners are raising a separate issue on the DMP, rather than simply addressing LDEQ’s RTC on this point, this claim is also denied on procedural grounds. CAA § 505(b)(2), 42 U.S.C. § 7661d(b)(2).

With regard to Petitioners’ statements regarding work practice standard and BACT, the applicable statute and regulations define BACT, in general, as an “emissions limitation (including a visible emissions standard)” and provide that if there are “technological or economic limitations on the application of measurement methodology to a particular emissions unit that would make the imposition of emission standard infeasible, a design, equipment, work practice, operational standard, or combination thereof, may be prescribed instead to satisfy the requirement” for BACT.<sup>36</sup> Thus, Petitioners’ statement that specific emission limits must be added to the permit does not address or analyze these provisions and does not demonstrate that the conditions for imposing a work practice standard are not met here. Notably, Petitioners cite to no applicable requirement for support of that statement. 2010 Petition at 55. Also, since fugitive sources, by definition, do not emit through stacks, vents, or other functionally equivalent openings, as an engineering matter, there would be no structure available to allow for a stack test or other direct measurement technique (e.g., Reference Test Method 5<sup>37</sup> or PM Continuous Emission Monitoring System – PM CEMS) to be conducted for such emissions units. The Petitioners did not cite or discuss any applicable requirements or guidance or describe any engineering or other factors that could lead a permitting authority or the EPA to provide different compliance provisions in a permit for these emission units. Further, Petitioners do not appear to recognize that information provided in this permit includes state-of-the-art ambient monitoring for fugitive emissions, such as additional monitoring and deposition gauges that can be conducted when there is no stack or equivalent structure available. *See, e.g.*, 2010 RTC at 163-166.<sup>38</sup> The permit imposes specific monitoring for fugitive emissions sources through the requirements of the DMP and LDEQ explains in the 2010 RTC how that monitoring is intended to provide for information from which numeric ambient emission levels can be calculated and application of work practices assured. *See, e.g.*, 2010 RTC at 165. These are all points raised by LDEQ in the 2010 RTC, which Petitioners state are inadequate, but Petitioners provide no further explanation as to why LDEQ’s reasoning is flawed as to that monitoring or how the permit fails to assure compliance with an applicable requirement.

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<sup>35</sup> AP-42 refers to an EPA document, *Compilation of air Pollutant Emissions Factors*, available at <http://www.epa.gov/ttnchie1/ap42/>.

<sup>36</sup> *See* CAA § 169(3), 42 U.S.C. § 7479(3); *see also* 40 C.F.R. § 51.166(b)(12), LAC 33:III.509.B.

<sup>37</sup> *See, e.g.*, 40 C.F.R. Part 60, Appendix A. Hereafter we refer to this as “Test Method 5.”

<sup>38</sup> In addition, the EPA notes that the DMP now includes ambient dust monitoring requirements (deposition gauges and visible inspection) in § 11.1 (See DMP attached to the DRI title V permit).



Concerning haul roads and storage piles, the Petitioners contend that the work practice standards selected by LDEQ are not adequate to ensure that control efficiencies are actually met. For example, Petitioners appear to suggest that handheld monitors of “unknown quality” used by a person of “unknown skill” would not be reliable. 2010 Petition at 57. In response to comments, LDEQ explained that in addition to the monitoring,<sup>39</sup> the DMP applies during construction and operation of the facility, and requires actual monitoring of dust with deposition gauges, portable monitors, and visual inspections. *See* §§ 9.0 - 12.0 of the DMP. The record also shows that these haul roads are required as BACT to be paved, where practicable, watering and sweeping are required on paved roads, along with reduced speed limits. *See* Permit No. 2560-0081-V0 at 71. Unpaved roads are required as BACT to utilize water sprays or dust suppression chemicals and reduced speeds of 15 mph will be enforced. *Id.* For storage piles, the Petitioners focus on enclosing such piles without explaining why the existing conditions to control emissions from the piles are inconsistent with applicable requirements. Thus, Petitioners make various claims but do not explain how the permit fails to assure compliance with the applicable requirements in light of LDEQ’s response and the permit terms and conditions, including the DMP.

The EPA concludes that the Petitioners have not met their burden of demonstrating that the conditions in the permit are not in compliance with the Act, or that LDEQ lacked a reasoned basis for the work practice standards and associated compliance conditions established in the title V permit for the listed fugitive emission units. For these reasons, the EPA denies these claims.

### **3. Testing Once Over Facility Lifetime Is Inadequate**

*Petitioners’ Claims.* The Petitioners provide a list of seven emission units<sup>40</sup> and eleven applicable permit conditions for which they claim the permit requires only an initial stack test. 2010 Petition at 58. Petitioners state that such testing is not consistent with ensuring that limits are met on a continuous basis. *Id.* Petitioners additionally assert that the monitoring provisions mentioned in LDEQ’s RTC do not measure emissions and thus do not assure compliance with emission rates expressed in pounds per hour such as those used in air quality monitoring. *Id.* at 59. Petitioners do not agree with using MACT or Compliance Assurance Monitoring (CAM) to assure compliance with PSD emission limits because they are separate statutory programs. *Id.* With regard to using parameters in monitoring, Petitioners assert that such monitoring is not adequate unless the permit explicitly requires this monitoring and makes it enforceable, and states that an exceedance of an indicator is a violation of the underlying requirement. 2010 Petition at 60.

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<sup>39</sup> The handheld monitors referenced in the Nucor permit record represent a state-of-the-art monitoring device intended to provide real-time, numeric data for fugitive emissions. These monitors are considered to provide more reliable data than other fugitive monitoring devices. Petitioners do not explain why they believe such devices are not reliable. A later revision of the DRI permit revised the DMP to remove the deposition gauges and replace them with fenceline ambient monitors. *See* “Technical Review Comments, EDMS Document ID 916627. The fenceline monitors are another kind of ambient monitor for fugitive dust.

<sup>40</sup> The EPA recognizes that the pig iron title V permit has been modified since the Petitions. To the extent that none of the current Nucor title V permits any longer contain an emission unit identified by the Petitioners that is subject to a “grant” in this Order, LDEQ could respond to the “grant” in this Order associated with a unit by simply explaining that the unit no longer exists at the Nucor facility and identifying the permitting action that eliminated that unit from the Nucor permits.

These claims were re-raised in the 2012 Petition, Att. B at 58-60.

*EPA's Response.* For the reasons provided below, the EPA grants these claims.

In response to comments, LDEQ explained that the Petitioners' contention ignores the other provisions applicable to these sources. 2010 RTC at 219. For example, LDEQ points out that the coke battery process area units (COK-110 and 210) are subject to 40 C.F.R. Subparts L and CCCCC, which regulate particulate emissions and require additional monitoring recordkeeping and reporting. *Id.* Slag baghouse vents (SLG-403 and 409) are controlled via baghouses subject to CAM. Slag Mill process area units (SLG-103 and 203) are subject to monitoring and recordkeeping for flow rate and slag diverted during each event and required to keep records of such, as well as opacity limits and daily monitoring under 40 C.F.R. Part 63, Subpart FFFFF. *Id.* The PCI mill vent (PCI-101) is subject to 40 C.F.R. 60, Subpart Y and baghouse monitoring conditions and it regulates particulate matter and contains additional monitoring, recordkeeping and reporting. *Id.* While LDEQ's response identifies additional monitoring, recordkeeping and reporting, it does not explain how the monitoring in the permit for each of these units assures compliance with the emissions limits in the permit – as was discussed earlier in response to Petition Claim III.B.1. The EPA therefore grants the Petition on this issue and directs LDEQ to explain how the monitoring, recordkeeping and reporting included in the permit are adequate to assure compliance with the numeric emission limits in the permit. As discussed previously in response to Petition Claim III.B.1., the EPA's understanding is that these federally enforceable limits apply at all times. As the EPA is seeking additional clarification from LDEQ regarding how the monitoring in the permit for each of these units assures compliance with the emissions requirements in the permit, the EPA is not addressing the Petitioners' additional statements relating to NESHAP, CAM, or parametric monitoring. However, the EPA notes that it has addressed similar monitoring claims in prior title V orders. *See, e.g., In the Matter of Wisconsin Pub. Serv. Corp. JP Pulliam Power Plant*, Order on Petition No. V-2012-01 (Jan. 7, 2013) at 13-15 ( "Whether a permit contains adequate monitoring to assure compliance is fact-specific, depending on all of the relevant monitoring provisions in each title V permit. The fact that certain indicator ranges in one title V permit must be enforceable to assure compliance with an applicable requirement in that permit does not necessarily speak to whether indicator ranges in other title V permits must be enforceable."). As LDEQ considers this issue, if LDEQ determines that the permit does not include the necessary monitoring requirements, the EPA directs LDEQ to include such requirements in the permit. *See, e.g., Wheelabrator Order* at 10. For these reasons, the EPA grants these claims.

#### **4. Testing Every 2.5 or 5 Years Is Inadequate**

*Petitioners' Claims.* The Petitioners provide a list of 11 emission units and 22 permit conditions for which they claim that the draft title V permit only requires testing initially and then every five years. 2010 Petition at 60. Petitioners claim that it is feasible to "monitor" pollutants from these sources more frequently than once every five years. *Id.* at 61. The Petition lists two additional units and four additional conditions for which they claim the permit requires testing only every 2.5 years and state that clarification is needed to understand the timing for the testing. *Id.* Again, Petitioners claim that more frequent monitoring is feasible for these units. *Id.* For both

sets of emission units, the Petitioners state that unless LDEQ modifies the permit to require monitoring sufficient to assure compliance at these sources, the permit will be unlawful. *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 60-61.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

In its response to comments, LDEQ pointed out specific testing requirements that do apply to the identified units in the response to comments. 2010 RTC at 220-221. Specifically, LDEQ clarified that “Nucor must test the sources in question *every* 2.5 or 5 years, not just twice.” *Id.* Further, LDEQ then provided a unit-by-unit response detailing the monitoring and testing applicable to the various units identified by Petitioners. LDEQ pointed out that most of these units are subject to additional monitoring, recordkeeping and reporting requirements under 40 C.F.R. Part 63, Subparts Y, CCCCC, FFFFF, and D. 2010 RTC at 221. Further, LDEQ pointed out that the MEROS System Vent Stack (unit SIN-101) is subject to SO<sub>2</sub> continuous emission monitoring among other monitoring. *Id.*; *see also* pig iron title V permit at 31 (unit EQT-0031).

The claim, in one sentence, references the response to comments (on the frequency of testing), but provides no substantive response to LDEQ's statements. In particular, the Petitioners provide no reply to LDEQ's unit-by-unit response explaining the monitoring for the identified units. In addition, Petitioners appear to simply reiterate their testing frequency claim without regard to LDEQ's clarifying explanation in the 2010 RTC. Petitioners' claims appear focused on the performance testing and do not appear to consider any of the other testing and monitoring for the identified sources. Petitioners' claims begin with a discussion of “testing” and conclude by finding that the “monitoring” is inadequate – without consideration of the monitoring required at each of the identified units. Instead, these claims are conclusory in nature and do not provide the EPA with any information indicating the specific basis for the objection, such as an explanation of why the Petitioners believe more frequent testing or monitoring would be required for these emission units. *See, e.g., Murphy Oil Order* at 12 (denying a title V petition claim where petitioners did not cite any specific applicable requirement that lacked required monitoring). As discussed earlier, the burden is on the petitioner to address the permitting authority's final decision, and the permitting authority's final reasoning (including the RTC). *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Nucor II Order* at 7. For these reasons, the Petitioners fail to explain how LDEQ's rationale was deficient or how the permit fails to assure compliance with an applicable requirement. For these reasons, the EPA denies these claims.

## **5. Continuous Emission Monitoring System (CEMS) Requirements Are Unclear**

*Petitioners' Claims.* The Petitioners contend that certain draft title V permit conditions related to CEMS requirements for Coke Battery Flue Gas desulfurization (FGD) stacks and the sinter plant FGD stacks are “unclear as to whether the CEMS data will be used to determine compliance and if so, exactly how and with what limitations.” 2010 Petition at 61. The Petitioners provide a specific example of the SO<sub>2</sub> CEMS condition for the Coke Battery 1 and 2 FGD Stacks (Conditions 216 and 245) and claim that the permit conditions regarding CEMS are unclear whether inlet and outlet concentrations be monitored, which they assert is required to determine compliance with the SO<sub>2</sub> control efficiency used as BACT. *Id.* at 62. Petitioners also state that

the permit is silent as to whether the CEMS data will be used to determine compliance with any emission limits, or be reported to the agency, contending that the CEMS data should be used to determine compliance with all relevant emissions limits and submitted quarterly in an electronic file and reported to LDEQ. *Id.* Petitioners contend that all required emissions limits must be clearly mandatory. *Id.* Petitioners contend that the provision associated with the CEMS for the MEROS System Sinter Vent Stack (Condition 325) are ambiguous because it requires continuous recordkeeping by CEMS, rather than monitoring on a specific timeframe. *Id.* at 62-63. Finally, Petitioners assert that LDEQ's response to the comments was not on point because it "failed to show how the CEMS data will be used to determine continuous compliance with the SO<sub>2</sub> emission rates in the Criteria Pollutant Emission Rate table." *Id.* at 63.

These claims were re-raised in the 2012 Petition, Att. B at 61-63.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

In response to comments, LDEQ provided direct answers to the Petitioners' claims. 2010 RTC at 222-223. Specifically, LDEQ explained that "CEMS data will be used to determine compliance with all relevant SO<sub>2</sub> emission limits and conditions." *Id.* As part of this response, LDEQ also cited the EPA's Credible Evidence Rule, 62 *Fed. Reg.* 8314 (Feb. 24, 1997) and General Conditions under LAC, including certain requirements relating to recordkeeping and reporting. LDEQ provided cross-references to the LAC provisions and permit conditions responsive to Petitioners' claims, pointing out specific answers and responding to inaccurate statements made by Petitioners. 2010 RTC at 223. LDEQ also responded to Petitioners' statement about the CEMS for the MEROS System Sinter Vent Stack (unit SIN-101), stating that emissions must be monitored once every fifteen minutes per LAC 33:III.1511.A. 2010 RTC at 223. As noted by LDEQ, the CEMS and other monitoring for the units identified by Petitioners will provide sufficient information to determine compliance with the limits.

Petitioners' claims do not appear to respond to the direct and reasoned responses provided by LDEQ in the 2010 RTC; rather, Petitioners reference the response by saying it is "not on point." 2010 Petition at 63. This statement is simply inaccurate – LDEQ directly responds to Petitioners' claims in the 2010 RTC and provides additional explanation regarding the issues identified by Petitioners. Petitioners additionally state that LDEQ "failed to show how the CEMS data will be used to determine continuous compliance with the SO<sub>2</sub> emission rates in the Criteria Pollutant Emission Rate table." *Id.* However, they do not explain why LDEQ's statements, including the statement in the RTC that "CEMS data will be used to determine compliance with all relevant SO<sub>2</sub> emission limits and conditions," 2010 RTC at 222, are inadequate to address this concern. Further, Petitioners do not cite or analyze any applicable requirements for support of their specific claims regarding monitoring and CEMS. As discussed earlier, the burden is on the petitioner to address the permitting authority's final decision, and the permitting authority's final reasoning (including the RTC). *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments and has looked to whether they have provided relevant citations and analyses to support the claim). Accordingly, the Petitioners fail to explain how LDEQ's rationale was deficient or how the permit fails to assure compliance with an applicable requirement. For these reasons, the EPA denies these claims.

## 6. Filter Manufacturer's Certifications

*Petitioners' Claims.* The Petitioners state that BACT for PM<sub>10</sub> and PM<sub>2.5</sub> for many sources was determined to be fabric filter baghouses designed to meet a particular control efficiency, and contend that the draft title V permit does not require any testing to demonstrate that the BACT control efficiency (and sometimes corresponding grain loadings) are met each day but rather requires only a filter manufacturer's initial certification. 2010 Petition at 63. Petitioners explain that baghouse performance can degrade over time and that operations can modify the performance of the baghouse. *Id.* Citing to the NSR Manual, Petitioners conclude that a vendor certification does not assure continuous compliance with a BACT limit expressed as a control efficiency. *Id.* Petitioners contend that testing would verify compliance but the draft title V permit does not require any testing to verify the baghouse control efficiencies, except for some units for which initial outlet testing using the EPA's Test Method 5 is provided. *Id.* at 64. Petitioners state that simultaneous testing at both the baghouse inlet and outlet would be required to verify compliance with the control efficiency. *Id.* Petitioners then identify 23 permit conditions which Petitioners state rely only on a manufacturer's certification for compliance with the control efficiency. *Id.* Petitioners recognize that some of these units must also undergo testing under the EPA's Test Method 5, but Petitioners appear to contend this is not adequate to determine control efficiency because it does not measure either PM<sub>10</sub> or PM<sub>2.5</sub>. *Id.*

These claims were re-raised in the 2012 Petition, Att. B at 63-65.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.<sup>41</sup>

In response to comments, LDEQ explained that the permit requires Nucor to maintain purchase orders or manufacturer certifications showing that the installed filters meet the manufacturer's specifications for particulate matter removal efficiency, or the Minimum Efficiency Reporting Value (MERV) rating, as applicable. 2010 RTC at 225. Further, LDEQ explained the basis for its BACT decision by pointing to an EPA-issued PSD permit in which compliance with a permit condition was demonstrated by certification of the engine manufacturer. *Id.* LDEQ also explained why a MERV rating is not the equivalent of a traditional vendor guarantee because it represents the worst case performance, it can assure performance when a maximum particle count must be maintained over the filter's entire life. *Id.* Finally, LDEQ notes that additional monitoring is associated with the baghouses – such as daily visible emissions monitoring, baghouse (including gasket) inspections every six months, and good air pollution control practices. *Id.* The EPA additionally notes that the Permit itself includes numerous parameter monitoring requirements for the units identified by Petitioners, including monitoring for temperature, pressure drop, and visible emissions – all parameters which provide information regarding the operations of a baghouse. *See, generally, EPA Air Pollution Control Cost Manual*, Sixth Edition, EPA/452/B-02-001, January 2002, Section 6 Particulate Matter Controls, Chapter 1 Baghouse Controls and Filters, Section 1.2.6, Fabric Filtration Theory (available online at <http://www.epa.gov/ttn/catc/dir1/cs6ch1.pdf>).

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<sup>41</sup> Petitioners' claims as to the adequacy of Test Method 5 measurements to ensure that the baghouse is performing at the BACT level for PM<sub>10</sub> and PM<sub>2.5</sub> are addressed in the response to Claim III.E below.

In the Petition, Petitioners do not acknowledge LDEQ's response nor address why LDEQ's response in the 2010 RTC is unreasonable or why the monitoring and inspection measures described in the permit fail to assure compliance with the BACT requirement for control of PM with baghouses. In addition, while Petitioners focus on the need for requirements for inlet and outlet testing of control efficiency, this would not necessarily provide information concerning the proper operation and maintenance of the filters that would be superior to that gained from the provisions in the permit for parameter monitoring of pressure drop and visible emissions, and regular inspection of filter conditions to check for bag leaks and filter condition. The Petition neither discusses LDEQ's response nor provides any citation to or analysis of any relevant requirements to support the contentions. As discussed earlier, the burden is on the petitioner to address the permitting authority's final decision, and the permitting authority's final reasoning (including the RTC). *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments and has looked to whether they have provided relevant citations and analyses to support the claim). For these reasons, the Petitioners fail to explain how LDEQ's rationale was deficient or how the permit fails to assure compliance with an applicable requirement. For the reasons described above, the EPA denies these claims.

#### **7. There Is Inadequate Monitoring for PM<sub>10</sub> or PM<sub>2.5</sub>.**

*Petitioners' Claims.* Petitioners contend that the permit lacks adequate monitoring to assure compliance with the PM<sub>10</sub> and PM<sub>2.5</sub> BACT analyses. 2010 Petition at 65. Pointing to numerous monitoring provisions in the permit, Petitioners state that all of the monitoring provisions in the draft title V permit require the use of the EPA's Test Method 5 to determine compliance with the PM<sub>10</sub> and PM<sub>2.5</sub> limits. *Id.* Petitioners explain that Test Method 5 is not adequate because it excludes condensable particulate matter, which is a component of both PM<sub>10</sub> and PM<sub>2.5</sub>, and because it measures all sizes of particulate matter. *Id.* Petitioners state that the draft title V permit therefore requires no testing for any PM<sub>10</sub> and PM<sub>2.5</sub> BACT limits and seem to indicate that the permit should require use of the EPA's Test Methods 201, 202 and Other Test Method (OTM) 27. *Id.* Further, Petitioners contend that LDEQ failed to respond to Petitioners' PM<sub>10</sub> comment and state that the permit does not include a requirement to test condensables for PM<sub>10</sub>. *Id.* at 66.

These claims were re-raised in the 2012 Petition, Att. B at 65-66.

*EPA's Response.* For the reasons provided below, the EPA denies these claims.

As a preliminary matter, many of the Petitioners' statements in this part of the Petition appear to be inaccurate. For example, Petitioners state that "the draft permit requires no testing for any BACT limits for PM<sub>10</sub> or PM<sub>2.5</sub>." 2010 Petition at 65. However, the permit itself includes numerous monitoring and testing requirements associated with PM. For example, the permit includes monitoring for visible emissions, pressure drop, and other parameters to assure that PM control devices are functioning effectively. *See, e.g.,* pig iron title V permit (COK-100), ARE-0001.

With regard to Petitioners' contention that LDEQ "failed to respond" to Petitioners PM<sub>10</sub> "argument," and citing to the 2010 RTC (LDEQ Response 258.E) (2010 Petition at 66), the information in the RTC appears to directly contravene Petitioners' contention. In the 2010 RTC

at 226-228, LDEQ provides a response to Petitioners' comments. In response to comments, LDEQ explained that due to the surrogacy demonstration it provided for PM<sub>10</sub> being a surrogate for PM<sub>2.5</sub>, LDEQ "believes it is reasonable for Nucor to assume all particulate matter emissions constitute PM<sub>10</sub>." 2010 RTC at 226. LDEQ also noted that Nucor may use other methods with prior approval from LDEQ. *Id.* Apart from the surrogacy issue that is addressed elsewhere in this Order, the Petitioners do not address the substantive points that LDEQ made in the RTC regarding monitoring for PM<sub>10</sub>, but rather simply state that PM<sub>10</sub> includes filterable and condensable components and that the permit only requires testing of the filterable component by Test Method 5, "which overestimates," but is silent as to the requirement to test condensables for PM<sub>10</sub>. 2010 Petition at 66.

As discussed earlier, the burden is on the petitioner to address the permitting authority's final decision, and the permitting authority's final reasoning (including the RTC). *See, e.g., MacClarence*, 596 F.3d at 1132-33; *Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments and has looked to whether they have provided relevant citations and analyses to support the claim). The language in the Petition appears to essentially be a copy of the comments submitted to LDEQ, with no substantive engagement with LDEQ's response, which includes citations to applicable legal requirements and explains the basis for the approach in the pig iron permit and why it was not required to address condensable PM at the time the pig iron permit was issued.

With regard to condensable PM, LDEQ explained the status of the applicable requirements, quoting from excerpts from both final and proposed rulemakings, which explain why Nucor was not obligated to include condensable PM in its analysis of compliance with the PM limits in the pig iron title V permit. 2010 RTC at 226 (citing to the PM<sub>2.5</sub> NSR Implementation Rule and the EPA's proposed rule titled, "*Methods for Measurement of Filterable PM<sub>10</sub> and PM<sub>2.5</sub> and Measurement of Condensable Particulate Matter Emissions from Stationary Sources*," 74 *Fed. Reg.* 12970 (March 25, 2009)). The Petition, however, fails to address the statements by LDEQ in the RTC explaining that the EPA will not require "states to address condensable PM in establishing enforceable emissions limits for either PM<sub>10</sub> or PM<sub>2.5</sub> in NSR permits until the completion of a transition period, currently scheduled to end on January 1, 2011," which is after the pig iron permits were issued. 2010 RTC at 226.

For the above reasons, the Petitioners fail to explain how LDEQ's rationale was deficient or how the permit fails to assure compliance with an applicable requirement. For these reasons, the EPA denies these claims. As is discussed below, the EPA is also denying in part (and granting in part on a separate issue) the Petition on the issues raised by Petitioners regarding PM<sub>10</sub> and PM<sub>2.5</sub> surrogacy with respect to the pig iron permit.

## **8. Cooling Tower BACT Limits Do Not Assure Compliance.**

*Petitioners' Claims.* Petitioners raise two main issues associated with the cooling tower total dissolved solids (TDS) concentration. 2010 Petition at 66. Petitioners contend that they commented that cooling water with a TDS concentration of less than 500 parts per million (ppm) was feasible. *Id.* Additionally, Petitioners contend that LDEQ did not respond to the substance of this comment and that the BACT determination is therefore deficient. *Id.* Second, Petitioners

contend that the title V permit fails to assure that the cooling tower BACT limits are enforceable. *Id.* Petitioners explain that LDEQ did respond to these comments, and even made changes to the permit in response to the comments, but that the changes fall short of what Petitioners contend was necessary. *Id.* With regard to Conditions 846, 849, and 855, Petitioners now contend that these should state, “BACT is the use of a cooling tower equipped with a 0.0005 percent efficient drift eliminator,” rather than “mist eliminating baffles.” 2010 Petition at 67. With regard to enforceability, Petitioners contend the permit should be modified to require at least an initial test to confirm the drift efficiency, mandatory maintenance and work practice standards to assure the BACT drift efficiency is met, and continuous flow rate monitoring, and that these values should be used in the calculation to determine compliance rather than vendor guarantees, stating that “‘vendor guarantee[s] alone are not sufficient justification that a control option will work.’” *Id.* (quoting the NSR Manual at B.20).

These claims were re-raised in the 2012 Petition, Att. B at 66-67.

*EPA’s Response.* For the reasons provided below, the EPA and denies these claims.

LDEQ responded to these issues in two portions of the response to comments document. First, in response to Comment No. 115, LDEQ explained that it conducted a top-down BACT analysis to reach the BACT related determinations associated with the cooling towers. 2010 RTC at 101. LDEQ also explained that its analysis of a lower TDS, which was informed by additional information submitted by Nucor (which is cited to in the 2010 RTC), did not result in any changes to the previous BACT determination. *Id.* The additional information, titled, “Addendum to Part 70 and PSD Permit Application,” and dated January 6, 2009, discusses the BACT analysis for the TDS on pages 7-8 (EDMS Document ID. No. 6271972) (hereafter referred to as “Addendum”). This document explains Nucor’s position that the BACT analysis was based on a fundamental design of the facility – that it would be a zero wastewater discharge facility. Addendum at 7. Specifically, Nucor explained, “[i]n considering BACT for the coke quench towers, control options were identified within the constraints of the zero-discharge facility design. The nature of this facility-wide water system prohibits the use of quench water with a TDS concentration below 1,100 ppm. The concentration of dissolved solids in the water may be reduced by water treatment, but such treatment creates a concentrated water blow downstream, which must be discharged. Additionally, fresh make-up water requirements of the tower increase.” *Id.* at 7-8. Nucor then concluded that it “considers the requirement for use of an ultra-low TDS quench water, or the use of a once-through quench water system, to be technically infeasible within the constraints of the fundamental facility design.” *Id.* at 8.

In a later portion of the 2010 RTC (in response to Comment No. 258.F), LDEQ provided additional information responsive to Petitioners’ comments. 2010 RTC at 228. Specifically, LDEQ explained that BACT limitations such as those suggested by the commenter (e.g., exhaust gas concentration or percent reduction) are not appropriate due to technical aspects of the particulate emissions at issue. *Id.* at 228-229. LDEQ also explained that it would add conditions to the permit, in response to Petitioners’ comments asking LDEQ to provide for more clarity surrounding the established design drift efficiency, compliance, and monitoring. *Id.* Among other things, LDEQ included additional monitoring involving the circulating water rate and also additional recordkeeping. *Id.*



The record shows that LDEQ did provide a response to the Petitioners' comment about a 500 ppm TDS limit on the cooling water. In the RTC, LDEQ stated that it had done a top-down BACT determination which led to the selection of "a combination of less than or equal to 1,000 milligrams per liter TDS concentration in the cooling water and drift eliminators employing a drift maximum of 0.0005%" as BACT. 2010 RTC at 101. LDEQ further stated that "Nucor submitted additional explanation regarding the use of a lower TDS value specifically addressing collateral environmental impacts," and then stated that "LDEQ's review of the additional information concluded that the original determination of BACT was correct." *Id.* Thus, LDEQ did respond to Petitioners' comment about a 500 ppm TDS limit on cooling water. Moreover, while LDEQ's response to the comment is not as clear or detailed as it could be, the basis for LDEQ's conclusion can reasonably be discerned from the response. *See, Alaska Dep't of Env'tl. Conservation v. EPA*, 540 U.S. 461, 497 (2004) (a decision of "less than ideal clarity" should be upheld "if the agency's path may reasonably be discerned") (quoting *Bowman Transp., Inc. v. Arkansas-Best Freight System, Inc.*, 419 U.S. 281, 285-286 (1974)). As discussed above, the information submitted by Nucor explained that achieving a lower TDS level in the quench water would have collateral environmental impacts. This option would require treatment of the quench water to remove TDS and thus produce a discharge of wastewater that was otherwise avoided by Nucor's zero-discharge design. The discharge of water would also require additional consumption of fresh water to make up for the water lost to the discharge. LDEQ's response shows that the agency considered the additional information submitted by Nucor on collateral environmental impacts and then determined the original BACT determination was correct on the basis of this information. The Petitioners do not provide any information or analysis to demonstrate that this response, or LDEQ's determination, is deficient or unreasonable.

With regard to the remaining issues raised in this portion of the Petition, the EPA denies those issues. In the remaining issues of the Petition, Petitioners take issue with specific changes made by LDEQ to the permit, but fail to cite to or analyze any relevant requirement indicating that the changes now requested by Petitioners are required to assure compliance with an applicable requirement. In addition, Petitioners do not address all the changes made by LDEQ in response to the comments, including particularly relevant ones. For example, the Petitioners continue to raise the need for continuous monitoring of circulating water flow rate (2010 Petition at 67), but do not discuss the monitoring in the permit for the cooling water. *See, e.g.*, modified pig iron title V permit, unit EQT-0060, condition 746. While the EPA understands that the Petitioners may prefer different wording or conditions, Petitioners have not demonstrated that the permit as drafted fail to assure compliance with an applicable requirement. For example, as to the drift efficiency, Petitioners state that the title V permit should include "mandatory maintenance and work practice standards to assure the BACT drift efficiency is maintained" but do not acknowledge or address LDEQ's change to the permit to require that the cooling tower drift eliminators be maintained consistent with the manufacturer's recommendation as described in the operating manual for the cooling tower, including a requirement to maintain a log of maintenance activity performed on the drift eliminators as well as the additional monitoring on the cooling water. 2010 RTC at 229.

Similarly, the Petitioners state that the vendor guarantees alone do not assure compliance over the lifetime of the equipment and state that the title V permit should contain "continuous flow rate monitoring," but they do not acknowledge or address LDEQ's explanation that "[u]se of the

design cooling tower circulating water rate will result in conservative emission estimates and negates the need to monitor this parameter.” *Id.* The Petition does not provide information demonstrating that LDEQ’s response or approach was deficient or unreasonable, or that the permit fails to assure compliance with a requirement of the Act. As discussed earlier, the burden is on the petitioner to address the permitting authority’s final decision, and the permitting authority’s final reasoning (including the RTC). *See MacClarence*, 596 F.3d at 1132-33; *See Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state’s final decision, including response to comments and has looked to whether they have provided relevant citations and analyses to support the claim).

For these reasons, the EPA denies claims.<sup>42</sup>

## **V. EPA DETERMINATIONS ON ISSUES ORIGINALLY RAISED BY THE PETITIONERS ON MODIFIED PIG IRON AND DRI PERMITS FROM THE 2011 PETITION**

### **A. The EPA Must Object to the Title V Permit For the Pig Iron Process Because the Permit Fails to Apply MACT Standards for the Topgas Boilers**

These claims are addressed above. *See* Petitioners’ Claims and the EPA Response (deny) in Section IV.A.1, *supra* at 11-13. *See* 2011 Petition at 7; 2012 Petition, Att. C at 7; *Public Comment Response Summary, Part 70 Operating Permit 3086-VO and Prevention of Significant Deterioration (PSD) Permit PSD-LA-751, and Part 70 Operating Permit Modification 2560-0028-VI*, January 27, 2011, EDMS Document ID 7806737 (hereafter the “2011 RTC”), 2011 RTC at 8.

### **B. The EPA Must Reject the Permits Because LDEQ Failed to Include Emission Limits for PM<sub>2.5</sub>**

*Petitioners’ Claims.* The Petitioners contend that LDEQ failed to include limits for PM<sub>2.5</sub> emissions in either the title V permit for the pig iron process or the PSD permit for the DRI process and failed to provide an appropriate analysis for PM<sub>2.5</sub>. The Petitioners state that the EPA

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<sup>42</sup> In addition, with respect to the 2010 Petition as a whole, the EPA notes that the Petitioners have generally incorporated by reference comments offered by certain commenters during the public comment period into the 2010 Petition. 2010 Petition at 1. The EPA notes that the scope of the intended incorporation is not clear because the Petitioners state that they “incorporate by reference their comments” but several commenters are named in the preceding sentences, including Sierra Club and LEAN, to whom this reference could refer. *Id.* In addition, the mere incorporation of comments into the Petition, without any attempt to explain how these comments relate to the argument in the Petition and without any attempt to address the state’s final permitting decisions and the reasoning supporting those decisions, including any response to comments by the state, is not sufficient to meet the demonstration standard. *See MacClarence*, 596 F.3d at 1132-33; *see generally Nucor II Order* at 7 (discussing demonstration burden). The Petitioners do not assert that LDEQ failed to respond to the incorporated comments, nor do they point to any flaw in any of LDEQ’s responses. The EPA also notes that this Petition states that it “adopt[s] and incorporate[s] by reference, as if fully set forth herein, the comments, facts, and arguments set forth in the Petition for EPA Objection filed by Zen-Noh Grain Corporation on June 25, 2010.” 2010 Petition at 1, 4. To the extent that Petitioners have incorporated by reference Zen-Noh’s 2010 Petition, the EPA has already responded to that petition, granting an objection in the *Zen-Noh Order*, and LDEQ has issued a response to the EPA’s objection. Thus, the EPA need not further address Zen-Noh’s 2010 Petition in this Order.

must object to the title V permit for each plant for the failure to include such limits as they are applicable requirements for PSD. 2011 Petition at 8-9. The Petitioners state that LDEQ concluded that PM<sub>10</sub> is an adequate surrogate for PM<sub>2.5</sub>, but failed to provide a case-specific demonstration that the use of PM<sub>10</sub> as a surrogate is reasonable under the facts and circumstances of the permits. *Id.* at 9. The Petitioners explain that in 1997, the EPA set forth an interim policy that allowed permitting authorities to use PM<sub>10</sub> as a surrogate for PM<sub>2.5</sub> where it proved administratively impracticable to address PM<sub>2.5</sub> due to technical and information deficiencies, but that in 2008, the EPA announced that the technical difficulties has been largely resolved. *Id.* at 9. For support, Petitioners explain that in 2009, the EPA issued an order that permitted the continued use of the PM<sub>10</sub> surrogate policy where the permit applicant provided a case specific demonstration that such use is reasonable under the facts and circumstances of the case. *Id.* at 9 (citing *LG&E Order* at 42, 44. In order to use the PM<sub>10</sub> surrogate policy, the Petitioners explain that the demonstration must include: “(1) a showing of sufficient correlation between the plant’s PM<sub>10</sub> and PM<sub>2.5</sub> emissions so as to provide ‘confidence that the statutory requirements will be met for PM<sub>2.5</sub> using the controls selected through a PM<sub>10</sub> NSR analysis’ and (2) a showing ‘that the degree of control of PM<sub>2.5</sub> by the control technology selected in the PM<sub>10</sub> BACT analysis will be at least as effective as the technology that would have been selected of a BACT analysis specific to PM<sub>2.5</sub> had been considered.’” *Id.* (citing *LG&E Order* at 45). For additional support, the Petitioners cited to several court decisions addressing surrogacy, as well as statements by the EPA. *Id.* (citing *LG&E Order* at 42, 44; *National Lime Assoc. v. EPA*, 233 F.3d 625, 637 (D.C. Cir. 2000); *Mossville Envtl. Action Now v. EPA*, 370 F.3d 1232, 1242–43 (D.C. Cir. 2004); Letter from Stephen L. Johnson to Paul Cort (Jan. 14, 2009) at 3).

These claims were re-raised in the 2012 Petition, Att. C at 8-10.

*EPA’s Response.* For the reasons provided below, the EPA grants in part and denies in part these claims.

The EPA issued the first PM<sub>2.5</sub> NAAQS in a 1997 revision to the suite of particulate matter NAAQS. 62 *Fed. Reg.* 39852 (July 28, 1997). That same year, the EPA issued a memorandum outlining what came to be known as the PM<sub>10</sub> Surrogate Policy, wherein sources would be allowed to use implementation of a PM<sub>10</sub> program as a surrogate for meeting PM<sub>2.5</sub> NSR requirements until certain technical difficulties could be resolved. *See* Memo from J. Seitz to EPA Division Directors, *Interim Implementation for the New Source Review Requirements for PM<sub>2.5</sub>* (Oct. 23, 1997). In the PM<sub>2.5</sub> NSR Implementation Rule, the EPA acknowledged that many of the technical difficulties associated with implementing NSR for the PM<sub>2.5</sub> NAAQS had been largely resolved. *Id.* at 28, 340. However, in order to permit states sufficient time to adopt the revisions promulgated in the 2008 rule, the EPA explained that states with SIP-approved NSR permitting programs could continue to implement the PM<sub>10</sub> surrogate policy during the SIP development period. *Id.* at 28, 340-41.<sup>43</sup>

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<sup>43</sup> In May 2011, the EPA issued a final rule repealing the federal grandfathering provision that allowed sources permitted pursuant to the federal PSD program to rely on the PM<sub>10</sub> Surrogate Policy. 76 *Fed. Reg.* 28646. In the preamble to that rulemaking, the EPA also explained that “the 1997 PM<sub>10</sub> Surrogate Policy may not be used for any state PSD permits after the 3 years allowed for SIP development (ending May 16, 2011). With the end of the 1997 PM<sub>10</sub> Surrogate Policy in SIP-approved states on May 16, 2011, and the repeal of the grandfather provision in this final action, the 1997 PM<sub>10</sub> Surrogate Policy may not be relied on for any pending or future applications.” *Id.* at 28,648. The permits in this case were issued before the final repeal of the PM<sub>10</sub> Surrogate Policy.

The Petitioners correctly point out that the EPA issued guidance in 2009 in the *LG&E* Order which further clarified the use of the PM<sub>10</sub> Surrogate Policy. In that order, the EPA explained that “[a]pplicants and state permitting authorities seeking to rely on the PM<sub>10</sub> Surrogate Policy should consider [applicable court opinions regarding the use of surrogacy] in determining whether PM<sub>10</sub> serves as an adequate surrogate for meeting the PM<sub>2.5</sub> requirements in the case of the specific permit application at issue.” *LG&E* Order at 43. The EPA further explained its belief that “the overarching legal principle from these decisions is that a surrogate may be used only after it has been shown to be reasonable (such as where the surrogate is a reasonable proxy for the pollutant or has a predictable correlation to the pollutant).” *Id.* The EPA concluded that the cases addressing PM surrogacy in particular “demonstrate the need for permit applicants and permitting authorities to determine whether PM<sub>10</sub> is a reasonable surrogate for PM<sub>2.5</sub> under the facts and circumstances of the specific permit at issue, and not proceed on a general presumption that PM<sub>10</sub> is always a reasonable surrogate for PM<sub>2.5</sub>.” *Id.* at 44.

The EPA continued by suggesting two steps that could be used as a possible approach to making an appropriate surrogacy demonstration for the use of PM<sub>10</sub> as a surrogate for PM<sub>2.5</sub>. First, the EPA explained that the source or permitting authority should establish in the permit record a strong statistical relationship between PM<sub>10</sub> and PM<sub>2.5</sub> emissions from the proposed unit in order to demonstrate confidence that the statutory requirements will be met for PM<sub>2.5</sub> using the controls selected through a PM<sub>10</sub> analysis. *Id.* at 45. The EPA explained that this step should give reasonable consideration to “whether and how the PM<sub>2.5</sub>:PM<sub>10</sub> ratio may vary with source operating conditions, including variations in the fuel rate and in control equipment condition and operation.” *Id.* Second, the EPA explained that the source or permitting authority should demonstrate “that the degree of control of PM<sub>2.5</sub> by the control technology selected in the PM<sub>10</sub> BACT analysis will be at least as effective as the technology that would have been selected if a BACT analysis specific to PM<sub>2.5</sub> emissions had been conducted.” *Id.* The Petitioners correctly identified these factors, but they are incorrect to state that these factors are mandatory. Rather, the EPA explained that:

these two steps are not intended to be the exclusive list of possible demonstrations that a source or permitting authority would make to show that PM<sub>10</sub> is a reasonable surrogate for PM<sub>2.5</sub>. Sources and permitting authorities are encouraged to carefully consider the case law and the limits of the Surrogate Policy to determine what information and analysis would need to be included in the permit application and record before relying on the Surrogate Policy.

*Id.* at 46.

The Petitioners challenge the adequacy of the surrogacy demonstration made with respect to two different permits evaluated by LDEQ. The EPA will consider each permit in turn.

#### *The Modified Pig Iron Permit*

The EPA denies the Petitioners’ claims as to the modified title V permit for the pig iron process. The Petitioners’ objections as to this permit apply to provisions in the pre-existing pig iron title V permit that LDEQ did not change in the final modified title V permit. In particular, the BACT

analysis and surrogacy demonstration made as to the pig iron process are not related to the title V permit modification action. The EPA interprets its regulations to limit the scope of petitions to object in modification actions to issues that are directly related to the permit modification action. *See In the Matter of Wisconsin Public Service Corporation – Weston Generating Station*, Order responding to Petition number V-2006-4, 11-17 (Dec. 19, 2007) (“*Weston Order*”). Because the Petitioners’ petition on this claim is not directly related to the permit modification action, the EPA denies the petition on this issue. As the EPA explained in the *Weston Order*, the EPA interprets its Title V regulations at 40 C.F.R. Part 70 to limit petitions on significant modifications to issues directly related to those modifications. *Id.* “Therefore, in evaluating a petition objecting to a significant modification permit, EPA will determine based on the facts whether the issues raised by the petitioner are directly related to the permit modification action.” *Id.* at 17.

In this case, the record shows that the final modified title V permit for the pig iron process did not include any changes to the requirements for PM<sub>2.5</sub> or PM<sub>10</sub> contained in the NSR permit previously issued by LDEQ. Rather, in the modified title V permit, some units were eliminated entirely; permitted rates were changed for others to reflect (a) the elimination of those units, (b) changes in material throughput from the addition of the DRI units, and (c) the addition of SCR; and some units were transferred to the DRI permit. *See* modified pig iron title V permit, (Air Permit Briefing Sheet at 3). LDEQ explained in response to comments, its view that the modified title V permit reflects a significant decrease in PM<sub>10</sub> and PM<sub>2.5</sub> emissions, and did not propose new physical changes or changes in the method of operation of the pig iron process. 2011 RTC at 53; *see also* modified pig iron title V permit, (Air Permit Briefing Sheet at 3). Thus, in this permit modification, LDEQ did not revisit the BACT analysis and the surrogacy demonstration conducted for PM<sub>10</sub> and PM<sub>2.5</sub> in the previously-issued NSR permit. Accordingly, the Petitioners’ objection is not directly related to the modified title V permit.

In their petition, the Petitioners contend the modified title V permit for the pig iron process did not include PM<sub>2.5</sub> emission limits, but did not explain how this issue directly relates to the permit modification action. Therefore, the Petitioners have failed to demonstrate that this claim is appropriately raised at this time, and the EPA therefore denies these claims.

### *The DRI Permit*

The EPA grants the Petitioners’ claims as to the title V permit for the DRI process. The final permit contains an analysis of the appropriate control technology for both PM<sub>2.5</sub> and PM<sub>10</sub>. However, the emissions limits derived from this analysis are expressed only in terms of PM<sub>10</sub>. We note that Petitioners are not challenging the choice of control technology; rather, their claim is that the permit should also contain limits expressed in terms of PM<sub>2.5</sub> emissions. We are granting this claim because LDEQ has neither included PM<sub>2.5</sub> BACT emissions limits in the DRI permits nor provided a reasonable explanation for the use of the PM<sub>10</sub> emission limits as surrogates for compliance with PM<sub>2.5</sub> requirements.

As an initial matter, the EPA is unable to discern from the permitting record whether or not LDEQ intended to rely upon the PM<sub>10</sub> Surrogate Policy in the final DRI PSD permit. At one point in the permitting record, LDEQ acknowledged that the *LG&E Order* stated that, in order to

use the PM<sub>10</sub> Surrogate Policy, permit applicants and permit authorities should determine whether PM<sub>10</sub> is a reasonable surrogate for PM<sub>2.5</sub> under the specific facts and circumstances of the permitting action. *See Basis for Decision, Part 70 Operating Permit No. 3086-VO and Prevention of Significant Deterioration Permit No. PSD-LA-751*, EDMS Document ID 7806731, (January 27, 2011)(hereafter “2011 Basis for Decision”) at 15. LDEQ then stated that it had “addressed PM<sub>2.5</sub> emissions directly by determining the best available control technology for PM<sub>2.5</sub> and determining the proposed source’s impact on currently monitored PM<sub>2.5</sub> concentrations in relation to the current PM<sub>2.5</sub> NAAQS.” *Id.* This might suggest that LDEQ determined that it would not use the PM<sub>10</sub> Surrogate Policy in the final permit but instead address BACT for PM<sub>2.5</sub> directly. However, at another point in the permitting record LDEQ stated that “the PM<sub>10</sub> Surrogate Policy has been used to address PM<sub>2.5</sub> emissions” and that “additional research was performed in order to address BACT for PM<sub>2.5</sub>.” *See* DRI PSD permit at 12. LDEQ does not further describe this additional research or its role in the BACT analysis that followed.

The EPA commented on the draft title V and PSD permits, noting that “LDEQ’s record should justify why PM<sub>10</sub> is an adequate surrogate for PM<sub>2.5</sub> in this case.” 2011 RTC at 55-56. In response, LDEQ noted that “Nucor provided a top-down BACT analysis for PM<sub>2.5</sub> and the requisite modeling analyses to demonstrate that the facility’s emissions will not result in violations of the annual and 24-hour PM<sub>2.5</sub> NAAQS. As discussed during LDEQ’s conference call with EPA on December 14, 2010, LDEQ has agreed to include PM<sub>2.5</sub> limitations in the final permits.” *Id.* Accordingly, LDEQ’s response suggests that it did not intend to rely on the PM<sub>10</sub> Surrogate Policy in the final permit but that it would instead include PM<sub>2.5</sub> BACT emission limits.

The record demonstrates that Nucor conducted separate top-down BACT analyses for each unit at the DRI process for both PM<sub>10</sub> and PM<sub>2.5</sub>. *See* the permit application for the pig iron process, *Nucor Steel Louisiana Direct Iron Reduction Facility Part 70 Initial Permit and Authorization to Construct and PSD Permit Application*, Section 3.0 (August 2010)(EDMS Document ID 6952414). The state concluded in these analyses that the same control technology was appropriate for both PM<sub>10</sub> and PM<sub>2.5</sub> at each unit. *See id.* LDEQ subsequently conducted a combined BACT analysis for both PM<sub>10</sub> and PM<sub>2.5</sub> for each unit that resulted in the same conclusions regarding the appropriate BACT technology for each unit. *See* the DRI PSD permit (Preliminary Determination Summary) at 11-72; Specific Conditions at 76-78. The Petitioners have not raised specific objections to the BACT technology chosen for each unit as a result of these analyses. However, the EPA agrees with the Petitioners that the BACT emission limits associated with these units only address PM<sub>10</sub> as there are no emission limits listed for PM<sub>2.5</sub>. *See* Specific Conditions at 79-80.

Section 165(a)(4) of the CAA prohibits construction of a new or modified major source unless it “is subject to the best available control technology for each pollutant subject to regulation under the” Act. The Act further defines “best available control technology” in relevant part to mean an “emission limitation based on the maximum degree of reduction of each pollutant subject to regulation under the” Act which is emitted from the source. CAA § 169(3). Thus, in order to satisfy the statutory requirement, it is not sufficient for LDEQ to only identify the appropriate BACT technology for each pollutant. Rather, the permitting agency must also determine the appropriate emission limitation for each pollutant that would constitute BACT, including

emission limitations for both PM<sub>10</sub> and PM<sub>2.5</sub>.<sup>44</sup> As explained in the *LG&E* Order, a permitting authority may only use the PM<sub>10</sub> BACT determination to satisfy the PM<sub>2.5</sub> requirements if an appropriate surrogacy demonstration has been made.

If LDEQ does not include a BACT emission limitation for PM<sub>2.5</sub> as required by the statute, LDEQ must provide a reasonable surrogacy demonstration. However, the EPA was unable to identify such a demonstration in the record. The record does not contain a specific discussion that is described as LDEQ's basis for concluding that PM<sub>10</sub> is an adequate surrogate for PM<sub>2.5</sub> in the context of this particular permit. The record explains that the EPA has allowed states to continue using PM<sub>10</sub> as a surrogate for PM<sub>2.5</sub>, but does not appear to explain why it is appropriate to use PM<sub>10</sub> as a surrogate for PM<sub>2.5</sub> in this case at this facility. The record does seem to reflect one element of the surrogacy demonstration that the EPA recommended in the *LG&E* Order. Nucor conducted a top-down BACT analysis for PM<sub>2.5</sub> that showed the control technology selected through the PM<sub>10</sub> BACT analysis is "physically the same as what is selected through the PM<sub>2.5</sub> BACT analysis." See, *LG&E* Order at 45. However, the permitting record contains no further discussion of the relationship between PM<sub>10</sub> and PM<sub>2.5</sub> emissions from the DRI process such as whether and how the PM<sub>2.5</sub>:PM<sub>10</sub> ratio may vary with source operating conditions at each unit. While LDEQ asserts, for example, that "[c]ompliance with the limit for PM is deemed compliance with the BACT limit for both PM, PM<sub>10</sub>, and PM<sub>2.5</sub>," 2011 RTC at 92-94,<sup>45</sup> such statements are conclusory without further explanation regarding the relationship between PM<sub>10</sub> and PM<sub>2.5</sub> emissions at the DRI process. There is no other explanation as to why PM<sub>10</sub> would serve as an adequate surrogate for PM<sub>2.5</sub> in this case.

For the foregoing reasons, the EPA grants the Petitioners' claims as to the DRI title V permit. The EPA directs LDEQ to either include PM<sub>2.5</sub> BACT emission limitations or provide an appropriate demonstration, consistent with court decisions referenced in the *LG&E* order that PM<sub>10</sub> is a reasonable surrogate for PM<sub>2.5</sub> under the facts and circumstances of this permit.

### **C. The Limit For Natural Gas Consumption Is Not the BACT for GHG Emissions from the DRI Process**

*Petitioners' Claims.* The Petitioners contend that neither LDEQ's DRI PSD permit nor Nucor's GHG BACT analyses contain any documentation for certain statements in Step 4 of the BACT analysis (including the conclusions that natural gas consumption is the most relevant parameter that can be measured and that minimization of natural gas consumed is the most effective means

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<sup>44</sup> In response to several comments, LDEQ disagrees with a commenter's assertion that "the permits are insufficient because LDEQ must establish BACT for every pollutant and that PM<sub>10</sub> and PM<sub>2.5</sub> are separate pollutants." See, e.g., 2011 RTC at 91-92. LDEQ continues to explain that "[t]he commenter is in error. The pollutant is particulate matter; PM<sub>10</sub> and PM<sub>2.5</sub> are merely indicators of the pollutant." *Id.* (citing *Prairie State*). While the *Prairie State* order does explain that PM<sub>10</sub> and PM<sub>2.5</sub> are both indicators of particulate matter, the EPA does not agree to the extent LDEQ suggests that this fact absolves the permitting authority of the requirement to set emission limitations for both indicators. The PSD BACT requirement applies to "each regulated NSR pollutant." 40 C.F.R. § 51.166(j)(2). Under the EPA's regulations, PM<sub>2.5</sub> and PM<sub>10</sub> are separate regulated NSR pollutants. 40 C.F.R. § 51.166(b)(49)(i); 73 *Fed. Reg.* 28321, 28324 (May 16, 2008) ("this action addresses PM<sub>2.5</sub> as a regulated NSR pollutant"); 75 *Fed. Reg.* 64864, 64871 (Oct. 20, 2010) ("the promulgation of a NAAQS for PM<sub>2.5</sub> established a NAAQS for an additional pollutant"); *LG&E* Order at 42-46.

<sup>45</sup> In response to the comment, LDEQ does not explain where in the permit it is stated that compliance with the BACT limit applies to both PM<sub>10</sub> and PM<sub>2.5</sub> and the EPA is unable to locate such a condition.

of reducing GHG generation). 2011 Petition at 11 (quoting statements from the BACT analysis in the proposed DRI PSD permit that was provided in the public notice in advance of the public hearing (citing EDMS Doc. ID 7731649<sup>46</sup>, p. 107)). The Petitioners further contend that neither the PSD permit nor Nucor's GHG BACT analysis contain any documentation for the conclusion that the limit selected (13 MMBtu/tonne<sup>47</sup> DRI produced) is BACT. *Id.* at 11, 12.<sup>48</sup>

The Petitioners further claim that the GHG BACT determination is inadequate for several reasons, including that the PSD Permit incorrectly identifies this limit not for the entire facility but rather for the Reformer/Main Flue Gas Stack (DRI 108) in Train #1 of the DRI process and that the Title V Permit fails to state that this is a BACT limitation for GHG. *Id.* at 11–12.<sup>49</sup>

These claims were re-raised in the 2012 Petition, Att. C at 11-12.

*EPA's Response.* For the reasons explained below, the EPA denies these claims. Issues raised under the Petition headings Claim IV.A and Claim IV.B of the 2011 Petition and Attachment C of the 2012 Petition are addressed below.

In response to concerns raised in public comments about the amount of documentation in the record, LDEQ explained that “limited data is currently available regarding control of greenhouse gases.” 2011 RTC at 25. LDEQ explained that the EPA's GHG Permitting Guidance was released after Nucor submitted its DRI GHG BACT analysis on October 22, 2010, and that LDEQ's review of Nucor's submittal occurred in large part without the benefit of that guidance. *Id.* LDEQ further noted that the EPA's technical white paper for the iron and steel industry on control techniques or measures to reduce GHG emissions does not address such controls or measures for facilities that produce DRI, except as an emerging technology. *Id.* LDEQ also stated that the iron and steel sector is not addressed in the EPA's Greenhouse Gas Mitigation Strategies Database, nor are relevant data included in the EPA's RACT/BACT/LAER Clearinghouse. *Id.* Elsewhere in the response to comments, LDEQ notes that its GHG BACT limit is “likely the first of its kind.” 2011 RTC at 35.

The Petitioners do not acknowledge or address LDEQ's response explaining the limitations in the available data for documentation. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). Moreover, Nucor submitted an addendum to its DRI permit application to support its GHG BACT analysis. *See Nucor's Direct Reduced*

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<sup>46</sup> EDMS Document ID 7731649 is identified as “Material associated with the proposed permit for Public Review; Nucor -DRI Permit #3086-VO & PSD-LA-751 (November 24, 2010)(hereafter “Public Hearing Materials”).

<sup>47</sup> “MMBtu/ tonne” means one million British thermal units per metric ton. A metric ton is about 2,204.6 pounds.

<sup>48</sup> Although part of this claim is presented under Claim IV.A, it is related to issues raised under Claim IV concerning documentation of conclusions underlying the BACT determination, so we are addressing these claims together here.

<sup>49</sup> In addition to these allegations, two other problems with the GHG BACT determination are briefly asserted in this claim: (1) the limit for natural gas consumption for DRI production is considerably higher than reported in the literature; (2) this limit is not supported by the values for natural gas consumption used by Nucor for calculation of criteria pollutant emissions from the DRI facility. 2011 Petition at 12. The Petitioners provide further argument and elaboration of the first point under Petition heading of Claim IV.A and, thus, that point is addressed as part of the discussion of that claim below. Similarly, the Petitioners provide further argument and elaboration of the second point under the Petition heading of Claim IV.B and, thus, that point is addressed as part of the discussion of that claim below.



*Facility GHG BACT Analysis*, October 22, 2011, EDMS Document ID 7718227. LDEQ also set forth reasoning to support the GHG BACT determination in the permit record for the PSD permit, including in the Preliminary Determination Summary and in the RTC, and the limits are included in the final permits, along with a statement that this limit reflects BACT for GHG carbon dioxide equivalent (CO<sub>2</sub>e) emissions, as explained below. See DRI title V permit, at 32 (Specific Requirements 380, 383-384, 386-387); *see also* DRI PSD permit at 47-50, 80. This claim does not provide any explanation of why additional documentation would be needed to support LDEQ's GHG BACT determination or analysis. The Petitioners have not demonstrated that the quoted statements in the BACT analysis lack a reasoned basis or are clearly incorrect. Thus, with respect to the documentation provided for the GHG BACT limit, the Petitioners have not shown LDEQ failed to comply with its SIP-approved regulations governing PSD permitting or that the state's exercise of discretion under such regulations was unreasonable or arbitrary. *See, e.g., 2012 Cash Creek Order* at 4-5.

The Petitioners contend that the PSD Permit incorrectly identifies the GHG BACT limit not for the entire facility but rather for the Reformer/Main Flue Gas Stack (DRI 108) in Train #1 of the DRI process. 2011 Petition at 12. LDEQ's Response to Comments agrees that the BACT limit would be more appropriately attributed to the entire facility and that the "permit will be modified accordingly." 2011 RTC at 38 (referenced by 2011 RTC at 31, which responds to the comment raising the specific claim in the Petition). In the Specific Requirements of the final DRI title V permit, the GHG BACT limit no longer appeared under the requirements for DRI Unit No. 1 Reformer Main Flue Gas Stack (DRI 108), but instead appeared under requirements for the "DRI Facility - Direct Reduction Iron Facility" (unit UNF 0002). DRI title V permit, Specific Requirement 384, at 32 (placing the GHG BACT limit under UNF 0002); *see also id.*, Specific Requirements 380, 383, 386-387 at 32-33 (GHG BACT recordkeeping and monitoring requirements under UNF-002); *compare* Specific Requirements 90-105 at 8-9 (requirements for DRI 108). This change is reflected in a later modification of the DRI PSD permit, *see* Permit No. PSD-LA-751(M-1), Specific Requirement 229, as well as a later modification of the DRI title V permit, *see* Permit No. 3086-V2, Specific Requirement 444. Thus, this point is moot. *See Chevron Order* at 6 (denying title V petition as moot where the permit had been corrected). Moreover, the Petitioners do not acknowledge or reply to LDEQ's response and these changes to the permits, nor do they provide any explanation why these changes would not address their concerns. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34).

The Petitioners contend that the title V permit fails to state that the limit is a BACT limit for GHG but provide no additional detail or explanation for this point. 2011 Petition at 12. In the 2011 RTC, LDEQ explained that it was apparent that the limit was established to limit GHG (CO<sub>2</sub>e) emissions when the BACT limit in the proposed title V permit was read with the PSD permit, but additionally stated that the final title V permit would note this fact. 2011 RTC at 32. Specific Requirement 384 of the final DRI title V permit states: "BACT for greenhouse gas (CO<sub>2</sub>e) emissions: Limit Natural gas <= 13 MM BTU (HHV) per tonne of Direct Reduced Iron (DRI) produced." *See* DRI title V permit, Permit No. 3086-V0, Specific Requirement 384, at 32; *see also id.*, Specific Requirements 380, 383, 386-387 at 32-33 (recordkeeping and monitoring requirements labeled as "BACT for greenhouse gas (CO<sub>2</sub>e) emissions"). Thus, this point is

moot based on the changes that LDEQ made to the final title V permit. *See Chevron Order* at 6 (denying title V petition as moot where the permit had been corrected). Furthermore, the Petitioners do not acknowledge or reply to LDEQ's response and these changes to the permit, nor do they provide any explanation why these changes would not address their concerns. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34).

For these reasons and for the reasons described in the discussion that follows, the EPA hereby denies these claims.

### **1. Lower Natural Gas Consumption for DRI Production Is Reported in the Literature**

*Petitioners' Claims.* The Petitioners challenge the GHG BACT limit of 13 MMBtu per tonne DRI produced, claiming that lower values for natural gas consumption are reported in the literature for other DRI facilities and other DRI production processes. 2011 Petition at 12. The Petitioners include a table summarizing reported values for natural gas consumption and electricity consumption (where available) for two other DRI facilities (one in the US and one in Australia) and for other DRI processes, and contend that Nucor's value of 13 MMBtu per tonne DRI produced is considerably higher than these reported values, which range from 7.3 to 11.55 MMBtu per tonne DRI produced. *Id.* at 12-13. The Petitioners contend that the Essar Minnesota Steel facility, which was then under construction and expected to be operational in 2012, is estimated to have a natural gas consumption value of 7.3 to 8.2 MMBtu per tonne DRI. *Id.* at 14. The Petition states that this facility was to be the first fully-integrated mine through steel-making facility in North America and its DRI production would be 56 percent of Nucor's proposed DRI process. *Id.* The Petitioners conclude that 13 MMBtu per tonne of DRI is clearly not BACT. *Id.*

These claims were re-raised in the 2012 Petition, Att. C at 12-14.

*EPA's Response.* For the reasons explained below, the EPA denies these claims.

The Petitioners have not demonstrated that LDEQ's BACT analysis lacked a reasoned basis or was flawed. LDEQ's response to comments explained that the Petitioners' comments on this point did not provide enough information to determine if the process-specific natural gas consumption rates were comparable to Nucor's. 2011 RTC at 34. LDEQ notes that natural gas consumption increases with increasing metallization and carbon content of the product, and that it could not assess from the information submitted whether products of the same metallization and carbon content were being compared. *Id.* LDEQ additionally notes that the references cited do not substantiate that the performance claims are achievable over extended periods and appear to exclude startup, shutdown and off-spec production. *Id.* Also, LDEQ notes that it is not clear if the natural gas combustion rates are based on higher or lower heating value (generally a 10 percent difference), and that Nucor uses higher heating value. *Id.* In addition, LDEQ quoted statements from the EAB reflecting that a permitting authority has some discretion in determining whether a particular control efficiency provides an appropriate basis for a BACT determination and emission limitation, and to consider factors such as whether the technology is

relatively unproven or whether the available data demonstrate whether the emission rate at issue has been achieved over the long term. *Id.* at 34-35 (quoting *In re Newmont Nev. Energy Inv., LLC*, 12 E.A.D. 429, 442 (EAB 2005)).

The Petitioners do not acknowledge or address LDEQ's response to comments. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). For example, the Petitioners have not responded to LDEQ's point that the comment did not provide sufficient information to determine if the values for natural gas consumption were comparable to the value used for Nucor's on material parameters.

In addition, the Petitioners' claim disregards the fact that BACT determinations are case-by-case, site-specific determinations. *See* CAA § 169(3); L.A.C. 33:III.509.B (definition of "Best Available Control Technology"). As noted by LDEQ, the EPA has long recognized that permitting authorities "retain discretion to set BACT levels that 'do not necessarily reflect the highest possible control efficiencies but, rather, will allow permittees to achieve compliance on a consistent basis.'" *In Re Pio Pico Energy Center*, PSD Appeal Nos. 12-04 through 12-06, Slip. Op. at 78 (Aug. 2, 2013) (quoting *In re Newmont Nev. Energy Inv., LLC*, 12 E.A.D. 429, 442 (EAB 2005)). Thus, the mere fact that a lower value for natural gas consumption has been calculated and reported for another facility or another type of DRI process in the literature does not establish that LDEQ lacked a reasoned basis in establishing an enforceable emissions limit that must be met on a continuous basis for Nucor as it did. The Petitioners do not demonstrate that the "lower values" reported in literature for these facilities were enforceable emissions limitations imposed by a permit or other legal requirement. The Petitioners have not provided any analysis to demonstrate that the reported levels they cite from the literature were achievable on a consistent basis by the facilities in question. Nor have they provided any analysis to demonstrate that these levels would be appropriate for Nucor and achievable on a continuous basis at the facility at issue here. *See Spurlock Order* at 21 (denying title V petition claim challenging a BACT determination where the petitioner provided examples of lower limits established at similar sources throughout the country but failed to provide any analysis to demonstrate that these BACT limits were appropriate for the particular unit at issue in the petition). Thus, the Petitioners have failed to establish that LDEQ's GHG BACT determination lacked a reasoned basis, or is otherwise not in compliance with the applicable CAA requirements. *Id.*

For these reasons, the EPA hereby denies these claims.

## **2. The Sum of Values for Natural Gas Consumption Used by Nucor for Calculation of Criteria Pollutant Emissions From the DRI Process is Less Than Half the BACT Limit**

*Petitioners' Claims.* The Petitioners contend that the GHG BACT limit should be lower than the limit LDEQ imposed of 13 MMBtu per tonne DRI produced. 2011 Petition at 16. In support of this contention, the Petitioners present calculations that they claim show that "natural gas consumption on a per unit basis" for the DRI process is lower than 13 MMBtu per tonne DRI. *Id.* Those calculations appear to be based on multiplying the maximum (average) firing rates that

Nucor used for a number of emissions units in its calculations for criteria pollutant emissions by the maximum annual hours of operation for those units, using these figures to estimate total annual gas consumption for the DRI process, and then dividing that total by the maximum annual production of DRI. *Id.* 15-16. The Petitioners contend that according to those calculations, “unless there are other major natural gas-consuming processes that the permits did not disclose, BACT for natural gas consumption as a parameter for GHG emissions for the facility is 6.0 MMBtu/tonne of DRI.” *Id.* at 16.

Noting LDEQ’s statement in the RTC that the Petitioners’ calculations, which were also provided during the comment period, did not account for the generation of reducing gas, the Petitioners state that LDEQ failed to “provide an estimate of how much reducing gas is required to determine the total natural gas consumption.” *Id.* at 16. The Petitioners also claim that based on information from MIDREX typical natural gas consumption would range from 9.3 MMBtu per tonne of DRI at the lower end of metallization and carbon content to 10.6 MMBtu per tonne of DRI at the higher end of metallization and carbon content. *Id.* The Petitioners state that these values are on the same order of magnitude discussed in the Petitioners’ comments and far below the natural gas consumption of 13 MMBtu per tonne DRI with unspecified metallization and carbon content. *Id.* Finally, the Petitioners contend that the GHG BACT limit of the 13 MMBtu per tonne DRI is not supported, *id.* at 16, and that “LDEQ must provide product and raw material specifications backed by vendor information and demonstrate how it derived the 13 MMBtu/tonne DRI natural gas consumption figure.” *Id.* at 17.

The claims were re-raised in the 2012 Petition, Att. C at 15-17.

*EPA’s Response.* For the reasons explained below, the EPA denies these claims.

In response to the Petitioners’ calculations and estimates presented during the comment period to argue that the GHG BACT limit should be 6.0 MMBtu per tonne of DRI, LDEQ responded that the comment did not “account[] for the fact that natural gas is not only used as a fuel, but also to generate reducing gas.” 2011 RTC at 36. LDEQ further explained that at high temperatures natural gas dissociates into a reducing gas rich in CO and hydrogen, which are the primary reductants for the DRI process. *Id.*

LDEQ’s Response to Comments provides an explanation for why the Petitioners’ estimate of natural gas consumption of 6.0 MMBtu per tonne DRI in the comments was different from the level imposed as BACT—that natural gas is consumed to generate reducing gas, in addition to firing combustion sources to produce process heat. *Id.*; *see also* the DRI PSD Permit, at 9 (“Reducing gas is generated initially from natural gas, which is heated and reformed in the reformer at an elevated temperature.”). In responding to LDEQ’s points, the Petitioners do not show that the calculations supporting their estimate include natural gas used to generate reducing gas. Thus, the Petitioners have not shown that their calculations or estimates include all the natural gas included in LDEQ’s value. *See, e.g.,* the DRI PSD permit, at 50 (“natural gas is consumed in the DRI process as both a raw material (for the formation of reducing gas) and as a fuel (for heating to reaction temperatures). All sources of natural gas consumption at the Reformer should be included in the analysis.”). Instead, the Petitioners provide additional estimates of typical natural gas consumption based on information from MIDREX, a vendor of

DRI units, which are substantially higher than the 6.0 MMBtu per tonne DRI estimate and which are more in the range of the value used by LDEQ. Also, to the extent that the Petitioners intended to contend that the GHG BACT limit must be directly related to emissions of GHG or criteria pollutant at Nucor, the EPA notes that the BACT limit for GHG at Nucor is an energy efficiency limit based on MMBtu/ tonne of DRI produced for which compliance can be assured without calculating the emissions of either GHG or criteria pollutants (instead compliance is determined based on records of actual natural gas consumption and tonnes of DRI produced). DRI title V permit, Specific Requirement 387, at 33.

With respect to the Petitioners' contention that LDEQ must provide additional product and material specifications and demonstrate how it derived the 13 MMBtu per tonne DRI natural gas consumption figure, the Petitioners' appear more focused on forwarding their calculations than demonstrating that LDEQ's evaluation was flawed. The Petitioners have not identified or analyzed any statutory or regulatory provision, nor any guidance, to support their contention that the information they identified must be provided, in addition to the explanation LDEQ provided in the record. *See, e.g., Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA § 505(b)(2)). In addition, LDEQ also explained in response to comments concerning the amount of documentation for the GHG BACT limit in the record that there was at the time limited data concerning control of greenhouse gas emissions, including techniques to reduce GHG emissions from facilities that produce DRI. *See* 2011 RTC at 24-25. This claim of the Petition does not acknowledge or address that point in LDEQ's response. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34). The Petitioners have not shown LDEQ failed to comply with its SIP-approved regulations governing PSD permitting or that the state's exercise of discretion under such regulations was unreasonable or arbitrary. *See, e.g., 2012 Cash Creek Order* at 4-5.

In sum, the Petitioners have not demonstrated that LDEQ's GHG BACT determination lacked a reasoned basis, or is otherwise not in compliance with the applicable CAA requirements. *See, supra*, at 5, "Raising PSD Issues in a Petition." For these reasons, the EPA hereby denies these claims.

#### **D. The Permits Must Specify Procedures for Estimating GHGs**

*Petitioners' Claims.* The Petitioners claim that "[t]he PSD permit must clearly specify the procedure for making the mass balance calculation for carbon in the DRI production process." 2011 Petition at 17 (citing the EPA's *Review of Proposed Title V Permits for Florida Power & Light* (1997), Enclosure 3, at 2) (*Florida Power & Light Letter*). The Petitioners further allege that Specific Requirement #82 in the proposed DRI part 70 permit provided in the public notice requires calculating DRI production rates and natural gas consumption using the provisions of both Subpart C and Subpart Q from the GHG Mandatory Reporting Rule, and that the provision is inadequate, asserting that Subpart Q "does not provide a calculation procedure for DRI production and the reference is therefore moot." *Id.* at 17 (citing Specific Requirements #81 and #82 in EDMS Document ID 7731649 (hereafter "Public Hearing Materials")). The Petitioners

conclude that the “EPA must require LDEQ to develop a calculation procedure for DRI production and present it for public review.” *Id.*

The Petitioners additionally contend that this calculation should account for the considerable variation in pipeline-grade natural gas for carbon content and heating values, as well as CO<sub>2</sub>-fuel efficiency coefficients. *Id.* at 17-18 (citing Energy Information Administration, *Emissions of Greenhouse Gases in the United States 1987–2002*, Fig. A-1; U.S. Dep’t of Energy, Voluntary Reporting of Greenhouse Gases Program, Fuel Emission Coefficients). Therefore, the Petitioners assert that Nucor should use “facility-specific values for carbon content and heating value” to determine GHG emissions from natural gas combustion wherever possible. *Id.* at 18. The Petitioners claim that such information should be available from suppliers of the fuel or Material Data Safety Sheets for the purchased fuel, and should be confirmed with fuel analysis. *Id.*

With respect to LDEQ’s response to comments, the Petitioners assert that the LDEQ’s response did not lay out the procedure for estimating GHG emissions or specify the CO<sub>2</sub>-fuel efficiency coefficient for pipeline natural gas, as discussed in Petitioners’ comments. *Id.* at 18–19.

These claims were re-raised in the 2012 Petition, Att. C at 17-19.

*EPA’s Response.* For the reasons explained below, the EPA denies these claims.

LDEQ’s response to comments explained that the monitoring provisions associated with the BACT limit did not require quantification of CO<sub>2</sub> emissions from the facility, and that, therefore, performing a mass balance calculation and monitoring parameters such as carbon content of the natural gas and DRI product is not necessary. 2011 RTC at 35-38 (cited by 2011 RTC at 38-39, which reflects LDEQ’s response to the comment raising the issues in this claim). It additionally stated that the only necessary parameters to monitor compliance with the 13 MMBtu/tonne DRI BACT limit were the amount of natural gas consumed by the process, including its heating value, and the amount of DRI product produced. *Id.* LDEQ also explained that requiring quantification of CO<sub>2</sub> emissions from the facility is the role of the GHG Reporting Rule under 40 C.F.R. 98. *Id.* LDEQ additionally stated in the response to comments that it was deleting Specific Requirements 82 and 235 from the proposed permit and replacing them with requirements for: (1) monitoring the total DRI natural gas and energy consumption, (2) recordkeeping of total DRI natural gas and energy consumption, (3) recordkeeping of total DRI production, and (4) determining compliance with the GHG BACT limit. *Id.*

Although the Petitioners state that LDEQ’s response “does not lay out a procedure for estimating GHG emissions,” the Petitioners do not address LDEQ’s point in the response to comments that such a procedure is not required to determine compliance with the GHG BACT limit, which is expressed in terms of MMBtu/tonne DRI. The only citation that the Petition provides for the assertion that a procedure for calculating carbon is required is a letter from the EPA Region 4 objecting to title V permits issued to Florida Power and Light. *Florida Power & Light Letter*, Enc. 3 at 2. In this letter, the EPA Region 4 stated that a particular condition in the permit must be revised to specify the procedure for calculating the sulfur content of the oil where there was ambiguity in the permit language that could allow for two different methods of determining compliance with the annual average sulfur content limit, one of which was not appropriate for

showing the limit would be met. The Petitioners do not provide any explanation of why Nucor's GHG BACT limit, which is based on MMBtu/tonne DRI, would merit analogous treatment as the permit condition in Florida Power & Light's permit, which limited the weight percent of sulfur in oil. *See id.* The Petition does not provide any other legal citation or analysis to support the assertion that a procedure for estimating GHG emissions was needed; nor do the Petitioners provide any explanation as to why LDEQ's statements in the response to comments are unreasonable or how the permit fails to comply with the Act. *See, e.g., Nucor II Order* at 7 (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34, and has looked to whether they have provided relevant citations and analyses to support the claim).

The Petitioners also do not address the changes that LDEQ made to the permit. For example, Specific Requirement 82 in the proposed DRI title V permit required Nucor to track DRI production and natural gas consumption "using a mass balance approach similar to Subpart Q for iron and steelmaking from the promulgated Mandatory Reporting of Greenhouse Gases rule." Public Hearing Materials at 37-38. LDEQ deleted that specific requirement, however, and replaced it with other requirements for monitoring, recordkeeping, and compliance demonstration. *See* DRI title V permit, DRI title V permit, Specific Requirements at pp. 7-8 (reflecting deletion of requirement to use an approach similar to Subpart Q), and pp.32-33 (reflecting addition of new requirements); *see also* 2011 RTC at 39. To the extent that the Petitioners believed that a mass balance calculation or other procedure for estimating GHG emissions was needed because of Specific Requirement 82 as it appeared in the proposed DRI title V permit, any such argument would be moot because that provision and its requirements were removed from the final DRI title V permit. For the same reason, the Petitioners' arguments relating to the inadequacies of Specific Requirement 82 and referring to Subpart Q are also moot. In addition, the Petitioners do not acknowledge or address the changes that LDEQ made to the permit or reply to LDEQ's explanation in the 2011 RTC of the permit changes it had made. *See, e.g., Nucor II Order* (explaining that the EPA expects title V petitioners to engage with the state's final decision, including response to comments, citing *MacClarence*, 596 F.3d at 1132-34).

With respect to the CO<sub>2</sub> fuel efficiency coefficients, both the Petition and the comments submitted on behalf of the Petitioners stated that facility specific values for carbon content and heating value should be used to determine GHG emissions because CO<sub>2</sub> fuel efficiency coefficients vary for pipeline natural gas. 2011 Petition at 18; *Comments on the Draft PSD Permit and Draft Title V Permit Best Available Control Technology Analyses for Greenhouse Gas Emissions for the Nucor Direct Reduced Iron Facility and Pig Iron Facility*, Submitted by Pless Environmental, Inc. via the Tulane Environmental Law Clinic (Jan. 3, 2011)(EDMS Document ID 7781475)(hereafter "2011 Pless Comments") at 23-24. The 2011 Petition also contends that LDEQ's response to comments "does not specify the CO<sub>2</sub> fuel efficiency coefficient for pipeline natural gas, as discussed in Petitioners' comments." 2011 Petition at 19. The Petitioners' comments, however, do not state that a CO<sub>2</sub> fuel efficiency coefficient should be established for pipeline natural gas. *See 2011 Pless Comments* at 23-24.<sup>50</sup> The discussion in the

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<sup>50</sup> The Petition does not cite or identify any particular discussion in the Petitioners' comments to support the claim; however, the discussion of CO<sub>2</sub> efficiency limits on p. 24 of the 2011 Pless Comments appears to be the intended discussion because it appears identical to the discussion originally presented in the 2011 Petition and because it is

2011 Petition and the comments of the CO<sub>2</sub> fuel efficiency coefficients is in the context of determining GHG emissions, and neither document provides any other reason why such a coefficient would be needed. *Id.* As explained above, the Petitioners have not demonstrated that quantification of GHG emissions was required in order to assure compliance with the GHG BACT limit, expressed in terms of MMBtu/tonne DRI, and have not identified or analyzed anything that would have required LDEQ to establish a procedure for estimating GHG emissions. *See, e.g., Nucor II Order* at 7 (explaining that the EPA has looked at whether title V petitioners have provided the relevant citations and analyses to support its claim in determining whether it has a duty to object under CAA § 505(b)(2)). Nor have they demonstrated that LDEQ's decision not to establish such a procedure was unreasonable or otherwise not in compliance with the applicable CAA requirements. *See, e.g., 2012 Cash Creek Order* at 4-5.

For these reasons, the EPA hereby denies these claims.<sup>51</sup>

## VI. CONCLUSION

For the reasons set forth above and pursuant to CAA § 505(b)(2) and 40 C.F.R. § 70.8(d), I hereby grant in part and deny in part, as described herein, issues originally raised in the 2010 Petition and the 2011 Petition, which were re-raised in the 2012 Petition. As explained more fully above, this order in conjunction with the June 19, 2013 Order responds to LEAN and Sierra Club's petitions requesting that the EPA object to certain title V permits issued to Nucor.

Dated: 1/30/14

  
Gina McCarthy  
Administrator

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the only discussion of CO<sub>2</sub> efficiency limits that the EPA located in the 2011 comments submitted by counsel for the Petitioners at Tulane Environmental Law Clinic.

<sup>51</sup> The EPA also notes that the 2011 Petition states that it “adopt[s] and incorporate[s] by reference Zen-Noh Grain’s petition asking the EPA to object to the modified title V permit for the pig iron plant and the initial title V permit for the DRI plant.” 2011 Petition at 2. To the extent that Petitioners have incorporated by reference Zen-Noh’s 2011 Petition, the EPA has already responded to that petition, granting an objection in the *Zen-Noh Order*, and LDEQ has issued a response to the EPA’s objection. Thus, the EPA need not further address Zen-Noh’s 2011 Petition in this Order.



# IN RE KNAUF FIBER GLASS, GMBH

PSD Appeal Nos. 99-8 through 99-72

## ***ORDER DENYING REVIEW***

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Decided March 14, 2000

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### Syllabus

This decision addresses the remaining petitions for review that have challenged the revised prevention of significant deterioration ("PSD") permit issued by Shasta County, California, Air Quality Management District ("SCAQMD") to Knauf Fiber Glass, GmbH, a corporation that plans to construct a new fiberglass manufacturing facility in the City of Shasta Lake, California. This is the second time a SCAQMD PSD permit decision for the proposed Knauf facility has come before the Environmental Appeals Board. In the first round of petitions, the Board issued a decision that denied review of many issues raised on appeal but remanded SCAQMD's permit decision on two issues: the best available control technology ("BACT") determination for PM<sub>10</sub> and environmental justice. *See In re Knauf Fiber Glass, GmbH*, 8 E.A.D. 121 (EAB 1999) ("*Knauf I*"). SCAQMD completed the remand proceedings on August 17, 1999, and issued a revised permit decision for the Knauf facility. The second round of petitions for review followed. Some of the petitions for review were previously dismissed on grounds of timeliness and standing. *See In re Knauf Fiber Glass, GmbH*, PSD Appeal Nos. 99-8 through 99-72 (EAB, Jan. 3, 2000) (Order Dismissing Certain Appeals on Timeliness and Standing).

Petitioners challenge the revised BACT determination for PM<sub>10</sub> and the environmental justice analysis, as well as several miscellaneous issues.

Held: Review is denied of the petitions for review for the following reasons:

- Many of the petitions for review fail to meet the Board's requirement that issues be raised with specificity. (Section II.A.1.)
- Most of the miscellaneous issues raised in the petitions for review are outside the scope of review for this post-remand appeal. The Board's decision in *Knauf I* was final as to all issues associated with the PSD permit for the proposed Knauf facility, with the exception of two: BACT for PM<sub>10</sub> and environmental justice. The only exception to the limitation on the scope of review is for issues pertaining to permit conditions that were modified during the remand period. (Section II.A.3)
- In contrast to the documentation in the administrative record for *Knauf I*, the supplemental BACT analysis and revised BACT determination provide ample support for SCAQMD's final decisions on BACT and the revised permit conditions on PM<sub>10</sub> emissions. On remand, SCAQMD revised the PM<sub>10</sub> BACT emission limitation downward from 5.37 lbs/ton to 3.5 lbs/ton and from 43.6 lbs/hr to 28.4 lbs/hr.

SCAQMD adequately explained how it reached its decisions regarding PM<sub>10</sub> control technology and the PM<sub>10</sub> emission limitation. (Section II.B.)

- The environmental justice analysis prepared during the remand period concludes that the proposed Knauf facility will not have disproportionately high or adverse human health or environmental effects on a minority or low-income population. None of the petitioners have shown that the anticipated PM<sub>10</sub> emissions from the proposed facility would in fact lead to an adverse impact. With regard to petitioners' contentions regarding public participation in this permit process, the Board notes that the public's involvement was effective in securing an environmental benefit through a lower PM<sub>10</sub> emission limitation. (Section II.C.)
- The new National Emissions Standards for Hazardous Air Pollutants ("NESHAP") rule applicable to the fiberglass manufacturing industry was appropriately cross-referenced in a revised permit condition in this instance. (Section II.D.)

***Before Environmental Appeals Judges Scott C. Fulton, Ronald L. McCallum, and Kathie A. Stein.***

***Opinion of the Board by Judge McCallum:***

This case is an appeal of an air permitting decision made by the Shasta County, California, Air Quality Management District ("SCAQMD"). The SCAQMD issued a preconstruction permit and authority to construct under the federal Clean Air Act prevention of significant deterioration ("PSD") program to Knauf Fiber Glass, GmbH, a corporation that plans to construct a new fiberglass manufacturing facility in the City of Shasta Lake, California. This is the second time a SCAQMD PSD permit for the proposed Knauf facility has come before the Environmental Appeals Board ("Board"). In a previous appeal, the original PSD permit issued by SCAQMD was challenged by several private citizens, citizens' groups, and by EPA Region IX. The Board issued a decision in that case in February 1999, denying review of many issues raised on appeal, but also remanding SCAQMD's permit decision on two issues. *In re Knauf Fiber Glass, GmbH*, 8 E.A.D. 121 (EAB 1999) ("*Knauf I*"). On August 17, 1999, SCAQMD completed the remand proceedings and issued a new permit decision for the Knauf facility. The Board subsequently received sixty-five (65) petitions for review of the August 1999 permit decision. Those petitions constitute the present appeal.

## **I. BACKGROUND**

The SCAQMD processes permit applications and issues permits in Shasta County, California, under the federal PSD program pursuant to a delegation

agreement with the U.S. EPA.<sup>1</sup> The PSD permit program is an element of the Clean Air Act (“CAA”) that requires preconstruction review and approval for new and modified major stationary sources. CAA § 165, 42 U.S.C. § 7475. As outlined in our previous decision regarding the planned Knauf facility, the PSD review process involves several technical analyses and determinations as well as specific procedural requirements designed to implement the CAA’s emphasis on public participation and input. *Knauf I*, 8 E.A.D. at 123-24.

The PSD review process for the proposed Knauf facility officially began in March 1997, when Knauf first submitted a PSD permit application to SCAQMD. The proposed facility is subject to PSD review due to its anticipated emissions of particulate matter less than 10 micrometers in diameter (“PM<sub>10</sub>”).<sup>2</sup> During the course of the original review process, SCAQMD conducted analyses of best available control technology (“BACT”) and air quality impacts relating to PM<sub>10</sub>. In addition, SCAQMD solicited comment on the terms of a draft permit for the proposed facility and held a public hearing. *See Knauf I*, 8 E.A.D. at 125 (providing details of SCAQMD’s administrative review of the Knauf permit application in 1997-1998). After issuing a final permit decision in March 1998, several individuals and entities filed petitions for review with the Board, seeking our review of SCAQMD’s permit decision and elements of its review process.

*Knauf I* examined several aspects of SCAQMD’s original PSD review process. We denied review of all of the issues raised in the appeal with the exception of two items for which we felt that SCAQMD’s decisions were not adequately justified on the record. *Knauf I*, 8 E.A.D. at 174-75. The two items that warranted a grant of review were: (1) the PM<sub>10</sub> BACT determination, and (2) conclusions regarding environmental justice. *Id.* The Board remanded the PSD permit to SCAQMD to provide supplemental analyses of these items and to make the analyses available for public comment. *Id.* The Board specifically limited the scope of the remand to these two issues. Although the Board expressly allowed for appeals upon conclusion of the remand procedures, we also cautioned that “[t]he subject matter of any such appeal must be limited to the issues identified in the remand order.” *Id.* at 73.

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<sup>1</sup> U.S. EPA delegated authority to the SCAQMD to administer the federal PSD program in 1985. The permits that SCAQMD issues pursuant to that delegation are considered federal permits subject to federal permitting procedures, including the potential for review by the Environmental Appeals Board under 40 C.F.R. § 124.19. *See In re RockGen Energy Center*, 8 E.A.D. 536, 537 n.1 (EAB 1999); *Knauf I*, 8 E.A.D. at 123; 40 C.F.R. § 124.41 (“when EPA has delegated authority to administer [permitting] regulations to another agency \* \* \*, the term *EPA* shall mean the delegate agency and the term *Regional Administrator* shall mean the chief administrative officer of the delegate agency.”)

<sup>2</sup> PSD review is triggered for PM<sub>10</sub> if a source has the potential to emit 15 tons per year or more of PM<sub>10</sub> emissions. 40 C.F.R. § 52.21(b)(23)(i). The annual PM<sub>10</sub> emissions from the proposed Knauf facility are well above this threshold.

During the remand period, SCAQMD prepared and/or obtained the supplemental analyses required by the Board's order. SCAQMD also prepared a revised draft permit, and made the revised permit, along with the supplemental analyses, available for public comment in April 1999. On June 2, 1999, SCAQMD held a public hearing on the revised permit. SCAQMD issued a final revised permit along with two response to comments documents on August 17, 1999. *See* Federal Prevention of Significant Deterioration (PSD) Authority to Construct (Aug. 17, 1999) ("Revised Permit"); Response to Comments, Written Comments Submitted During Public Comment Period ("Resp. to Comments"); Response to Comments, Public Hearing 6/2/99 ("Public Hear. Resp.").

During September 1999, the Board received sixty-five (65) petitions for review regarding the revised permit for the proposed Knauf facility.<sup>3</sup> Sixty-four (64) of these petitions were filed by citizens or citizens' groups who oppose the Knauf facility. One petition was filed by another fiberglass manufacturer, CertainTeed Corporation. Most of the citizen petitions request that the Board deny the permit issued to Knauf.

At the Board's request, SCAQMD prepared responses to each of the petitions for review.<sup>4</sup> Petitioners were subsequently granted the opportunity to file replies to the SCAQMD responses. Notice to All Petitioners and Order Granting Motions for Leave to File Reply Briefs (Nov. 16, 1999). EPA Region IX, which was a petitioner in *Knauf I*, but did not file a petition for review of the revised permit decision, sought permission to file an amicus brief in this proceeding. The Board granted that request. Order Granting Motion for Leave to File Amicus

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<sup>3</sup> The petitioners (and corresponding appeal numbers) are: Robert Rollins (99-8), Colleen Leavitt (99-9), Mary Scott (99-10), David Nigro & Paula Hetzler (99-11), Debra Kaut (99-12), Betty Doty (99-13), Dorothy Kearsley (99-14), Walter May (99-15), Citizens for Cleaner Air et al. (99-16), Arnold Erickson (99-17), Russ Wade (99-18), Earl Hastings (99-19), Doreen Hastings (99-20), Ivan Hall (99-21), Barbara Frisbie (99-22), Stuart Oliver (99-23), Stuart Oliver & Jonathan McInter (99-24), Radley Davis (99-25), Judy Sills (99-26), James Sills (99-27), April Frank (99-28), Warren Teel (99-29), Sharon Bellomo (99-30), Dwight Bailey (99-31), William Caraway (99-32), Vicki Caraway (99-33), Dara Caraway (99-34), Joanna Caul & Richard Sanford (99-35), Robert DiGiulio (99-36), Robert & Constance Hegge (99-37), Heidi Silva (99-38), Suzanne Auteni-Tony (99-39), Rhonda Posey (99-40), Gloria Zeller (99-41), Jim Price (99-42), Judy Hansen (99-43), Barbara Condon (99-44), Elizabeth Ballou (99-45), Joseph & Lillian Hernandez (99-46), Bonnie Rule (99-47), Cindy Christie (99-48), Aracelia Briggs (99-49), Rebecca Christie (99-50), Becky Wilson (99-51), Ron Pearsall (99-52), George McArthur (99-53), Georgette McArthur (99-54), James Melby (99-55), Carolyn Singelmann (99-56), Fulton Doty (99-57), Nadine Stutsman (99-58), Patricia Cogburn (99-59), Bryan Jones (99-60), Orville & Juanita Vanderzanden (99-61), Doreen Melby (99-62), Linda Andrews (99-63), Jeffrey Lewellyn (99-64), Barbara Jo Garner (99-65), CertainTeed Corp. (99-66), Justin Jones (99-67), Hans Ortlieb (99-68), Tillie Smith (99-69), Laurie O'Connell & Ed Barger (99-70), Joy Newcom (99-71), Fulton Doty (99-72). Specific petitions are cited herein as "Petition [#]."

<sup>4</sup> SCAQMD's responses are cited herein as "Resp. to Petition [#]." We refer to specific pages within the response by the administrative record page number, i.e., (AR #).

Brief (Nov. 10, 1999). The amicus brief represents the views of Region IX, EPA's Office of Air and Radiation, and EPA's Office of Environmental Justice. Amicus Brief of EPA Region IX, EPA Office of Air and Radiation, and EPA Office of Environmental Justice in Support of Shasta County, California, Air Quality Management District's Response to Petitioners ("EPA Amicus Brief").

Through its responses to the petitions for review, SCAQMD challenged several petitions on the threshold regulatory requirements of timeliness and standing. The Board reviewed all of the petitions for compliance with the timeliness and standing requirements and issued an order dismissing several of the petitions for review on timeliness and standing grounds. Order Dismissing Certain Appeals on Timeliness and Standing (Jan. 3, 2000).<sup>5</sup>

## II. DISCUSSION

### A. Scope of Review

#### 1. Preliminary Requirements

In determining whether to grant review of a petition for review of a PSD permit, the Board first looks to whether the petition meets the threshold procedural requirements of the permit appeal regulations. *See* 40 C.F.R. § 124.19; *In re Sutter Power Plant*, 8 E.A.D. at 685 (EAB 1999). The threshold procedural requirements include timeliness, standing, and preservation of an issue for review.

As discussed above, the Board issued an earlier order dealing with timeliness and standing.<sup>6</sup> In that order, we noted that we also expect petitions for review to meet a minimum standard of specificity. *See* Order Dismissing Certain Appeals on Timeliness and Standing at 2 n.1 (Jan. 6, 2000); *citing In re Envotech, L.P.*, 6 E.A.D. 260, 267 (EAB 1996). To meet the specificity requirement, petitioners must include specific information supporting their allegations. Petitions for review may not simply repeat objections made during the comment period; instead they must demonstrate why the permitting authority's response to those objections warrants review. *Sutter*, 8 E.A.D. at 687 (EAB 1999); *In re Encogen Cogeneration Facility*, 8 E.A.D. at 244, 251-52 (EAB 1999).

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<sup>5</sup> The January 3 order disposed of the following petitions for review in their entirety: 99-12, 99-25 through 99-28, 99-31, 99-39 through 99-52, 99-55, 99-56, 99-60 through 99-62, 99-64, 99-65, 99-67, 99-70. Supplemental letters in support of petition numbers 99-17 and 99-38 were also dismissed.

<sup>6</sup> The threshold procedural requirement that issues be properly preserved for review is not contested in this case.

As we explained in *Knauf I*, the Board broadly construes petitions filed by persons unrepresented by legal counsel. *Knauf I*, 8 E.A.D. at 127. While the Board expects such petitions to meet the requirement of specificity, it does not expect those petitions to contain sophisticated legal arguments or to employ precise technical or legal terms. *Id.*; *Sutter*, 8 E.A.D. at 687. For purposes of specificity, the Board expects such petitions to clearly identify the issue being raised and to provide some supportable reason as to why review is warranted. *Knauf I*, 8 E.A.D. at 127; *Sutter*, 8 E.A.D. at 687-88.

In this case, many of the petitions for review fall far short of even this generous approach to specificity. Most of the petitions do not identify even one particular permit condition as a basis for an appeal. While many of the petitions for review allude to the two issues that were the subject of the Board's remand order, i.e., BACT and environmental justice, few of them discuss why SCAQMD's written responses on these issues are incorrect or inadequate. It is clear from reading all of the petitions for review, that the petitioning citizens and citizens' groups feel strongly that the Knauf facility, at least as currently designed, is inappropriate for the Shasta Lake community. We respect the petitioners' right to voice their objections, but for us to fairly and accurately examine the merits of this appeal, we must insist that minimum specificity standards are adhered to.

There are nonetheless, approximately one dozen petitions for review that satisfy the preliminary requirements, including specificity. These petitions fairly represent the overall collection of petitions for review filed by citizens and citizens' groups. Of these, certain petitions for review do a particularly good job of highlighting the issues and objections to SCAQMD's responses. For purposes of brevity and clarity, we will refer only to selected petitions in our discussion of the merits on the issues before us. We view the petitions cited herein as representative of the entire collection of citizens' petitions meeting the preliminary requirements.

## *2. Standard of Review for a Grant or Denial of Review in a Permit Appeal*

If the preliminary requirements have been satisfied, the Board will determine whether a petition for review shows that the permit decision in question was based on a clearly erroneous finding of fact or conclusion of law, or if the decision involves an important policy consideration or exercise of discretion that warrants review. 40 C.F.R. § 124.19(a); *Knauf I*, 8 E.A.D. at 126-27. If either of these conditions is met, the Board will grant review and potentially remand the permit decision. If neither of the conditions is met, the Board denies review of the petition.

The above standard of review is applied stringently in practice, in keeping with the directive in the preamble to section 124.19 that the "power of review should be only sparingly exercised" and "most permit conditions should be finally

determined at the [permitting authority] level.” 45 Fed. Reg. 33,290, 33,412 (May 19, 1980). Thus, it is infrequent that the Board will grant review in a permit appeal. The Board exercises this authority only when the petitions for review and the administrative record are abundantly persuasive that the Board’s active involvement in the matter is warranted.

### *3. Limitations on Scope of Review Established by the Remand Order*

In this case, the potential for a grant of review is also limited by the *Knauf I* decision. That decision was final as to all issues associated with the PSD permit for the proposed Knauf facility, with the exception of two: BACT and environmental justice. Those are the issues that were the subject of our remand order to SCAQMD, and are the focus of this decision. As noted above, the *Knauf I* decision explicitly limited any post-remand appeals to those two issues. *Knauf I*, 8 E.A.D. at 175-76.

Therefore, we decline review of the abundance of miscellaneous issues raised in the petitions for review. Some of the issues outside the scope of review for this post-remand appeal are issues that were specifically addressed and for which review was denied in *Knauf I*. This category includes issues such as: concerns about federal and state air quality standards, permit limits on hazardous air pollutant emissions, the PM<sub>10</sub> mitigation plan, the desire for an environmental impact statement under the National Environmental Policy Act, and use of local landfills for waste disposal. In addition, the petitions for review raise some new issues that were not before us in *Knauf I*. Such issues may not be raised at this juncture because the scope of the remand was expressly limited. All other issues pertaining to this PSD permit should have been raised at the time of the first appeal. Issues raised outside of the appeals period on the original permit are considered untimely. *See Knauf I*, 8 E.A.D. at 126 n.9 (new issues raised in reply briefs are equivalent to late filed appeals and must be denied as untimely).

The only exception to the limitation on the scope of review as established by the remand order is for issues pertaining to permit conditions that were modified during the remand period. Such permit conditions may qualify for review because the conditions have not been previously subject to the appeal process. In this case, an issue has been raised regarding SCAQMD’s modification of the permit in light of the National Emission Standards for Hazardous Air Pollutants (“NESHAP”) for the fiberglass manufacturing industry. *See* 64 Fed. Reg. 31,695 (June 14, 1999). The fiberglass NESHAP was promulgated in June 1999, during the remand period, and we may examine the issue raised in the petitions for review regarding the permit’s consistency with this regulation.

The next section of the Discussion describes the revised BACT determination reached by SCAQMD during the remand period and addresses issues raised

in the petitions for review regarding this determination. Following that, we address the arguments regarding environmental justice and how that concept relates to this case. Last, we look at the issue of how the revised permit addresses the fiberglass NESHAP.

## B. BACT

The Clean Air Act and the PSD regulations require that “best available control technology” be employed on facilities subject to PSD review. CAA § 165(a)(4), 42 U.S.C. § 7475(a)(4); 40 C.F.R. § 52.21(j)(2). These requirements are implemented through a BACT analysis and, ultimately, a BACT determination issued by the permitting authority. The BACT determination typically consists of selecting an emission limitation based on a specified control technology for control of a particular air pollutant. *Knauf I*, 8 E.A.D. at 128-29.

In *Knauf I*, we remanded SCAQMD’s original BACT determination for PM<sub>10</sub> because we found deficiencies in how the control technology and emission limits for the proposed Knauf facility were selected. *Knauf I*, 8 E.A.D. at 141. The documentation on SCAQMD’s BACT determination did not demonstrate that SCAQMD had fully considered the PM<sub>10</sub> control technologies and emission limits at other fiberglass manufacturing facilities. *Id.* In addition, commenters in the original permit proceeding had raised questions regarding configuration and size of the particular pollution control equipment selected. We held that SCAQMD had not adequately considered these comments and had not convinced us that either the particular design of the control technology or the specified emission limit constituted BACT. *Id.*

Our remand order instructed SCAQMD to identify multiple PM<sub>10</sub> control options and to thoroughly document its analysis of the potential control options. *Knauf I*, 8 E.A.D. at 175. During the remand period, Knauf prepared and submitted a supplemental BACT analysis to SCAQMD. Mostardi-Platt ass’n., Supplemental Best Available Control Technology Analysis for PM<sub>10</sub> (Feb. 1999) (“Supp. BACT Analysis”). SCAQMD subsequently revised its BACT determination. Authority to Construct/PSD Permit Evaluation (Apr. 1999) (“Revised Evaluation”).

In contrast to the documentation in the administrative record for *Knauf I*, these new documents provide ample support for SCAQMD’s final decisions on BACT and the revised permit conditions on PM<sub>10</sub> emissions. The supplemental BACT analysis, for example, identifies PM<sub>10</sub> control technologies and emission limits for five other fiberglass manufacturing facilities. Supp. BACT Analysis at 10. Knauf’s original permit application identified only one other facility for comparison purposes, a Knauf plant located in Alabama. *See Knauf I*, 8 E.A.D. at 134. The supplemental analysis also addresses the technical feasibility of six types of control options. Supp. BACT Analysis at 11-14. The original permit application contained no technical feasibility discussion at all. *See Knauf I*, 8 E.A.D. at 134.



SCAQMD's documentation of its BACT determination is also much improved. SCAQMD details the PM<sub>10</sub> control technologies used by five other fiberglass manufacturing facilities. Revised Evaluation at 14-16. SCAQMD also assesses the efficiencies of the various control options, *Id.* at 18-19, and concludes that energy, environmental, and economic impacts would not justify selection of a control option other than the top option. *Id.* at 20. None of these features were included in SCAQMD's previous evaluation document. *See Knauf I*, 8 E.A.D. at 134-35 (description of SCAQMD's justification for its original BACT determination).

The supplemental BACT analysis and SCAQMD's revised evaluation resulted in revised permit conditions governing PM<sub>10</sub> emissions from the main stack of the proposed Knauf facility. Table 1 compares the PM<sub>10</sub> control technology and emission limits as expressed in the original and revised permits.

TABLE 1  
Comparison of PM<sub>10</sub> Permit Limits

	Control Technology	PM <sub>10</sub> Emission Limit	Source
SCAQMD Permit Decision (3/30/1998)	7 venturi scrubbers; WEP*	43.6 lbs/hr 5.37 lbs/ton <sup>7</sup>	¶ 48a, 53
SCAQMD Revised Permit Decision (8/17/1999)	Knauf process technology; 7 venturi scrubbers; WEP	28.4 lbs/hr 3.5 lbs/ton	¶ 47, 52

\*Wet Electrostatic Precipitator

The revised BACT determination, as reflected in the revised permit, differs from the original in two ways. First, SCAQMD has chosen to list "Knauf process technology" as a component of BACT. SCAQMD noted that each of the fiberglass facilities considered in the course of the supplemental BACT analysis use proprietary process controls, which have some effect on the amount of PM<sub>10</sub> emissions generated prior to any add-on pollution control technology.<sup>8</sup> Revised Evalu-

<sup>7</sup> Emission limits for the fiberglass industry are commonly expressed in pounds per ton of glass pulled or "lbs/ton." The permit expresses the PM<sub>10</sub> emission limit in units of both lbs/hour and lbs/ton.

<sup>8</sup> The definition of BACT encompasses "production processes \* \* \*, systems, and techniques," as well as add-on pollution devices. *See* CAA § 169(3), 42 U.S.C. § 7479(3); 40 C.F.R. § 52.21(b)(12).

ation at 17. Second, the revised permit lowers the PM<sub>10</sub> emission limit from 5.37 lbs/ton to 3.5 lbs/ton, and from 43.6 lbs/hr to 28.4 lbs/hr.

A lower PM<sub>10</sub> emission limit was proposed by Knauf in the supplemental BACT analysis. *See* Supp. BACT Analysis at 23-24. The lower limit is made possible by two factors. First, Knauf improved the efficiency of its process technology and conducted stack tests at its Alabama plant to derive an actual emission rate that is lower than the rate used in the original permit application.<sup>9</sup> This rate reflects the expected emissions without a wet electrostatic precipitator (WEP), a type of add-on pollution control equipment. Second, Knauf obtained a slightly higher guaranteed WEP control efficiency from its WEP vendor. The combination of these two factors yielded a proposed PM<sub>10</sub> emission limit of 3.9 lbs/ton. Supp. BACT Analysis at 24.

SCAQMD tightened Knauf's proposed emission limit even further by requiring Knauf's process efficiency efforts at the new Shasta Lake facility to match the more recent performance of the Alabama plant. SCAQMD recalculated expected PM<sub>10</sub> emissions prior to treatment by the WEP by using the three best stack test results from the Alabama facility. Revised Evaluation at 21. SCAQMD ultimately selected a PM<sub>10</sub> emission limit of 3.5 lbs/ton. *Id.* at 22. This value was included in the revised permit. Revised Permit ¶ 52.

With this background, we now turn to the petitioners' objections to the revised BACT determination. The petitions for review pose many of the same arguments set forth in *Knauf I* to challenge the adequacy of the revised BACT determination. These arguments were persuasive in the prior appeal because support for the BACT determination in the administrative record was weak. The more thorough justification now before us adequately addresses petitioners' arguments and the questions we posed in *Knauf I*. *See Knauf I*, 8 E.A.D. at 140-41 (identification of open questions that need to be addressed in order to assess SCAQMD's BACT determination).

### 1. Availability of Proprietary Process Technology

One of the arguments raised in *Knauf I* was that Knauf ought to be required to obtain and employ a fiberglass manufacturing process technology from one of its competitors, i.e., CertainTeed Corporation. *See Knauf I*, 8 E.A.D. at 142. We noted that while "inherently lower-polluting processes" should be considered during the BACT selection process, *see id.* at 129, and there must be "serious consid-

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<sup>9</sup> The stack tests from Knauf's Alabama plant can provide relevant data for the proposed Shasta Lake facility because the Alabama plant uses the same process technology as planned for Shasta Lake as well as wet/venturi scrubbers for emission control. The Alabama plant does not have a WEP.

eration of pollution control designs for other facilities that are a matter of public record,” *id.* 8 E.A.D. at 142 n.34, the permit applicant does not have an obligation to pursue its competitors’ trade secrets. *Id.* Petitioners in the present appeal suggest that efforts by Knauf and SCAQMD to assess the availability of other process technologies were inadequate. *See, e.g.,* Petition 99-29 at 5.

The administrative record indicates that Knauf made several attempts to obtain information about process technologies used by other fiberglass manufacturers, including reviewing federal databases, reviewing permits issued to other fiberglass manufacturing plants, and filing Public Records Act (CA) and Freedom of Information Act (federal) requests for information. Supp. BACT Analysis at 11-12. SCAQMD also contacted air quality agencies that had issued permits to other fiberglass plants to seek information on process technologies. Revised Evaluation at 18. Information on process technology has been historically treated as proprietary and confidential by the fiberglass industry, and this position was again asserted in response to the inquiries by Knauf and SCAQMD. *See* Supp. BACT Analysis app. I (contains documentation of legal action or threats of legal action against Knauf and Knauf’s attorneys by competitor companies). Faced with this information, SCAQMD concluded that use of a competitor’s process technology was not a feasible control option for the proposed Knauf plant. Revised Evaluation at 18.

In *Knauf I*, we described the sequential elements of a BACT selection process. *Knauf I*, 8 E.A.D. at 129-32. The process begins with an investigation of a variety of potential control technologies, consisting of both process technologies or practices and add-on controls. While BACT selection often focuses on add-on controls, we noted that it is legitimate to expect a permitting authority to also include process technologies in the list of available control options if any are available. *Id.* 8 E.A.D. at 142 n.34. Process technology that is treated as proprietary and confidential, however, will not likely qualify as “available” for purposes of BACT. If that is the case, such technologies may be eliminated from the BACT consideration process. In this case, Knauf and SCAQMD investigated the availability of process technology used by other fiberglass manufacturers, learned that it was treated as proprietary and confidential, and concluded that such technology was not available for purposes of BACT. SCAQMD’s decision on the non-availability of alternative process technology is adequately justified by the record.<sup>10</sup>

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<sup>10</sup> A decision that alternative process technology is not available does not exempt a permit applicant and permitting authority from fully investigating add-on pollution controls. *Knauf I*, 8 E.A.D. at 142 n.34. On remand, Knauf and SCAQMD documented an investigation of add-on controls, and as described in this section, this supplemental search and analysis was satisfactorily performed.

## 2. Size of Wet Electrostatic Precipitator

A wet electrostatic precipitator (WEP) is an add-on pollution control device for PM<sub>10</sub> that uses electrical forces to remove PM<sub>10</sub> from an emission gas stream and deposits the particulate onto collection plates. Supp. BACT Analysis at 14. According to SCAQMD's analysis, a WEP is the most effective add-on control device available for PM<sub>10</sub> emissions from a fiberglass manufacturing plant.<sup>11</sup> Revised Evaluation at 18. Petitioners have not challenged the selection of WEP technology as the most stringent add-on emission control. However, the petitions question the size of the WEP planned for the Knauf facility. *See, e.g.*, Petition 99-37 at 6 (challenging SCAQMD's determination that a larger WEP is not available). We posed a similar question in *Knauf I*, 8 E.A.D. at 141.

The supplemental BACT analysis contains an economic and removal efficiency analysis associated with increasing the size of the WEP as designed for the Knauf facility. Supp. BACT Analysis at 16-17. The analysis examines the additional costs and PM<sub>10</sub> removals for WEPs 1.5 and 2 times larger than the WEP as designed. The analysis indicates that the PM<sub>10</sub> emission rate can be reduced by 0.2 lbs/ton with a WEP one and half times larger than the current design and by 0.3 lbs/ton with a WEP that is twice as large. *Id.* at 16. The price for these incremental emissions reductions is estimated at between \$43,000 and \$54,000 per ton of additional PM<sub>10</sub> removed. *Id.*

The economic and removal efficiency analysis presented in the supplemental BACT analysis is largely a hypothetical discussion, because no vendor has proposed to provide a WEP of the magnitudes suggested. Of the three vendor proposals received, Knauf chose the largest WEP offered. Supp. BACT Analysis at 17. Petitioners argue that just because a larger WEP was not offered by a vendor does not mean that a larger WEP would not have been available. Petition 99-37 at 6. While it may be physically possible to construct a larger WEP, we believe that SCAQMD's decision to require the WEP as recommended in the supplemental BACT analysis is reasonable given the low incremental PM<sub>10</sub> removal and high incremental costs associated with a larger device.

## 3. Multiple WEPs

The petitions for review filed during *Knauf I* pointed out that fiberglass manufacturing facilities owned by CertainTeed Corporation use multiple WEPs

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<sup>11</sup> Other add-on control technologies discussed in the supplemental BACT analysis and the revised evaluation included wet scrubbers, spray towers, and baghouses. Supp. BACT Analysis at 13; Revised Evaluation at 14. SCAQMD ranked the available add-on control technologies in order of stringency as follows: (1) wet scrubbers followed by a WEP; (2) wet scrubbers only; (3) spray towers. Revised Evaluation at 18. Baghouses were eliminated as technically infeasible because they are used only where PM<sub>10</sub> exhaust is dry. *Id.*

for PM<sub>10</sub> control. *See Knauf I*, 8 E.A.D. at 138. We questioned why SCAQMD had not investigated whether the use of a different WEP configuration would result in better emissions reduction. *Id.* at 141. Petitioners raise this point again in this proceeding. *See, e.g.*, Petition 99-10 at 2; Petition 99-21 at 4-6; Petition 99-37 at 6-7.

SCAQMD noted that CertainTeed manufacturing facilities in Chowchilla, California, and Kansas City, Kansas, use multiple WEPs, but that these devices “are not used in series or in any other configuration . . . that would improve emission control efficiency.” Revised Evaluation at 17. SCAQMD further explained in the response to comments that the CertainTeed WEPs are each treating a portion of the air flow from its manufacturing process, whereas the WEP designed for Knauf will treat the entire process air flow.<sup>12</sup> Resp. to Comments at 23. SCAQMD concluded that the exhaust air from the CertainTeed facilities and from the proposed Knauf facility would receive the same emission control. *Id.* The decision of whether to approve, as BACT, the use of one WEP that treats the entire emission stream rather than multiple WEPS, each of which treats a portion of the emissions stream is one that we can comfortably leave to the technical expertise of the permitting authority. We are satisfied that SCAQMD investigated and considered other control technology configurations used in practice and documented the reasons for its decisions in the administrative record.

#### 4. Selection of the PM<sub>10</sub> Emission Limit

Many of the petitions for review raise objections to the PM<sub>10</sub> emission limit in the revised permit. Even though the PM<sub>10</sub> limit was revised downwards to 28.4 lbs/hr and 3.5 lbs/ton, petitioners are dissatisfied because PM<sub>10</sub> emission limits at CertainTeed facilities in California and Kansas are lower still.<sup>13</sup> *See, e.g.*, Petition

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<sup>12</sup> SCAQMD’s statement that CertainTeed’s “WEPs are sized only for a reduced portion of the air flow” elicited an objection from CertainTeed in its petition for review. Petition 99-66 at 1. CertainTeed objects to the characterization of its pollution control devices as sized to handle only a portion of process exhaust. CertainTeed states, “[e]ach of CertainTeed’s WEPS \* \* \* are sized properly to handle all of the exhaust from the corresponding processes, not just a portion.” *Id.* In response to CertainTeed’s petition, SCAQMD attempts to clarify that its use of the phrase “reduced portion of the air flow,” was simply intended to distinguish CertainTeed’s method of treating portions of air flow by individual WEPs from Knauf’s proposed method of treating the entire exhaust stream with wet scrubbers and one large WEP. Resp. to Petition 99-66 (AR 11,355). We believe that the issue raised by CertainTeed in its petition is largely one of semantics rather than substance and does not merit a grant of review.

<sup>13</sup> The materials filed with this appeal contain a fair amount of debate and discussion regarding what the emission limitations for the CertainTeed facilities actually are. The permits for the CertainTeed facilities express PM<sub>10</sub> limits differently from the way PM<sub>10</sub> limits are expressed in the revised permit for the proposed Knauf facility. We need not address the various issues regarding how CertainTeed’s PM<sub>10</sub> limits compare to Knauf’s because we find that SCAQMD’s explanation of the reasons for the differences is adequate.

99-10 at 2; Petition 99-33 at 2; Petition 99-37 at 10.

We noted in *Knauf I* that emission limits for different facilities may differ, even if identical control technology is applied. *Knauf I*, 8 E.A.D. at 143. For example, the two CertainTeed facilities in question each use a combination of wet scrubbers and WEPs for PM<sub>10</sub> control, yet their permitted PM<sub>10</sub> emission limits differ. In fact, the Kansas City facility has a higher PM<sub>10</sub> emission limit than the Chowchilla facility although the Kansas City facility is newer. *See id.* at 23; Supp. BACT Analysis at 24-25; Revised Evaluation at 16; Resp. to Comments at 12.

Here, SCAQMD explains the difference between the CertainTeed limits and the limit it set for the proposed Knauf facility by pointing to the underlying proprietary processes used by each of the companies. SCAQMD notes that the process technologies and product blends for individual fiberglass manufacturing facilities differ, and these differences will yield emission limitations that are specific to a particular facility. Resp. to Comments at 12; Resp. to Petition 99-37 (AR 11,316). We agree that numerical emission limitations under the PSD program are individualized for specific facilities and we believe that SCAQMD has adequately justified the emission limitations in the revised permit for the proposed Knauf plant as compared to other fiberglass manufacturing facilities.

A few of the petitioners not only question the PM<sub>10</sub> emission limit as compared the PM<sub>10</sub> limits at the CertainTeed facilities, but also question the factors used to derive the numerical limit of 3.5 lbs/ton. *See, e.g.*, Petition 99-10 at 5; Petition 99-29 at 6.

SCAQMD used the following equation to derive the PM<sub>10</sub> emission limitation for the proposed Knauf facility:

$$\text{Actual PM}_{10} \text{ emissions (Lanett)} \times 1.25 \text{ (safety factor)} \times \text{WEP efficiency factor}$$

Revised Evaluation at 22. The equation begins with an average value for PM<sub>10</sub> emissions from Knauf's facility in Lanett, Alabama. That value is multiplied by a safety factor to take into account process variability and then multiplied again by the WEP efficiency factor as guaranteed by the WEP manufacturer. Petitioners question use of the Lanett emissions level and the safety factor.

Petition 99-29 objects to the use of emissions data from Knauf's Lanett, Alabama facility as a basis for the emission limitation in the proposed Shasta Lake plant. Petition 99-29 at 6. This petitioner believes that the Lanett emissions levels may be inflated so as to obtain a more generous emission limit for Shasta Lake. *Id.* The petitioner also contends that it is improper for SCAQMD to set emission limits based on Knauf's historical performance when Knauf has no incentive to lower its emission levels. *Id.* SCAQMD responds that the stack tests from which the emissions data were produced were witnessed by Alabama officials who provide "third-party objectivity" to the data. Resp. to Petition 99-29 (AR

11,306). SCAQMD also defends its approach of setting an emission limit based on actual emission tests from a similar facility. *Id.* SCAQMD believes that use of actual emissions data from the very same process that will be employed in the proposed facility is the best way to set an emission limitation.

The petitioner may be correct that Knauf has no incentive to lower emissions from its Lanett, Alabama facility. But in fact, the data collected from Lanett show PM<sub>10</sub> emissions significantly lower than the permitted emission limitation for that facility. Supp. BACT Analysis app. M. Thus, even without an incentive, Knauf achieved lower emissions at Lanett. SCAQMD reasonably decided to require the same level of performance at the proposed facility in Shasta Lake.

Another petitioner questions the need for a 25% safety factor (represented as 1.25 in the above equation) for process variability. The safety factor essentially incorporates a margin of error in the calculation of an emission limitation. The petitioner suggests that the PM<sub>10</sub> emission limitation should be set without regard to a safety factor. Petition 99-10 at 5. SCAQMD responds that a 25% “variability” factor is appropriate in light of potential variations in the fiberglass insulation manufacturing process, which is affected by glass pull rates, temperature, and humidity. In addition, a safety factor can be used to protect against test method variability. Resp. to Comments at 24; Resp. to Petition 99-10 (AR 11,284).

There is nothing inherently wrong with setting an emission limitation that takes into account a reasonable safety factor. The resulting emission limitation is still an enforceable cap on PM<sub>10</sub> emissions. The inclusion of a reasonable safety factor in the emission limitation calculation is a legitimate method of deriving a specific emission limitation that may not be exceeded. SCAQMD adequately explained why it set the limit where it did, and Petitioners did not meet their burdens of showing why SCAQMD’s decision in this case was clearly erroneous or an abuse of discretion.

In sum, we deny review of all issues raised in the petitions for review regarding the revised BACT determination for the proposed Knauf facility.

### *C. Environmental Justice*

The issue of environmental justice as presented in this case refers to allegations made by members of the public that the issuance of a PSD permit for the proposed Knauf facility may disproportionately impact a low-income population. This issue was invoked through reference to an Executive Order that instructs federal agencies to address, as appropriate, “disproportionately high and adverse human health or environmental effects of [their] programs, policies, and activities on minority and low-income populations \* \* \*.” Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations, Exec. Order 12,898, 59 Fed. Reg. 7629 (Feb. 16, 1994) (“Executive Order”).

Our treatment of environmental justice in *Knauf I* was largely on procedural grounds. The administrative record indicated that the issue of environmental justice pursuant to the Executive Order had been properly raised before SCAQMD, but there was no documentation of a substantive response or analysis of the issue. *Knauf I*, 8 E.A.D. at 174-175. SCAQMD asserted that EPA Region IX had taken responsibility for addressing environmental justice and SCAQMD relied on the Region's representations. Our remand order instructed SCAQMD to obtain documentation of the Region's alleged environmental justice analysis, to include it in the administrative record, and make it available for public comment. *Id.* 8 E.A.D. at 175.

SCAQMD obtained documentation of Region IX's environmental justice analysis, which consists of two memoranda analyzing the demographics of the area surrounding the proposed Knauf facility and assessing whether the emissions from the facility will have a disproportionately high and adverse impact on human health or the environment. Memorandum from Willard Chin, Region 9 Environmental Justice Team, to Michael Kussow, Shasta County Air Pollution Control Officer (Mar. 18, 1999) (AR 8220) ("EJ Memorandum"); Addendum to the EJ Review Memorandum (Apr. 7, 1999) (AR 8369) ("EJ Addendum").<sup>14</sup>

Both the EJ Memorandum and the EJ Addendum were made available during the public comment period on the revised permit. EPA Region IX prepared a response to comments on the environmental justice analysis. EPA's Response to Public Comments on the Knauf Environmental Justice Review (Aug. 12, 1999) (AR 9644) ("EPA Resp. to Comments on EJ").

In each of the three documents prepared by Region IX regarding environmental justice, the Region concluded that the proposed Knauf facility will not have disproportionately high and adverse human health or environmental effects on a minority or low-income population. EJ Memorandum at 6; EJ Addendum at 2; EPA Resp. to Comments on EJ at 2. The Region's adverse impacts conclusion is based on its finding that the Shasta County area has been designated as an attainment area for PM<sub>10</sub> and that the additional PM<sub>10</sub> from the proposed Knauf facility will not exceed the federal NAAQS or PSD increment for PM<sub>10</sub>. EJ Memorandum at 4; EJ Addendum at 2. The Region states, "the air quality within the area surrounding the proposed site would remain well within the levels determined to [be] healthful and environmentally acceptable." EJ Memorandum at 4. In response to a comment regarding potential impacts on sensitive subpopulations, the Region noted that the NAAQS are designed to protect public health. EPA Resp. to Comments on EJ at 7. *See also* 40 C.F.R. § 50.2(b) (NAAQS are set at

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<sup>14</sup> The EJ Addendum was prepared because the EJ Memorandum incorrectly identified the location of the proposed facility, placing it approximately two miles northeast of the actual location.



levels that EPA has determined are necessary to protect the public health and welfare).

Petitioners object to numerous aspects of the Region's environmental justice analysis, including the methodology and data used for the demographic analysis, and the scope of the adverse impact analysis. *See, e.g.*, Petition 99-10 at 7; Petition 99-29 at 3-4; Petition 99-37 at 13-20. None of the petitioners, however, have shown that the Region's conclusion regarding the lack of adverse impacts from PM<sub>10</sub> emissions is clearly erroneous. As there has been no serious contention that the additional PM<sub>10</sub> emissions from the proposed facility would in fact lead to an adverse impact, and as the Executive Order concerns itself with effects that are "adverse," we find it unnecessary to address petitioners' other objections, including those relating to the demographic analysis.

Several petitioners raised issues about the quantity and quality of the public participation in SCAQMD's permitting process. *See, e.g.*, Petition 99-9 at 8; Petition 99-13 at 10; Petition 99-29 at 4; Petition 99-33 at 5. While the petitioners uniformly assert that meaningful opportunities for public participation were lacking, SCAQMD represents that it engaged in proactive community involvement. In light of the disconnect between the impressions of the community and the permitting authority, it is no surprise that this case led to two Board appeals. Our review of the public participation record here shows that SCAQMD fulfilled the applicable regulatory obligations, even if it did not go beyond those requirements.

We note, however, that the public's involvement over the course of this permitting process has had a significant role in shaping the conditions of the PSD permit that was ultimately issued to Knauf. The PM<sub>10</sub> emission limit in the revised permit is less than half the level of PM<sub>10</sub> emissions proposed by Knauf in its initial permit application. From our review of the record, it appears that these reductions are largely attributable to the active community interest and involvement in the permit process. *See In re AES Puerto Rico L.P.*, 8 E.A.D. 324, 351 (EAB 1999) (identifying specific conditions that were incorporated into a PSD permit as a result of concerns raised during the public comment period), *aff'd Sur Contra La Contaminacion v. EPA*, 202 F.3d 443 (1st Cir. 2000) ("That the permit issued here is particularly stringent may be due in large part to the participation of the area residents."). Thus, although petitioners may not be fully satisfied with the type of public participation that occurred here, it was, in fact, effective in securing an environmental benefit through lower emissions.

We deny review of both the substantive and procedural environmental justice issues raised in this appeal.

#### *D. Permit Compliance with NESHAP Rule*

In June 1999, EPA promulgated a final National Emissions Standards for Hazardous Air Pollutants (“NESHAP”) rule for the fiberglass manufacturing industry. 64 Fed. Reg. 31,695 (June 14, 1999). This rule sets hazardous air pollutant emissions standards for fiberglass manufacturing facilities such as the proposed Knauf facility. Petitioners argue that the requirements of the NESHAP should be specifically enumerated in the PSD permit. Petition 99-37 at 25. SCAQMD points out that the permit has a specific provision noting that the Knauf facility will be subject to the new NESHAP. Revised Permit ¶ 10; Resp. to Petition 99-37 (AR 11,316). The permit condition further notes that emission limits in the NESHAP “do not supersede more stringent limits found in other conditions of this permit.” Revised Permit ¶ 10. SCAQMD believes that it would be “unnecessary and impracticable to enumerate all of the requirements of the NESHAP” in the PSD permit. Resp. to Petition 99-37 (AR 11,316). Based on the circumstances presented here, we agree. The permit condition that cross-references the NESHAP is sufficient to incorporate all applicable provisions of the new rule into the PSD permit. Moreover, the NESHAP is independently enforceable. Review is denied on this issue.

### **III. CONCLUSION**

The petitions for review of the revised PSD permit decision issued by SCAQMD for the proposed Knauf facility are denied. SCAQMD has complied with the Board’s remand order in *Knauf I*. Documentation of the revised BACT determination provides adequate justification for SCAQMD’s selection of PM<sub>10</sub> control technology and emission limitations. SCAQMD also made an environmental justice analysis available to the public in accordance with our earlier order. That analysis concludes that the PSD permit for the proposed Knauf facility will not cause any disproportionately high or adverse human health or environmental effects on a low-income or minority population. Finally, there is no need for review of the revised permit condition regarding applicability of the fiberglass NESHAP.

So ordered.<sup>15</sup>

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<sup>15</sup> This decision constitutes final agency action for purposes of judicial review. See 40 C.F.R. § 124.19(f)(1)(i). Region IX shall make sure that notice of this decision is published in the Federal Register in accordance with 40 C.F.R. § 124.19(f)(2).