Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft

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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 Trends Stations
NCEA	National Center for Environmental Assessment
NCore	National Core
NO_2	Nitrogen dioxide
NO_X	Oxides of nitrogen
O_3	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
$PM_{2.5}$	In general terms, particulate matter with an aerodynamic diameter less than or equal to a nominal 2.5 μm; a measurement of fine particles
	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
PM_{10}	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)
	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
$PM_{10-2.5}$	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 coarse particulate matter or the coarse fraction of PM_{10}
	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
	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_2	Sulfur dioxide
	SO_X	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, External Review Draft* (hereafter referred to as draft *PA*), presents the draft 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 Policy Assessment (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 $PM_{2.5}$ and PM_{10}, 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.[1]

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.

[1] 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.[2] 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 draft PA, we[3] take into account the available scientific evidence, as assessed in the external review draft *Integrated Science Assessment for Particulate Matter* (draft ISA [U.S. EPA, 2018]), and additional policy-relevant analyses of air quality and risks. Our approach to considering the available evidence and analyses in this draft 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. The final PA will be informed by the advice and recommendations received from the CASAC during its review of this 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.

[2] 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.

[3] 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."[4] 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."[5]

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

[4] 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).

[5] 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 periodic review 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 periodically review and, if appropriate, revise the NAAQS, based on the revised air quality criteria.[6]

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 committee, which is to be composed of "seven members including at least one member of the

[6] This section of the Act requires the Administrator to complete these reviews and make any revisions that may be appropriate "at five-year intervals."

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.[7]

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.

[7] 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^3 (primary) 150 µg/m^3 (secondary)	Not to be exceeded more than once per year
		Annual	75 µg/m^3 (primary) 60 µg/m^3 (secondary)	Annual geometric mean
1987	PM_{10}	24-hour	150 µg/m^3	Not to be exceeded more than once per year on average over a 3-year period
		Annual	50 µg/m^3	Annual arithmetic mean, averaged over 3 years
1997	$PM_{2.5}$	24-hour	65 µg/m^3	98th percentile, averaged over 3 years
		Annual	15.0 µg/m^3	Annual arithmetic mean, averaged over 3 years[a]
	PM_{10}	24-hour	150 µg/m^3	99th percentile, averaged over 3 years[b]
		Annual	50 µg/m^3	Annual arithmetic mean, averaged over 3 years
2006	$PM_{2.5}$	24-hour	35 µg/m^3	98th percentile, averaged over 3 years
		Annual	15.0 µg/m^3	Annual arithmetic mean, averaged over 3 years[c]
	PM_{10}	24-hour[d]	150 µg/m^3	Not to be exceed more than once per year on average over a 3-year period
2012	$PM_{2.5}$	24-hour	35 µg/m^3	98th percentile, averaged over 3 years
		Annual	12.0 µg/m^3 (primary) 15.0 µg/m^3 (secondary)	Annual mean, averaged over 3 years[e]
	PM_{10}	24-hour	150 µg/m^3	Not to be exceeded more than once per year on average over 3 years

Note: When not specified, primary and secondary standards are identical.

[a] The level of the 1997 annual $PM_{2.5}$ 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).

[b] 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).

[c] 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).

[d] The EPA revoked the annual PM_{10} NAAQS in 2006 (71 FR 61144, October 17, 2006).

[e] In the 2012 decision, the EPA eliminated the option for spatial averaging (78 FR 3086, January 15, 2013).

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).[8] 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 (μm) (referred to as total suspended particulates or TSP). The primary standards were set at 260 $\mu g/m^3$, 24-hour average, not to be exceeded more than once per year, and 75 $\mu g/m^3$, annual geometric mean. The secondary standards were set at 150 $\mu g/m^3$, 24-hour average, not to be exceeded more than once per year, and 60 $\mu g/m^3$, 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 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.[9] The level of the 24-hour standards (primary and secondary) was set at 150 $\mu g/m^3$, 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 $\mu g/m^3$, 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 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 PM_{10} standards. The EPA added new standards, using $PM_{2.5}$ as the indicator for fine particles (with $PM_{2.5}$ referring to particles with a nominal mean aerodynamic diameter less than or equal to 2.5 μm). The new primary standards were as follows: (1) an annual standard with a level of 15.0 $\mu g/m^3$, based on the 3-year average of annual arithmetic mean

[8] 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.

[9] PM_{10} refers to particles with a nominal mean aerodynamic diameter less than or equal to 10 μm. More specifically, 10 μm is the aerodynamic diameter for which the efficiency of particle collection is 50 percent.

$PM_{2.5}$ concentrations from single or multiple community-oriented monitors;[10] and (2) a 24-hour standard with a level of 65 μg/m^3, based on the 3-year average of the 98th percentile of 24-hour $PM_{2.5}$ concentrations at each monitor within an area. Also, the EPA established a new reference method for the measurement of $PM_{2.5}$ 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_{10} (referred to as thoracic coarse particles or $PM_{10-2.5}$; 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_{10} standard and revised the form of the 24-hour primary PM_{10} standard to be based on the 99th percentile of 24-hour PM_{10} 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_{10} standards, concluding that the EPA had not provided a reasonable explanation justifying use of PM_{10} 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_{10} standards, and the pre-existing 1987 PM_{10} 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

[10] The 1997 annual $PM_{2.5}$ 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 $PM_{2.5}$ 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).[11] 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).

[11] 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 μg/m^3, retained the level of the annual $PM_{2.5}$ standards at 15.0 μg/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 μg/m^3, and revoked the annual standards.[12] 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 $PM_{10\text{-}2.5}$ 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

[12] In the 2006 proposal, the EPA proposed to revise the 24-hour PM_{10} standard in part by establishing a new $PM_{10\text{-}2.5}$ indicator for thoracic coarse particles (i.e., particles generally between 2.5 and 10 μm in diameter). The EPA proposed to include any ambient mix of $PM_{10\text{-}2.5}$ 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 $PM_{10\text{-}2.5}$ 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 [$PM_{10\text{-}2.5}$] 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,[13] 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 $PM_{2.5}$, the EPA revised the level of the annual $PM_{2.5}$ standard[14] to 12.0 $\mu g/m^3$ and retained the 24-hour $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$. For the primary PM_{10} 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., $PM_{10-2.5}$). With regard to the secondary PM standards, the EPA generally retained the 24-hour and annual $PM_{2.5}$ standards[15] and the 24-hour PM_{10} 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 $PM_{2.5}$ and PM_{10} NAAQS and issued a call for information

[13] The history of the NAAQS review process, including revisions to the process, is discussed at *http://www3.epa.gov/ttn/naaqs/review2.html*.

[14] The EPA also eliminated the option for spatial averaging.

[15] 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 EPA staff in developing a draft IRP, which was reviewed by the CASAC 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 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 announcing the Agency's intention to conduct this review of the PM NAAQS in such a manner as to ensure that any necessary revisions are finalized by December 2020 (Pruitt, 2018). Consistent with this intention, the EPA released the draft ISA in October 2018 (83 FR 53471, October 23, 2018). The draft ISA was reviewed by the 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, 2019). The Administrator's response to this CASAC advice was provided in a letter to the CASAC chair dated July 25, 2019.[16] Consistent with that letter, we anticipate releasing the final ISA, reflecting consideration of CASAC advice and public input on the draft ISA, in December 2019. In addition, following the review of this draft PA by the CASAC and the public, and our consideration of CASAC advice and public input, we anticipate issuing a final PA in early 2020, followed by a proposed rulemaking in early 2020 and a final rulemaking in late 2020.

[16] Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/Signed+CASAC+response+letter+7.25.19.pdf

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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 (μm) in diameter (U.S. EPA, 2018, section 2.2). When describing PM, subscripts are used to denote the aerodynamic diameter[1] of the particle size range in micrometers (μm) of 50% cut points of sampling devices. The EPA defines $PM_{2.5}$, also referred to as fine particles, as particles with aerodynamic diameters generally less than or equal to 2.5 μm. The size range for $PM_{10\text{-}2.5}$, also called coarse or thoracic coarse particles, includes those particles with aerodynamic diameters generally greater than 2.5 μm and less than or equal to 10 μm. PM_{10}, which is comprised of both fine and coarse fractions, includes those particles with aerodynamic diameters generally less than or equal to 10 μm. 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 μm based on physical size, thermal diffusivity or electrical mobility (U.S. EPA, 2018, section 2.2).

[1] Aerodynamic diameter is the size of a sphere of unit density (i.e., 1 g/cm^3) that has the same terminal settling velocity as the particle of interest (U.S. EPA, 2018, section 4.1.1).

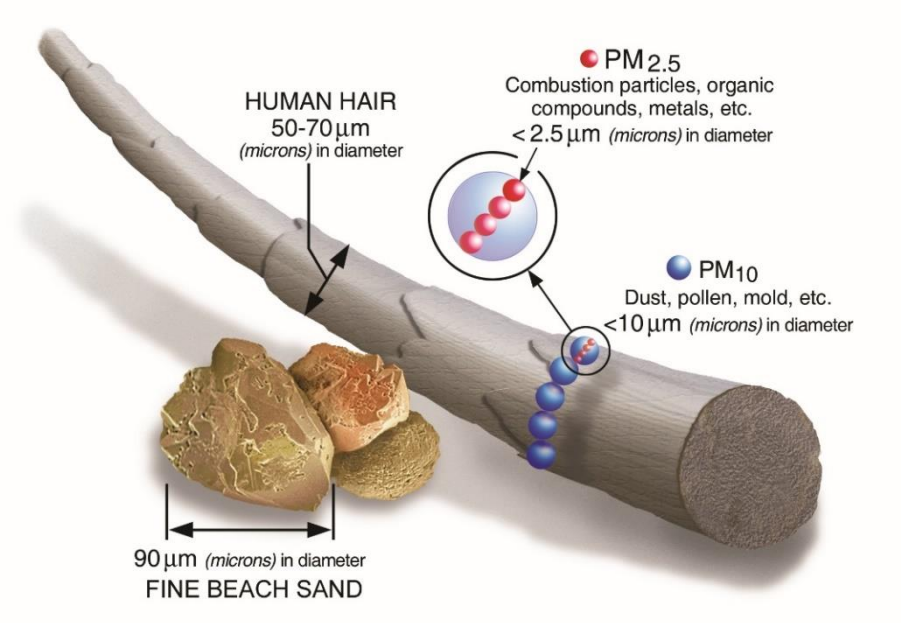

Figure 2-1.Comparisons of $PM_{2.5}$ and PM_{10} diameters to human hair and beach sand.
(Adapted from: *https://www.epa.gov/pm-pollution/particulate-matter-pm-basics*)

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, 2018, 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 µm 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 $PM_{2.5}$ mass and surface area, though only a minor contributor to particle number (U.S. EPA, 2018, section 2.2). PM2.5 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 µm 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 $PM_{10–2.5}$ sampling, but small fractions of coarse mode mass can be smaller than 2.5 µm or greater than 10 µm in diameter (U.S. EPA, 2018, 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 $PM_{2.5}$, which often remains in the atmosphere for days to weeks (U.S. EPA, 2018, Table 2-1) before being removed by wet or dry deposition. In contrast, atmospheric lifetimes for UFP and $PM_{10–2.5}$ 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. $PM_{10–2.5}$ are also generally removed from the atmosphere within hours, through wet or dry deposition (U.S. EPA, 2018, 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, 2018, 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_2) that contribute to the secondary formation of PM in the atmosphere (U.S. EPA, 2018, 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_2 (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, 2018, section 2.3.3).

Generally, the sources of PM for different size fractions vary. While $PM_{2.5}$ in ambient air is largely emitted directly by sources such as those described above or through secondary PM formation in the atmosphere, $PM_{10\text{-}2.5}$ 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, 2018, 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 $PM_{2.5}$ and $PM_{10-2.5}$ emissions into ambient air, respectively, based on the U.S. EPA 2014 National Emissions Inventory (NEI).[2] 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 $PM_{2.5}$ 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 US 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ emissions nationally. Fires include wildfires, prescribed fires, and agricultural fires, with wildfires and prescribed fires accounting for most of the fire-related primary $PM_{2.5}$ emissions nationally (U.S. EPA, 2018, section 2.3.1.1). Other lesser-contributing anthropogenic sources of $PM_{2.5}$ emissions nationally include stationary fuel combustion and agriculture sources (e.g., agricultural tilling).

[2] 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 2-3. 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*.

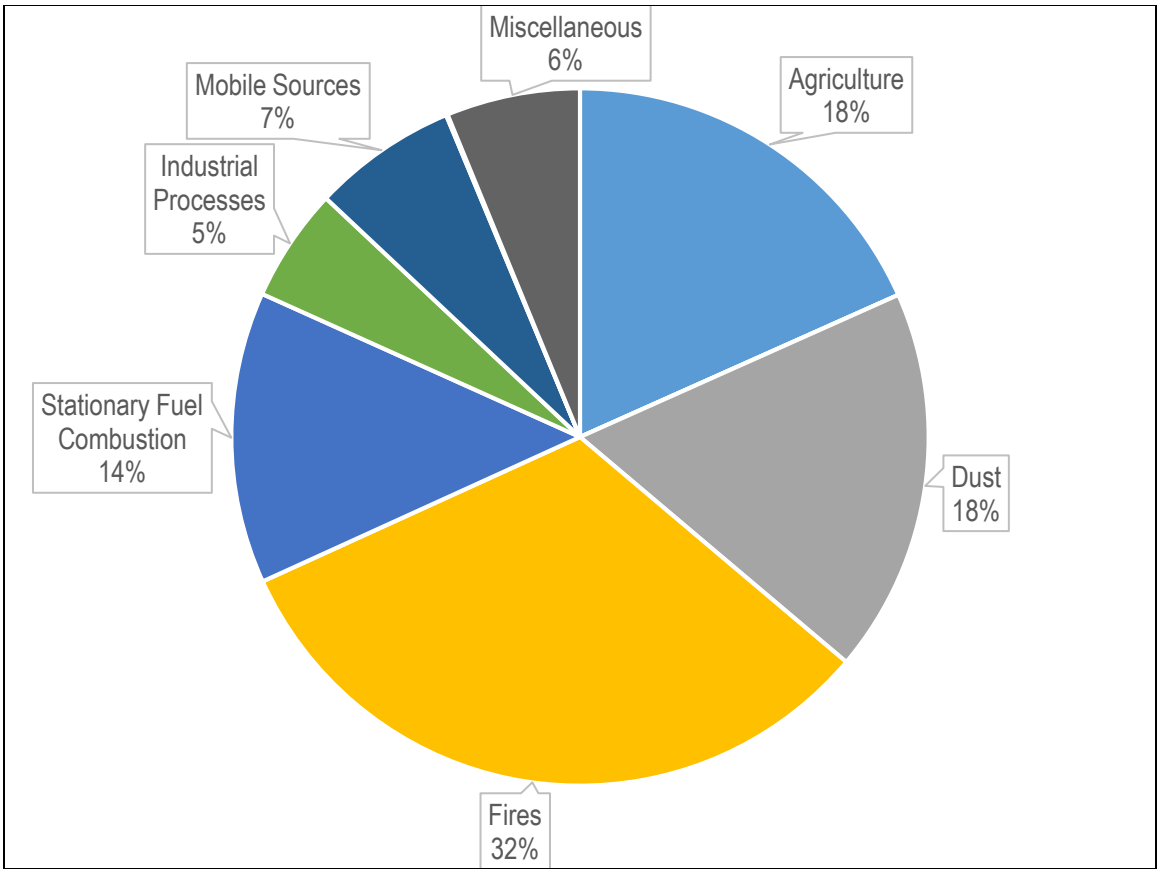

Figure 2-2.Percent contribution of $PM_{2.5}$ emissions by national source sectors. (Source: 2014 NEI)

The relative contributions of specific sources to annual emissions of primary $PM_{2.5}$ can vary from location to location, with a notable difference in contributions of sources of $PM_{2.5}$ emissions in urban areas compared to national emissions. For example, the draft ISA illustrates this variation of primary $PM_{2.5}$ emissions with data from five urban counties in the U.S. (U.S. EPA, 2018, Figure 2-3).[3] Across the majority of these urban areas, the largest $PM_{2.5}$-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, 2018, section 2.3.1.2, Figure 2-3). While primary $PM_{2.5}$ from mobile sources are a dominant contributor in some urban areas, accounting for an estimated 13 to 30% of the total primary $PM_{2.5}$ emissions, mobile sources contribute only about 7% to total primary $PM_{2.5}$ emissions nationally as shown in Figure 2-2.

[3] The five counties included in the draft ISA analysis include Queens County, NY, Philadelphia County, PA, Los Angeles County, CA, Sacramento County, CA, and Maricopa County (Phoenix), AZ (U.S. EPA, 2018, section 2.3.1.2).

2.1.1.2 Sources Contributing to Primary $PM_{10-2.5}$ Emissions

Although the NEI does not estimate emissions of $PM_{10-2.5}$ specifically, estimates of PM_{10} emissions can provide insight into sources of coarse particles. Thus, the discussion below focuses on PM_{10} emissions. The relative contributions of key sources to national PM_{10} emissions, based on the 2014 NEI, are shown in Figure 2-3. Total PM_{10} emissions are estimated to be about 13 million tons. National emissions of PM_{10} 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 $PM_{10-2.5}$ 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, 2018, section 2.3.3).

As with $PM_{2.5}$, the relative contributions of particular sources to total PM_{10} emissions varies from location to location (e.g., depending on local climate, geography, degree of urbanization, etc.). However, unlike with $PM_{2.5}$, the sectors included in Figure 2-3 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_{10} emissions.

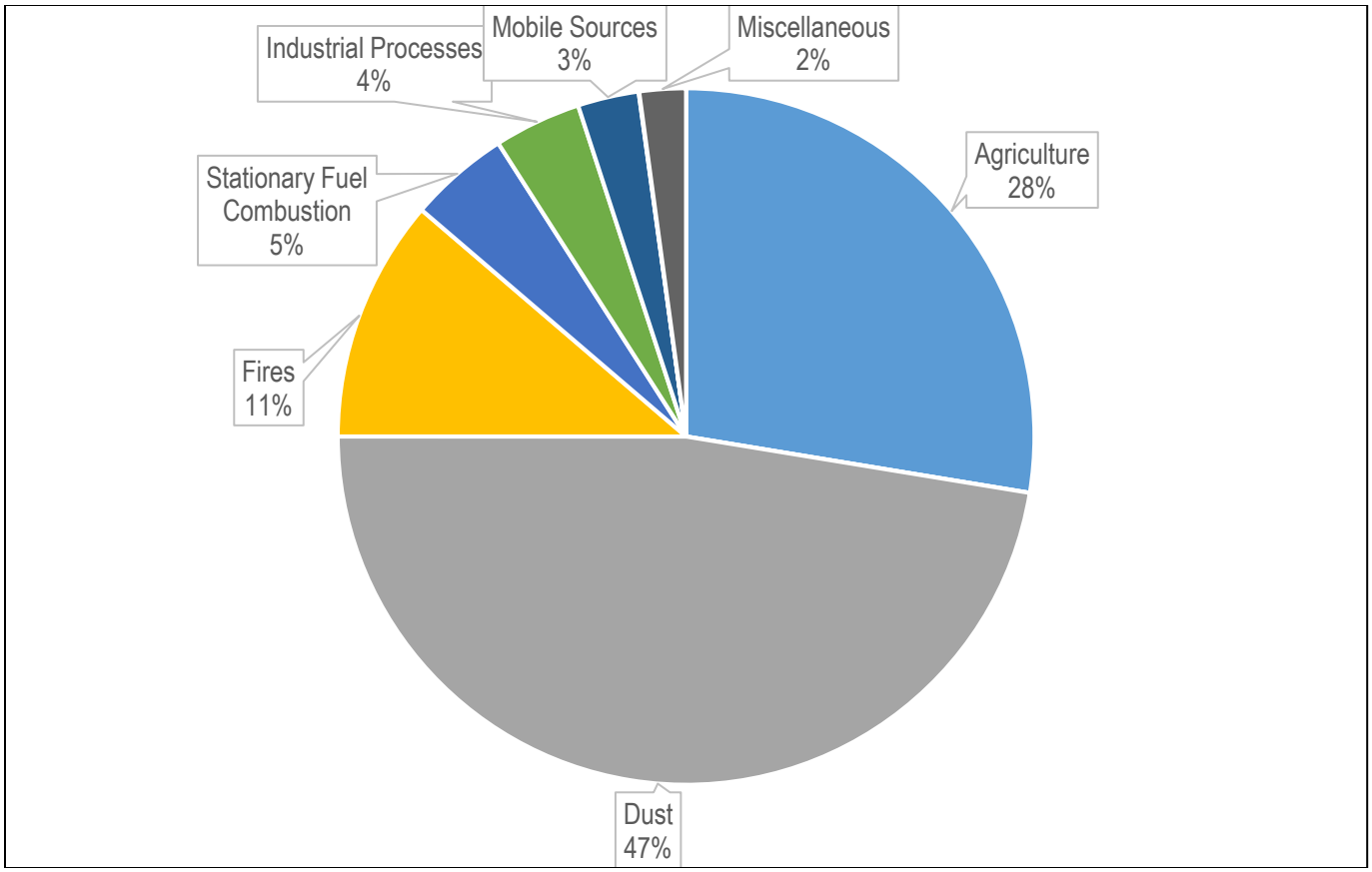

Figure 2-3.Percent contribution of PM_{10} emissions by national source sectors. (Source: 2014 NEI)

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 $PM_{2.5}$ mass include sulfate (SO_4^{2-}), nitrate (NO_3^-), 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 $PM_{2.5}$ 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 $PM_{2.5}$, emissions of elemental (or black) carbon and organic carbon often make up the largest percentage of directly emitted $PM_{2.5}$ mass. Figure 2-4 illustrates the sources that contribute to national emissions of elemental and organic carbon based on the 2014 NEI. The top panel of Figure 2-4 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 2-4 shows that fires and mobile sources (mostly diesel sources) contribute 80% of the estimated 431,000 tons of particulate EC in the 2014 NEI.

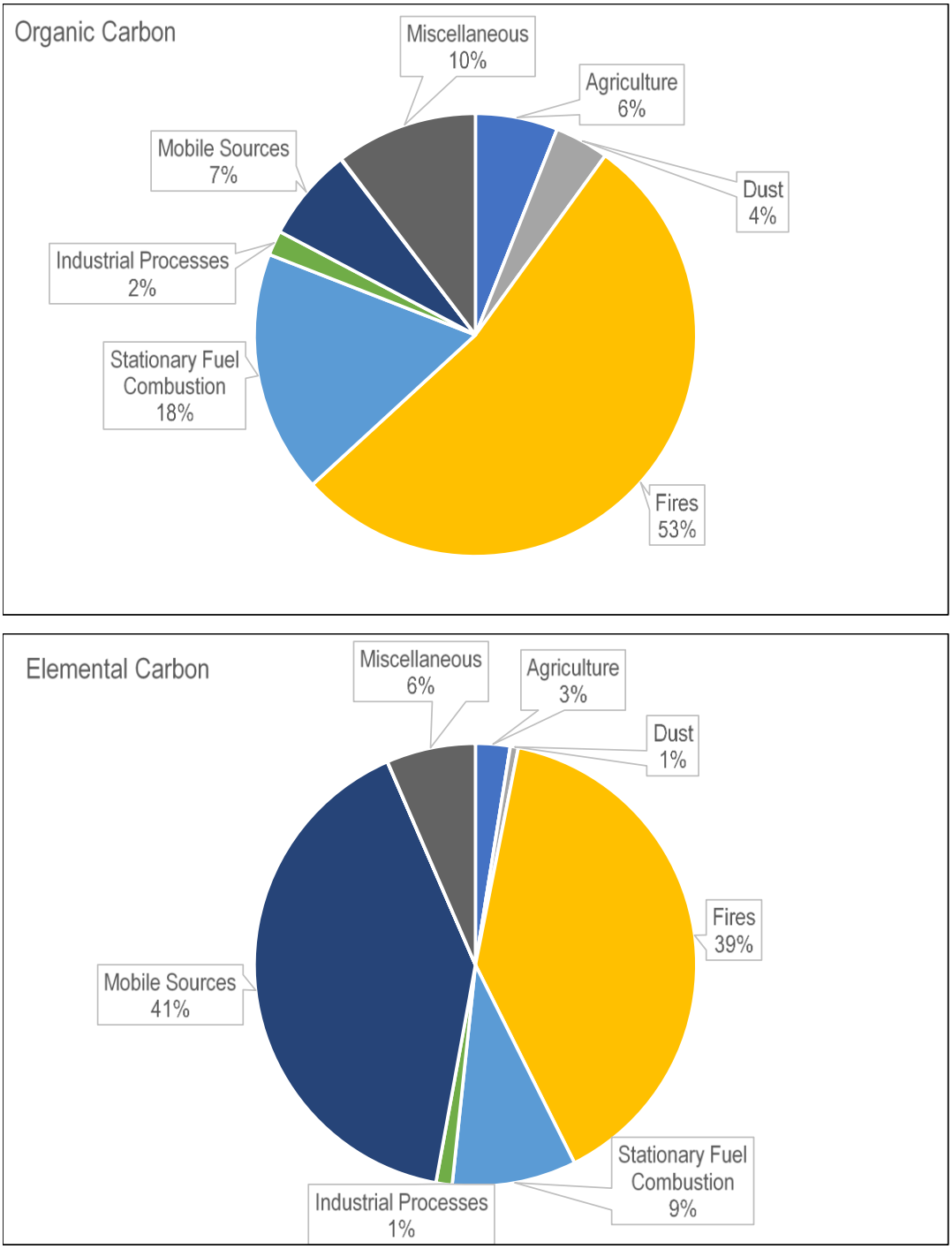

Figure 2-4.Percent contribution to organic carbon (top panel) and elemental carbon (bottom panel) national emissions by source sectors. (Source: 2014 NEI)

2.1.1.3.2 Sources Contributing to Emissions of Precursor Gases

As discussed further in the draft ISA (U.S. EPA, 2018, 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_2, NO_X, and volatile organic compound (VOC) gases of anthropogenic or natural origin (U.S. EPA, 2018, section 2.3.2.1). Anthropogenic SO_2 and NO_X are the predominant precursor gases in the formation of secondary $PM_{2.5}$, 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, 2018, 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 2-5 illustrates the source contributions at the national level for these $PM_{2.5}$ precursor gases. As shown in Panel A in Figure 2-5, stationary fuel combustion sources contribute nearly 80% of the estimated total of 4.8 million tons of national SO_2 national emissions. Within this source category, nearly all of the SO_2 emitted to the atmosphere comes from electricity generating units, or EGUs. NO_X 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_X emitted. Of the total estimate of 3.6 million tons of ammonia (NH_3) emissions shown in panel C of Figure 2-5, NH_3 emissions are dominated by the agriculture source categories. In these categories, NH_3 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_3 emissions (U.S. EPA, 2018, 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.

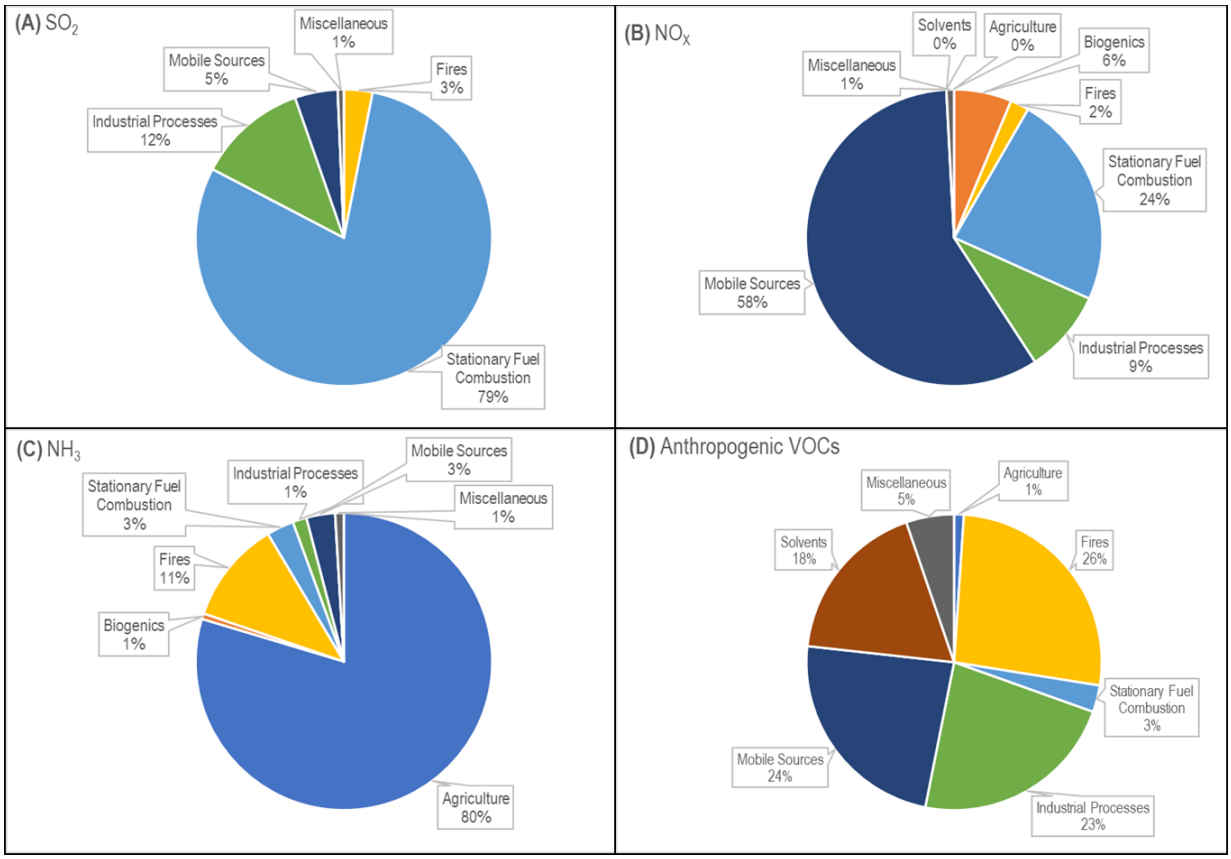

Figure 2-5.Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B), ammonia (panel C), and anthropogenic volatile organic compounds (panel D) national emissions by source sectors. (Source: 2014 NEI)

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)[4] for both PM_{10} and $PM_{2.5}$ (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.

[4] 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*).

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).[5]

The EPA and monitoring agencies manage and operate robust national networks for both PM_{10} and $PM_{2.5}$, as these are the two measurement programs directly supporting the PM NAAQS. PM_{10} measurements are based on gravimetric mass, while $PM_{2.5}$ measurements include gravimetric mass and chemical speciation. A smaller network of stations is operating and reporting data for $PM_{10\text{-}2.5}$ 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.[6] Section 2.2.1 provides information on monitoring for total suspended particulates (TSP), section 2.2.2 provides information on monitoring for PM_{10}, section 2.2.3 provides information on monitoring $PM_{2.5}$, section 2.2.4 provides information on monitoring for $PM_{10\text{-}2.5}$, 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 2-6 below illustrates the changes in PM monitoring stations reporting to the EPA's AQS database by size fraction since 1970.

[5] 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.

[6] More information on ambient monitoring networks can be found at *https://www.epa.gov/amtic/*.

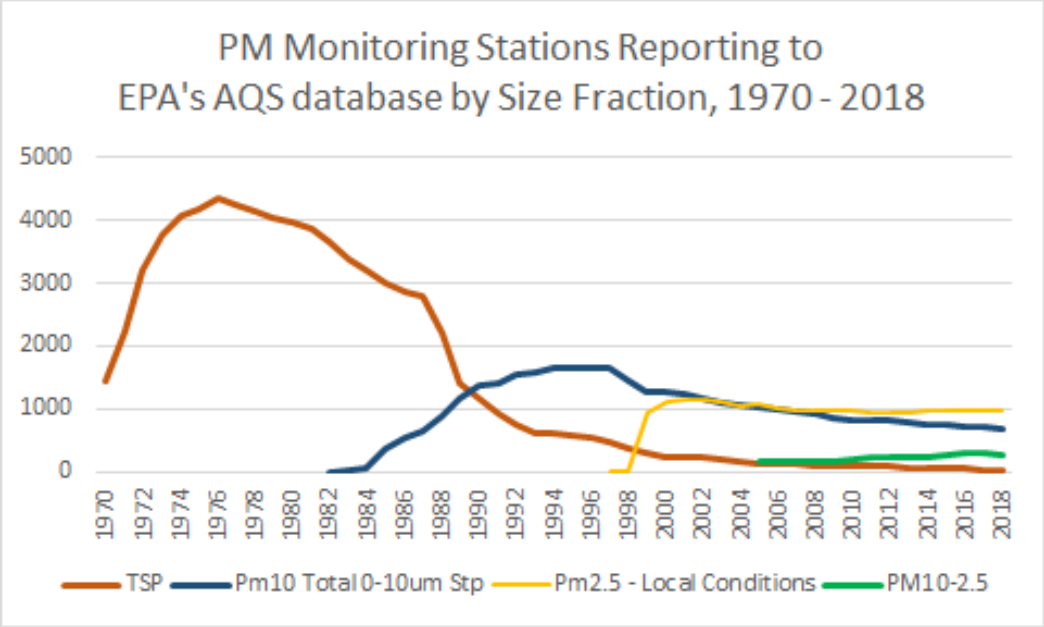

Figure 2-6.PM Monitoring stations reporting to EPA's AQS database by PM size fraction, 1970-2018.

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 the original standards was the high-volume sampler, which collects PM up to a nominal size of 25 to 45 μm (referred to as total suspended particles or TSP). TSP was replaced by 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 PM_{10} Monitoring

To support the 1987 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 PM_{10} network consisting of mainly high-volume samplers. The network design criteria emphasize monitoring at

middle[7] and neighborhood[8] scales to effectively characterize the emissions from both mobile and stationary sources, although not ruling out microscale[9] monitoring in some instances (40 CFR Part 58 Appendix D, 4.6 (b)). The PM_{10} monitoring network peaked in size in 1995 with 1,665 stations reporting data.

In 2018, there were 714 PM_{10} stations in operation to support comparison of the PM_{10} data to the NAAQS, trends, and reporting and forecasting of the AQI. Though the PM_{10} network is relatively stable, monitoring agencies may continue divesting of some of the PM_{10} monitoring stations where concentration levels are low relative to the NAAQS.

While the PM_{10} network is national in scope, there are areas of the west, such as California and Arizona, with substantially higher PM_{10} station density than the rest of the country. In the PM_{10} mass network, 365 of the stations operate automated continuous mass monitors approved as FEMs and 391 operate FRMs. About 40 of the PM_{10} stations have collocation with both continuous FEMs and FRMs. About two thirds of the PM_{10} stations with

[7] For PM_{10}, middle-scale is defined as follows: Much of the short-term public exposure to 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 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 PM_{10}, unpaved or seldomly swept parking lots associated with these sources could be an important source in addition to the vehicular emissions themselves.

[8] For 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 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.

[9] For PM_{10}, 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_{10} 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.

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 $PM_{2.5}$ Monitoring

To support the 1997 $PM_{2.5}$ NAAQS, the first PM standard with $PM_{2.5}$ as an indicator, the EPA and states implemented a $PM_{2.5}$ 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 $PM_{2.5}$ monitoring program remains one of the major ambient air monitoring programs operated across the country.

For most urban locations $PM_{2.5}$ monitors are sited at the neighborhood scale,[10] where $PM_{2.5}$ concentrations are reasonably homogeneous throughout an entire urban sub-region. In each CBSA with a monitoring requirement, at least one $PM_{2.5}$ 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 $PM_{2.5}$ NAAQS.

There are three main components of the current $PM_{2.5}$ monitoring program: FRMs, $PM_{2.5}$ 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 $PM_{2.5}$ continuous mass monitors. $PM_{2.5}$ 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 $PM_{2.5}$ monitoring program and of recent changes to $PM_{2.5}$ monitoring requirements.

2.2.3.1 Federal Reference Method and Continuous Monitors

As noted above, the $PM_{2.5}$ monitoring network began operation in 1999 with nearly 1,000 monitoring stations operating FRMs. The $PM_{2.5}$ FRM network peaked in operation in 2001 with over 1,150 monitoring stations. In the $PM_{2.5}$ network, in 2018 there were 624 FRM filter-based

[10] For $PM_{2.5}$, 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 $PM_{2.5}$ 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. $PM_{2.5}$ 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 $PM_{2.5}$ monitoring in urban areas should have this scale.

samplers that provide 24-hour $PM_{2.5}$ mass concentration data. Of these operating FRMs, 70 are providing daily $PM_{2.5}$ data, 422 every third day, and 132 every sixth day.

As of 2018, there are 940 continuous $PM_{2.5}$ mass monitors that provide hourly data on a near real-time basis reporting across the country. A total of 579 of the $PM_{2.5}$ 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 $PM_{2.5}$ continuous monitors were largely purchased prior to the availability of $PM_{2.5}$ continuous FEMs.

The first method approved as a continuous $PM_{2.5}$ FEM was the Met One BAM 1020. This method, approved in 2008, accounts for just over 50% of the operating $PM_{2.5}$ continuous FEMs in the country. The EPA has approved a total of 11 $PM_{2.5}$ continuous methods as FEMs. Other methods approved as continuous $PM_{2.5}$ 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.

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 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 $PM_{2.5}$ CSN, long-term measurements are made at about 76 largely urban locations comprised of either the STN or the National Core (NCore) network.[11] NCore is a

[11] 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.

multipollutant network measuring particles, gases, and basic meteorology that has been in formal operation since January 1, 2011. Particle measurements made at NCore include $PM_{2.5}$ filter-based mass, which is largely the FRM, except in some rural locations that utilize the IMPROVE program $PM_{2.5}$ mass filter-based measurement; $PM_{2.5}$ speciation using either the CSN program or IMPROVE program; and $PM_{10\text{-}2.5}$ 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.[12] 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.[13]

Specific components of fine particles are also measured through the IMPROVE monitoring program[14] 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

[12] See http://www.epa.gov/ttn/amtic/speciepg.html for more information on the $PM_{2.5}$ speciation monitoring program.

[13] 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*.

[14] 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*.

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.[15] 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[16] to support reviews of the secondary PM NAAQS.

2.2.3.3 Recent Changes to $PM_{2.5}$ Monitoring Requirements

Key changes made to the EPA's monitoring requirements as a result of the 2012 PM NAAQS review included the addition of $PM_{2.5}$ monitoring at near-road locations in core-based statistical areas (CBSAs) over 1 million in population; the clarification of terms used in siting of $PM_{2.5}$ monitors and their applicability to the NAAQS; and the provision of flexibility on data uses to monitoring agencies where their $PM_{2.5}$ continuous monitors are not providing data that meets the performance criteria used to approve the continuous method as an FEM. The addition of $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ FRM or FEM operating are 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 $PM_{2.5}$ 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 $PM_{10\text{-}2.5}$ Monitoring

In the 2006 PM NAAQS review, the EPA promulgated a new FRM for the measurement of $PM_{10\text{-}2.5}$ mass in ambient air. Although the standard for coarse particles uses a PM_{10} indicator,

[15] See the Chemical Speciation Network Assessment Interactive Website at: *https://www.sdas.battelle.org/CSNAssessment/html/Default.html*.

[16] 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*.

a new FRM for $PM_{10-2.5}$ 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 $PM_{10-2.5}$ 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_{10} and $PM_{2.5}$ low-volume FRMs operating across the country that are essentially providing the $PM_{10-2.5}$ FRM measurement by the difference method.

$PM_{10--2.5}$ 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 $PM_{10-2.5}$ method. For urban NCore stations and other State and Local Air Monitoring Stations (SLAMS) the method employed is either a $PM_{10-2.5}$ FRM, which is performed using a low-volume PM_{10} FRM collocated with a low volume $PM_{2.5}$ FRM of the same make and model, or FEMs for $PM_{10-2.5}$, including filter-based dichotomous methods and continuous methods of which several makes and models are approved. Filter-based $PM_{10-2.5}$ 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. $PM_{10-2.5}$ filter-based methods at other SLAMS typically operate every third or sixth day. For IMPROVE, which is largely a rural network, $PM_{10-2.5}$ measurements are made with two sample channels; one each for PM_{10} and $PM_{2.5}$. All IMPROVE program samplers operate every third day. All together there were 279 stations in 2018 where $PM_{10-2.5}$ 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_2 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.[17] 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, 2019). 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

[17] 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.

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.[18]

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 $PM_{2.5}$ 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.[19] As illustrated in Figure 2-7, from 1990 to 2014, SO_2 emissions have undergone the largest declines while NH_3 emissions have undergone the smallest change. Declining SO_2 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, 2018, section 2.3.2.1). In more recent years (i.e., 2002 to 2014), emissions of SO_2 and NO_X have undergone the largest declines, while direct $PM_{2.5}$ and NH_3 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 2-7 and Table 2-1.[20] For example, Hand et al. (2012) studied reductions in EGU-related annual SO_2 emissions during the 2001−2010 period and found that while SO_2 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.

[18] 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*.

[19] 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/*.

[20] 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*.

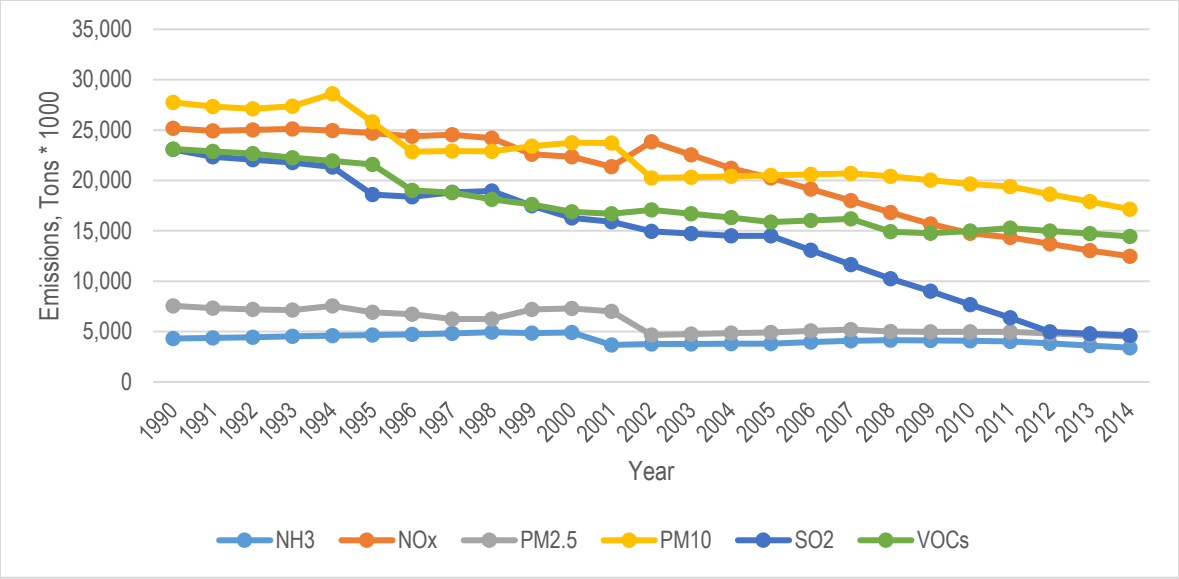

Figure 2-7.National emission trends of $PM_{2.5}$, PM_{10}, and precursor gases from 1990 to 2014.[21]

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_3	-21%	-10%	Agricultural Sources (Fertilizer and Livestock Waste), Fires
NO_X	-50%	-48%	EGUs, Mobile Sources
SO_2	-80%	-69%	EGUs, other Stationary Sources
VOCs	-38%	-15%	Solvents, Fires, Mobile Sources
$PM_{2.5}$	-40%	-4%	Dust, Fires
PM_{10}	-38%	-15%	Dust, Fires

2.3.2 Trends in Monitored Ambient Concentrations

2.3.2.1 National Characterization of $PM_{2.5}$ Mass

At long-term monitoring sites in the U.S., annual $PM_{2.5}$ concentrations from 2015 to 2017 averaged 8.0 $\mu g/m^3$ (ranging from 3.0 to 18.2 $\mu g/m^3$) and the 98th percentiles of 24-hour concentrations averaged 20.9 $\mu g/m^3$ (ranging from 9.2 to 111 $\mu g/m^3$). Figure 2-8 (top panels) shows that the highest ambient $PM_{2.5}$ concentrations occur in the west, particularly in California and the Pacific northwest. Much of the eastern U.S. has lower ambient concentrations, with

[21] Emission trends in Figure 2-7 do not include wildfire emissions.

annual average concentrations generally at or below 12.0 $\mu g/m^3$ and 98^{th} percentiles of 24-hour concentrations generally at or below 30 $\mu g/m^3$.

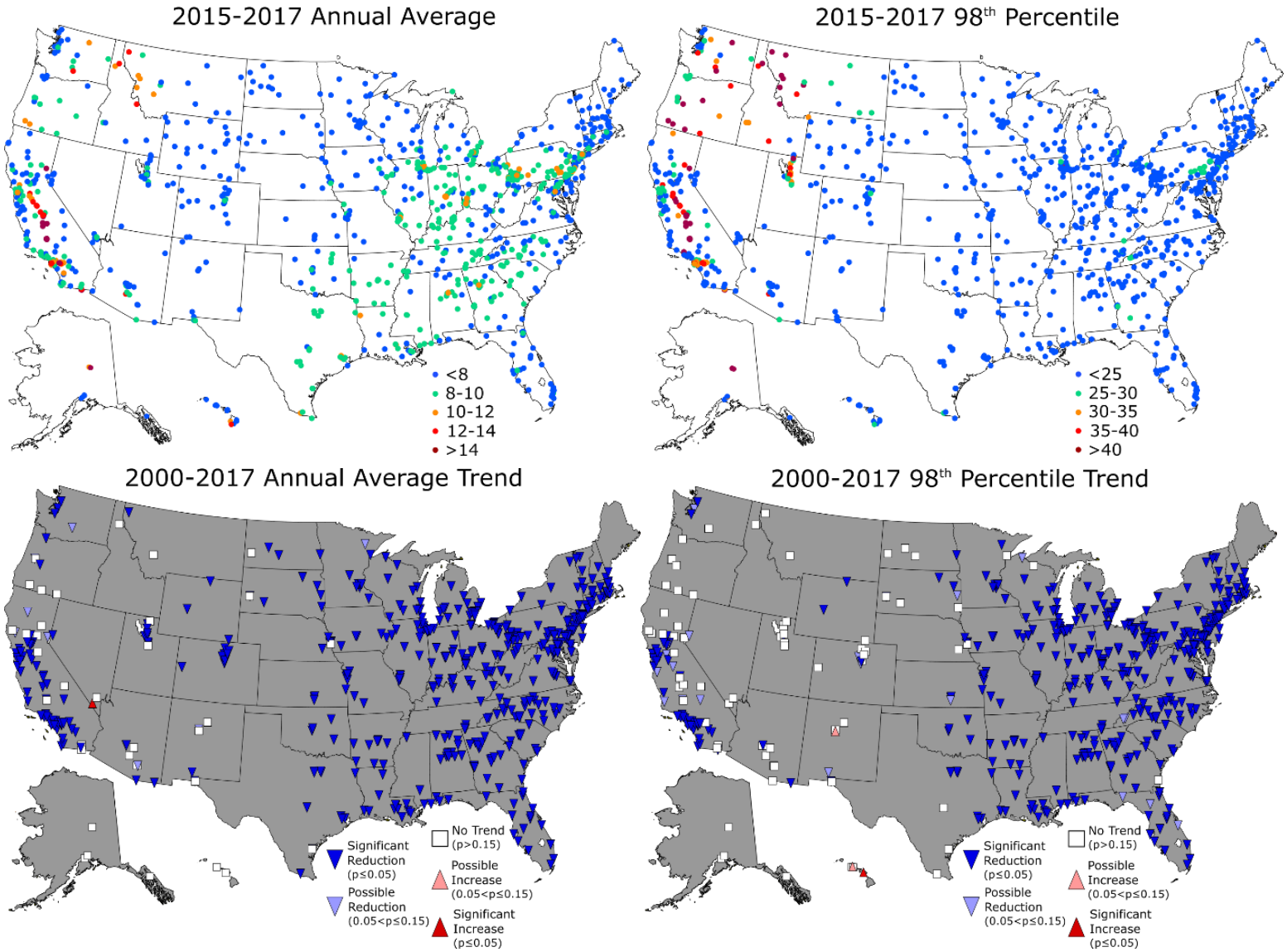

Figure 2-8.Annual average and 98th percentile $PM_{2.5}$ concentrations (in μg/m^3) from 2015-2017 (top) and linear trends and their associated significance (based on p-values) in $PM_{2.5}$ concentrations from 2000-2017 (bottom).

Analysis of monthly data indicate distinct peaks in national ambient $PM_{2.5}$ concentrations during the summer and the winter (U.S. EPA, 2018, Figure 2-22). Through 2008, the summer peaks reflected the highest national average $PM_{2.5}$ concentrations. These summer peaks in ambient $PM_{2.5}$ concentrations were largely a consequence of summertime peaks in SO_2 emissions from power plants in the eastern U.S., and subsequent sulfate formation. However, substantial reductions in SO_2 emissions (see above and U.S. EPA, 2018, sections 2.5.1.1.1 and 2.5.2.2.1) have changed this pattern. Starting in 2009, winter peaks in national average $PM_{2.5}$ concentrations have been higher than those in the summer (U.S. EPA, 2018, section 2.5.2.2.1). This pattern is illustrated by data from 2013 to 2015, when average winter $PM_{2.5}$ concentrations were about 11 $\mu g/m^3$, average summer concentrations were about 9 $\mu g/m^3$, and average spring and fall concentrations were about 7 $\mu g/m^3$ (Chan et al., 2018).

The ambient $PM_{2.5}$ concentrations in Figure 2-8 reflect the substantial reductions that have occurred across much of the U.S. over recent years (Figure 2-8, bottom panels and Figure 2-9). From 2000 to 2017, national annual average $PM_{2.5}$ concentrations have declined from 13.5 $\mu g/m^3$ to 8.0 $\mu g/m^3$, a 41% decrease (Figure 2-9).[22] These declines have occurred at both urban and rural monitoring sites, although urban $PM_{2.5}$ concentrations remain consistently higher than those in rural areas (Chan et al., 2018).

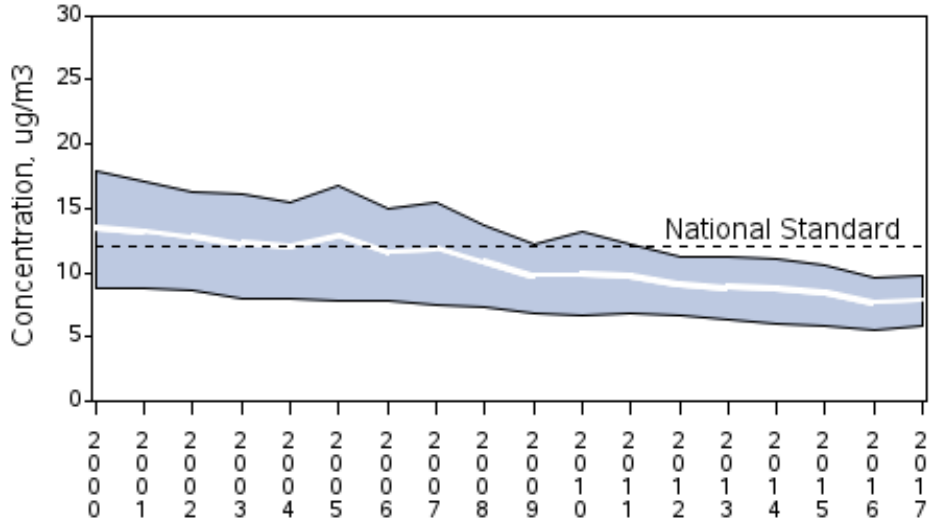

Figure 2-9.Seasonally-weighted annual average $PM_{2.5}$ concentrations in the U.S. from 2000 to 2017 (429 sites).

Analyses at individual monitoring sites indicate that declines in ambient $PM_{2.5}$ concentrations have been most consistent across the eastern U.S. and in parts of coastal California, where both annual average and 98th percentiles of 24-hour concentrations have

[22] 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.

declined significantly (Figure 2-8, bottom panels). In contrast, trends in ambient $PM_{2.5}$ 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 98th percentiles of 24-hour concentrations (Figure 2-8, bottom panels). Trends in annual average $PM_{2.5}$ concentrations have been highly correlated with trends in 98th percentiles of 24-hour concentrations at individual sites (Figure 2-10). 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).

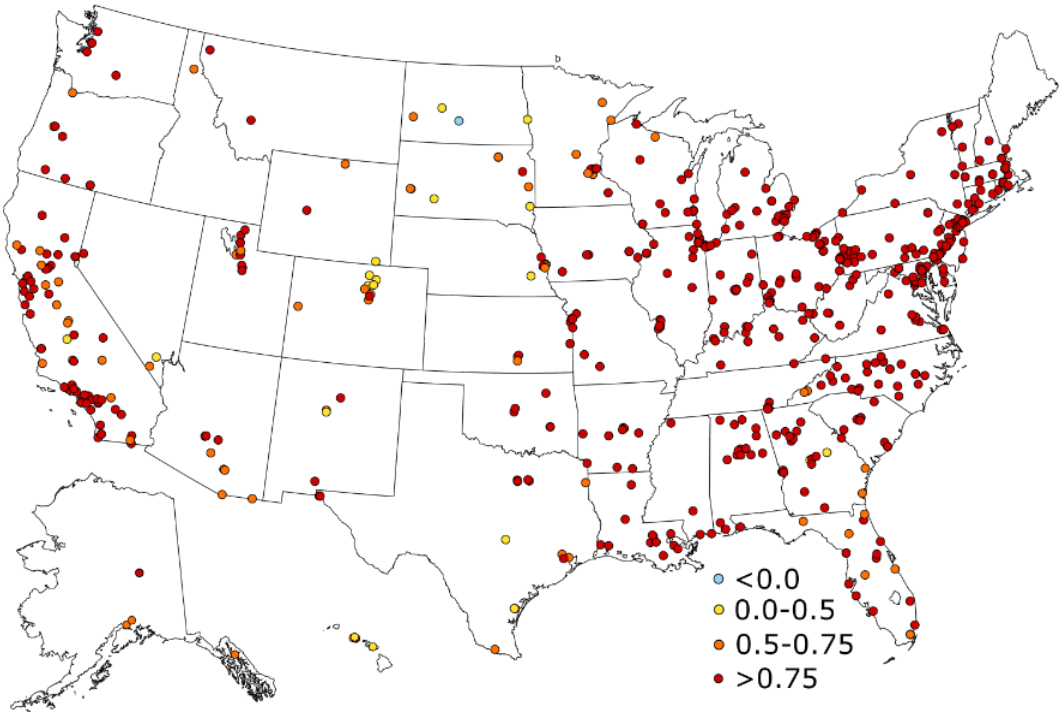

Figure 2-10.Pearson's correlation coefficient between annual average and 98th percentile of 24-hour $PM_{2.5}$ concentrations from 2000-2017.

2.3.2.2 Characterization of $PM_{2.5}$ 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 $PM_{2.5}$ design values within a CBSA are positively correlated with some noticeable regional variability (Figure 2-11). The regions that cluster outside of the typical annual/daily design value ratio line in Figure 2-11 are the Southeast and Northwest U.S. In the Southeast U.S., the annual design values are high relative to the daily design values due to the lack of seasonality in the concentrations and infrequent impacts of episodic events like wildfire or dust storms. On the other hand, the

Northwest U.S. has very high daily design values relative to the annual design values. This is due to episodically high PM2.5 concentrations that affect the region, both from wintertime stagnation events and summer/fall wildfire smoke events.[23] The relatively small population and low emissions in the region result in much lower $PM_{2.5}$ concentrations during the other parts of the year not affected by these episodes.

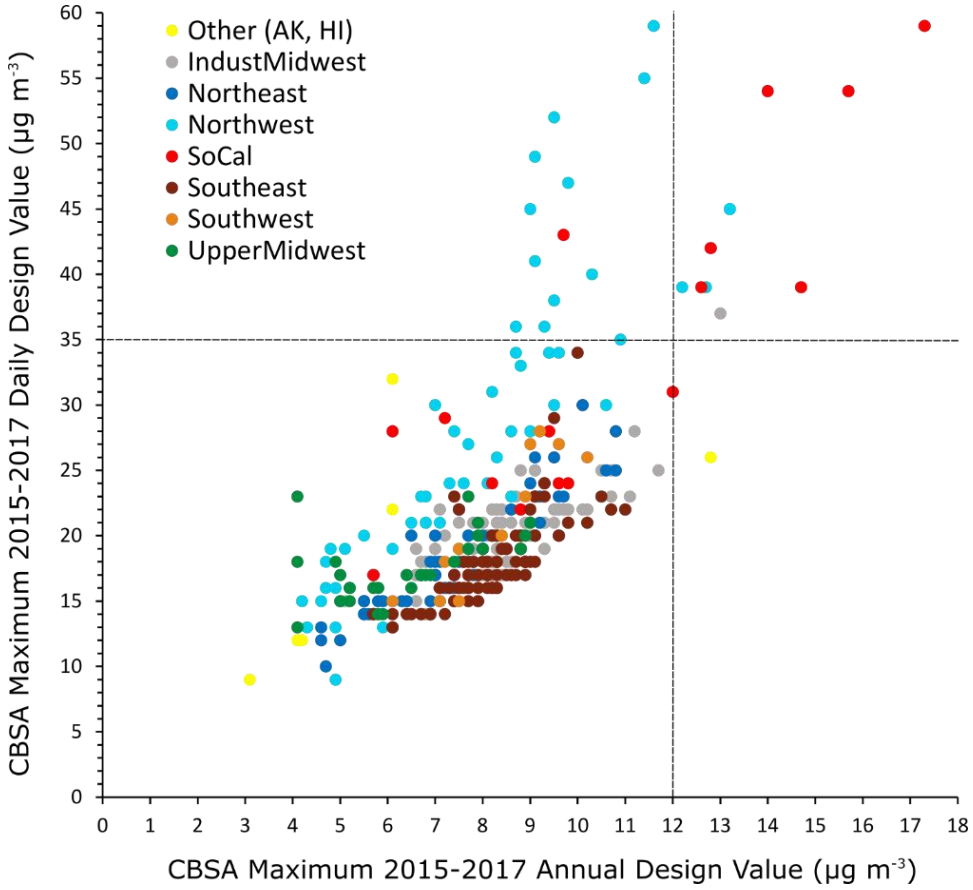

Figure 2-11.Scatterplot of CBSA maximum annual versus daily design values (2015-2017).

2.3.2.2.2 $PM_{2.5}$ Near Major Roadways

Because of its longer atmospheric lifetime (U.S. EPA, 2018, section 2.2), $PM_{2.5}$ is expected to exhibit less spatial variability on an urban scale than UFP or $PM_{10–2.5}$ (U.S. EPA, 2018, section 2.5.1.2.1). Analyses in the 2009 ISA for PM indicated that correlations between $PM_{2.5}$ monitoring sites up to a distance of 100 km from each other were greater than 0.75 in most

[23] Due to the recent time period shown in Figure 2-11, 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 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, 2018, section 2.5.1.2.1). The recent deployment of $PM_{2.5}$ 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 $PM_{2.5}$, along with NO_2 and CO, near major roads in CBSAs with populations greater than 1 million. $PM_{2.5}$ 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 $PM_{2.5}$ concentrations and the concentrations at other sites in the same CBSA) was 1.2 $\mu g/m^3$ for 2014 to 2015. The near-road increment has a diurnal cycle, with a peak during the morning rush hour (Figure 2-12).

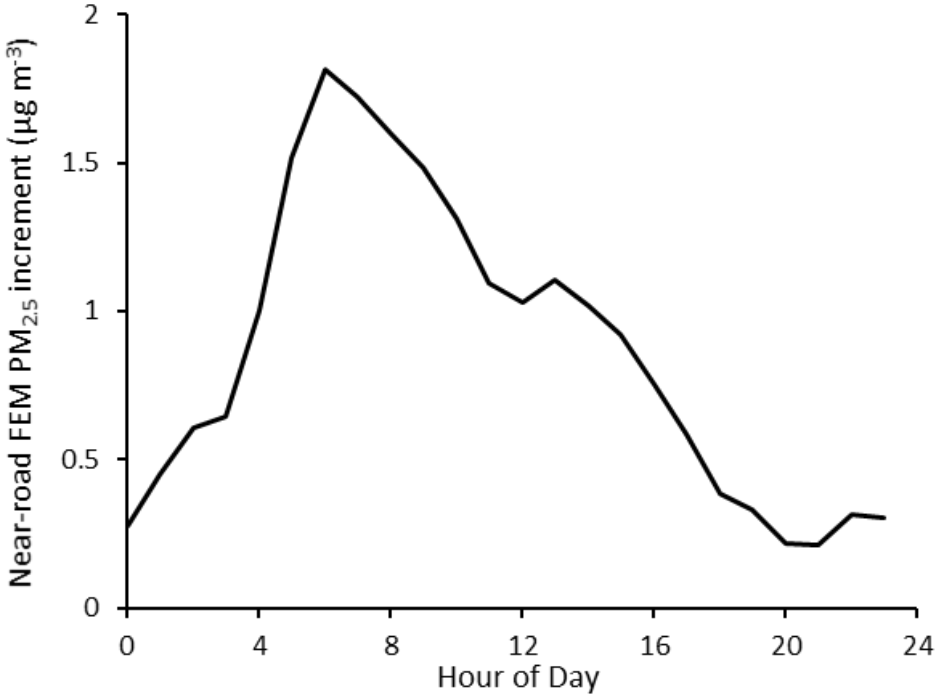

Figure 2-12.Network-wide average of the hourly near-road $PM_{2.5}$ increment through 2017.

Analyses of recent data indicate that, of the 25 CBSAs with valid design values[24] at the near-road site(s) from 2015 to 2017, 52% measured the highest annual design value at the near-road site while 24% measured the highest 24-hour design value at the near-road site (Table 2-2).

[24] 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 $PM_{2.5}$ design values for the 2015-2017 period.

Of the CBSAs with highest annual design values at near-road sites, those design values were, on average, 0.7 μg/m^3 higher than at the highest measuring non-near-road sites (range is 0.1 to 2.0 μg/m^3 higher at near-road sites).

Table 2-2.Daily and annual $PM_{2.5}$ 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 $PM_{2.5}$ concentrations, analyses of the data at one near-road-like site in Elizabeth, NJ, [25] show that the annual average increment has generally decreased between 1999 and 2017 from about 2.0 μg/m^3 to about 1.3 μg/m^3 (Figure 2-13). The trend in the near-road increment of elemental carbon at the Elizabeth, NJ site has shown a similar reduction, with

[25] 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.

values of ~1.0 μg/m^3 in 2000 decreasing to ~0.5 μg/m^3 in 2017. These data are consistent with the timing of EPA emission standards for motor vehicles.[26] 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 $PM_{2.5}$ increments.

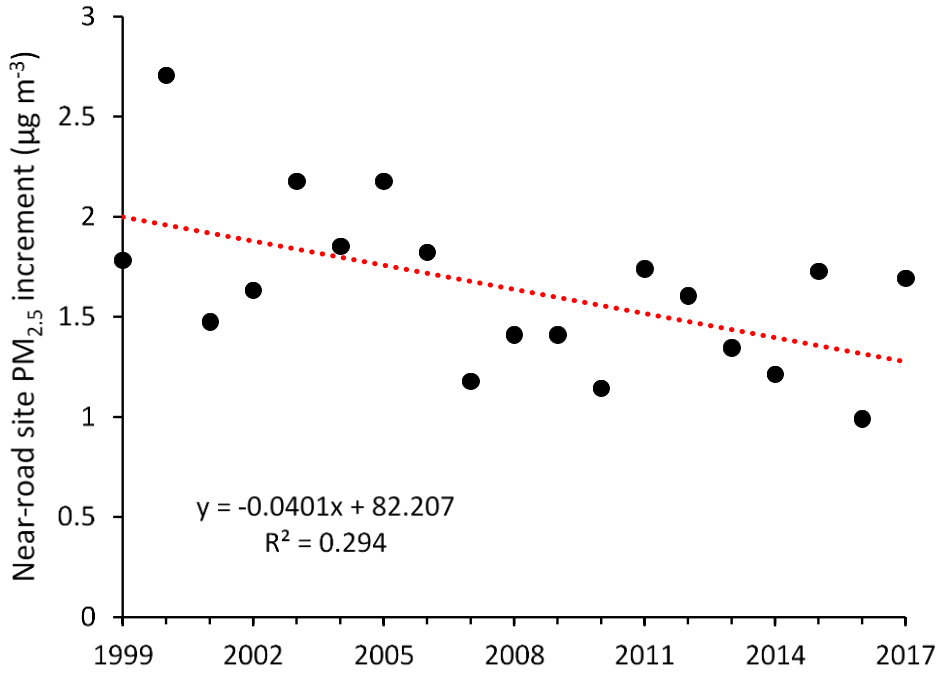

Figure 2-13.Annual average near-road increment for $PM_{2.5}$ at the Elizabeth, NJ site.

2.3.2.2.3 Sub-Daily Concentrations of $PM_{2.5}$

Ambient $PM_{2.5}$ concentrations can exhibit a diurnal cycle that varies due to impacts from intermittent emission sources, meteorology, and atmospheric chemistry. The $PM_{2.5}$ monitoring network in the U.S. has an increasing number of continuous FEM monitors reporting hourly $PM_{2.5}$ mass concentrations that reflect this diurnal variation. 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). Because a focus on annual average and 24-hour average $PM_{2.5}$ 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 $PM_{2.5}$ concentrations across the U.S. Figure 2-14 below presents the frequency distribution of 2-hour average $PM_{2.5}$ mass concentrations from all

[26] See *https://www.epa.gov/diesel-fuel-standards/diesel-fuel-standards-and-rulemakings#nonroad-diesel*.

FEM $PM_{2.5}$ monitors in the U.S. for 2015-2017.[27] At sites meeting the current primary $PM_{2.5}$ standards, these 2-hour concentrations generally remain below 11 µg/m^3, and virtually never exceed 32 µg/m^3. Two-hour concentrations are higher at sites violating the current standards, generally remaining below 19 µg/m^3 and virtually never exceeding 69 µg/m^3.

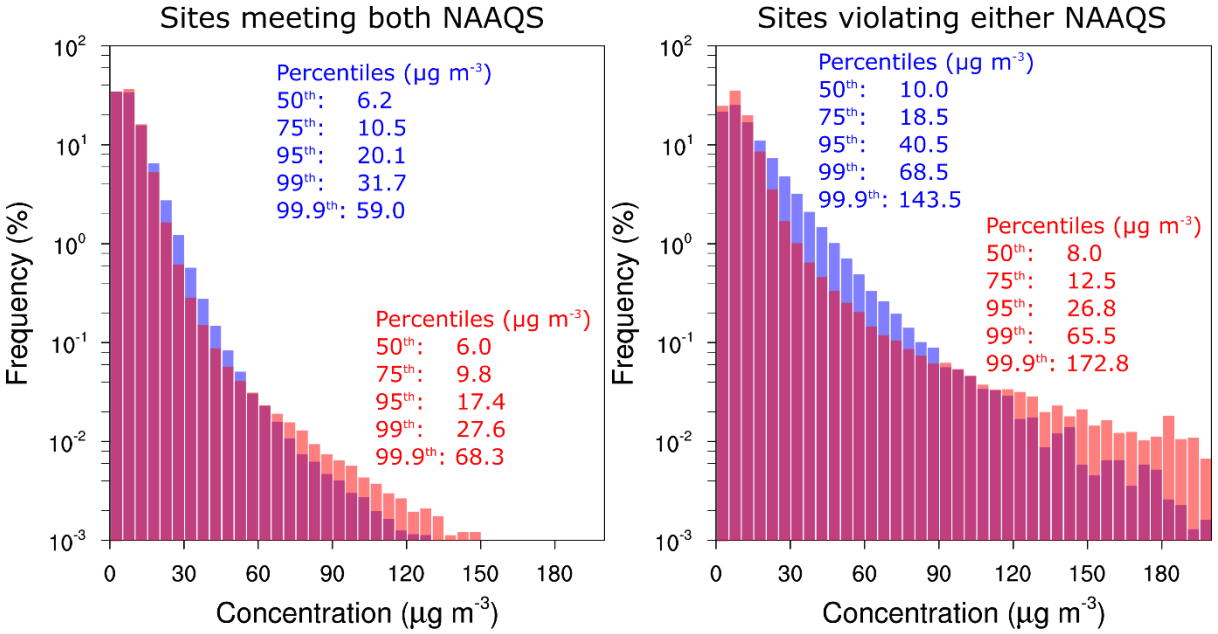

Figure 2-14.Frequency distribution of 2015-2017 2-hour averages for sites meeting or violating the annual $PM_{2.5}$ NAAQS for October to March (blue) and April to September (red).

The extreme upper end of the distribution of 2-hour $PM_{2.5}$ concentrations is shifted higher during the warmer months (red in Figure 2-14), 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 (see Appendix A, Figure A-1). When the period of peak wildfire frequency is excluded from the analysis (blue in Figure 2-14), the extreme upper end of the distribution is reduced.

2.3.2.3 Chemical Composition of $PM_{2.5}$

Based on recent air quality data, the major chemical components of $PM_{2.5}$ 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

[27] As discussed further in section 3.2, $PM_{2.5}$ controlled human exposure studies often examine 2-hour exposures. Thus, when evaluating those studies in the context of the current primary $PM_{2.5}$ standards, it is useful to consider the distribution of 2-hour $PM_{2.5}$ concentrations.

high concentrations of organic carbon are widespread across most of the Continental U.S., as shown in Figure 2-15. 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.

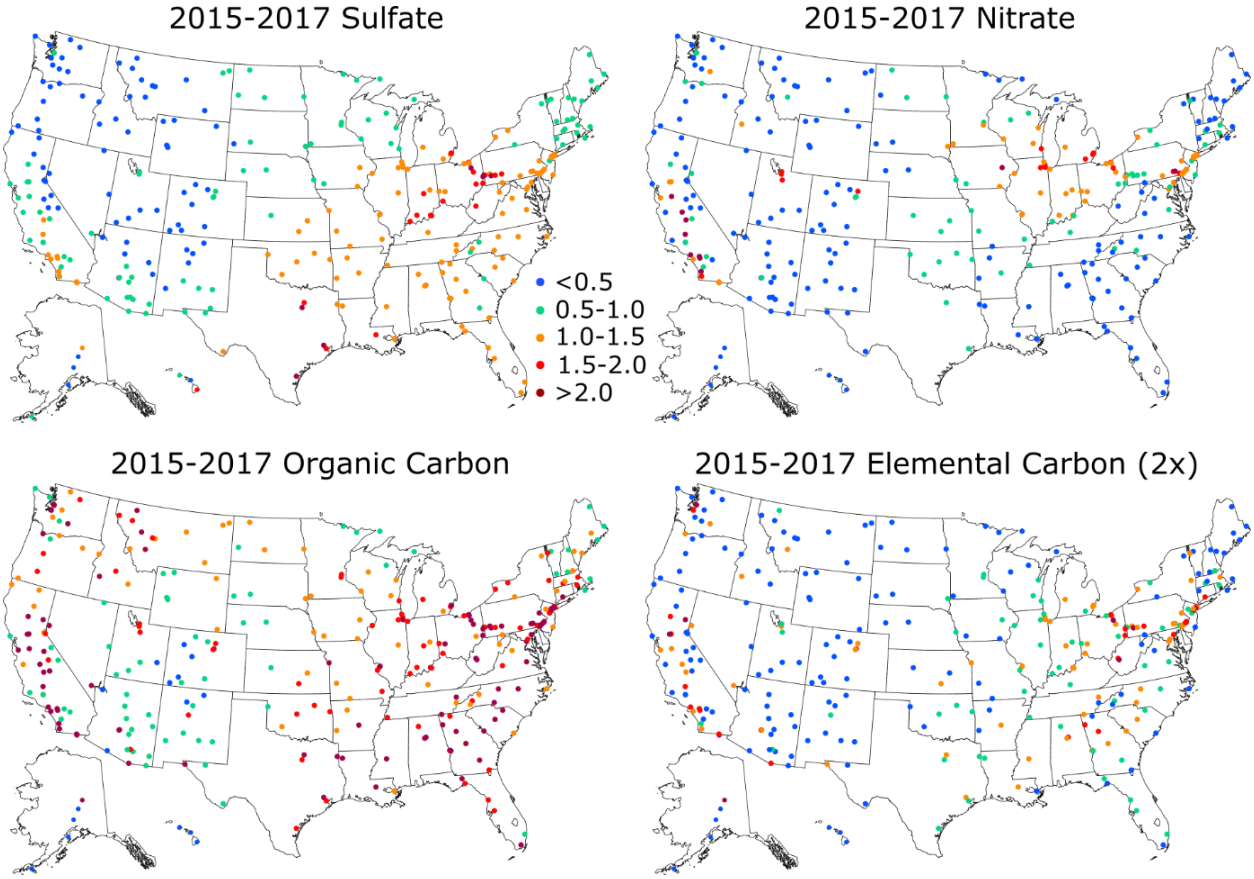

Figure 2-15.Annual average $PM_{2.5}$ sulfate, nitrate, organic carbon, and elemental carbon concentrations (in µg/m^3) from 2015-2017.

An examination of $PM_{2.5}$ composition trends can provide insight into the factors contributing to overall reductions in ambient $PM_{2.5}$ concentrations. The biggest change in $PM_{2.5}$ composition that has occurred in recent years is the reduction in sulfate concentrations due to reductions in SO_2 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 $PM_{2.5}$ mass in many locations (U.S. EPA, 2018, Figure 2-19). The overall reduction in sulfate concentrations has contributed substantially to the decrease in national average $PM_{2.5}$ concentrations as well as the decline in the fraction of PM_{10} mass accounted for by $PM_{2.5}$ (U.S. EPA, 2018, section 2.5.1.1.6; section 2.3.1 above).

2.3.2.4 National Characterization of PM_{10} Mass

At long-term monitoring sites in the U.S., the 2015-2017 average of 2nd highest 24-hour PM_{10} concentration was 56 μg/m^3 (ranging from 18 to 173 μg/m^3) (Figure 2-16, top panels).[28] The highest PM_{10} concentrations tend to occur in the western U.S. Seasonal analyses indicate that ambient PM_{10} 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, 2018, Table 2-5). This is due to fact that the major PM_{10} emission sources, dust and agriculture, are more active during the warmer and drier periods of the year.

[28] The form of the current 24-hour PM_{10} standard is one-expected-exceedance, averaged over three years.

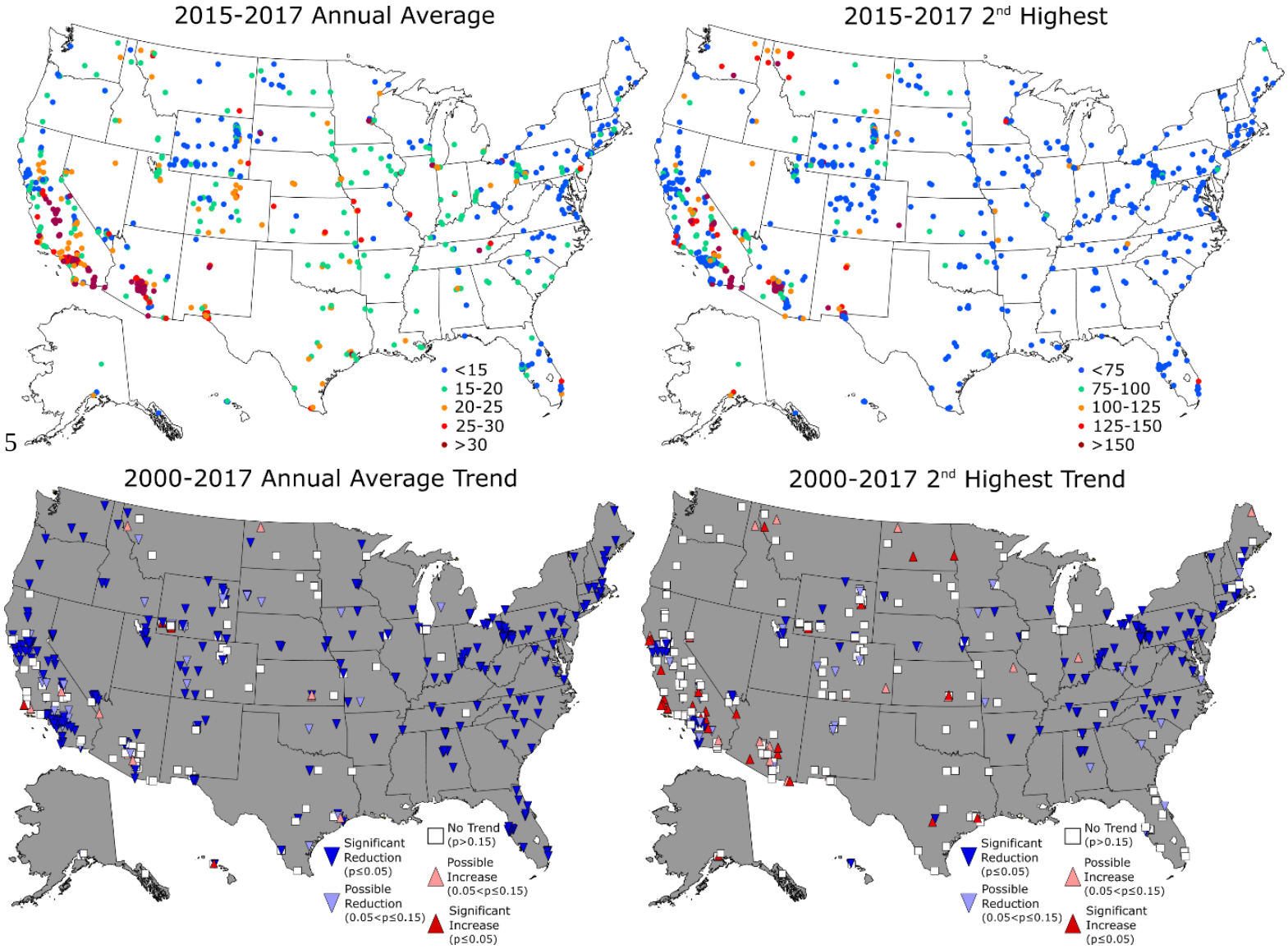

Figure 2-16.Annual average and 2nd highest PM_{10} concentrations (in µg/m^3) from 2015-2017 (top) and linear trends and their associated significance in PM_{10} concentrations from 2000-2017 (bottom).

Recent ambient PM_{10} concentrations reflect reductions that have occurred across much of the U.S. (Figure 2-16, bottom panels). From 2000 to 2017, 2nd highest 24-hour PM_{10} concentrations have declined by about 30% (Figure 2-17).[29] Analyses at individual monitoring sites indicate that annual average PM_{10} concentrations have declined at most sites across the U.S., with much of the decrease in the eastern U.S. associated with reductions in $PM_{2.5}$ concentrations. Annual second highest 24-hour PM_{10} concentrations have generally declined in the eastern U.S., while concentrations in the much of the midwest and western U.S. have remained unchanged or increased since 2000 (Figure 2-16, bottom panels).

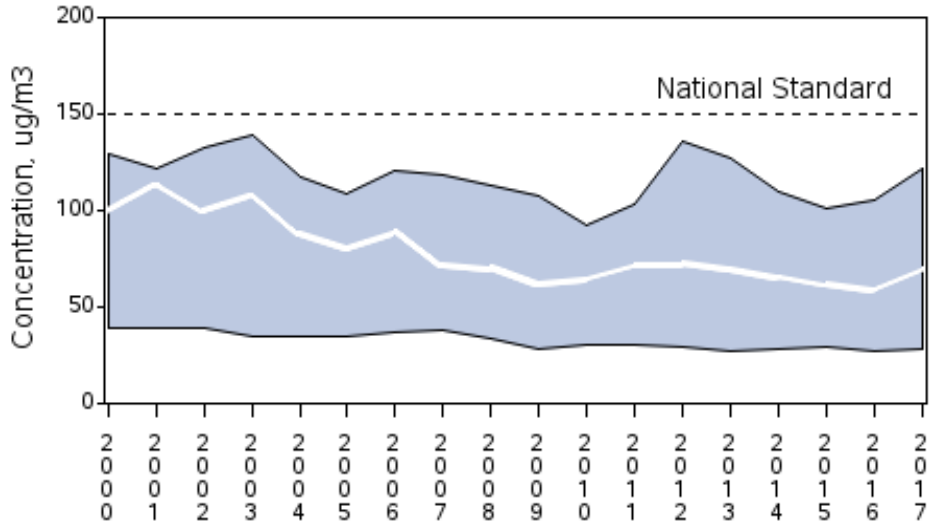

Figure 2-17.National trends in Annual 2nd Highest 24-Hour PM_{10} concentrations from 2000 to 2017 (131 sites).

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 $PM_{2.5}$ and $PM_{10–2.5}$ to PM_{10} mass. $PM_{2.5}$ generally contributes more to annual average PM_{10} mass in the eastern U.S. than the western U.S. (Figure 2-18). At most sites in the eastern U.S., the majority of PM_{10} mass is comprised of $PM_{2.5}$. Similar east-west patterns are observed for both urban/suburban and rural sites. As ambient $PM_{2.5}$ concentrations have declined in the eastern U.S. (section 2.3.2.2, above), the ratios of $PM_{2.5}$ to PM_{10} have also declined.

[29] For more information, see *https://www.epa.gov/air-trends/particulate-matter-pm10-trends#pmnat.*

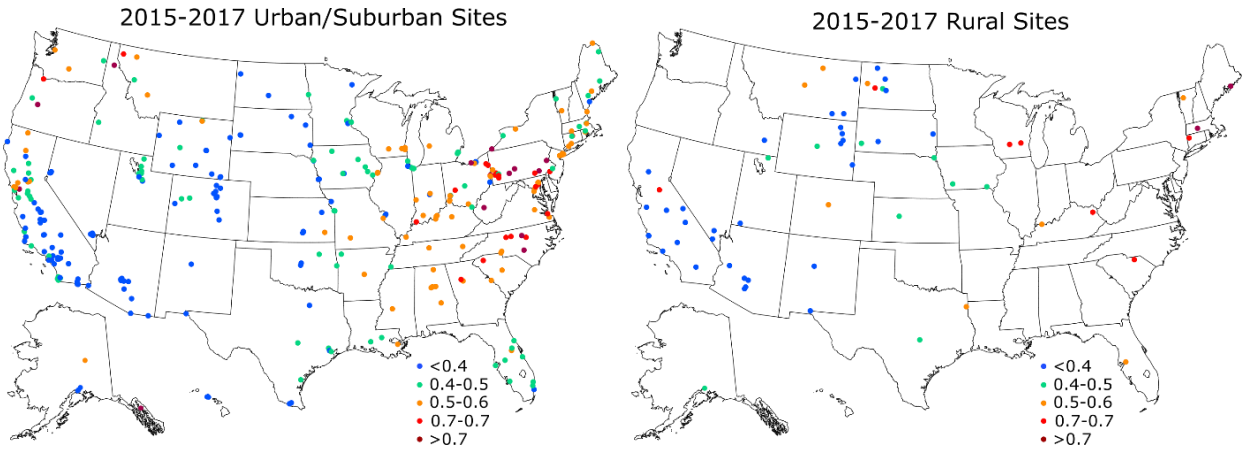

Figure 2-18.Annual average $PM_{2.5}/PM_{10}$ ratio for 2015-2017.

For days with very high PM_{10} concentrations (Figure 2-19), 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 $PM_{2.5}$.

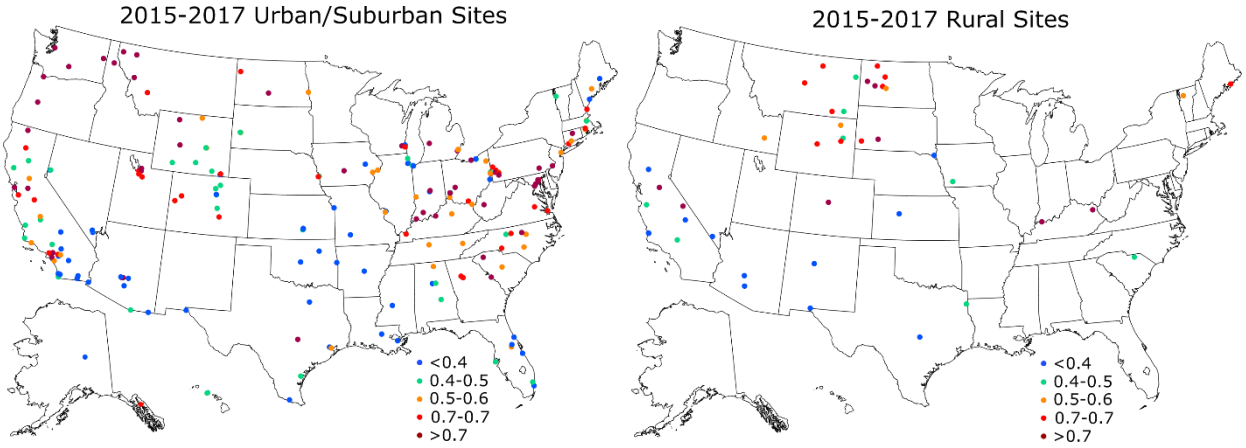

Figure 2-19.$PM_{2.5}/PM_{10}$ ratio for the second highest PM_{10} concentrations for 2015-2017.

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 2-20 (top panels), annual average and 98th 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 2-20, lower panels).

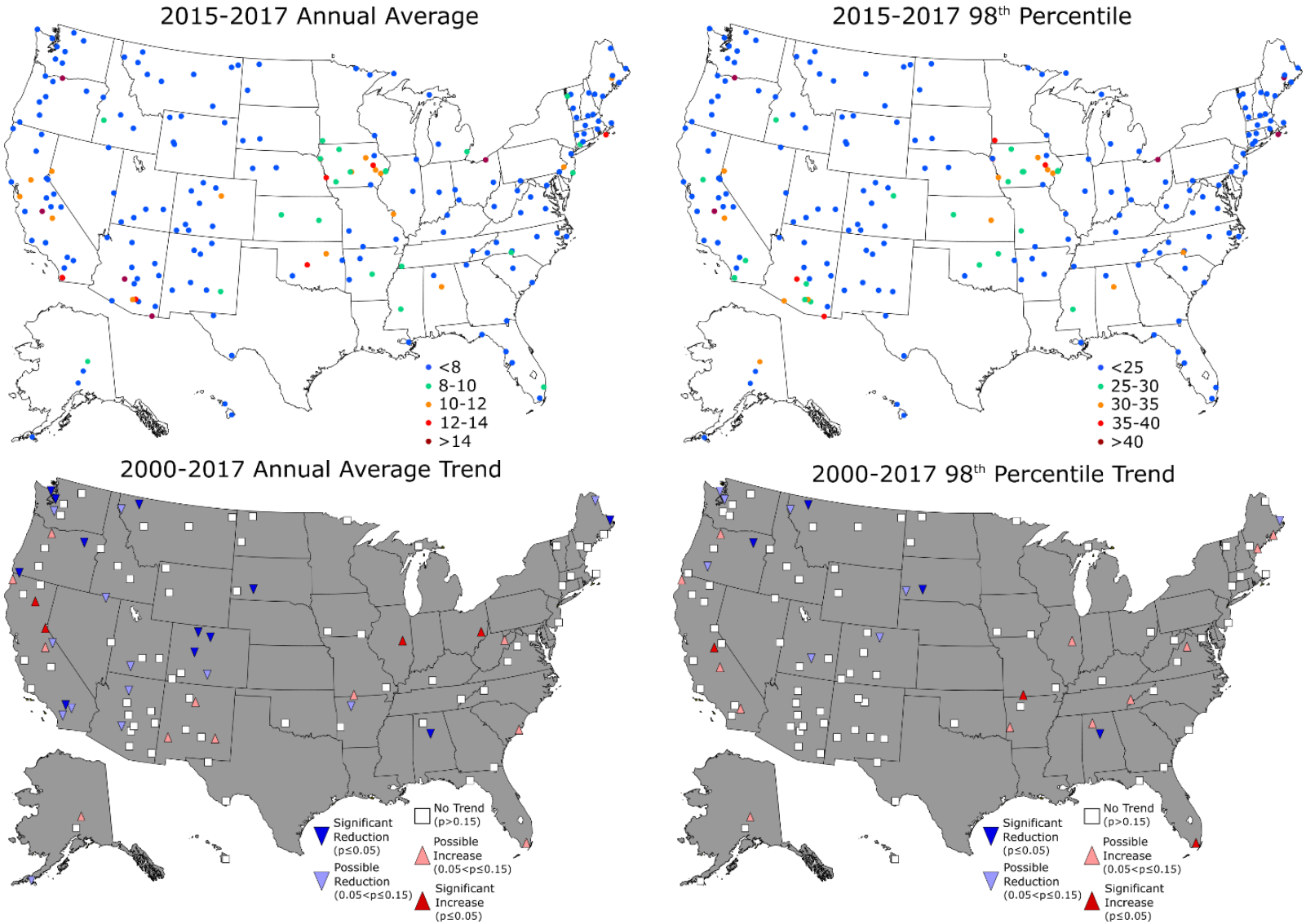

Figure 2-20.Annual average and 98th percentile $PM_{10-2.5}$ concentrations (μg/m^3) from 2015-2017 (top) and linear trends and their associated significance in $PM_{10-2.5}$ concentrations from 2000-2017 (bottom).

2.3.2.6 Characterization of the Ultrafine Fraction of $PM_{2.5}$ Mass

Compared to $PM_{2.5}$ 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^{-3} have been reported in U.S. cities (U.S. EPA, 2018). 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 2-21; U.S. EPA, 2018, 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.

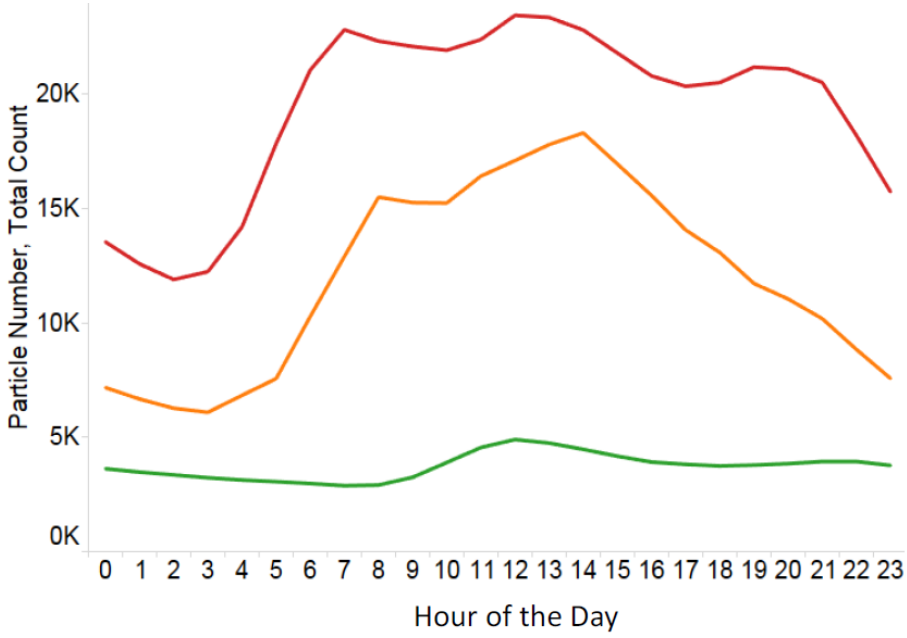

Figure 2-21.Average hourly particle number concentrations from three locations in the State of New York for 2014 to 2015 (green is Steuben County, orange is Buffalo, red is New York City). (Source: Figure 2-18 in U.S. EPA, 2018).

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^{-3} for 0.01 to 0.05 μm particles and 1,838 cm^{-3} 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 μm 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, 2018, 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 $PM_{2.5}$ mass over that same period (Figure 2-22, below). Particle number concentrations at this site are closer to those of the background site in Figure 2-21 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 $PM_{2.5}$ associated with SO_2 emission reductions (Saha et al., 2018). However, the relationship between changes in ambient $PM_{2.5}$ and UFPs cannot be comprehensively characterized due to the high variability and limited monitoring of UFPs.

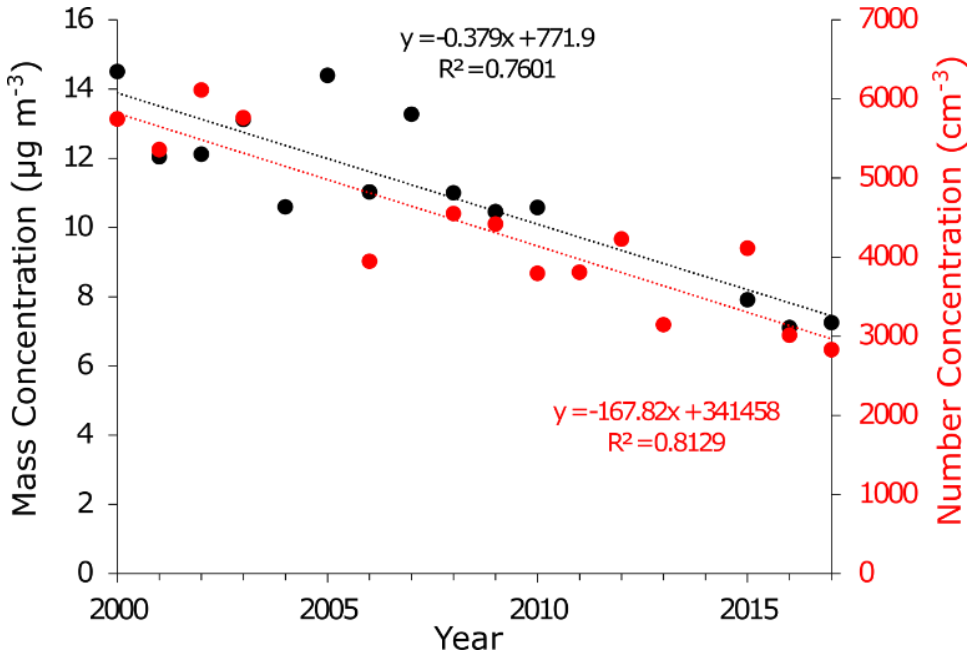

Figure 2-22.Time series of annual average mass and number concentrations (left) and scatterplot of mass vs. number concentration (right) between 2000-2017 in Bondville, IL.

2.3.3 Predicted Ambient $PM_{2.5}$ Based on Hybrid Modeling Approaches

Ambient concentrations of $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2018, chapter 3). Yet data averaging methods may not adequately represent the spatial heterogeneity of $PM_{2.5}$ within an area and are not practical for large unmonitored areas or time periods. As a result, additional methods have been developed to improve $PM_{2.5}$ 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, 2018, chapter 3). A number of recent studies have employed such methods to estimate $PM_{2.5}$ air quality concentrations across the U.S. and Canada, and to estimate population exposures for use in epidemiologic analyses (U.S. EPA, 2018, 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 $PM_{2.5}$ concentrations and rely on the moderate degree of spatial autocorrelation in $PM_{2.5}$ in many areas of the U.S. Interpolation methods often use inverse-distance or inverse-distance-squared weighted averaging of monitoring data to predict $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ predictions or AOD estimates on monitoring data. The resulting relationships are then used to develop a gridded $PM_{2.5}$ field from the CTM or AOD input field. Bayesian downscalers have been applied to develop gridded daily $PM_{2.5}$ 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 $PM_{2.5}$ at high resolution over Colorado during the fire season (Geng et al., 2018).

Surface $PM_{2.5}$ concentrations can also be predicted based on satellite retrievals of AOD and the relationship between surface $PM_{2.5}$ 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 $PM_{2.5}$ concentration for the 2001-2010 period that was combined with information from radiometrically stable satellite instruments (Boys et al., 2014) to develop global $PM_{2.5}$ 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 $PM_{2.5}$ monitoring networks to improve performance. Specifically, geographically weighted regression (GWR) of residual $PM_{2.5}$ (i.e., the difference between monitored $PM_{2.5}$ and predictions based on satellite-derived AOD) with land-use and other variables is performed to improve $PM_{2.5}$ 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 $PM_{2.5}$ fields globally and for North America at about 1-km resolution. However, the developers caution that $PM_{2.5}$ gradients may not be fully resolved at 1-km resolution due to the influence of coarser-scale data used in the model[30] and report that mean error variance decreases when averaging the 1-km fields to coarser resolution (van Donkelaar et al., 2019).

Daily $PM_{2.5}$ fields based on non-parametric (i.e., machine learning) methods have also been developed to characterize $PM_{2.5}$ over the U.S. Non-parametric methods facilitate the use of large numbers of predictor variables that may have complex nonlinear relationships with $PM_{2.5}$ concentrations that would be challenging to specify with a parametric method. For example, a neural network algorithm was used to predict daily $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ models have recently been reviewed in Chapter 3 of the draft ISA (U.S. EPA, 2018).

[30] See 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 $\mu g\ m^{-3}$ for daily $PM_{2.5}$ 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 $\mu g\ m^{-3}$ for daily $PM_{2.5}$ 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 $\mu g\ m^{-3}$ in cross validation for longer-term $PM_{2.5}$ predictions at sites in North America (van Donkelaar et al., 2015b). The Bayesian downscalers had weaker performance in cross validation (e.g., national R2: 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentration (e.g., Figure 2-23) could be due in part to the relatively sparse monitoring in remote areas, where $PM_{2.5}$ 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).

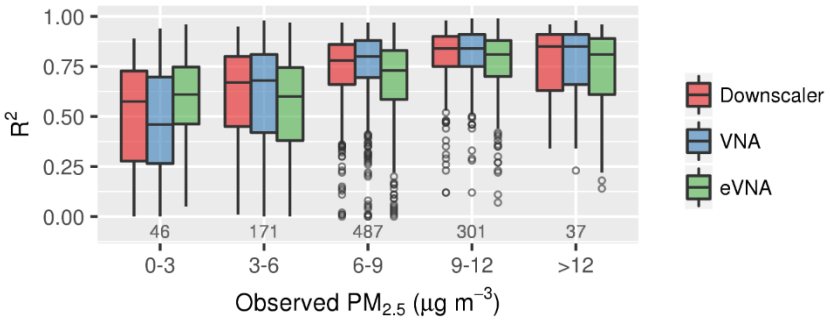

Figure 2-23.R^2 for ten-fold cross-validation of daily $PM_{2.5}$ predictions in 2015 from three methods for individual sites as a function of observed concentration. Text indicates the number of monitors in the $PM_{2.5}$ concentration range. Downscaler: Bayesian downscaler of CMAQ predictions; VNA: Voronoi Neighbor Averaging; eVNA: enhanced-VNA. From Kelly et al. (2019).

A limited number of studies have intercompared concentration predictions based on different $PM_{2.5}$ characterization methods. Huang et al., 2018 compared $PM_{2.5}$ 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 $PM_{2.5}$ concentrations from fourteen approaches of varying complexity for developing $PM_{2.5}$ 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^2 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 $PM_{2.5}$ Fields Across Approaches

To illustrate features of the spatial fields reported in the literature, the annual mean $PM_{2.5}$ concentrations for 2011 from four methods is shown in Figure 2-24, 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 $PM_{2.5}$ estimates (VD2019; van Donkelaar et al., 2019). Annual mean concentrations were developed from daily $PM_{2.5}$ predictions in the downscaler, DI2016, and HU2017 cases and from monthly $PM_{2.5}$ 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 $PM_{2.5}$ concentration for the VD2019 case (7.06 μg m^{-3}) is slightly lower than those of the other cases (7.36-7.44 μg m^{-3}), possibly because the VD2019 fields were developed using monthly (rather than daily) $PM_{2.5}$ 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 $PM_{2.5}$ concentrations predicted by the four methods in nine U.S. climate regions (Karl and Koss, 1984) are provided in Table 2-3.

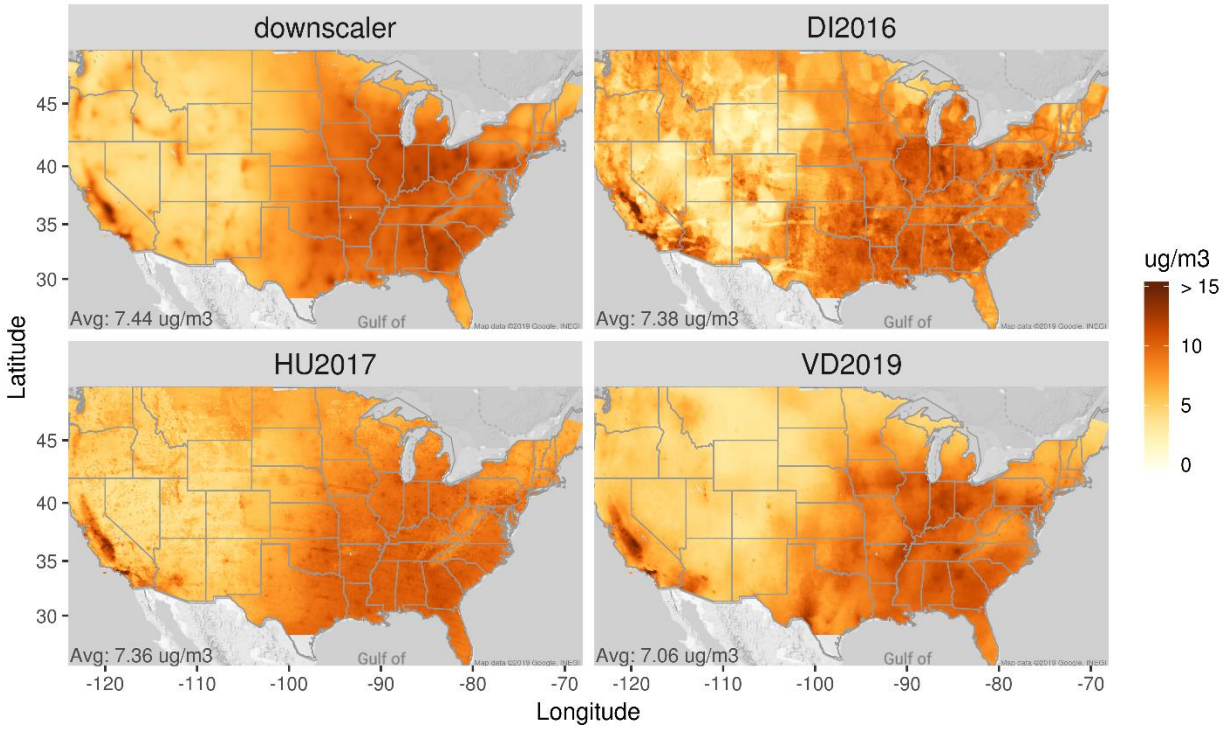

Figure 2-24.Comparison of 2011 annual average $PM_{2.5}$ concentrations from four methods. (Note: These four methods include: downscaler (Berrocal et al., 2012), DI2016 (Di et al., 2016), HU2017 (Hu et al., 2017), and VD2019 (van Donkelaar et al., 2019). Predictions have been averaged to a common 12-km grid for this comparison.

Table 2-3.Mean 2011 $PM_{2.5}$ concentration by region for predictions in Figure 2-24

Region[1]	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

[1] U.S. climate region: *https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php*.

In Figure 2-25, $PM_{2.5}$ 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 2-25, panels a and c) than the eastern region centered on New Jersey (Figure 2-25, 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 $PM_{2.5}$ concentration field, and the 1-km neural network (DI2016) produces the field with the greatest variance. Some of the largest differences in $PM_{2.5}$ 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 2-25, panel c).

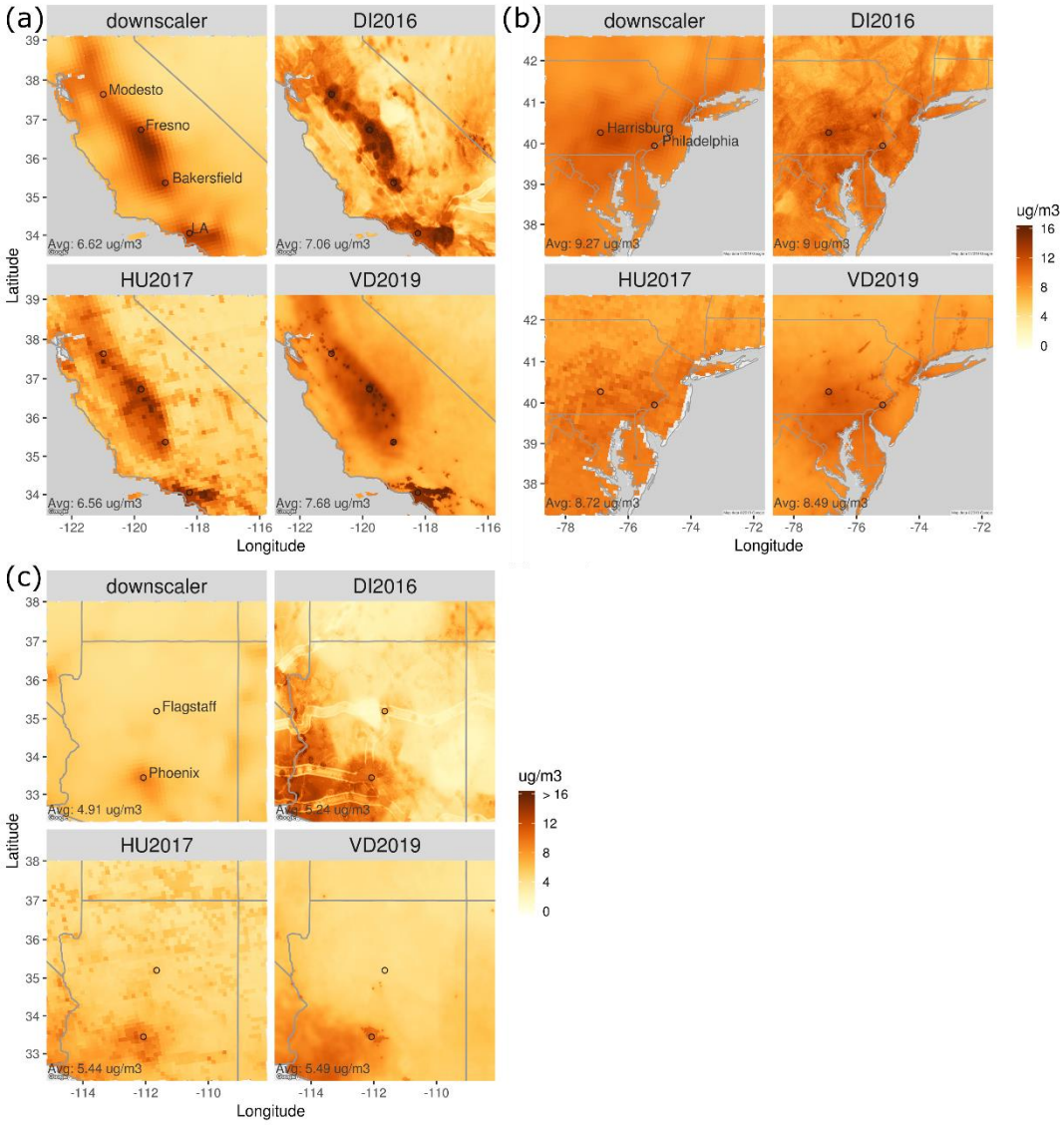

Figure 2-25.Comparison of 2011 annual average $PM_{2.5}$ concentrations from four methods for regions centered on the (a) California (b) New Jersey, and (c) Arizona. Predictions are shown at their native resolution (i.e., about 1-km for DI2016 and VD2019 and 12-km for downscaler and HU2017).

In Figure 2-26, 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 $PM_{2.5}$ 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).

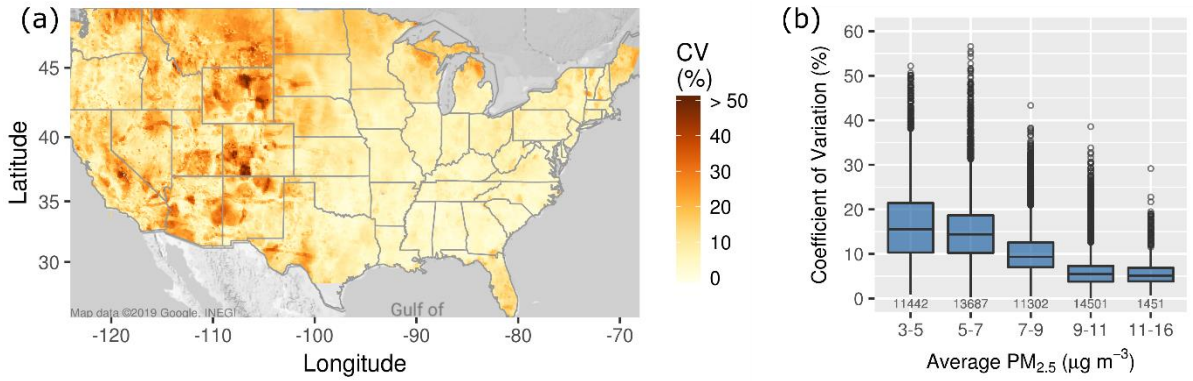

Figure 2-26.(a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in percentage units for the four models in Figure 2-24. (b) Boxplot distributions of CV for grid cells binned by the average $PM_{2.5}$ concentration for the four models. (Note: The box brackets the interquartile 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.)

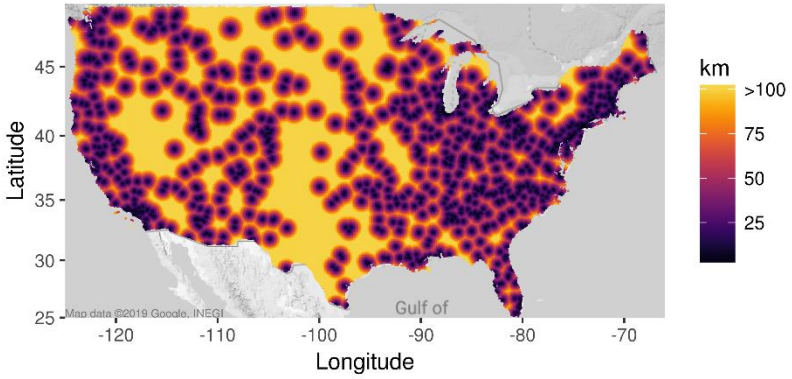

Figure 2-27.Distance from the center of the 12-km grid cells to the nearest $PM_{2.5}$ monitoring site for $PM_{2.5}$ measurements from the AQS database and IMPROVE network.

Concentrations less than 5 μg 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 2-28, top row). Concentrations between 5 and 7 μg 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 2-28, second row). The CV among methods increases with decreasing concentration (Figure 2-26 above, panel b), and the median CV is about 15% for grid cells with mean concentrations less than 7 μg m^{-3}. As illustrated by Figure 2-26 and Figure 2-28, 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.

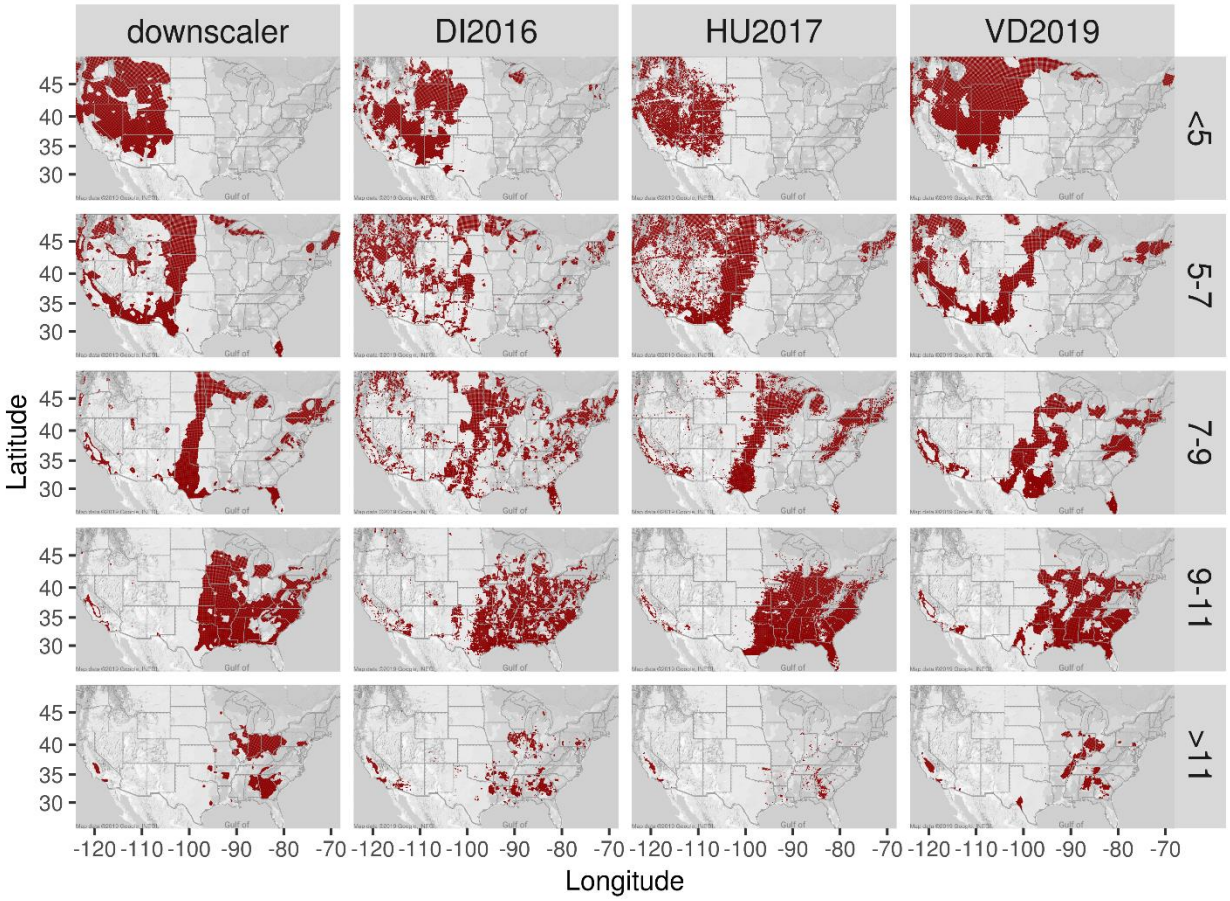

Figure 2-28.Location of $PM_{2.5}$ predictions by range in annual average concentration for the four prediction methods at their native resolution. (Note: Concentration ranges: < 5 μg/m^3, 5-7 μg/m^3, 7-9 μg/m^3, 9-11 μg/m^3, and >11 μg/m^3.**)**

The comparison of $PM_{2.5}$ 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, $PM_{2.5}$ concentrations have declined over the U.S. in the last several decades. Annual mean $PM_{2.5}$ concentrations predicted by the VD2019 method for 2011 are compared with predictions for 2001, 2006, and 2016 in Figure 2-29. The VD2019 fields capture the trend of decreasing $PM_{2.5}$ over the U.S. during this period, and the areas with annual mean $PM_{2.5}$ concentration greater than 11 μg m^{-3} in 2016 are limited to California and southwest Arizona.

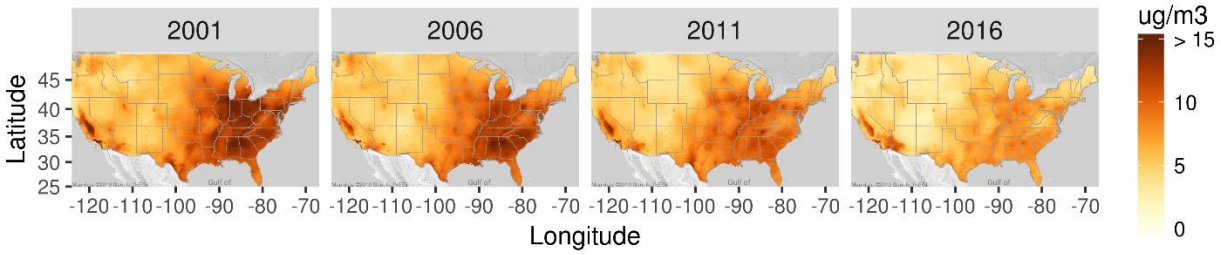

Figure 2-29.Annual mean $PM_{2.5}$ from the VD2019 method (van Donkelaar et al., 2019) for 2001, 2006, 2011, and 2016.

2.3.3.1.4 Summary

Hybrid $PM_{2.5}$ modeling methods have improved the ability to estimate $PM_{2.5}$ 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 $PM_{2.5}$ exposure in many applications. As discussed in Chapter 3 of this draft 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 $PM_{2.5}$ fields (e.g., averaging daily $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations were estimated to range from 0.5 - 3 $\mu g/m^3$ 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. 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 ($PM_{2.5}$) and coarse (PM_{10}) 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_2 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, 2018, 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.

Soil dust and sea salt have been estimated to account for less than 10% of urban $PM_{2.5}$ 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_{10} concentrations were greater than 5,000 $\mu g/m^3$, with area-wide average hourly concentrations ranging from a few hundred to a few thousand $\mu g/m^3$ (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, 2018, 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 $\mu g/m^3$) 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, 2018, 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 2-30 and Figure 2-31 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[31] image from August 4th 2017 (Figure 2-30) 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 2-31), where daily fine PM concentrations were increased from a typical baseline of less than 10 µg/m^3 to ~100 µg/m^3 during this time.

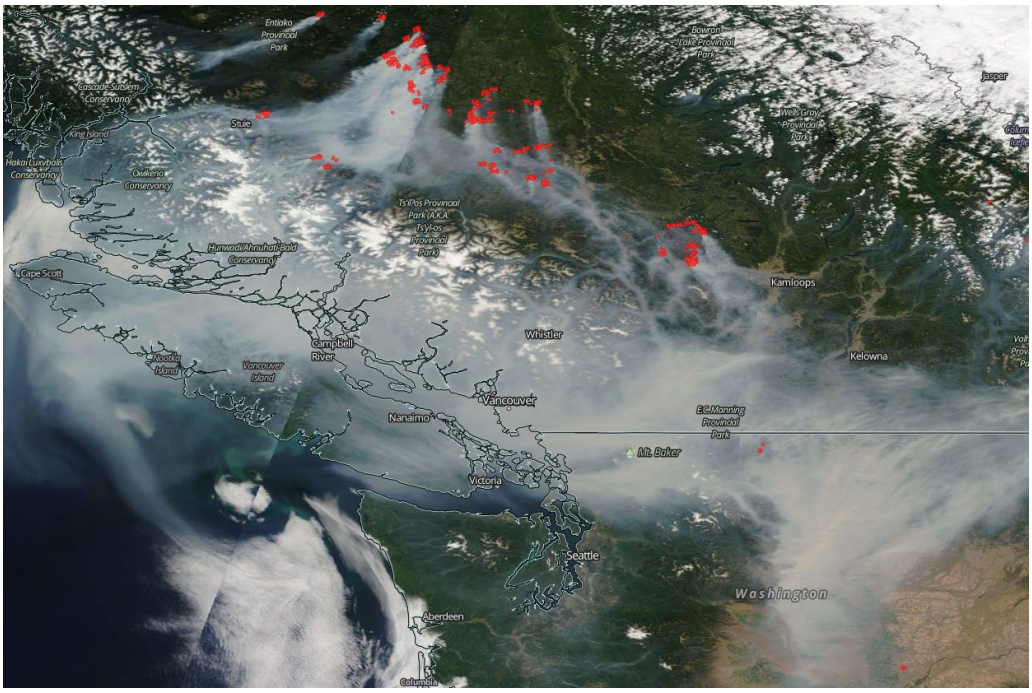

Figure 2-30.Smoke and fire detections observed by the MODIS instrument onboard the Aqua satellite on August 4th, 2017 accessed through NASA Worldview.

[31] Available from *https://worldview.earthdata.nasa.gov*.

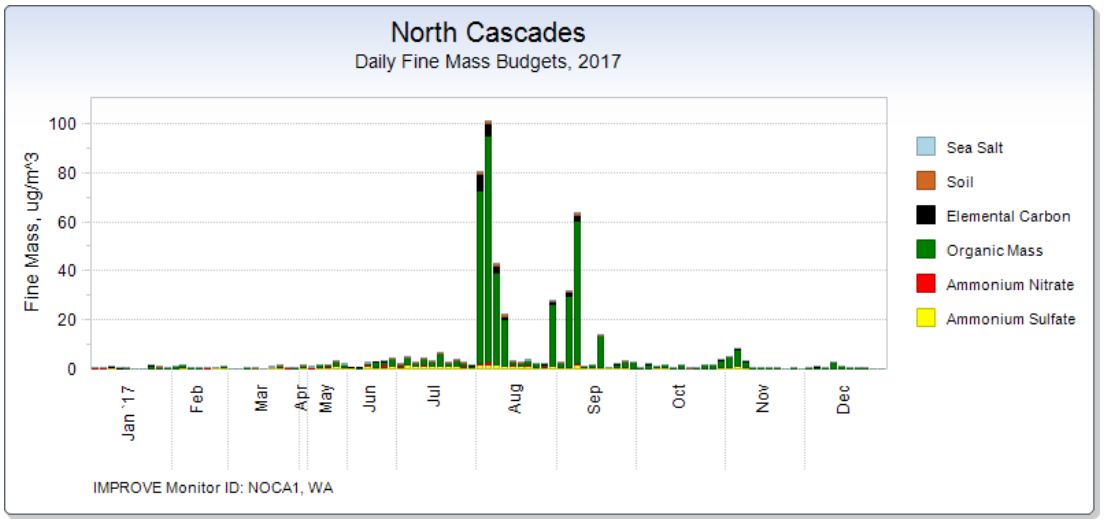

Figure 2-31.Fine PM mass time series during 2017 from the North Cascades IMPROVE site in north central Washington state.[32]

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.

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;

[32] Available at *http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_PmHazeComp*.

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_X emissions from Canada and Mexico accounted for 6% and 4% respectively of total daily inorganic $PM_{2.5}$ in the U.S. These authors also estimated that SO_X 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 $\mu g/m^3$ of sulfate $PM_{2.5}$ in the Northwestern U.S. in spring, and Leibensperger et al. (2011) estimated intercontinental contributions from Asian anthropogenic SO_2 and NO_X emissions of 0.1 - 0.25 $\mu g/m^3$ 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 $\mu g/m^3$ 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, 1999b; Prospero, 1999a; 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 μg/m^3 to annual $PM_{2.5}$ 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 μg/m^3 to daily 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 μg/m^3 (Prospero, 1999a; Prospero, 1999b).[33]

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 2-32 shows observed annual average $PM_{2.5}$ in 2004 and 2016 at the same remote monitors examined in the last ISA. The comparisons show decreases in both total $PM_{2.5}$ and ammonium sulfate across all sites examined, consistent with decreases in anthropogenic 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 μg/m^3 of the annual average $PM_{2.5}$ 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 μg/m^3 at most of the sites in 2016. The largest contribution from nitrate occurs at the BRIG1 monitor in New Jersey and is likely anthropogenic given the high density of NO_X 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 2-32 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

[33] Sample collection began in 1974, before network PM_{10} and $PM_{2.5}$ samplers were developed, and no size cut was specified (Prospero, 1999a).

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 2-32 will also include the influence of fires for some monitors. The highest organic matter contribution for any of the sites shown in Figure 2-32, including the three Southeast monitors, is approximately 2 $\mu g/m^3$. 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 $\mu g/m^3$ 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.

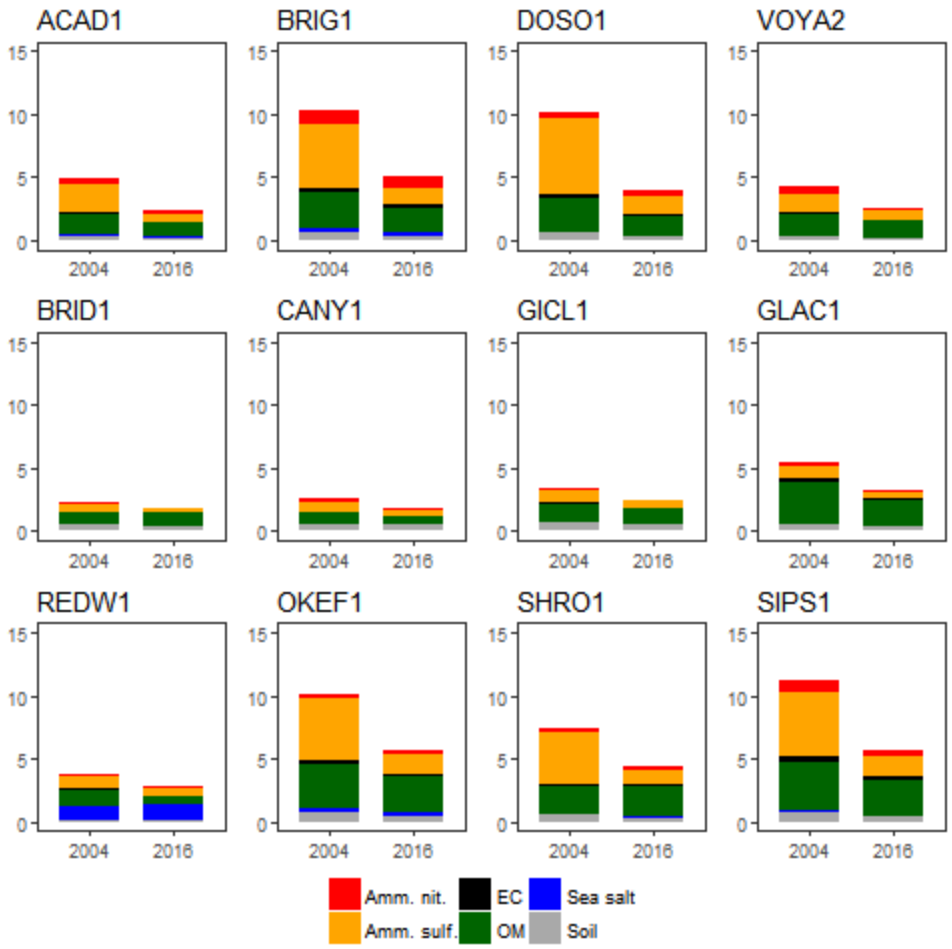

Figure 2-32.Speciated annual average IMPROVE $PM_{2.5}$ in µg/m^3 at select remote monitors during 2004 and 2016. (Note: Monitor locations are shown in Figure 2-33.)

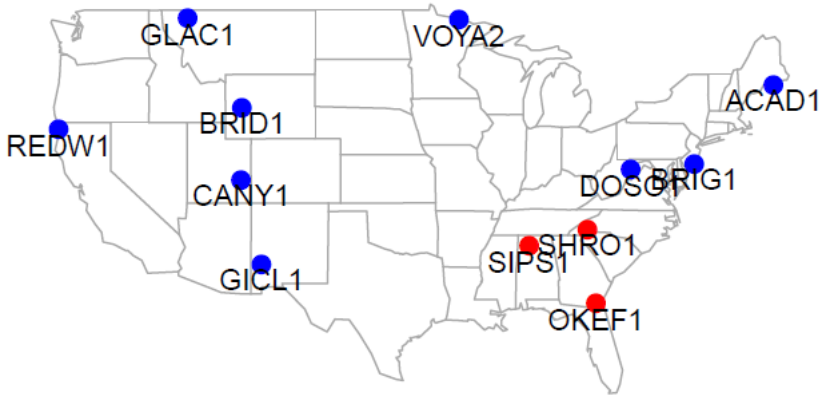

Figure 2-33.Site locations for the IMPROVE monitors in Figure 2-32. (Note: Monitors also assessed in the 2009 ISA are shown in blue. Monitors only examined in this assessment are shown in red.)

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3 REVIEW OF THE PRIMARY STANDARDS FOR $PM_{2.5}$

This chapter presents our key policy-relevant considerations and preliminary conclusions regarding the public health protection provided by the current suite of primary $PM_{2.5}$ standards and the protection that could be provided by potential alternative standards. These considerations and preliminary 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 $PM_{2.5}$ 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 $PM_{2.5}$. Sections 3.2 and 3.3 present our consideration of the available scientific evidence and our consideration of information from the $PM_{2.5}$ risk assessment, respectively. Section 3.4 summarizes our preliminary conclusions regarding the adequacy of the public health protection provided by the current primary $PM_{2.5}$ standards and the protection that could be provided by potential alternative standards. Section 3.5 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 $PM_{2.5}$ standard from 15.0 to 12.0 $\mu g/m^3$,[1] and retained the existing 24-hour $PM_{2.5}$ standard with its level of 35 $\mu g/m^3$. The 2012 decision to strengthen the suite of primary $PM_{2.5}$ standards was based on the Administrator's consideration of the extensive body of scientific

[1] The Agency also eliminated spatial averaging provisions as part of the form of the annual standard.

evidence assessed in the 2009 ISA (U.S. EPA, 2009); the quantitative risk analyses presented in the 2010 HREA (U.S. EPA, 2010);[2] the advice and recommendations of the CASAC (e.g., Samet, 2009; Samet, 2010a; 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 $PM_{2.5}$" (78 FR 3120, January 15, 2013). This included epidemiologic studies reporting health effect associations based on long-term average $PM_{2.5}$ concentrations ranging from about 15.0 $\mu g/m^3$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standards was necessary in order to provide increased public health protection. Specifically, she concluded that the then-existing suite of primary $PM_{2.5}$ 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, 2010a).

The Administrator next considered what specific revisions to the existing primary $PM_{2.5}$ standards were appropriate, given the available evidence and quantitative risk information. She considered both the annual and 24-hour $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ indicator and remained too limited to support a distinct

[2] 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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).[3] With regard to $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2010c, p. 12). In light of the evidence and the CASAC's advice, the Administrator concluded that it was "appropriate to retain $PM_{2.5}$ 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 $PM_{2.5}$ standard to provide protection from health effects associated with long- and short-term exposures to $PM_{2.5}$, 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 $PM_{2.5}$ standards, with a focus on evaluating support for the existing annual and 24-hour

[3] 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., $PM_{2.5}$, PM_{10}, $PM_{10-2.5}$, 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 $PM_{2.5}$ standard with an averaging time shorter than 24-hours (U.S. EPA, 2011, p. 2-57).[4] With regard to potential support for a seasonal averaging time, few studies were available to deduce a general pattern in $PM_{2.5}$-related risk across seasons, and these studies did not provide information on health effects associated with season-long exposures to $PM_{2.5}$ (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, 2010c, 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard as an annual arithmetic mean, averaged over 3 years, from single or multiple community-oriented monitors.[5] 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

[4] 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).

[5] 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 $PM_{2.5}$ concentration.

averaging)[6] (62 FR 38671 to 38672, July 18, 1997). In the 1997 review, the EPA also established the form of the 24-hour $PM_{2.5}$ standard as the 98th 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).[7]

In the last review, the EPA's consideration of the form of the annual $PM_{2.5}$ standard again included a focus on the issue of spatial averaging. An analysis of air quality and population demographic information indicated that the highest $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2010a, 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, 2010c, 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard should be revised to eliminate spatial averaging provisions (78 FR 3124, January 15, 2013). Thus, the level of the annual $PM_{2.5}$ 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.

[6] 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).

[7] 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 $PM_{2.5}$ standard. The Agency recognized that the existing 98th percentile form for the 24-hour standard was originally selected to provide a balance between limiting the occurrence of peak 24-hour $PM_{2.5}$ concentrations and identifying a stable target for risk management programs.[8] Updated air quality analyses in the last review provided additional support for the increased stability of the 98th percentile $PM_{2.5}$ concentration, compared to the 99th 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 98th percentile form for the 24-hour $PM_{2.5}$ standard (78 FR 3127, January 15, 2013).

3.1.1.4 Level

The EPA's approach to considering alternative levels of the $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations (e.g., areas with important local or seasonal sources) and for $PM_{2.5}$-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

[8] 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 $PM_{2.5}$-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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations to consider in judging the need for public health protection.[9] In doing so, the Administrator recognized that

[9] This information characterized the distribution of health events in the studies, and the corresponding long-term mean $PM_{2.5}$ concentrations (78 FR 3130 to 3134, January 15, 2013). The additional population-level data helped

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 $\mu g/m^3$, the Administrator noted that such a level was below the long-term mean $PM_{2.5}$ 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 $\mu g/m^3$ generally corresponded to the lower portions (i.e., about the 25th 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 $PM_{2.5}$ 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 $\mu g/m^3$ 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).[10]

In conjunction with a revised annual standard with a level of 12.0 $\mu g/m^3$, the Administrator concluded that the evidence supported retaining the 35 $\mu g/m^3$ level of the 24-hour $PM_{2.5}$ standard. Specifically, she judged that by lowering the level of the annual standard, the distribution of 24-hour $PM_{2.5}$ concentrations would be lowered as well, affording additional protection against effects associated with short-term $PM_{2.5}$ exposures.[11] She noted that the existing 24-hour standard, with its 35 $\mu g/m^3$ level and 98th percentile form, would to provide supplemental protection, particularly for areas with high peak-to-mean ratios possibly associated with strong local or seasonal sources and for areas with $PM_{2.5}$-related effects that may be associated with shorter than daily exposure periods (78 FR 3163, January 15, 2013).

The Administrator recognized that uncertainties remained in the scientific information. She specifically noted uncertainties related to understanding the relative toxicity of the different

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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).

[10] With respect to cancer, mutagenic, and genotoxic effects, the Administrator observed that the $PM_{2.5}$ 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).

[11] 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).

components in the fine particle mixture, the role of $PM_{2.5}$ in the complex ambient mixture, exposure measurement errors in epidemiologic studies, and the nature and magnitude of estimated risks related to relatively low ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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 $\mu g/m^3$ 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 $\mu g/m^3$, combined with a 24-hour standard with a level of 35 $\mu g/m^3$. 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 $\mu g/m^3$ and a 24-hour standard level from 35 to 30 $\mu g/m^3$ (Samet, 2010a). Taken together, the Administrator concluded that the revised annual $PM_{2.5}$ standard, with its level of 12.0 $\mu g/m^3$ 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 $PM_{2.5}$ 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 draft ISA, U.S. EPA, 2018), along with quantitative assessments of $PM_{2.5}$-associated health risks and analyses of $PM_{2.5}$ air quality, 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. The final ISA and PA developed in this review will provide the basis for addressing a series of key policy-relevant questions, meant to inform the Administrator's decisions as to

whether to retain or revise the primary $PM_{2.5}$ standards. In the 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 $PM_{2.5}$ 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 $PM_{2.5}$ standards, and the protection that could be provided by alternatives, we emphasize health outcomes for which the draft ISA determines that the evidence supports either a "causal" or a "likely to be causal" relationship with $PM_{2.5}$ exposures (U.S. EPA, 2018). We consider the $PM_{2.5}$-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 $PM_{2.5}$ 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 draft 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 $PM_{2.5}$ standards is complicated by the fact that such studies evaluate associations between distributions of ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2018, 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 $PM_{2.5}$ 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 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), generally falling around the middle of the distribution (i.e., rather than at the extreme upper or lower ends of the distribution). When uncertainty is quantitatively evaluated, these studies report that confidence intervals around concentration-response functions tend to be narrowest near the overall means of the $PM_{2.5}$ concentrations examined, likely reflecting high data density in this part of the distribution (i.e., reflecting the numerous "typical" daily or annual $PM_{2.5}$ exposures estimated around the overall means). Thus, as described in greater detail in section 3.2.3.2.1, in applying this approach to considering information from epidemiologic studies we focus on the overall mean $PM_{2.5}$

concentrations reported by key studies, and the daily and annual average $PM_{2.5}$ concentrations around such means (i.e., where the bulk of the data supporting reported health effect associations generally fall).

A key uncertainty in using study-reported mean $PM_{2.5}$ concentrations to inform conclusions on the primary $PM_{2.5}$ standards is that they reflect the averages of daily or annual $PM_{2.5}$ exposure estimates in the study population over the years examined by the study, and are not the same as the $PM_{2.5}$ design values used by the EPA to determine whether areas meet the NAAQS (section 3.2.3.2.1).[12] 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 $PM_{2.5}$ design values (i.e., referred to in this draft 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 $PM_{2.5}$ standards.

To the extent the application of these two approaches indicates that health effect associations are based on $PM_{2.5}$ air quality likely to have met the current or alternative standards, those standards are likely to allow the daily or annual average $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standards. Controlled human exposure studies examine short-term $PM_{2.5}$ exposures (i.e., up to several hours) under carefully controlled laboratory conditions. Drawing from the draft ISA, such studies report $PM_{2.5}$-induced changes in markers of cardiovascular function and provide strong support for the biological plausibility of the more serious cardiovascular-related outcomes observed in

[12] 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.

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 $PM_{2.5}$ exposures to concentrations that typically correspond to the upper end of the $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standards.[13]

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 $PM_{2.5}$ 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 98th 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 $PM_{2.5}$ air quality designed to meet an annual standard would likely result not only in lower short- and long-term $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98th percentile form, would result not only in fewer and lower peak 24-hour $PM_{2.5}$ concentrations, but also in lower annual average $PM_{2.5}$ 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 98th percentile form) is more directly tied to short-term peak $PM_{2.5}$ concentrations than to the more typical concentrations that make up the middle portion of the air 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

[13] 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 $PM_{2.5}$ exposures and doses that cause those effects, to human populations.

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 $PM_{2.5}$ concentrations that comprise the middle portion of the air quality distribution, affording protection against the daily and annual $PM_{2.5}$ exposures that strongly support associations with the most serious $PM_{2.5}$-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 $PM_{2.5}$ 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, 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^{th} percentile form, as a means of providing supplemental protection against the short-term exposures to peak $PM_{2.5}$ concentrations that can occur in areas with strong contributions from local or seasonal sources, even when overall mean $PM_{2.5}$ 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.

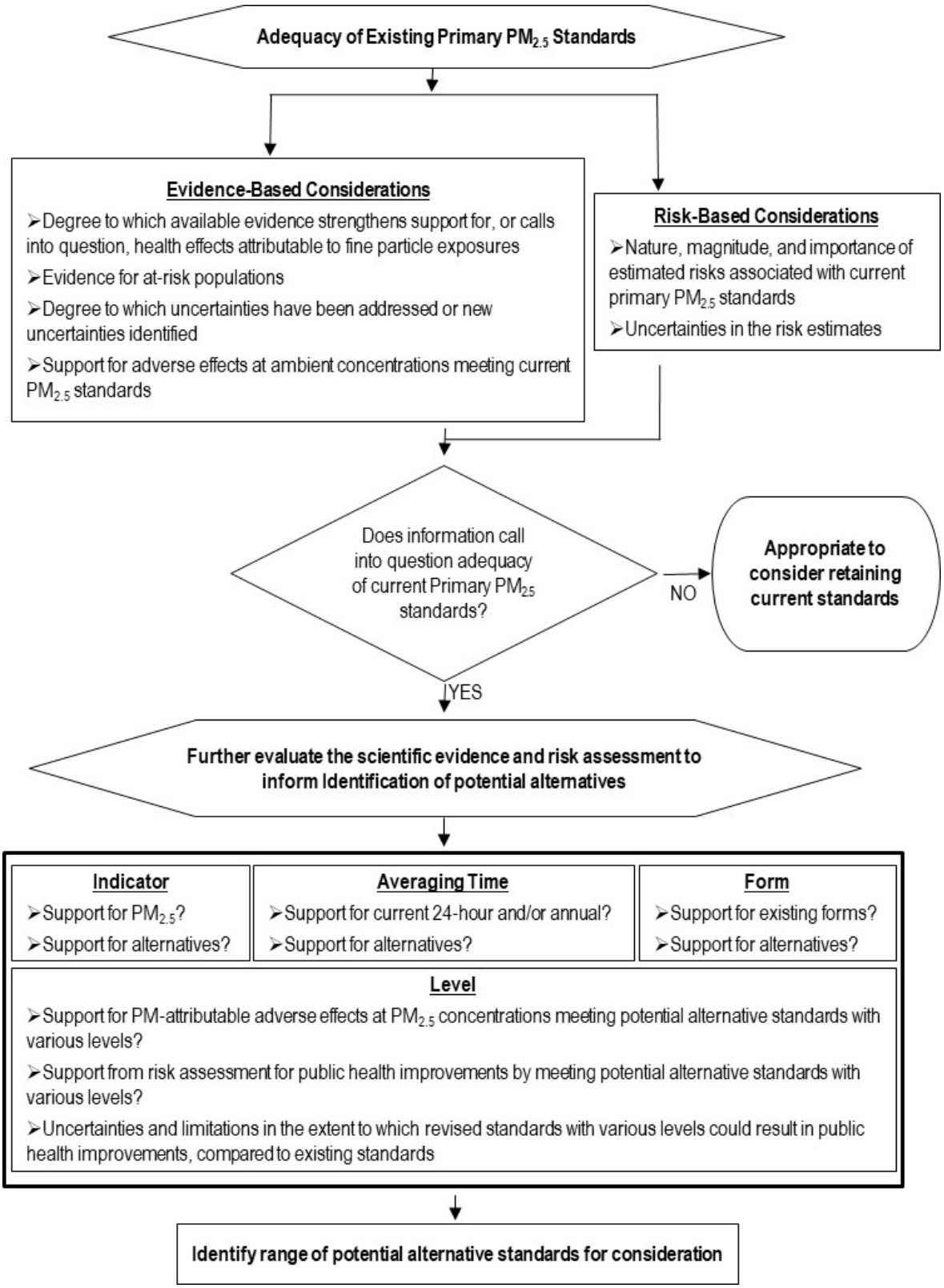


Figure 3-1. Overview of general approach for review of primary $PM_{2.5}$ standards.

In adopting the approach outlined above, we recognize that decisions on the primary $PM_{2.5}$ 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 draft 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 draft ISA (U.S. EPA, 2018) to consider the following policy-relevant question:

- **To what extent does the currently available scientific evidence, as assessed in the draft ISA, support or call into question the public health protection afforded by the current suite of $PM_{2.5}$ standards?**

The draft 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 draft 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 draft 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 draft ISA to demonstrate a "causal" or a "likely to be causal" relationship with PM exposures. The draft ISA defines these causality determinations as follows (U.S. EPA, 2018, p. p-18):

- 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures presented in the draft 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 draft 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 $PM_{2.5}$, the draft ISA also assesses the evidence for the ultrafine fraction of $PM_{2.5}$ (ultrafine particles or UFP), generally considered as particulates with a diameter less than or equal to

0.1 μm[14] (typically based on physical size, thermal diffusivity or electrical mobility) (U.S. EPA, 2018, Preface, p. xi). Table 3-1 lists the health outcomes for which the draft ISA concludes the evidence supports either a causal, a likely to be causal, or a suggestive relationship (adapted from U.S. EPA, 2018, Table 1-5).

[14] 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 μm in diameter in some studies of UFP-related health effects.

Table 3-1. Key causality determinations for $PM_{2.5}$ and UFP exposures. [15]

Health Outcome	Size Fraction	Exposure Duration	2009 PM ISA	2018 draft PM ISA
Mortality	***$PM_{2.5}$***	***Long-term***	***Causal***	***Causal***
		Short-term		
Cardiovascular effects	***$PM_{2.5}$***	***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	***$PM_{2.5}$***	***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	***$PM_{2.5}$***	***Long-term***	***Suggestive of, but not sufficient to infer***	***Likely to be causal***
Nervous System effects	***$PM_{2.5}$***	***Long-term***	***---***	***Likely to be causal***
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
	UFP	**Long-term**	**---**	***Likely to be causal***
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
Metabolic effects	$PM_{2.5}$	Long-term	---	Suggestive of, but not sufficient to infer
		Short-term	---	Suggestive of, but not sufficient to infer
Reproduction and Fertility	$PM_{2.5}$	Long-, Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Pregnancy and Birth Outcomes				

[15] Based on its review, the CASAC questioned several of the causality determinations in the draft ISA. Specifically, the CASAC found that "the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term $PM_{2.5}$ exposure and nervous system effects; between long-term ultrafine particulate (UFP) exposure and nervous system effects; or between long-term $PM_{2.5}$ exposure and cancer" (Cox, 2019). Thus, while the causality determinations for these health outcome categories are listed as "likely to be causal" in Table 3-1, 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.

Table 3-1 lists the health outcomes for which the draft ISA concludes the evidence supports either a causal, a likely to be causal, or a suggestive relationship. For other health outcomes, the draft ISA concludes the evidence is inadequate to infer a causal relationship (U.S. EPA, 2018, Table 1-5).

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 draft ISA's causality determinations reflect the combined evidence for both short- and long-term exposures (U.S. EPA, 2018, Chapter 9).

Sections 3.2.1.1 to 3.2.1.5 summarize the evidence supporting the draft ISA's "causal" and "likely to be causal" determinations for $PM_{2.5}$ (bold, italics in Table 3-1). Section 3.2.1.6 briefly summarizes the evidence supporting the draft 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 $PM_{2.5}$ exposures

In the last review, the 2009 PM ISA reported that the evidence was "sufficient to conclude that the relationship between long-term $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures and mortality. These studies add support for associations with total and non-accidental mortality,[16] as well as with specific causes of death, including cardiovascular disease and respiratory disease (U.S. EPA, 2018, 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 $PM_{2.5}$ exposures and mortality (U.S. EPA, 2018, section

[16] The majority of these studies examined non-accidental mortality outcomes, though some Medicare studies lack cause-specific death information and, therefore, examine total mortality.

11.2.2.1; Figures 11-17 and 11-18). Adding to recent evaluations of the ACS and Six Cities cohorts, studies conducted in other cohorts also demonstrate consistent, positive associations between long-term $PM_{2.5}$ 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, 2018, sections 11.2.2.2, 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).

A recent series of "accountability" studies has additionally tested the hypothesis that reductions in ambient $PM_{2.5}$ concentrations would be associated with increased life expectancy or a decreased mortality rate (U.S. EPA, 2018, section 11.2.2.6). 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 $\mu g/m^3$ decrease in long-term $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. In this follow-up study, a decrease in long-term $PM_{2.5}$ 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 $\mu g/m^3$ decrease in long-term $PM_{2.5}$ 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 $PM_{2.5}$ are associated with improvements in longevity (U.S. EPA, 2018, section 11.2.2.6).

The draft ISA specifically evaluates the degree to which recent studies that examine the relationship between long-term $PM_{2.5}$ 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 draft ISA concludes that positive associations between long-term $PM_{2.5}$ exposures and mortality are robust across recent analyses using various approaches to estimate $PM_{2.5}$ exposures (e.g., based on monitors, modeling, satellites, or hybrid methods that combine information from multiple sources) (U.S. EPA, 2018, section 11.2.5.1), across statistical models (U.S. EPA, 2018, section 11.2.5.2), across diverse geographic regions and populations, and across a range of temporal periods including the periods of declining PM concentrations (U.S. EPA, 2018, section 11.2.5.3). Recent evidence further demonstrates that (1) associations with mortality remain robust in analyses of potential confounding by copollutants (U.S. EPA, 2018, section 11.2.3); (2) associations persist in analyses restricted to long-term exposures below 12 $\mu g/m^3$ (Di et al., 2017b) or 10 $\mu g/m^3$ (Shi et al., 2016) (i.e., indicating that risks are not disproportionately driven by the upper portions of the air quality distribution); and (3) concentration-response relationships remain linear over the distribution of ambient $PM_{2.5}$

concentrations with no evidence of a threshold, though uncertainty increases near the upper and lower ends of the $PM_{2.5}$ air quality distribution due to limited exposure and outcome data (U.S. EPA, 2018, section 11.1.10).

The biological plausibility of $PM_{2.5}$-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 draft ISA outlines the available evidence for plausible pathways by which inhalation exposure to $PM_{2.5}$ 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, 2018, section 6.2.1), and to metabolic disease and diabetes (U.S. EPA, 2018, section 7.3.1). The draft ISA notes "more limited evidence from respiratory morbidity" (U.S. EPA, 2018, p. 11-98) such as exacerbation of COPD (U.S. EPA, 2018, section 5.2.1) to support the biological plausibility of mortality due to long-term $PM_{2.5}$ exposures (U.S. EPA, 2018, 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures. The draft ISA concludes that, "collectively, this body of evidence is sufficient to conclude that a causal relationship exists between long-term $PM_{2.5}$ exposure and total mortality" (U.S. EPA, 2018, section 11.2.7; p. 11-99).

Short-term $PM_{2.5}$ exposures

The 2009 PM ISA concluded that "a causal relationship exists between short-term exposure to $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$-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 $PM_{2.5}$ exposure and cardiovascular mortality.

Recent multicity studies evaluated since the 2009 ISA continue to provide evidence of primarily positive associations between daily $PM_{2.5}$ exposures and mortality, with percent increases in total mortality ranging from 0.19% (Lippmann et al., 2013) to 2.80% (Kloog et al., 2013)[17] at lags of 0 to 1 days in single-pollutant models. These results are further supported by initial studies employing causal inference and quasi-experimental statistical approaches (U.S. EPA, 2018, section 11.1.2.1). For example, 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. Whereas most studies rely on assigning exposures using data from ambient monitors, some recent studies have also employed hybrid modeling approaches that use additional $PM_{2.5}$ 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). Recent studies expand the assessment of potential copollutant confounding. These studies provide additional evidence indicating that associations between short-term $PM_{2.5}$ exposures and mortality remain positive and relatively unchanged in copollutant models with both gaseous pollutants and $PM_{10-2.5}$ (U.S. EPA, 2018, Section 11.1.4). Additionally, the low ($r < 0.4$) to moderate correlations ($r = 0.4$-0.7) between $PM_{2.5}$ and gaseous pollutants and $PM_{10–2.5}$ increase the confidence in $PM_{2.5}$ having an independent effect on mortality (U.S. EPA, 2018, section 11.1.4).

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, 2018, 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 $PM_{10-2.5}$. This evidence further supports the copollutant analyses conducted for total mortality. The strong evidence for ischemic

[17] As detailed in the Preface to the draft ISA, risk estimates are for a 10 $\mu g/m^3$ increase in 24-hour avg $PM_{2.5}$ concentrations, unless otherwise noted (U.S. EPA, 2018).

events and heart failure, as detailed in the assessment of cardiovascular morbidity (U.S. EPA, 2018, Chapter 6), provides biological plausibility for $PM_{2.5}$-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 $PM_{2.5}$-related respiratory mortality (U.S. EPA, 2018, Chapter 5).

In addition to examining potential copollutant confounding, a number of studies also examine the influence of model specification, such as temporal trends and weather covariates, on the $PM_{2.5}$-mortality association. 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, 2018, section 11.1.5.1). Seasonal analyses continue to provide evidence that associations are larger in magnitude during warmer months, but it remains unclear if copollutants confound the associations observed. In addition to seasonal analyses, some studies also examine whether temperature modifies the $PM_{2.5}$-mortality relationship. Initial evidence indicates that the $PM_{2.5}$-mortality association may be modified by temperature, though results remain unclear overall (U.S. EPA, 2018, section 11.1.6.2).

In the 2009 ISA, one of the main uncertainties identified was the regional and city-to-city heterogeneity in $PM_{2.5}$-mortality associations observed in multicity studies. Recent studies examine both city-specific as well as regional characteristics to identify the underlying contextual factors that contribute to this heterogeneity (U.S. EPA, 2018, section 11.1.6.3). Analyses focusing on effect modification of the $PM_{2.5}$-mortality relationship by $PM_{2.5}$ components, regional patterns in $PM_{2.5}$ components and city-specific differences in composition and sources indicate some differences in the $PM_{2.5}$ composition and sources across cities and regions, but these differences do not fully explain the heterogeneity observed. Additional studies find that factors related to 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, 2018, section 11.1.6.3). Collectively, recent studies indicate that the heterogeneity in $PM_{2.5}$-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 (U.S. EPA, 2018, section 11.1.12).

A number of recent studies conducted systematic evaluations of the lag structure of associations for the $PM_{2.5}$-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 $PM_{2.5}$ exposures on mortality (U.S. EPA, 2018, 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 exposures below 25 $\mu g/m^3$ (Di et al., 2017a) or below 30 $\mu g/m^3$ (Shi et al., 2016), indicating that risks associated with short-term $PM_{2.5}$ 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 $PM_{2.5}$ (U.S. EPA, 2018, 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 $PM_{2.5}$-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, 2018). However, given the limited data available at the lower end of the distribution of ambient $PM_{2.5}$ concentrations, the shape of the concentration-response curve remains uncertain at these low concentrations and, to date, studies have not conducted extensive analyses exploring alternatives to linearity when examining the shape of the $PM_{2.5}$-mortality concentration-response relationship.

Overall, recent epidemiologic studies build upon and extend the conclusions of the 2009 ISA for the relationship between short-term $PM_{2.5}$ exposures and total mortality. Supporting evidence for $PM_{2.5}$-related cardiovascular morbidity, and more limited evidence from respiratory morbidity, provides biological plausibility for mortality due to short-term $PM_{2.5}$ 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 draft ISA states that, collectively, "this body of evidence is sufficient to conclude that a causal relationship exists between short-term $PM_{2.5}$ exposure and total mortality" (U.S. EPA, 2018, pp. 11-56 to 11-57).

3.2.1.2 Cardiovascular Effects

Long-term $PM_{2.5}$ exposures

The scientific evidence reviewed in the 2009 PM ISA was "sufficient to infer a causal relationship between long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposure and cardiovascular mortality (Pope et al., 2004, Krewski et al., 2009, Miller et al., 2007, Laden et al., 2006). Studies of long-term $PM_{2.5}$ 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 $PM_{2.5}$ 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, recent studies continue to provide strong support for a causal relationship between long-term exposure to $PM_{2.5}$ 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 $PM_{2.5}$ exposure and cardiovascular mortality (U.S. EPA, 2018, Figure 6-19) in evaluations conducted at varying spatial scales and employing a variety of exposure assessment and statistical methods (U.S. EPA, 2018, section 6.3.10). Positive associations between long-term $PM_{2.5}$ exposures and cardiovascular mortality are generally robust in copollutant models adjusted for ozone, NO_2, $PM_{10-2.5}$, or SO_2. 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 $PM_{2.5}$ exposures and do not identify a threshold below which effects do not occur (U.S. EPA, 2018, section 6.2.16; Table 6-52).[18]

Associations with cardiovascular morbidity are coherent with mortality findings, helping to support the biological plausibility of mortality findings by providing evidence of the progression of cardiovascular disease linked to $PM_{2.5}$ exposures. Positive associations with cardiovascular morbidity (e.g., coronary heart disease, stroke) and atherosclerosis progression are observed in several epidemiologic studies (U.S. EPA, 2018, 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 $PM_{2.5}$ collected from multiple locations across the U.S. (U.S. EPA, 2018, section 6.2.4.2). A small number of epidemiologic studies also report positive associations between long-term $PM_{2.5}$ exposure and heart failure, changes in blood pressure, and hypertension (U.S. EPA, 2018, 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 $PM_{2.5}$ exposure (U.S. EPA, 2018, section 6.2.5.2). Similarly, a limited number of animal toxicological studies demonstrating a relationship between long-term exposure to $PM_{2.5}$ and consistent increases in blood pressure in rats and mice are coherent with epidemiologic studies reporting positive associations between long-term exposure to $PM_{2.5}$ and hypertension.

[18] 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.

Longitudinal epidemiologic analyses also report positive associations with markers of systemic inflammation (U.S. EPA, 2018, section 6.2.12), coagulation (U.S. EPA, 2018, section 6.2.13), and endothelial dysfunction (U.S. EPA, 2018, section 6.2.14). These results are coherent with animal toxicological studies generally reporting increased markers of systemic inflammation, oxidative stress, and endothelial dysfunction (U.S. EPA, 2018, section 6.2.12.2 and 6.2.14).

In summary, the draft ISA concludes that there is consistent evidence from multiple epidemiologic studies illustrating that long-term exposure to $PM_{2.5}$ 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 $PM_{2.5}$ associations. Additional evidence of the independent effect of $PM_{2.5}$ on the cardiovascular system is provided by experimental studies in animals, which demonstrate biologically plausible pathways by which long-term inhalation exposure to $PM_{2.5}$ could potentially result in outcomes such as CHD, stroke, CHF and cardiovascular mortality. The combination of epidemiologic and experimental evidence results in the draft ISA conclusion that "a causal relationship exists between long-term exposure to $PM_{2.5}$ and cardiovascular effects" (U.S. EPA, 2018, section 6.2.18).

Short-term $PM_{2.5}$ exposures

The 2009 PM ISA concluded that "a causal relationship exists between short-term exposure to $PM_{2.5}$ 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 $PM_{2.5}$ 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.[19] Key uncertainties from the last review resulted from inconsistent results across disciplines with respect to the relationship between short-term exposure to $PM_{2.5}$ 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

[19] 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.

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 $PM_{2.5}$ exposure and cardiovascular effects. This includes generally positive associations observed in numerous 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 $PM_{2.5}$ exposures and hospital admissions for HF (U.S. EPA, 2018, section 6.1.3.1). Additional multicity studies conducted in the northeast U.S. report positive associations between short-term $PM_{2.5}$ exposures and emergency department visits or hospital admissions for IHD (U.S. EPA, 2018, section 6.1.2.1) while studies conducted in the U.S. and Canada reported positive associations between short-term $PM_{2.5}$ exposures and emergency department visits for HF. Epidemiologic studies conducted in single cities contribute some support, though associations reported in these studies are less consistent than in multicity studies (U.S. EPA, 2018, sections 6.1.2 and 6.1.3). When considered as a whole, the recent body of IHD and HF epidemiologic evidence further supports the evidence from previous ISAs reporting mainly positive associations between short-term $PM_{2.5}$ 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 $PM_{2.5}$ exposure could plausibly result in IHD or HF through pathways that include endothelial dysfunction, arterial thrombosis, and arrhythmia (U.S. EPA, 2018, 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 $PM_{2.5}$ exposure on measures of blood flow (U.S. EPA, 2018, 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, 2018, 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, 2018, section 6.1.4.3), changes in heart rate variability (HRV) (U.S. EPA, 2018, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2018, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2018, 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposure and heart failure (U.S. EPA, 2018, 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, 2018, section 6.1.13.3). Studies in animals also provide evidence for changes in a number of other cardiovascular endpoints following short-term $PM_{2.5}$ exposure. Although not entirely consistent, these studies provide some evidence of conduction abnormalities and arrhythmia (U.S. EPA, 2018, section 6.1.4.4), changes in HRV (U.S. EPA, 2018, section 6.1.10.3), changes in blood pressure (U.S. EPA, 2018, section 6.1.6.4), and evidence for systemic inflammation and oxidative stress (U.S. EPA, 2018, 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations and IHD and HF, and ultimately cardiovascular mortality. The draft ISA concludes that, overall, "there continues to be sufficient evidence to conclude that a causal relationship exists between short-term $PM_{2.5}$ exposure and cardiovascular effects" (U.S. EPA, 2018, p. 6-134).

3.2.1.3 Respiratory Effects

Long-term $PM_{2.5}$ exposures

The 2009 PM ISA concluded that "a causal relationship is likely to exist between long-term $PM_{2.5}$ exposure and respiratory effects" (U.S. EPA, 2009). This conclusion was based

mainly on epidemiologic evidence demonstrating associations between long-term $PM_{2.5}$ 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 $PM_{2.5}$ CAPs, which found impaired lung development. Epidemiologic evidence for associations between long-term $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2018, section 5.2.13). This relationship is further supported by a recent accountability study that reports an association between declining $PM_{2.5}$ concentrations and improvements in lung function growth in children (U.S. EPA, 2018, section 5.2.11). Epidemiologic studies also examine asthma development in children (U.S. EPA, 2018, 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, 2018, 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, 2018, section 5.2.13). Other epidemiologic studies report a $PM_{2.5}$-related acceleration of lung function decline in adults, while improvement in lung function was observed with declining $PM_{2.5}$ concentrations (U.S. EPA, 2018, section 5.2.11). A recent longitudinal study found declining $PM_{2.5}$ 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 $PM_{2.5}$ exposure (U.S. EPA, 2018, 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 draft 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, 2018, 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 $PM_{2.5}$ 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 draft ISA concludes that "the collective evidence is sufficient to conclude that a causal relationship is likely to exist between long-term $PM_{2.5}$ exposure and respiratory effects" **(**U.S. EPA, 2018, section 5.2.13).

Short-term $PM_{2.5}$ exposures

The 2009 PM ISA (U.S. EPA, 2009) concluded that a "causal relationship is likely to exist" between short-term $PM_{2.5}$ 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 $PM_{2.5}$-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 $PM_{2.5}$ associations with respiratory effects were robust to inclusion of CO or SO_2 in the model, but often were attenuated (though still positive) with inclusion of O_3 or NO_2. In addition to the copollutant models, evidence supporting an independent effect of $PM_{2.5}$ exposure on the respiratory system was provided by animal toxicological studies of $PM_{2.5}$ 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, $PM_{2.5}$ 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 $PM_{2.5}$ exposures.

Recent epidemiologic studies provide evidence for a relationship between short-term $PM_{2.5}$ exposure and several respiratory-related endpoints, including asthma exacerbation (U.S. EPA, 2018, section 5.1.2.1), COPD exacerbation (U.S. EPA, 2018, section 5.1.4.1), and combined respiratory-related diseases (U.S. EPA, 2018, section 5.1.6), particularly from studies examining emergency department visits and hospital admissions. The generally positive associations between short-term $PM_{2.5}$ 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, 2018, 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 $PM_{2.5}$ exposure and respiratory mortality provide evidence of consistent positive associations, demonstrating a continuum of effects (U.S. EPA, 2018, 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 $PM_{2.5}$ associations with asthma exacerbation, combined respiratory-related diseases, and respiratory mortality remain relatively unchanged in copollutant models with gaseous pollutants (i.e., O_3, NO_2, SO_2, with more limited evidence for CO) and other particle sizes (i.e., $PM_{10–2.5}$) (U.S. EPA, 2018, section 5.1.10.1).

The uncertainty related to whether there is an independent effect of $PM_{2.5}$ on respiratory health is also partially addressed by findings from animal toxicological studies. Specifically, short-term exposure to $PM_{2.5}$ 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, 2018, sections 5.1.2.4.3 and 5.1.4.4.2). 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 draft ISA concludes that "[t]he strongest evidence of an effect of short-term $PM_{2.5}$ 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 $PM_{2.5}$ effects" (U.S. EPA, 2018, p. 5-155). When taken together, the draft ISA concludes that this evidence "is

sufficient to conclude that a causal relationship is likely to exist between short-term $PM_{2.5}$ exposure and respiratory effects" (U.S. EPA, 2018, p. 5-155).

3.2.1.4 Cancer – Long-term $PM_{2.5}$ Exposures

The 2009 ISA concluded that the overall body of evidence was "suggestive of a causal relationship between relevant $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2018, section 10.2.5). Reanalyses of the ACS cohort using different years of $PM_{2.5}$ data and follow-up, along with various exposure assignment approaches, provide consistent evidence of positive associations between long-term $PM_{2.5}$ exposure and lung cancer mortality (U.S. EPA, 2018, 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, 2018, 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 $PM_{2.5}$ exposures (U.S. EPA, 2018, 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, 2018, section 10.2.5.1.2). Associations between long-term $PM_{2.5}$ 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, 2018, section 10.2.5.1.2).

The draft 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 $PM_{2.5}$ exposure and lung cancer mortality or incidence. The small number of such studies have generally focused on O_3 and report that $PM_{2.5}$ associations remain relatively unchanged in copollutant models (U.S. EPA, 2018, 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, 2018, 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, 2018, section 10.2.5.1.4).

In support of the biological plausibility of an independent effect of $PM_{2.5}$ on cancer, the draft ISA notes evidence from recent experimental and epidemiologic studies demonstrating that $PM_{2.5}$ exposure can lead to a range of effects indicative of mutagenicity, genotoxicity, and carcinogenicity, as well as epigenetic effects (U.S. EPA, 2018, section 10.2.7). For example, both in vitro and in vivo toxicological studies have shown that $PM_{2.5}$ exposure can result in DNA damage (U.S. EPA, 2018, 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, 2018, section 10.2.3.3), 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 $PM_{2.5}$ exposure contributing to genomic instability (U.S. EPA, 2018, section 10.2.3).

Epidemiologic evidence for associations between $PM_{2.5}$ and lung cancer mortality and incidence, together with evidence supporting the biological plausibility of such associations, contributes to the draft ISA's conclusion that the evidence "is sufficient to conclude that a causal

relationship is likely to exist between long-term $PM_{2.5}$ exposure and cancer" (U.S. EPA, 2018, section 10.2.7).[20]

3.2.1.5 Nervous System Effects

Long-term $PM_{2.5}$ exposures

Reflecting the very limited evidence available in the last review, the 2009 ISA did not make a causality determination for long-term $PM_{2.5}$ exposures and nervous system effects (U.S. EPA, 2009). Since the last review, this body of evidence has grown substantially (U.S. EPA, 2018). Recent animal toxicology studies report that long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposure is associated with reduced cognitive function (U.S. EPA, 2018, 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 $PM_{2.5}$ 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, 2018, 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, 2018, section 8.2.4), though epidemiologic studies provide only limited support for associations with Parkinson's disease (U.S. EPA, 2018, 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 $PM_{2.5}$ 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 $PM_{2.5}$ during the prenatal period and autism spectrum disorder (ASD) are observed in multiple epidemiologic studies (U.S. EPA, 2018, section 8.2.7.2), while studies of cognitive function provide little support for an association (U.S. EPA, 2018, 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

[20] As noted above, the CASAC found that "the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between… long-term $PM_{2.5}$ exposure and cancer" (Cox, 2019). The final ISA will reflect the EPA's consideration of this and other CASAC advice on the draft ISA.

corpus collosum and hippocampus, as well as ventriculomegaly (i.e., enlarged lateral ventricles) in young mice following prenatal exposure to $PM_{2.5}$ CAPs.

Taken together, the draft ISA concludes that recent studies indicate long-term $PM_{2.5}$ 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, 2018, 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 $PM_{2.5}$ 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 draft ISA's conclusion that "the collective evidence is sufficient to conclude that a causal relationship is likely to exist between long-term $PM_{2.5}$ exposure and nervous system effects" (U.S. EPA, 2018, section 8.2.9).[21]

Long-term UFP exposures

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, 2018, 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, 2018, 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, 2018, section 8.6.6.2). Persistent ventriculomegaly was observed in male, but not female, mice exposed postnatally to UFP (U.S. EPA, 2018, 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

[21] As noted above, the CASAC found that "the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term $PM_{2.5}$ exposure and nervous system effects" (Cox, 2019). The final ISA will reflect the EPA's consideration of this and other CASAC advice on the draft ISA.

exposures at the population level. Based primarily on the animal toxicological evidence of neurotoxicity and altered neurodevelopment, the draft ISA concludes that the evidence is "sufficient to conclude that a causal relationship is likely to exist between long-term UFP exposure and nervous system effects" (U.S. EPA, 2018, section 8.6.7).[22]

3.2.1.6 Other Effects

Compared to the health outcomes discussed above, the draft ISA concludes that there is greater uncertainty in the evidence linking $PM_{2.5}$ 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) $PM_{2.5}$ and UFP exposures.

3.2.1.6.1 Long-term Exposures

As indicated in Table 3-1 above, the draft ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship" between long-term $PM_{2.5}$ 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, 2018, Preface, p. xvii). The basis for these causality determinations is summarized briefly below.

$PM_{2.5}$ – Metabolic effects

There were no causality determinations for long-term $PM_{2.5}$ exposure and metabolic effects in the 2009 ISA (U.S. EPA, 2009). However, the literature pertaining to the effect of long-term exposure to $PM_{2.5}$ and metabolic effects has expanded substantially since the 2009 ISA, and consists of both epidemiologic and experimental evidence (U.S. EPA, 2018, section 7.2). Epidemiologic studies report positive associations between long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposure and metabolic effects from experimental studies demonstrating increased blood glucose, insulin resistance, and inflammation and visceral

[22] As noted above, the CASAC found that "the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between… long-term ultrafine particulate (UFP) exposure and nervous system effects" (Cox, 2019). The final ISA will reflect the EPA's consideration of this and other CASAC advice on the draft ISA.

adiposity but the experimental evidence was not entirely consistent. Based on this evidence, the draft ISA concludes that, "[o]verall, the collective evidence is suggestive of, but is not sufficient to infer, a causal relationship between long-term $PM_{2.5}$ exposure and metabolic effects" (U.S. EPA, 2018 p. 7-54).

$PM_{2.5}$ – Reproductive and developmental effects

The 2009 ISA determined that the evidence was "suggestive of a causal relationship" for the association between long-term $PM_{2.5}$ 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, 2018, sections 9.1.1, 9.1.2, 9.1.5).

Effects of $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures with decreased odds of becoming pregnant. Studies in rodents have shown ovulation and estrus are affected by $PM_{2.5}$ 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 $PM_{2.5}$ exposure. The draft ISA concludes that, "[c]ollectively, the evidence is suggestive of, but not sufficient to infer, a causal relationship between $PM_{2.5}$ exposure and male and female reproduction and fertility" (U.S. EPA, 2018, p. 9-42).

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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposure and various pregnancy-related pathologies, including gestational hypertension, pre-eclampsia and gestational diabetes is inconsistent. Biological plausibility for effects of $PM_{2.5}$ exposure and various pregnancy and birth outcomes is provided by studies showing that $PM_{2.5}$ 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 draft ISA concludes that the available evidence, including uncertainties that evidence, is "suggestive of, but not sufficient to infer, a causal relationship between exposure to $PM_{2.5}$ and pregnancy and birth outcomes" (U.S. EPA, 2018, p. 9-44).

3.2.1.6.2 Short-term Exposures

As indicated in Table 3-1 above, the draft ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship" between short-term $PM_{2.5}$ exposures and metabolic effects and nervous system effects. Additionally, the draft 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, 2018, Preface, p. xvii). The basis for these causality determinations is summarized briefly below.

$PM_{2.5}$ – Metabolic effects

There were no studies of the effect of short-term $PM_{2.5}$ exposure and metabolic effects reviewed in the 2009 ISA (U.S. EPA, 2009). New evidence for a relationship between short-term $PM_{2.5}$ 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, 2018, section 7.1). The draft ISA concludes that, overall, the collective evidence "is suggestive of, but not sufficient to infer, a causal relationship between short-term $PM_{2.5}$ exposure and metabolic effects" (U.S. EPA, 2018, p. 7-15).

$PM_{2.5}$ – Nervous system effects

The evidence reviewed in the 2009 ISA was characterized as "inadequate to infer" a causal relationship between short-term $PM_{2.5}$ 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 $PM_{2.5}$ can affect the nervous system (U.S. EPA, 2018, 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 $PM_{2.5}$ exposure with hospital admissions for Parkinson's disease was observed indicating the potential for exacerbation of neurological diseases. The draft ISA concludes that, overall, the collective evidence "is suggestive of, but not sufficient to infer, a causal relationship between short-term exposure to $PM_{2.5}$ and nervous system effects" (U.S. EPA, 2018, p. 8-20).

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, 2018, section 6.5.9.1) and markers of coagulation (U.S. EPA, 2018, 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, 2018, 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, 2018, section 1.4.3). When considered as a whole, the draft ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term exposure to UFPs and cardiovascular effects" (U.S. EPA, 2018, p. 6-298).

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, 2018, section 5.5). For example, associations persist in one epidemiologic study with adjustment for NO_2, 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, 2018, section 5.5). The draft 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, 2018, p. 5-300).

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, 2018, section 8.5). The draft 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, 2018, p. 8-93).

3.2.1.7 Summary

Based on the evidence assessed in the draft ISA (U.S. EPA, 2018), 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 $PM_{2.5}$ exposures.

Studies conducted since the 2009 ISA have broadened our understanding of the health effects that can result from long-term $PM_{2.5}$ exposures and have reduced key uncertainties identified in the last review. Recent epidemiologic studies consistently report positive associations between long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposures, statistical models, and long-term exposure windows (i.e., the exposure period that is associated with the health outcome). Recent evidence also includes "accountability" studies that demonstrate improvements in health outcomes, including increasing life expectancy, decreasing mortality, or decreasing respiratory effects, as a result of declines in ambient $PM_{2.5}$ concentrations over time. 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures could lead to effects on the cardiovascular and respiratory systems, effects on the nervous system, and to lung cancer.[23] [24] In addition to providing insight into potential mechanisms, experimental studies also demonstrate direct effects of $PM_{2.5}$ 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, 2018) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposures. Recent epidemiologic studies build upon and further

[23] 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.

[24] Animal studies also provide stronger support in this review for effects following exposures to UFP (section 3.2.1.5), 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.

reaffirm those studies evaluated in the 2009 PM ISA, providing evidence of positive associations across a range of effects. The independence of the $PM_{2.5}$ effects reported in such studies is further supported by the results of copollutant analyses indicating that associations with short-term $PM_{2.5}$ remain robust. Some recent studies report that associations persist in analyses that exclude short-term $PM_{2.5}$ 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 $PM_{2.5}$-associated mortality is supported by associations with cardiovascular and respiratory morbidity. Direct evidence for $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures and respiratory effects, particularly asthma and COPD exacerbations, and while animal toxicological studies provide biological plausibility for these findings, some uncertainty remains with respect to the independence of $PM_{2.5}$ effects. Thus, when taken together, the evidence available in this review (U.S. EPA, 2018) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of short-term $PM_{2.5}$ 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 draft ISA cites extensive evidence indicating that "both the general population as well as specific populations and lifestages are at risk for $PM_{2.5}$-related health effects" (U.S. EPA, 2018, p. 12-1). For example, in support of its "causal" and "likely to be causal" determinations, the draft ISA cites substantial evidence for:

- PM-related mortality and cardiovascular effects in older adults (U.S. EPA, 2018, sections 11.1, 11.2, 6.1, and 6.2);
- PM-related cardiovascular effects in people with pre-existing cardiovascular disease (U.S. EPA, 2018, section 6.1);
- PM-related respiratory effects in people with pre-existing respiratory disease, particularly asthma (U.S. EPA, 2018, section 5.1); and
- PM-related impairments in lung function growth and asthma development in children (U.S. EPA, 2018, sections 5.1 and 5.2; 12.5.1.1).

The draft 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 $PM_{2.5}$ exposures and in $PM_{2.5}$-related health risk. Such analyses indicate that minority populations such as Hispanic and non-Hispanic black populations have higher $PM_{2.5}$ exposures than non-Hispanic white populations, thus contributing to adverse health risk in non-white populations (U.S. EPA, 2018, 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 $PM_{2.5}$-related adverse health effects (U.S. EPA, 2018, Chapter 12).

Thus, the groups at risk of $PM_{2.5}$-related health effects represent a substantial portion of the total U.S. population. In evaluating the primary $PM_{2.5}$ standards, an important consideration is the potential $PM_{2.5}$-related public health impacts in these populations.

3.2.3 $PM_{2.5}$ Concentrations in Key Studies Reporting Health Effects

To inform conclusions on the adequacy of the public health protection provided by the current primary $PM_{2.5}$ standards, this section evaluates the $PM_{2.5}$ exposures and ambient concentrations (i.e., used as surrogates for exposures in epidemiologic studies) in studies reporting $PM_{2.5}$-related health effects. We specifically consider the following overarching questions:

- **What are the short- or long-term $PM_{2.5}$ 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 $PM_{2.5}$ standards?**

In addressing these questions, we emphasize health outcomes for which the draft 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 $PM_{2.5}$ exposures and cancer and nervous system effects associated with long-term $PM_{2.5}$ exposures. While the causality determinations in the draft ISA are informed by studies evaluating a wide range of $PM_{2.5}$ 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 $PM_{2.5}$ 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 draft ISA, the evidence for a particular $PM_{2.5}$-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, 2018, Preamble, p. 11).

In this section, we consider the $PM_{2.5}$ exposure concentrations shown to cause effects in controlled human exposure studies and in animal toxicology studies. We particularly consider the consistency of specific $PM_{2.5}$-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 $PM_{2.5}$ exposures likely to occur in areas meeting the current primary standards?**

Controlled Human Exposure Studies

As discussed in detail in the draft ISA (U.S. EPA, 2018, section 6.1), controlled human exposure studies have reported that $PM_{2.5}$ exposures lasting from less than one hour up to five hours can impact cardiovascular function.[25] The most consistent evidence from these studies is for impaired vascular function (U.S. EPA, 2018, section 6.1.13.2). In addition, although less consistent, the draft ISA notes that studies examining $PM_{2.5}$ exposures also provide evidence for

[25] In contrast, controlled human exposure studies provide little evidence for respiratory effects following short-term $PM_{2.5}$ exposures (U.S. EPA, 2018, section 5.1, Table 5-18). Therefore, this section focuses on cardiovascular effects evaluated in controlled human exposure studies of $PM_{2.5}$ exposure.

increased blood pressure (U.S. EPA, 2018, section 6.1.6.3), conduction abnormalities/arrhythmia (U.S. EPA, 2018, section 6.1.4.3), changes in heart rate variability (U.S. EPA, 2018, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2018, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2018, section 6.1.11.2). The draft ISA concludes that, when taken as a whole, controlled human exposure studies demonstrate that short-term exposure to $PM_{2.5}$ may impact cardiovascular function in ways that could lead to more serious outcomes (U.S. EPA, 2018, section 6.1.16). Thus, such studies can provide insight into the potential for specific $PM_{2.5}$ exposures to cause physiological changes that could increase the risk of more serious effects.

Table 3-2 below summarizes information from the draft ISA[26] on available controlled human exposure studies that evaluate effects on markers of cardiovascular function following exposures to $PM_{2.5}$, either as concentrated ambient particles (CAP) or in unfiltered versus filtered exhaust.[27]

[26] Table 3-2 includes the controlled human exposure studies, and the endpoints from each study, that are discussed in the draft ISA. In the final PA, Table 3-2 will be updated, as needed, to reflect changes made in the final ISA regarding the studies and/or endpoints that are discussed (e.g., in response to CASAC comments or public input).

[27] Table 3-2 identifies controlled human exposure studies included in the draft ISA that examine the potential for $PM_{2.5}$ exposures to alter markers of cardiovascular function. Studies that focus on specific components of $PM_{2.5}$ (e.g., endotoxin), or studies that evaluated $PM_{2.5}$ 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 $PM_{2.5}$ controlled human exposure studies.

Study	Population	Exposure Details (average concentration; duration)	Results
Bräuner et al., 2008	Healthy adults	10.5 µg/m^3 $PM_{2.5}$ (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^3 (unfiltered) vs 3.0 µg/m^3 (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^3 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^3 CAP; 2 h	No significant changes in heart rate variability
Devlin et al., 2003	Healthy older adults	99 µg/m^3 CAP[28]; 2 h	Decreased heart rate variability
Hazucha et al., 2013	Adult current and former smokers	109 µg/m^3 CAP; 2 h	No significant changes in markers of inflammation or coagulation
Ghio et al., 2000	Healthy young adults	120 µg/m^3 CAP; 2 h	Increased fibrinogen (coagulation)
Ghio et al., 2003	Healthy young adults	120 µg/m^3 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^3 CAP (higher exposure); 2 h	Increased blood inflammatory markers
Brook et al., 2009	Healthy adults	149 µg/m^3 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^3 CAP; 2 h	Decreased anti-oxidant/anti-inflammatory capacity when baseline capacity was low

[28] The published study reports an average CAP concentration of 41 µg/m^3, but communication with the study authors revealed an error in that reported concentration (Jenkins, 2016).

Sivagangabalan et al., 2011	Healthy adults	150 µg/m^3 CAP; 2 h	Increase in indicator of possible arrhythmia; no significant effect on heart rate
Kusha et al., 2012	Healthy adults	154 µg/m^3 CAP; 2 h	No significant effect on indicator of possible arrhythmia
Gong et al., 2003	Adults with and without asthma	174 µg/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 µg/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 µg/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 µg/m^3 CAP; 130 min	Increased blood pressure
Behbod et al., 2013	Healthy adults	~250 µg/m^3 CAP; 130 min	Increase in markers of inflammation
Tong et al., 2015	Healthy older adults	253 µg/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 µg/m^3 (unfiltered) vs 7.2 µg/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 µg/m^3 (unfiltered) vs 25 µg/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 $PM_{2.5}$ exposure concentrations at or above about 100 µg/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 $PM_{2.5}$ concentrations at and above about 120 µg/m^3, with less consistent evidence for effects following exposures to lower concentrations. Impaired vascular function, the effect identified in the draft

ISA as the most consistent across studies (U.S. EPA, 2018, section 6.1.13.2), is shown following 2-hour exposures to $PM_{2.5}$ concentrations at and above 149 $\mu g/m^3$. Mixed results are reported in the three studies that evaluate longer exposure durations (i.e., longer than 2 hours) and lower $PM_{2.5}$ concentrations, with significant effects on some outcomes reported following 5-hour exposures to 24 $\mu g/m^3$ 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 $\mu g/m^3$ in Bräuner et al. (2008).

To provide some insight into what these studies may indicate regarding the primary $PM_{2.5}$ standards, we consider the degree to which 2-hour ambient $PM_{2.5}$ 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 $PM_{2.5}$ concentrations from all FEM $PM_{2.5}$ monitors in the U.S. for 2015-2017. At sites meeting the current primary $PM_{2.5}$ standards, most 2-hour concentrations are below 11 $\mu g/m^3$, and almost never exceed 32 $\mu g/m^3$. The extreme upper end of the distribution of 2-hour $PM_{2.5}$ 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^{th} percentile of 2-hour concentrations is 68 $\mu g/m^3$ 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^{th} percentile of 2-hour concentrations is 59 $\mu g/m^3$).

Thus, while controlled human exposure studies support the plausibility of the serious cardiovascular effects that have been linked with ambient $PM_{2.5}$ exposures (U.S. EPA, 2018, Chapter 6), the $PM_{2.5}$ 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 provide limited insight into the occurrence of cardiovascular effects following $PM_{2.5}$ exposures likely to occur in the ambient air in areas meeting the current primary $PM_{2.5}$ standards and are of limited utility in informing conclusions on 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, 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 draft ISA relies on animal toxicology studies to support the plausibility of a wide range of $PM_{2.5}$-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 $PM_{2.5}$ 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 $PM_{2.5}$ standards.

Most of the animal toxicology studies assessed in the draft ISA have examined effects following exposures to $PM_{2.5}$ concentrations well-above the concentrations likely to be allowed by the current $PM_{2.5}$ standards. Such studies have generally examined short-term exposures to $PM_{2.5}$ concentrations from 100 to >1,000 $\mu g/m^3$ and long-term exposures to concentrations from 66 to >400 $\mu g/m^3$ (e.g., see U.S. EPA, 2018, 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 $PM_{2.5}$ concentration of 16.8 $\mu g/m^3$ (Mauad et al., 2008) and a study reporting increased carcinogenic potential following long-term exposures (i.e., 2 months) to an average $PM_{2.5}$ concentration of 17.7 $\mu g/m^3$ (Cangerana Pereira et al., 2011). These two studies demonstrate serious effects following long-term exposures to $PM_{2.5}$ concentrations similar to the ambient concentrations reported in some $PM_{2.5}$ epidemiologic studies (U.S. EPA, 2018, 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 $PM_{2.5}$ exposures (U.S. EPA, 2018), but have not evaluated $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures and mortality or morbidity represent a large part of the evidence base supporting several of the draft ISA's "causal" and "likely to be causal" determinations. In this section, we consider the ambient $PM_{2.5}$ concentrations present in areas where epidemiologic studies have evaluated associations with mortality or morbidity, and what

such concentrations may indicate regarding the primary $PM_{2.5}$ 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 $PM_{2.5}$ standards is complicated by the fact that such studies evaluate associations between distributions of ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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, 2018, 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ air quality likely to have met the current or alternative standards, such standards are likely to allow the daily or annual average $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standards are discussed below in section 3.4.

3.2.3.2.1 $PM_{2.5}$ Air Quality Distributions Associated with Mortality or Morbidity in Key Epidemiologic Studies

In this section, we consider the $PM_{2.5}$ 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 $PM_{2.5}$ concentrations across the air quality distribution, 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 (i.e., estimated exposures and/or health events). This is the case both for studies of daily $PM_{2.5}$ exposures and for studies of annual average $PM_{2.5}$ exposures.

Studies of daily $PM_{2.5}$ exposures examine associations between day-to-day variation in $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures, most of the estimated exposures reflect annual average $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures, indicating that "peak" $PM_{2.5}$ 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, 2018, Figure 6-26). For the years 1974 to 2009, Lepeule et al. (2012) report a positive and statistically significant association between estimated long-term $PM_{2.5}$ 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 $PM_{2.5}$ concentrations near the overall mean concentration reported in the study (i.e., 15.9 $\mu g/m^3$), indicating relatively high confidence in the reported association. Confidence intervals widen at lower and higher long-term $PM_{2.5}$ concentrations, particularly at concentrations $\leq \sim 10$ $\mu g/m^3$ and $\geq \sim 20$ $\mu g/m^3$. 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).

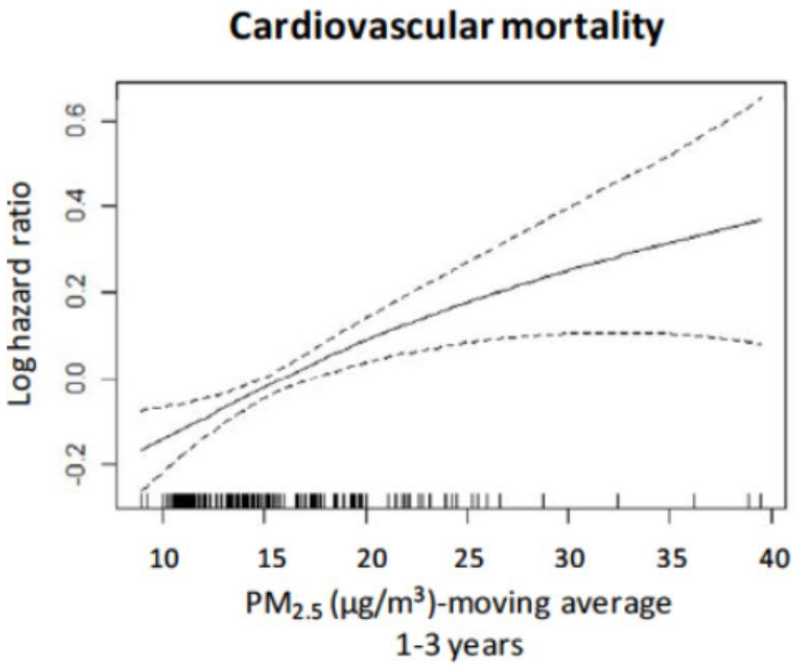


Figure 3-2. Estimated concentration-response function and 95% confidence intervals between $PM_{2.5}$ and cardiovascular mortality in the Six Cities Study (1974-2009) (from Lepeule et al., 2012, supplemental material, figure 1; Figure 6-26 in U.S. EPA, 2018).

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 near the overall mean $PM_{2.5}$ concentrations reported by those studies, likely reflecting high data density near mean concentrations (e.g., Crouse et al., 2015; Villeneuve et al., 2015; Shi et al., 2016 as discussed in U.S. EPA, 2018, section 11.2.4). Thus, as in the last review (78 FR 3161, January 15, 2013; U.S. EPA, 2011, sections 2.1.3 and 2.3.4.1), we recognize that the part of the air quality distribution corresponding to the bulk of the data in a given study generally falls around the overall mean concentration for that study, reflecting the mean of daily or annual exposure estimates over the study period, and that concentrations around (i.e., somewhat above to somewhat below) the overall mean correspond to the range over which studies generally provide the most confidence in reported health effect associations. As described further below, when considering the $PM_{2.5}$ air quality distributions in epidemiologic studies in this section, we focus on $PM_{2.5}$ concentrations around these overall means.

To evaluate the $PM_{2.5}$ air quality distributions in key studies in this review, we first identify the epidemiologic studies assessed in the draft ISA that have the potential to be most informative in reaching conclusions on the primary $PM_{2.5}$ 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 $PM_{2.5}$ exposures in the draft ISA. We focus on the health effect associations that are determined in the draft 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, 2018, section 11.1). 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.[29]

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 draft ISA and that meet these criteria. For each study, Figures 3-3 to 3-6 present the cohort and/or geographic area examined, the approach used to estimate $PM_{2.5}$ exposures (i.e., monitored versus predicted with hybrid modeling methods[30]), the study years during which health events occurred, the years of $PM_{2.5}$ air quality data used to estimate exposures, and the effect estimate[31] with 95% confidence intervals (per 5 $\mu g/m^3$ for long-term exposures; 10 $\mu g/m^3$ 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 $PM_{2.5}$ exposure estimates reported by the study.

Figure 3-3 and Figure 3-4 summarize information from studies of long-term $PM_{2.5}$ exposures. Figure 3-5 and Figure 3-6 summarize information from studies of short-term $PM_{2.5}$ exposures. Table 3-3 summarizes information from the smaller group of "accountability" studies that have evaluated the potential for improvements in public health as ambient $PM_{2.5}$ concentrations have declined over time. It is important to note that these retrospective studies tend to focus on time periods during which ambient $PM_{2.5}$ concentrations were substantially higher than those measured more recently (e.g., see Chapter 2, Figure 2-8).

[29] 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).

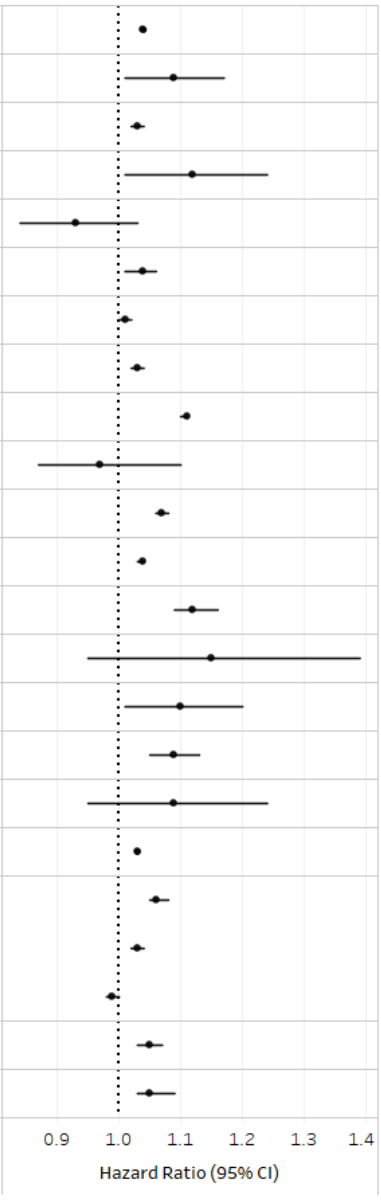
[30] As discussed further below, and in Chapter 2, hybrid methods incorporate data from several sources, often including satellites and models, in addition to ground-based monitors.

[31] The effect estimates presented in the forest plot figures (Figure 3-3 to Figure 3-6) show the associations of long or short-term $PM_{2.5}$ 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).

1

All-cause mortality

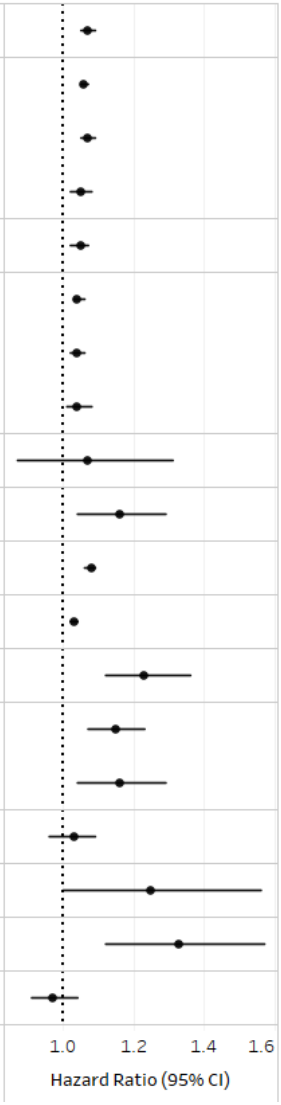
Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Di et al., 2017b	Medicare	2000-2012	2000-2012	11 (5th and 95th: 6.21- 15.64)
		Hart et al., 2015	Nurses Health	2000-2006	1999-2006	12.0 (NR)
		Pope et al., 2015	ACS CPS-II	1982-2004	1999-2004	12.6 (1.0–28.0)
		Puett et al., 2009	Nurses Health	1992-2002	1988-2002	13.9 (5.8–27.6)
		Puett et al., 2011	Health Professionals	1989-2003	1988-2003	17.8 (NR)
		Shi et al., 2016	Medicare	2003-2008	2003-2008	8.12 (0.8-20.22)
		Thurston et al., 2016	NIH-AARP	2000-2009	2000-2008	12.2 (2.9-28.0)
		Turner et al., 2016	ACS CPS-II	1982-2004	1999-2004	12.6 (1.4-27.9)
		Wang et al., 2017	Medicare	2000-2013	2000-2013	NR (Median: 10.7) (6.0-20.6)
		Weichenthal et al., 2014	Ag Health	1993-2009	2001-2006	Iowa: 8.8; North Carolina: 11.1 (NR)
	Canada	Crouse et al., 2012	CanCHEC	1991-2001	2001-2006	8.7 (1.9- 19.2)
		Crouse et al., 2015	CanCHEC	1991-2006	1984-2006	8.9 (0.9- 17.6)
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)
Monitor	U.S.	Goss et al., 2004	U.S. Cystic Fibrosis	1999-2000	2000	13.7 (NR)
		Hart et al., 2015	Nurses Health	2000-2006	2000-2006	12.7 (NR)
		Kiomourtzoglou et al., 2016	Medicare	2000-2010	2000-2010	12.0 (Mean Range: 9-13) (NR)
		Lepeule et al., 2012	Harvard Six-City	2001-2009	1979-2009	1974-2009: 15.9; 2000 onwards mean range: <15-<18 (NR)
		Lipfert et al., 2006	Veterans	1997-2001	1999-2001	14.3 (NR)
		Zeger et al., 2008	MCAPS	2000-2005	2000-2005	Central region: NR (Median: 10.7) (NR) Eastern region: NR (Median: 14.0) (NR) Western region: NR (Median: 13.1) (NR)
	Canada	Crouse et al., 2012	CanCHEC	1991-2001	1987-2001	11.2 (NR)
		Weichenthal et al., 2016a	CanCHEC	1991-2009	1998-2009	9.8 (4.74-13.62)



2

CVD mortality

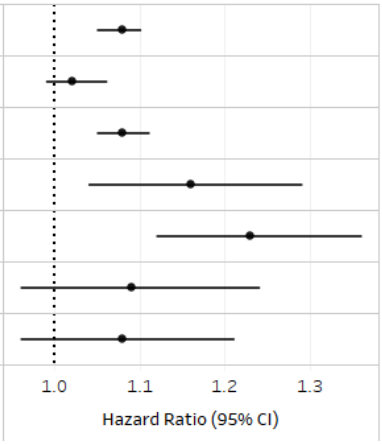
Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)	Health Outcome
Modelled	U.S.	Jerrett et al., 2016	ACS CPS-II	1982-2004	2002-2004	12 (1.5–26.6)	IHD mortality Age 30+
		Pope et al., 2015	ACS CPS-II	1982-2004	1999-2004	12.6 (1.0–28.0)	CVD mortality Age 30+
							IHD mortality Age 30+
							Other CVD-CBVD Age 30+
		Thurston et al., 2016	NIH-AARP	2000-2009	2000-2008	12.2 (2.9-28.0)	CVD mortality Age 50-71
		Turner et al., 2016	ACS CPS-II	1982-2004	1999-2004	12.6 (1.4-27.9)	CVD mortality Age 30+
							IHD mortality Age 30+
							Other CVD-CBVD Age 30+
		Weichenthal et al., 2014	Ag Health	1993-2009	2001-2006	Iowa: 8.8; North Carolina: 11.1 (NR)	CVD mortality
	Canada	Chen et al., 2016	EFFECT RCT	1999-2011	2001-2010	10.7 (NR)	CVD mortality Age 35+
		Crouse et al., 2012	CanCHEC	1991-2001	2001-2006	8.7 (1.9- 19.2)	CVD mortality Age 25+
		Crouse et al., 2015	CanCHEC	1991-2006	1984-2006	8.9 (0.9- 17.6)	CVD mortality Age 25-90
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)	CVD mortality Age 25-90
		Villeneuve et al., 2015	CNBSS	1980-2005	1998-2006	9.1 (1.3- 17.6)	CVD mortality Age 40-59
							IHD mortality Age 40-59
Monitor	U.S.	Hart et al., 2011	TrIPS	1985-2000	2000	14.1 (NR)	CVD mortality
		Lepeule et al., 2012	Harvard Six-City	2001-2009	1979-2009	1974-2009: 15.9; 2000 onwards mean range: <15-<18 (NR)	CVD mortality Age 25-74
		Miller, et al. 2007	WHI	1994-2002	2000	13.5 (3.4-28.3)	CVD mortality Age 50-79
	Canada	Weichenthal et al., 2016a	CanCHEC	1991-2009	1998-2009	9.8 (4.74-13.62)	IHD mortality Age 25-89



1

Respiratory mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Pope et al., 2015	ACS CPS-II	1982-2004	1999-2004	12.6 (1.0–28.0)
		Thurston et al., 2016	NIH-AARP	2000-2009	2000-2008	12.2 (2.9-28.0)
		Turner et al., 2016	ACS CPS-II	1982-2004	1999-2004	12.6 (1.4-27.9)
	Canada	Crouse et al., 2015	CanCHEC	1991-2006	1984-2006	8.9 (0.9- 17.6)
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)
Monitor	U.S.	Hart et al., 2011	TrIPS	1985-2000	2000	14.1 (NR)
	Canada	Weichenthal et al., 2016a	CanCHEC	1991-2009	1998-2009	9.8 (4.74-13.62)



Lung cancer mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Turner et al., 2016	ACS CPS-II	1982-2004	1999-2004	12.6 (1.4-27.9)
	Canada	Crouse et al., 2015	CanCHEC	1991-2006	1984-2006	8.9 (0.9- 17.6)
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)
		Villeneuve et al., 2015	CNBSS	1980-2005	1998-2006	9.1 (1.3- 17.6)
Monitor	U.S.	Hart et al., 2011	TrIPS	1985-2000	2000	14.1 (NR)
		Krewski et al., 2009	ACS CPS-II	1982-2000	1979-1983; 1999-2000	1979- 1983: 21.2; 1999- 2000: 14.0 (NR)
		Laden et al., 2006	Harvard Six-City	1974-1998	1979-1987; 1985-1998	16.4 (Mean Range: 10.2-29.0) (NR)
		Lepeule et al., 2012	Harvard Six-City	2001-2009	1979-2009	1974-2009: 15.9; 2000 onwards mean range: <15-<18 (NR)
		Thurston et al., 2013	ACS CPS-II	1982-2004	2000-2005	14.2 (NR)
	Canada	Weichenthal et al., 2016a	CanCHEC	1991-2009	1998-2009	9.8 (4.74-13.62)

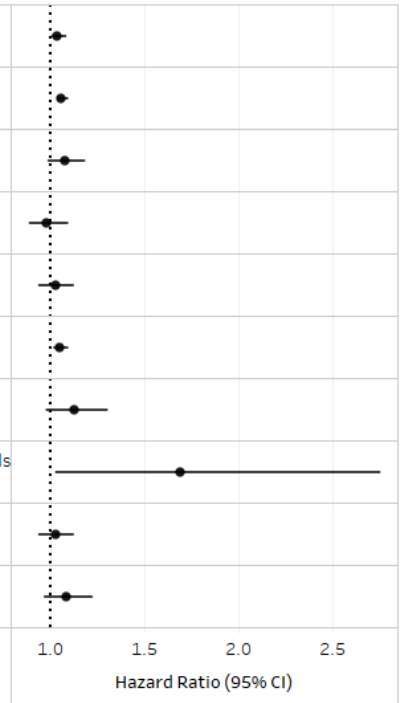
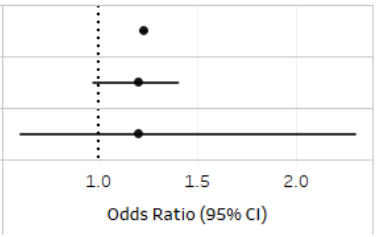


Figure 3-3. Epidemiologic studies examining associations between long-term $PM_{2.5}$ exposures and mortality.

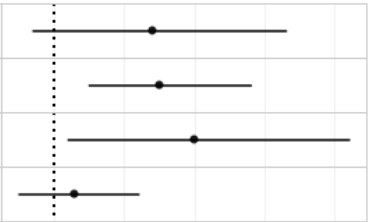
Asthma incidence

Exposure Proxy (group)	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	Canada	Tetreault et al., 2016	QICDSS	1996-2011	2001-2006	9.86 (NR)
Monitor	U.S.	McConnell et al., 2010	CHS	2003-2005	2003-2004	13.9 (6.3-23.7)
		Nishimura et al., 2013	GALA II/ SAGE II	1986-2003	1986-2003	Mean Range: 8.1-17.0 (NR)



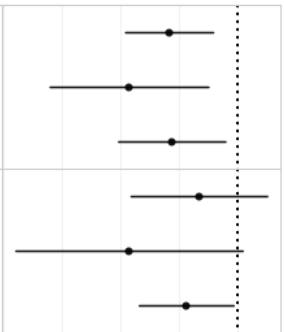
Lung cancer incidence

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	Canada	Hystad et al., 2013	NECSS	1994–1997	1975-1994	11.9 (NR)
		Tomczak et al., 2016	CNBSS	1980-2004	1998-2006	9.1 (1.3-17.6)
Monitor	U.S.	Gharibvand et al., 2016	AHSMOG-2	2002-2011	2000-2001	12.9 (NR)
		Puett et al., 2014	Nurses Health	1994-2010	1988-2007	13.1 (NR)



Lung development

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)	Health Outcome
Monitor	U.S.	Breton et al., 2011	CHS	1993-2000, 1996-2004	1994-2004	Mean Range: 6.0-28.0 (NR)	Lung Development (Change) - FVC Age 10-18
							Lung Development (Change) - MMEF Age 10-18
							Lung Development (Change)- FEV1 Age 10-18
		Gauderman et al., 2004	CHS	1993-2000	1994-2000	Mean Range: 6.0-28.0 (NR)	Lung Development (Change) - FVC Age 10-18
							Lung Development (Change) - MMEF Age 10-18
							Lung Development (Change)- FEV1 Age 10-18



Lung function

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)	Health Outcome
Monitor	U.S.	Urman et al., 2014	CHS	2002-2007	2002-2007	6.0-28.0 (NR)	Lung Function Decline (%)- FEV1 Age 5-7
							Lung Function Decline (%)- FVC Age 5-7

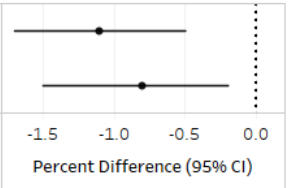
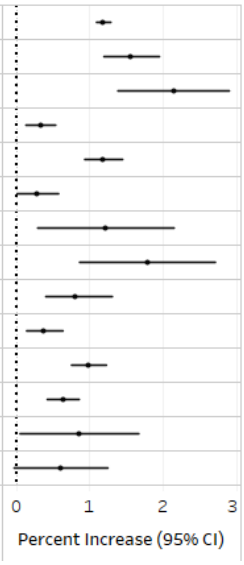


Figure 3-4. Epidemiologic studies examining associations between long-term $PM_{2.5}$ exposures and morbidity.

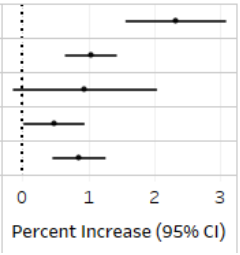
All-cause mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Di et al., 2017a	Medicare	2000-2012	11.6 (5th and 95th: 6.21- 15.64)
		Lee et al., 2015b	State Dept	2007-2011	11.1 (17.7- 69.9)
		Shi et al., 2016	Medicare	2003-2008	8.12 (0.8-20.22)
Monitor	U.S.	Baxter et al., 2017	NCHS/State Dept	2001-2005	Cluster Mean Range: 12.2-14.1 (NR)
		Dai et al., 2014	NCHS/State Dept	2000-2006	13.3 (NR)
		Dominici et al., 2007	NMMAPS	1999–2000	NR (NR)
		Franklin et al., 2007	NCHS/State Dept	1997-2002	15.6 (NR)
		Franklin et al., 2008	NCHS/State Dept	2000-2005	14.8 (NR)
		Klemm et al., 2003	Harvard Six-City	1979–1988	14.7 (Median: 9.0) (NR)
		Krall et al., 2013	NCHS/State Dept	2000–2005	13.6 (NR)
		Zanobetti and Schwartz, 2009	NCHS	1999-2005	13.2 (NR)
		Zanobetti et al., 2014	Medicare	1999-2010	Mean Range: 4.37-17.97 (NR)
	Canada	Burnett et al., 2003	Statistics Canada	1986-1996	13.3 (NR)
		Burnett et al., 2004	Statistics Canada	1981-1999	12.8 (NR)



CVD mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Lee et al., 2015b	State Dept	2007-2011	11.1 (17.7- 69.9)
Monitor	U.S.	Dai et al., 2014	NCHS/State Dept	2000-2006	13.3 (NR)
		Franklin et al., 2007	NCHS/State Dept	1997-2002	15.6 (NR)
		Franklin et al., 2008	NCHS/State Dept	2000-2005	14.8 (NR)
		Zanobetti and Schwartz, 2009	NCHS	1999-2005	13.2 (NR)



Respiratory mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Lee et al., 2015b	State Dept	2007-2011	11.1 (17.7- 69.9)
Monitor	U.S.	Dai et al., 2014	NCHS/State Dept	2000-2006	13.3 (NR)
		Franklin et al., 2007	NCHS/State Dept	1997-2002	15.6 (NR)
		Franklin et al., 2008	NCHS/State Dept	2000-2005	14.8 (NR)
		Zanobetti and Schwartz, 2009	NCHS	1999-2005	13.2 (NR)

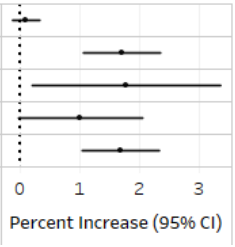
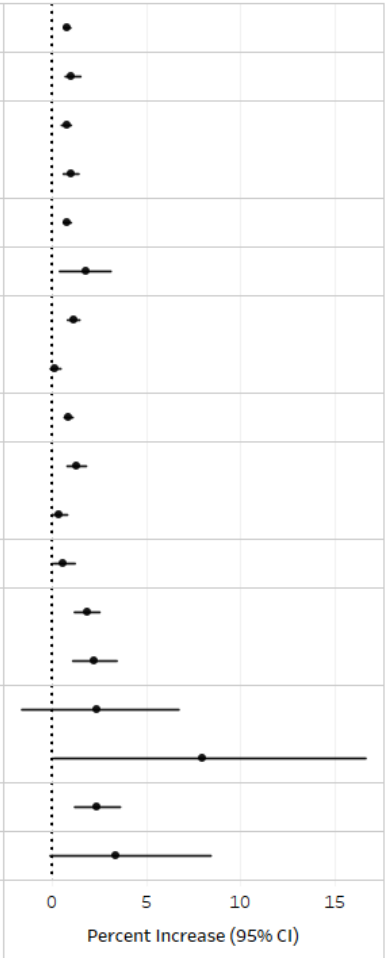


Figure 3-5. Epidemiologic studies examining associations between short-term $PM_{2.5}$ exposures and mortality.[32]

[32] As noted above, the overall mean $PM_{2.5}$ 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 $PM_{2.5}$ exposure estimates.

CVD morbidity

Exposure Proxy	Country	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)	Health Outcome
Modelled	U.S.	Bravo et al., 2017	Medicare	2002-2006	12.3 (NR)	CVD HA Age 65+
		Kloog et al., 2012	Medicare	2000-2006	9.6 (0.01-72.59	CVD HA Age 65+
		Kloog et al., 2014	Medicare	2000-2006	11.9 (NR)	CVD HA Age 65+
						IHD HA Age 65+
Monitor	U.S.	Bell et al., 2008	Medicare	1999-2005	12.9 (NR)	CVD HA Age 65+
		Bell et al., 2014	Medicare	2000-2004	14.0 (Median: 11.7) (NR)	CVD HA Age 65+
		Bell et al., 2015	Medicare	1999-2010	12.3 (6.4- 20.2)	Heart Failure HA Age 65+
						IHD HA Age 65+
		Bravo et al., 2017	Medicare	2002-2006	12.5 (NR)	CVD HA Age 65+
		Dominici et al., 2006	Medicare	1999-2002	13.4 (NR)	Heart Failure HA Age 65+
						IHD HA Age 65+
		Peng et al., 2009	Medicare	2000-2006	NR (Median: 11.8) (NR)	CVD HA Age 65+
		Zanobetti et al., 2009	Medicare	2000-2003	15.3 (NR)	Heart Failure HA Age 65+
						MI HA Age 65+
	Canada	Stieb et al., 2009	Hospital Database	1992-2003	8.2 (6.7-9.8)	Angina/MI ED
						Heart Failure ED
		Szyszkowicz et al., 2009	Hospital Database	1992-2003	8.3 (NR)	Angina ED
		Weichenthal et al., 2016b	NACRS	2004–2011	6.9 (NR)	MI ED



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Respiratory morbidity

Exposure Proxy	Country	Citation	Cohort	Health Data	Health Outcome	Reported PM Mean (Range)(ug/m3)
Modelled	U.S.	Kloog et al., 2014	Medicare	2000-2006	COPD HA Age 65+	11.9 (NR)
Monitor	U.S.	Alhanti et al., 2016	Hospital Database	1993-2009	Asthma ED Age 5-18	Mean Range: 11.1-14.1 (NR)
					Asthma ED Age 65+	Mean Range: 11.1-14.1 (NR)
		Bell et al., 2015	Medicare	1999-2010	Asthma HA Age 65+	12.3 (6.4- 20.2)
					COPD HA Age 65+	12.3 (6.4- 20.2)
		Dominici et al., 2006	Medicare	1999-2002	COPD HA Age 65+	13.4 (NR)
		Malig et al., 2013	Hospital Inpatient and Outpatient visits	2005-2008	Asthma ED & HA	Mean Range: 5.2-19.8 (NR)
					COPD ED & HA	Mean Range: 5.2-19.8 (NR)
		Ostro et al., 2016	Hospital Inpatient and Outpatient visits	2005-2009	Asthma ED & HA	16.5 (NR)
					COPD ED & HA	16.5 (NR)
		Yap et al, 2013	Hospital Admissions	2000-2005	Asthma HA Age 1-9: Central Valley	Mean Range: 12.8-20.8 (NR)
					Asthma HA Age 1-9: South Coast	Mean Range: 14.0-24.6 (NR)
	Canada	Stieb et al., 2009	Hospital Database	1992-2003	Asthma ED	8.2 (6.7-9.8)
					COPD ED	8.2 (6.7-9.8)
		Weichenthal et al., 2016c	NACRS	2004-2011	Asthma ED	7.1 (NR)
					COPD ED	7.1 (NR)

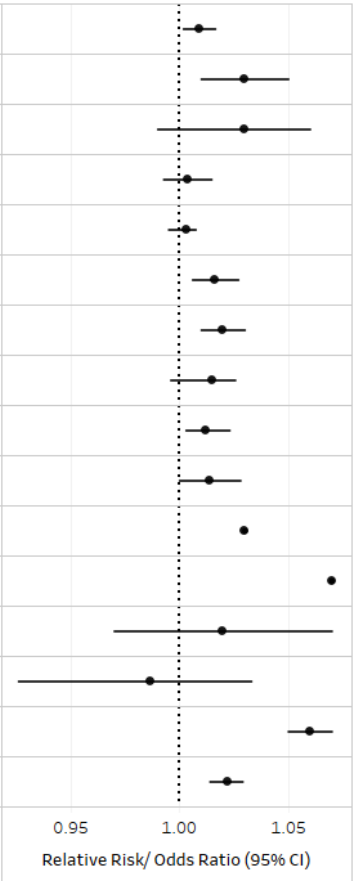


Figure 3-6. Epidemiologic studies examining associations between short-term $PM_{2.5}$ exposures and morbidity.

Table 3-3. Epidemiologic studies examining the health impacts of long-term reductions in ambient $PM_{2.5}$ concentrations.

Study Reference	Study Area	Years of $PM_{2.5}$ Air Quality (monitored)	Starting $PM_{2.5}$ Concentrations (mean)	Ending $PM_{2.5}$ concentrations (mean)	Study Results
Pope et al. (2009)	211 U.S. counties	1979-1983 compared to 1999-2000	20.6 μg/m^3	14.1 g/m^3	Statistically significant association between declining ambient $PM_{2.5}$ and increasing life expectancy
Correia et al. (2013)	545 U.S. counties	2000 compared to 2007	13.2 μg/m^3	11.6 g/m^3	Statistically significant association between declining ambient $PM_{2.5}$ 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^3	14.4 μg/m^3	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^3	11.9-17.8 μg/m^3	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 $PM_{2.5}$ exposures (short- or long-term) and mortality or morbidity across a wide range of ambient $PM_{2.5}$ concentrations. As discussed above, considering the $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures that provide strong support for reported associations.

In addition to overall mean $PM_{2.5}$ concentrations, for a subset of key epidemiologic studies with available information we also consider 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations corresponding to the 25th and 10th 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.[33]

To frame our evaluation of study-reported $PM_{2.5}$ concentrations, we specifically consider the following questions:

- **What are the overall mean $PM_{2.5}$ 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 $PM_{2.5}$ concentrations corresponding to the 25th and/or 10th percentiles of those data?**

To answer these questions, Figure 3-7 and Figure 3-8 below present information on the monitored (Figure 3-7) and hybrid model-predicted (Figure 3-8) ambient $PM_{2.5}$ concentrations used to estimate $PM_{2.5}$ exposures in key epidemiologic studies.

Drawing from the U.S. and Canadian multicity studies in Figure 3-3 to Figure 3-6 above,[34] the studies included in Figure 3-7 and Figure 3-8 are those that report overall mean (or median) $PM_{2.5}$ concentrations and for which the years of $PM_{2.5}$ air quality data used to estimate exposures overlap entirely with the years during which health events are reported. Regarding this latter issue, the $PM_{2.5}$ 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

[33] In the last review of the PM NAAQS, the PA identified the long-term $PM_{2.5}$ concentrations corresponding to the 25th and 10th 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 25th and 10th percentiles.

[34] Most of the studies included in Table 3-3 above (i.e., studies that examine relationships between declining ambient $PM_{2.5}$ concentrations and improving health) report mean ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that contributed to reported associations.[35]

Figure 3-7 highlights the overall mean (or median) $PM_{2.5}$ concentrations reported in key studies that use ground-based monitors alone to estimate long- or short-term $PM_{2.5}$ 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 $PM_{2.5}$ concentrations corresponding to the 25^{th} and 10^{th} percentiles of health events[36] (see Appendix B, Section B.2 for more information).

[35] This is an issue only for some studies of long-term $PM_{2.5}$ exposures. While this approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term $PM_{2.5}$ exposures, under the assumption that spatial patterns in $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that could have contributed to reported health outcomes.

[36] That is, 25% of the total health events occurred in study locations with mean $PM_{2.5}$ concentrations (i.e., averaged over the study period) below the 25^{th} percentiles identified in Figure 3-7 and 10% of the total health events occurred in study locations with mean $PM_{2.5}$ concentrations below the 10^{th} percentiles identified.

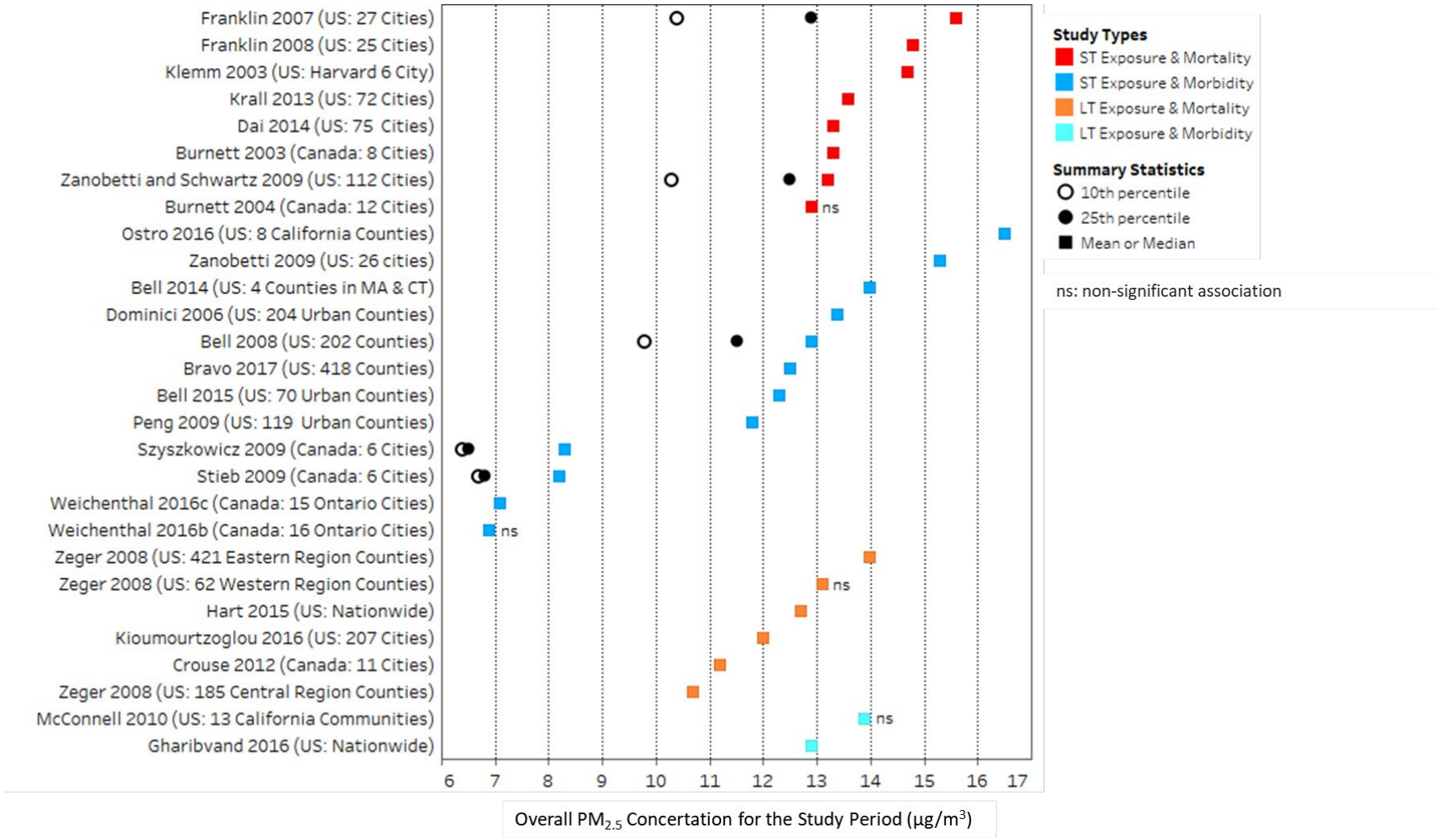


Figure 3-7. Monitored $PM_{2.5}$ concentrations in key epidemiologic studies.

We also consider the emerging body of studies that use predicted ambient $PM_{2.5}$ concentrations from hybrid modeling methods to estimate long- or short-term $PM_{2.5}$ exposures (Figure 3-8, below). As discussed in Chapter 2 of this draft 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 $PM_{2.5}$ exposures in areas with relatively sparse monitoring networks (U.S. EPA, 2018, sections 3.3.2 to 3.3.5).

Figure 3-8 presents overall means of predicted $PM_{2.5}$ concentrations for key studies, and the concentrations corresponding to the 25th and 10th percentiles of estimated exposures or health events[37] when available (see appendix B, section B.3 for additional information). As for the monitor-based studies highlighted above, Figure 3-8 focuses on multicity studies[38] that examine health outcomes supporting "causal" or "likely to be causal" determinations in the draft ISA and that use air quality data to estimate $PM_{2.5}$ exposures for the entire range of years during which health events occurred. 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 $PM_{2.5}$ exposures are estimated in the health study (e.g., regional or national 10-fold cross validation performance statistics reported for the same years that exposures are estimated).[39]

[37] For most studies in Figure 3-8, 25th percentiles of exposure estimates are presented. That is, 25% of short-term or long-term exposure estimates in the study population are below these concentrations. The exception is Di et al. (2017a), for which Figure 3-8 presents the short-term $PM_{2.5}$ exposure estimates corresponding to the 25th and 10th percentiles of deaths in the study population (i.e., 25% and 10% of deaths occurred at concentrations below these exposure estimates).

[38] All studies that meet the criteria for inclusion in Figure 3-8 were conducted in the U.S.

[39] For example, due to lack of spatial field availability before 1998, Crouse et al. (2015) use median annual $PM_{2.5}$ concentrations for the 1998-2006 time period (van Donkelaar et al., 2010; van Donkelaar et al., 2015a; van 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 $PM_{2.5}$ monitoring was sparse prior to 1999, with many of the available $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ based on TSP measurements and metropolitan indicator variables because only small number of $PM_{2.5}$ 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.

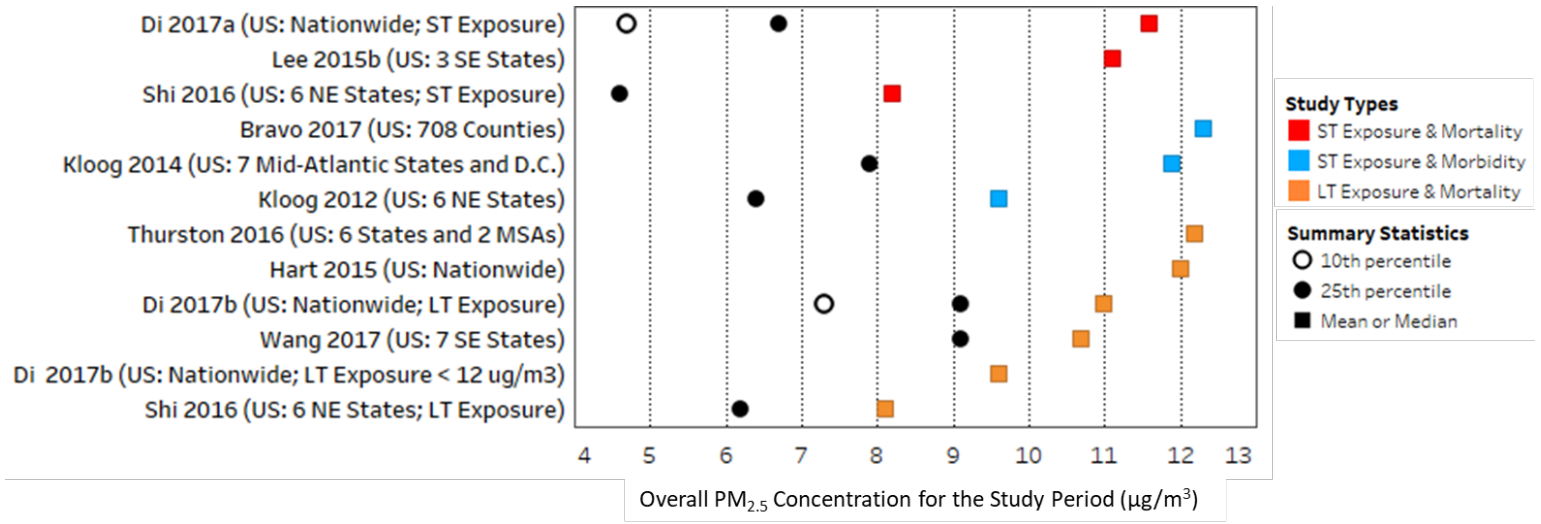


Figure 3-8. Hybrid model-predicted $PM_{2.5}$ concentrations in key epidemiologic studies.

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 $PM_{2.5}$ exposures (short- or long-term) and mortality or morbidity across a wide range of monitored or hybrid-model-predicted ambient $PM_{2.5}$ concentrations. With regard to these studies, we particularly note the following:

- For the large majority of key studies, the $PM_{2.5}$ air quality distributions that support reported associations are characterized by overall mean (or median) $PM_{2.5}$ concentrations ranging from just above 8.0 $\mu g/m^3$ to just above 16.0 $\mu g/m^3$. 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 $PM_{2.5}$ exposures, and all of the U.S. studies in this group, report overall mean $PM_{2.5}$ concentrations at or above 10.7 $\mu g/m^3$.
  - Four Canadian studies that use monitors alone report lower overall mean concentrations. Two of these studies report overall means just above 8.0 $\mu g/m^3$ (both report positive and statistically significant associations) and two studies report overall means around 7.0 $\mu g/m^3$ (positive and statistically significant association in one of these studies).
  - Most key studies that use hybrid modeling approaches to estimate $PM_{2.5}$ exposures report overall mean concentrations at or above 9.6 $\mu g/m^3$. 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 $PM_{2.5}$ concentrations reports overall means just above 8.0 $\mu g/m^3$ (i.e., Shi et al., 2016). This study reports positive and statistically significant health effect associations with both short- and long-term $PM_{2.5}$ exposures.[40]
- Three 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 $\mu g/m^3$"). In addition to this study, Di et al. (2017a) and Shi et al. (2016) also report positive and statistically significant associations in restricted analyses.
  - Di et al. (2017a) reports a positive and statistically significant association in an analysis restricted to 24-hour $PM_{2.5}$ exposure estimates < 25 $\mu g/m^3$. This study did not report an overall mean $PM_{2.5}$ concentration for the restricted analysis, though it was presumably somewhat below the mean reflected in Figure 3-8 (i.e., 11.6 $\mu g/m^3$).
  - Shi et al. (2016) report positive and statistically significant associations in analyses restricted to annual $PM_{2.5}$ exposure estimates < 10 $\mu g/m^3$ and in analyses restricted to 24-hour exposure estimates < 30 $\mu g/m^3$. This study does not report

[40] However, the authors report that, for associations with long-term $PM_{2.5}$ exposures, most deaths occurred at or above the 75th percentile of annual exposure estimates (i.e., 10 $\mu g/m^3$) (see Tables 1 and 2 in published manuscript). Authors did not report this information for their analysis of short-term $PM_{2.5}$ exposures.

the overall mean $PM_{2.5}$ concentrations in restricted analyses, though such means are presumably somewhat below those reflected in Figure 3-8 (i.e., 8.1 and 8.2 $\mu g/m^3$).

- For some key studies, information on the broader distributions of $PM_{2.5}$ exposure estimates and/or health events is available.
  - In U.S. studies that use monitors alone to estimate $PM_{2.5}$ exposures, 25th percentiles of health events correspond to mean $PM_{2.5}$ concentrations (i.e., averaged over the study period for each study city) at or above 11.5 $\mu g/m^3$ and 10th percentiles of health events correspond to mean $PM_{2.5}$ concentrations at or above 9.8 $\mu g/m^3$ (i.e., 25% and 10% of health events, respectively, occur in study locations with mean $PM_{2.5}$ concentrations below these values).
  - In the Canadian studies that use monitors alone to estimate $PM_{2.5}$ exposures, 25th percentiles of health events correspond to mean $PM_{2.5}$ concentrations at or above 6.5 $\mu g/m^3$ and 10th percentiles of health events correspond to mean $PM_{2.5}$ concentrations at or above 6.4 $\mu g/m^3$.
  - Of the key studies that use hybrid modeling approaches to estimate long-term $PM_{2.5}$ exposures, the ambient $PM_{2.5}$ concentrations corresponding to 25th percentiles of estimated exposures are 6.2 and 9.1 $\mu g/m^3$. In the one study with data available on the 10th percentile of $PM_{2.5}$ exposure estimates, the concentration corresponding to that 10th percentile is 7.3 $\mu g/m^3$.
  - In studies that use hybrid modeling approaches to estimate short-term $PM_{2.5}$ exposures, the ambient concentrations corresponding to 25th percentiles of estimated exposures, or health events, are generally at or above 6.4 $\mu g/m^3$. In the one study with lower concentrations, the ambient $PM_{2.5}$ concentration corresponding to the 25th percentile of estimated exposures is 4.7 $\mu g/m^3$.[41] In the one study with information available on the 10th percentile of health events, the ambient $PM_{2.5}$ concentration corresponding to that 10th percentile is 4.7 $\mu g/m^3$.

The information in Figure 3-7 and Figure 3-8 indicates consistent support for generally positive and statistically significant health effect associations for $PM_{2.5}$ air quality distributions characterized by overall mean (or median) concentrations above 8.0 $\mu g/m^3$, with most studies (and all but one U.S. study) reporting overall mean (or median) concentrations at or above 9.6 $\mu g/m^3$. While the ambient $PM_{2.5}$ concentrations around these overall means generally reflect the part of the air quality distribution over which studies provide the strongest support for reported $PM_{2.5}$ effect estimates, there are uncertainties in using these concentrations to inform conclusions on the primary $PM_{2.5}$ standards. These uncertainties are summarized below and their potential

[41] As noted above, in this study (Shi et al., 2016), the authors report that most deaths occurred at or above the 75th percentile of annual exposure estimates (i.e., 10 $\mu g/m^3$). The short-term exposure estimates accounting for most deaths are not presented in the published study.

implications for conclusions on the current and alternative standards are discussed further in section 3.4.

A key uncertainty in using study-reported mean $PM_{2.5}$ concentrations to inform conclusions on the primary $PM_{2.5}$ 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 $PM_{2.5}$ concentrations reported by key epidemiologic studies reflect averaging of short- or long-term $PM_{2.5}$ 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 $PM_{2.5}$ standard, design values are calculated as the annual arithmetic mean $PM_{2.5}$ concentration, averaged over 3 years. For the 24-hour standard, design values are calculated as the 98^{th} percentile of the annual distribution of 24-hour $PM_{2.5}$ 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 $PM_{2.5}$ concentrations (section 2.2.3), areas meeting a $PM_{2.5}$ standard with a particular level would be expected to have average $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations.[42] 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 3.4 below, when

[42] Given that higher $PM_{2.5}$ concentrations have been reported at some near-road monitoring sites, relative to the surrounding area (section 2.3.2.2.2), recent requirements for $PM_{2.5}$ 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.

evaluating what the mean $PM_{2.5}$ concentrations reported by key epidemiologic studies may indicate regarding the current or alternative $PM_{2.5}$ standards, we consider the broader relationships between mean $PM_{2.5}$ concentrations, averaged across space and over time, and $PM_{2.5}$ design values.[43]

Additional uncertainties in using the $PM_{2.5}$ concentrations reported by key epidemiologic studies to inform conclusions on the primary $PM_{2.5}$ standards include the following:

- Effects can occur over the full distributions of ambient $PM_{2.5}$ concentrations evaluated in epidemiologic studies, and the evidence does not identify a threshold concentration below which $PM_{2.5}$-associated effects no longer occur. Thus, while conclusions on primary standards can be informed by comparing the $PM_{2.5}$ 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 $PM_{2.5}$ exposures that result in health effects or exposures below which effects do not occur.
- For studies that use hybrid model predictions to estimate $PM_{2.5}$ 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 $PM_{2.5}$ concentrations, potentially accounting for the observations that model performance for hybrid models weaken by some metrics with decreasing $PM_{2.5}$ 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 preliminary conclusions on the current and alternative primary $PM_{2.5}$ standards are discussed below in section 3.4.

3.2.3.2.2 $PM_{2.5}$ Pseudo-Design Values in Locations of Key Epidemiologic Studies

In addition to considering the study-reported $PM_{2.5}$ concentrations discussed above, we also evaluate study area air quality using metrics more closely related to the design values employed by the EPA to determine whether areas meet or violate the primary $PM_{2.5}$ standards. To the extent these metrics suggest that reported health effect associations are based largely on $PM_{2.5}$ air quality that would have met the current or alternative standards during study periods, we have greater confidence that those standards would allow the $PM_{2.5}$ 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

[43] As discussed above in section 3.1.2, compared to the annual standard, the potential implications of overall mean $PM_{2.5}$ concentrations reported by key epidemiologic studies are less clear for the 24-hour $PM_{2.5}$ standard with its 98^{th} percentile form (section 3.4).

alternative standards, there is greater uncertainty in the degree to which those standards would allow the $PM_{2.5}$ exposures that provide the basis for reported associations.

To evaluate this issue, we calculate metrics similar to $PM_{2.5}$ design values (referred to here as "pseudo-design values") for the locations and time periods evaluated by key U.S. and Canadian epidemiologic studies. For each study, we first identify the locations included in the study that contained one or more $PM_{2.5}$ monitors during the study period, and that had sufficient monitoring data available to calculate pseudo-design values.[44] For key studies conducted in the U.S., study locations were defined as the counties that were included in the study. For key studies conducted in Canada, study locations were defined as the cities included in the study. For each monitored study location, we then identified the highest annual and 24-hour $PM_{2.5}$ pseudo-design values for each 3-year period of the study and calculated the study-period average of these highest values. We also identified 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.[45] 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 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-4 and B-5) and 24-hour (Appendix B, Figure B-9) $PM_{2.5}$ standards.[46]

In Figure 3-9, whiskers reflect annual $PM_{2.5}$ pseudo-design values corresponding to 5^{th} and 95^{th} percentiles of study area populations (or health events), boxes correspond to the 25^{th} and 75^{th} percentiles, and the vertical lines inside the boxes correspond to 50^{th} percentiles. The vertical dotted line in Figure 3-9 is drawn at 12.0 $\mu g/m^3$, the level of the current annual $PM_{2.5}$ standard. For studies with 25^{th} percentiles $\leq$ 12.0 $\mu g/m^3$, at least 25% of the study area population (i.e., in

[44] 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. 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).

[45] 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).

[46] As discussed below, among study locations with averaged $PM_{2.5}$ pseudo-design values (i.e., averaged over the study period) at or below 12.0 $\mu g/m^3$, almost all individual 3-year pseudo-design values are also at or below 12.0 $\mu g/m^3$ (i.e., 89% for Di et al., 2017b; 98% for Shi et al., 2016 – see Appendix B, section B.9).

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).[47] Similarly, for studies with 50th or 75th percentiles ≤ 12.0 μg/m^3, 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).

[47] As noted below, and discussed further in Appendix B (section B.9), among study locations with averaged $PM_{2.5}$ pseudo-design values at or below 12.0 μg/m^3 (i.e., averaged over the study period), almost all individual 3-year pseudo-design values during the study period are also at or below 12.0 μg/m^3.

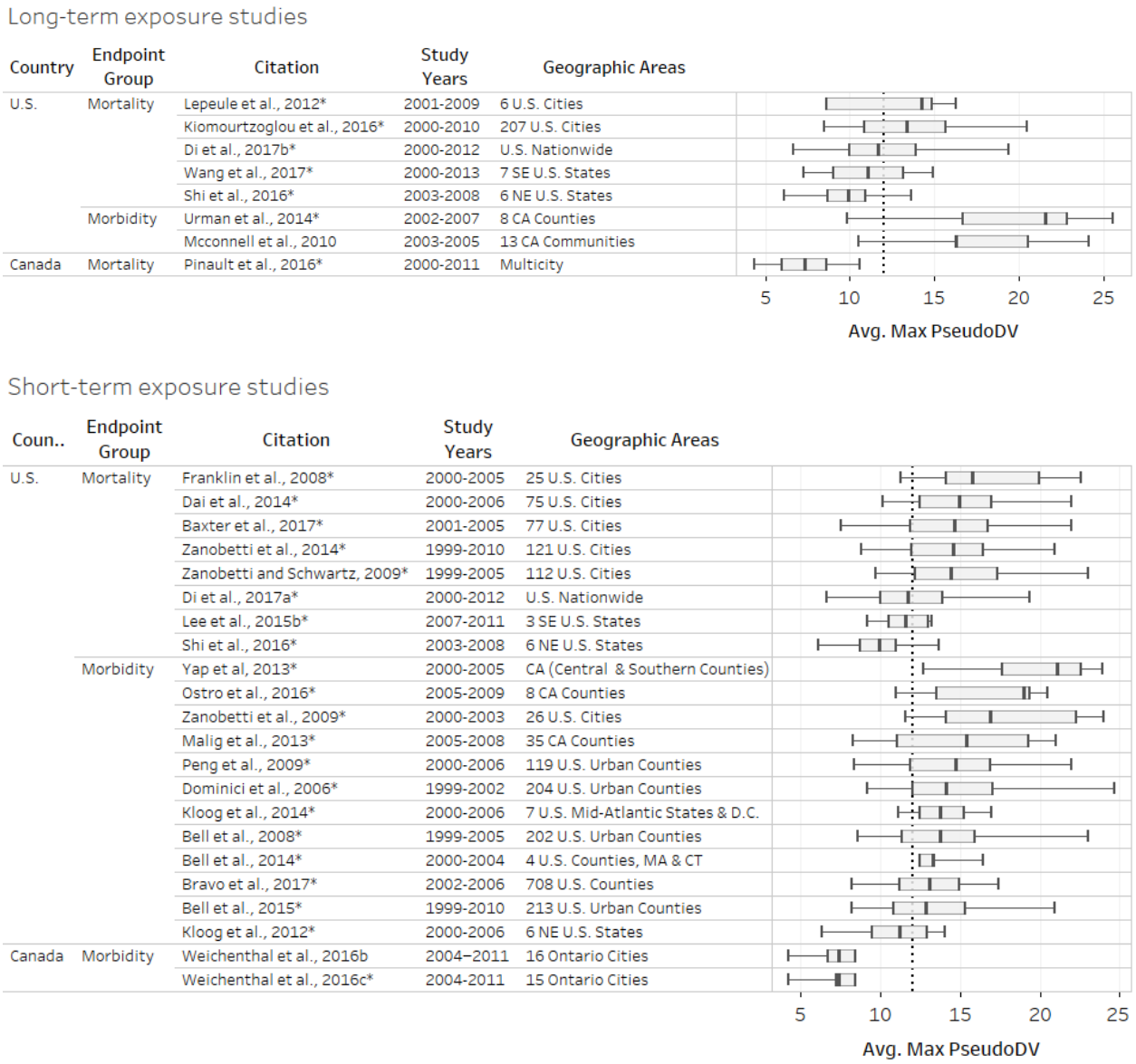


Figure 3-9. $PM_{2.5}$ annual pseudo-design values (in µg/m^3) corresponding to various percentiles[48] of study area populations or health events for studies of long-term and short-term $PM_{2.5}$ exposures.[49]

[48] Asterisks next to study citations denote statistically significant effect estimates.

[49] 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[50]), 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 $\mu g/m^3$ correspond to 25th 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 $\mu g/m^3$ correspond to 25th 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 $\mu g/m^3$ correspond to 50th 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 $\mu g/m^3$ correspond to 50th 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 $\mu g/m^3$ corresponds to the 75th percentile of the study area population.[51]
  - 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 $\mu g/m^3$ correspond to 75th 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

[50] Shi et al. (2016) separately examined long- and short-term $PM_{2.5}$ exposures and, therefore, is included twice in Figure 3-9 and Figure B-9.

[51] 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 ≤ 12.0 $\mu g/m^3$).

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 $PM_{2.5}$ standards, there are important uncertainties to consider when using such information to inform conclusions on the primary $PM_{2.5}$ 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 $PM_{2.5}$ concentrations below 12 $\mu g/m^3$ (Di et al., 2017b) or 10 $\mu g/m^3$ (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 $PM_{2.5}$ pseudo-design values (i.e., averaged over the study period) at or below 12.0 $\mu g/m^3$, almost all individual 3-year pseudo-design values are also at or below 12.0 $\mu g/m^3$ (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 $PM_{2.5}$ monitor. While this approach can account for the large majority of study area populations for studies that use monitors alone to estimate $PM_{2.5}$ exposures, some recent key epidemiologic studies use hybrid modeling approaches to predict ambient $PM_{2.5}$ concentrations in locations with and without nearby ground-based monitors (i.e., Figure 3-8, above). For these studies, $PM_{2.5}$ 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 $PM_{2.5}$ concentrations than monitored areas, our analyses of pseudo-design values could be biased toward the higher values present in monitored locations.

- $PM_{2.5}$ monitoring requirements have changed since the study periods covered by key studies. In particular, $PM_{2.5}$ pseudo-design values during study periods do not reflect the near-road $PM_{2.5}$ monitors that are now required in many large urban areas (discussed in section 2.3.2.2.1 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 $\mu g/m^3$ higher than at the highest measuring non-near-road sites (range is 0.1 to 2.0 $\mu g/m^3$ higher at near-road sites) (Table 2-2 above).

The potential implications of these and other uncertainties for the primary $PM_{2.5}$ standards are discussed in section 3.4 below.

3.2.3.3 Preliminary Conclusions from the Evidence

In reaching preliminary 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures in epidemiologic studies (U.S. EPA, 2018, Chapters 5 to 11). However, the $PM_{2.5}$ 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 $PM_{2.5}$ concentrations (Mauad et al. (2008); Cangerana Pereira et al. (2011),[52] though still above typical ambient concentrations observed in locations meeting the current standards. Thus, while experimental studies support the plausibility of serious $PM_{2.5}$-associated health effects, these studies provide limited insight into the occurrence of effects following $PM_{2.5}$ exposures likely to occur in the ambient air in areas meeting the current primary $PM_{2.5}$ 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

[52] 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 $\mu g/m^3$ $PM_{2.5}$. Hemmingsen et al. (2015b) reports cardiovascular effects in human volunteers following 5-hour exposures to an average of 24 $\mu g/m^3$ $PM_{2.5}$. 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 $PM_{2.5}$ concentration of 10.5 $\mu g/m^3$.

$PM_{2.5}$ exposures (short- or long-term) and mortality or morbidity across a broad range of ambient concentrations. These include associations based on $PM_{2.5}$ air quality distributions lower than those in key studies from the last review.[53] 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 $PM_{2.5}$ concentrations ranging from 8.1 $\mu g/m^3$ to 16.5 $\mu g/m^3$, with mean concentrations in most of these studies (and all but one key U.S. study) at or above 9.6 $\mu g/m^3$. 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 $PM_{2.5}$ exposures, and approaches to control for confounding. The evidence for associations at lower ambient concentrations (i.e., means < 8.0 $\mu g/m^3$) is more limited, with two studies conducted in Ontario reporting positive associations (statistically significant in one study) for $PM_{2.5}$ air quality distributions characterized by overall mean concentrations around 7.0 $\mu g/m^3$ (Weichenthal et al., 2016c; Weichenthal et al., 2016b).

Considering the $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures that support reported health effect associations. However, a limitation of considering study-reported mean $PM_{2.5}$ concentrations to inform conclusions on the primary $PM_{2.5}$ 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 $PM_{2.5}$ 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-8). These relationships suggest that areas with maximum annual $PM_{2.5}$ design values of 12.0 $\mu g/m^3$ (i.e., just meeting the current annual standard) are likely to have long-term mean $PM_{2.5}$ concentrations (i.e., averaged

[53] In the last review key epidemiologic studies supporting "causal" or "likely to be causal" determinations examined distributions of ambient $PM_{2.5}$ with overall mean concentrations at or above 12.8 $\mu g/m^3$ (U.S. EPA, 2011, Figure 2-8).

across space and over time) that are somewhat below 12.0 μg/m^3 but still higher than the overall means reported by a number of key epidemiologic studies reporting $PM_{2.5}$ health effect associations. This indicates that the current standards are likely to allow the distributions of short- and long-term $PM_{2.5}$ 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 $PM_{2.5}$ standards is to consider analyses of $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 μg/m^3 correspond to 50th 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 μg/m^3. For the Canadian studies with the lowest ambient $PM_{2.5}$ concentrations, annual pseudo-design values of about 7.3 to 7.4 μg/m^3 correspond to 50th percentiles of study area populations (or health events), and annual pseudo-design values from 8.4 to 8.6 μg/m^3 correspond to 75th percentiles.

When the information summarized above is taken together, along with the uncertainties discussed in section 3.2.3.2 above, we reach the preliminary conclusion that a number of key epidemiologic studies report positive and statistically significant $PM_{2.5}$ health effect associations for air quality distributions likely to be allowed by the current primary $PM_{2.5}$ standards. Our consideration of the evidence and air quality information to inform preliminary conclusions on the primary $PM_{2.5}$ standards is discussed further in section 3.4 below.

3.3 RISK-BASED CONSIDERATIONS

To inform conclusions regarding the primary $PM_{2.5}$ 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 $PM_{2.5}$-related health risks in locations with three-year annual $PM_{2.5}$ design values of 12.0 μg/m^3 and/or three-year 24-hour design values of 35 μg/m^3 (i.e., neither above nor below the levels of the current standards). Therefore, in addition to our evaluation of $PM_{2.5}$ 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 $PM_{2.5}$ air quality that has been adjusted to simulate "just meeting" the current standards (i.e., design values equal to 12.0 $\mu g/m^3$ and/or 35 $\mu g/m^3$). Given our preliminary conclusions based on the evidence (section 3.2.3.3), we also estimate risks associated with $PM_{2.5}$ 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 $PM_{2.5}$ exposures. Our consideration of estimated risks focuses on addressing the following policy-relevant questions:

- **What are the estimated $PM_{2.5}$-associated health risks for air quality just meeting the current primary $PM_{2.5}$ 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 $PM_{2.5}$-associated health risks combines concentration-response functions from epidemiologic studies with ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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.
  - *$PM_{2.5}$ 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 $PM_{2.5}$ standards. Inclusion of such areas in

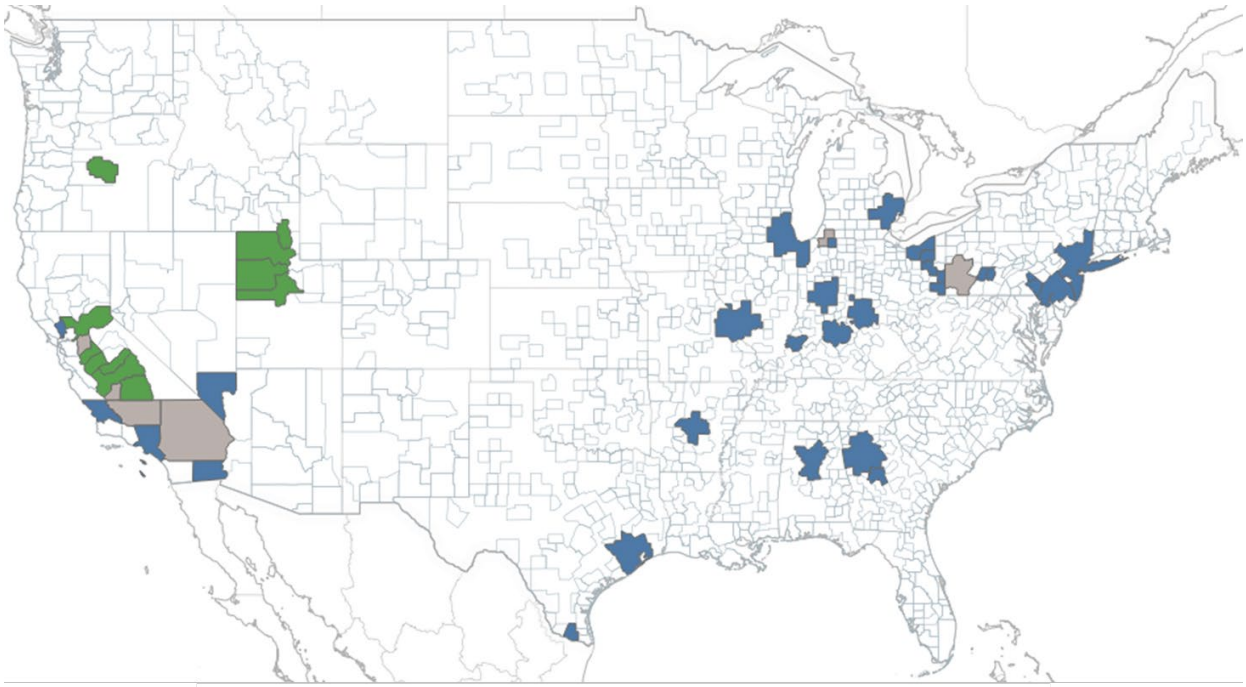
the risk assessment necessitates an upward adjustment to $PM_{2.5}$ air quality concentrations in order to simulate just meeting the current standards. Given uncertainty in how such increases could potentially occur, we select areas (i.e., CBSAs[54]) requiring either a downward adjustment to air quality or a relatively modest upward adjustment (i.e., no more than 2.0 $\mu g/m^3$ for the annual standard and 5 $\mu g/m^3$ 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,[55] 11 study areas where just meeting the current standards is controlled by the daily standard,[56] and 6 areas where the controlling standard differed depending on the air quality adjustment approach (Figure 3-10). [57]

[54] 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.

[55] 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.

[56] 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.

[57] In these 6 areas, the controlling standard depended on the air quality adjustment method used and/or the standard scenarios evaluated.



Number of Urban Study Areas (CBSAs)	Controlling Standard	Population (≥30 years old)
30	Annual (Blue)	~50M
11	Daily (Green)	~4M
6	Mixed (Grey)	~5M
Total: 47		**~60M**

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 $PM_{2.5}$ exposures and (b) total mortality associated with short-term $PM_{2.5}$ 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 draft ISA (U.S. EPA, 2018).
- **Concentration-response functions**: Concentration-response functions used in this risk assessment are from large, multicity U.S. epidemiologic studies that evaluate $PM_{2.5}$ 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 $PM_{2.5}$-associated risk.

Epidemiology Study	Study Population[a]	Age Range (years)	Mortality Categories Covered
Long-term mortality studies			
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
Short-term mortality			
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

[a]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.

- **$PM_{2.5}$ air quality scenarios evaluated**: We first estimate health risks associated with air quality adjusted to simulate "just meeting" the current primary $PM_{2.5}$ standards (i.e., the annual standard with its level of 12.0 $\mu g/m^3$ and the 24-hour standard with its level of 35 $\mu g/m^3$). We additionally evaluate the potential for alternative annual standards with levels of 9.0, 10.0 and 11.0 $\mu g/m^3$ 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 $PM_{2.5}$-associated health risks by limiting the $PM_{2.5}$ 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 3.4.2.4.2). 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 $\mu g/m^3$.[58]
- **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 $\mu g/m^3$ (annual) and 30 $\mu g/m^3$ (24-hour). The air quality modeling employs a hybrid approach that combines CMAQ-modeled surfaces[59] and ambient monitoring data to generate ambient $PM_{2.5}$ estimates for 2015 on a national grid with 12-km horizontal resolution (downscaler). The modeled 2015 $PM_{2.5}$ concentrations were then adjusted using one of two approaches[60] for each air quality scenario (discussed in detail in Appendix C, section C.1.4):

[58] We also estimate population risks for recent (i.e., unadjusted) ambient $PM_{2.5}$ concentrations (Appendix C).

[59] https://www.epa.gov/cmaq

[60] These two modeling approaches provided sensitivity analyses on key aspects of the HHRA and are not additive.

- *Reductions in primarily-emitted $PM_{2.5}$ (Pri-PM)*: This approach simulates air quality scenarios of interest by preferentially adjusting modeled directly emitted PM.[61]
- *Reductions in secondarily produced $PM_{2.5}$ (Sec-PM)*: This approach simulates air quality scenarios of interest by preferentially adjusting modeled SO_2 and NO_X precursor emissions to simulate changes in secondarily formed $PM_{2.5}$.[62]

- **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 $\mu g/m^3$) and 9.0 $\mu g/m^3$ (i.e., extrapolated from 12.0 and 10.0 $\mu g/m^3$), 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.

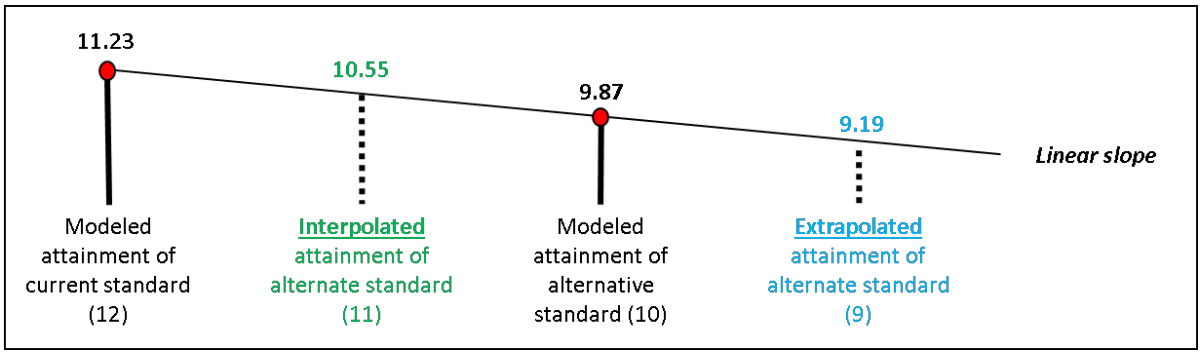


Figure 3-11. Illustration of approach to adjusting air quality to simulate just meeting annual standards with levels of 11.0 and 9.0 $\mu g/m^3$.

- **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).

[61] In locations for which air quality scenarios cannot be simulated by adjusting modeled directly emitted PM alone, modeled SO_2 and NO_X precursor emissions are additionally adjusted to simulate changes in secondarily formed $PM_{2.5}$ (Appendix C, section C.1.4).

[62] 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 $PM_{2.5}$-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*.[63] 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 $PM_{2.5}$ exposure.[64] In addition to tables, we also provide figures to illustrate how risks are distributed across annual average ambient PM_{25} concentrations. Figures present results for IHD mortality associated with long-term $PM_{2.5}$ 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 $PM_{2.5}$ standard is controlling (section 3.3.2.2), and the subset of 11 areas for which the 24-hour $PM_{2.5}$ 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 $PM_{2.5}$ 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^3)" 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 $\mu g/m^3$, is controlling. Risk estimates presented in the column labeled "Alternative 24-hour Standard (30 ug/m^3)" reflect the reductions estimated in the subset of study areas for which the alternative 24-hour standard,

[63] *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 $PM_{2.5}$-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.

[64] In other words, the percent of the effect associated with $PM_{2.5}$ exposure. For example, risk results estimate that 13-14% of all IHD mortality in 2015 was associated with $PM_{2.5}$ exposure (Table 3-5).

with a level of 30 μg/m^3, 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 $PM_{2.5}$-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/m^3)	CS (12/35) % of baseline**	Alternative Standard Absolute Risk	
					Alternative Annual (10 μg/m^3)	Alternative 24-hr (30 μg/m^3)
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 $PM_{2.5}$-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^3)**	CS-AS 24-hr Standard (30 µg/m^3)**	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 $PM_{2.5}$ standards
  - Long-term $PM_{2.5}$ 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.[65]
  - Short-term $PM_{2.5}$ 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

[65] 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 $\mu g/m^3$ or an alternative 24-hour standard with a level of 30 $\mu g/m^3$ (Table 3-6).[66]
- 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 $PM_{2.5}$-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 $PM_{2.5}$ 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 $\mu g/m^3$, 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.[67]

[66] In most study areas, the risk reductions presented for an annual standard with a level of 10.0 $\mu g/m^3$ reflect the difference between air quality with a maximum three-year design value of 12.0 $\mu g/m^3$ and air quality with a maximum three-year design value of 10.0 $\mu g/m^3$. Similarly, in most study areas, the risk reduction presented for a 24-hour standard with a level of 30 $\mu g/m^3$ reflects the difference between air quality with a maximum three-year design value of 35 $\mu g/m^3$ and air quality with a maximum three-year design value of 30 $\mu g/m^3$. However, in a small number of study areas, the "starting concentration" for the annual standard are below 12.0 $\mu g/m^3$ (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 $\mu g/m^3$ (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.

[67] As noted above, Figure 3-12 and Figure 3-13 present estimates of IHD mortality associated with long-term $PM_{2.5}$ exposures, based on the study by Jerrett et al. (2016).

Table 3-7. Estimates of $PM_{2.5}$-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 Standad Absolute Risk (12/35 µg/m^3)	CS (12/35 µg/m3) % of baseline**	Alternative Annual Standard (absolute risk)		
					11 µg/m^3	10 µg/m^3	9 µg/m^3
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 $PM_{2.5}$-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/m^3	12-10 µg/m^3	12-9 µg/m^3	12-11 µg/m^3	12-10 µg/m^3	12-9 µg/m^3
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.

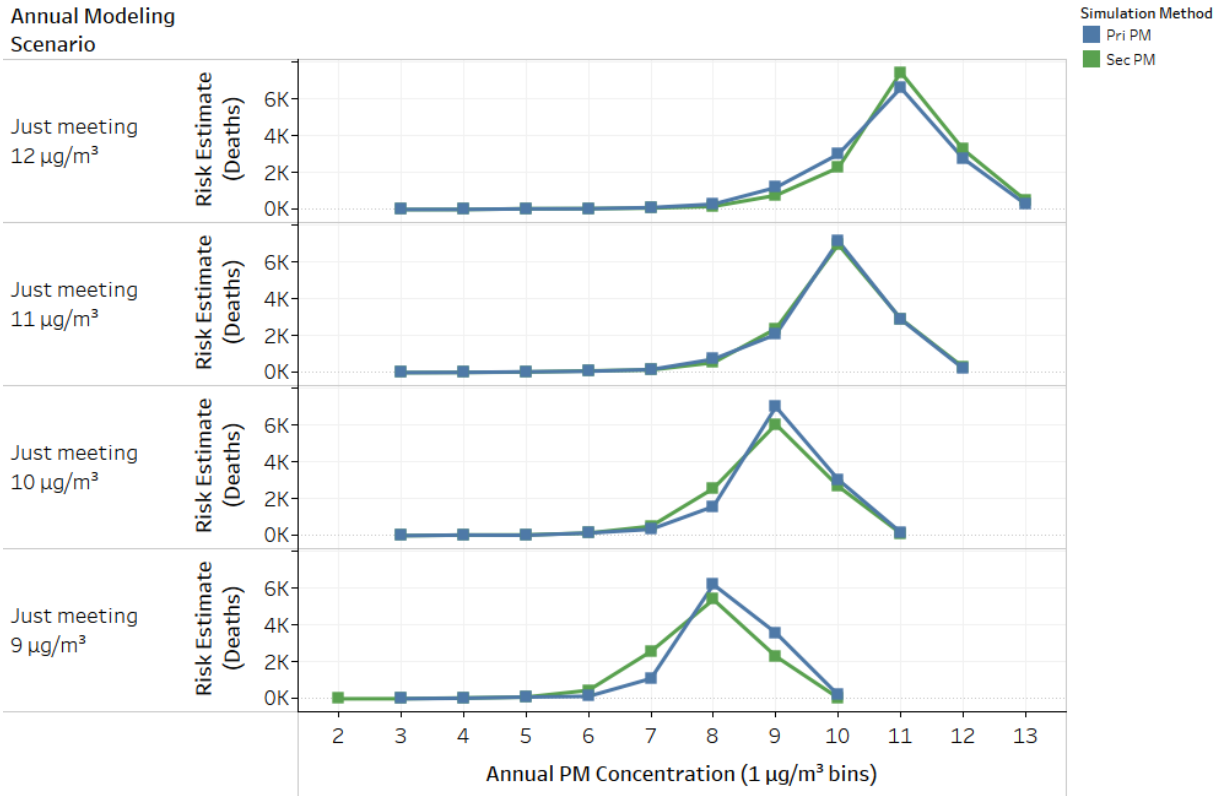


Figure 3-12. Distribution of absolute risk estimates ($PM_{2.5}$-associated mortality) for the current and alternative annual standards for the subset of 30 urban study areas where the annual standard is controlling (blue and green lines represent the Pri-$PM_{2.5}$ and Sec-$PM_{2.5}$ estimates, respectively).[68]

[68] In Figure 3-12, risk estimates are rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m³ includes risk occuring 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 µg/m³ bins)												Total
		2	3	4	5	6	7	8	9	10	11	12	13	
12-11 µg/m³	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 µg/m³	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 µg/m³	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 µg/m³) and alternative annual standards with levels of 11.0, 10.0, and 9.0 µg/m³ for the subset of 30 urban study areas where the annual standard is controlling.[69]

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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations from 10 to 12 µg/m³ (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 µg/m³; 14 to 18% reduction for a level of 10.0 µg/m³; 21 to 27% reduction for a level of 9.0 µg/m³) (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 $PM_{2.5}$ concentrations. Specifically, for air quality adjusted to simulate just meeting an annual standard with a level of 11.0 µg/m³, the majority of risk reduction occurs in grid cells with ambient $PM_{2.5}$ concentrations between 9 and 11 µg/m³; for air quality adjusted to simulate just meeting an annual standard with a level of 10.0 µg/m³, the majority of risk reduction occurs in grid cells with ambient $PM_{2.5}$ concentrations between 8 and 10 µg/m³; and for air quality adjusted to simulate just meeting an annual standard with a level of 9.0 µg/m³, the majority of risk reduction occurs in grid cells with ambient $PM_{2.5}$ concentrations between 7 and 9 µg/m³ [70] (Figure 3-13).

[69] Risks are presented as integers rounded to three significant digits and aggregated into 1 µg/m³ bins. Bins begin at the whole number value indicated and include values up to, but not including, the next whole number (e.g., risk occuring 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.

[70] Compared to adjusting primary $PM_{2.5}$ emissions, adjustment of PM precursor emissions resulted in substantially larger estimated risk reductions at 7 µg/m³.

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 $PM_{2.5}$ 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 $\mu g/m^3$ is associated with reductions in estimated risk of 14 to 18%.

Table 3-9. Estimates of $PM_{2.5}$-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. (2017) 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 $\mu g/m^3$ (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 $PM_{2.5}$ exposure and all-cause mortality based on effect estimates from Ito et al. (2013) versus long-term $PM_{2.5}$ 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 $PM_{2.5}$ 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.[71] Below, we identify those sources of uncertainty given at least a medium classification for the potential magnitude of impact on risk estimates. Refer to Appendix C, Table C-31 for additional details.
    - Simulating attainment of standard levels using the air quality modeling approach (medium)
    - Simulation of attainment of the alternative annual standards with levels of 9.0 and 11.0 $\mu g/m^3$ using linear interpolation/extrapolation (medium)
    - Representing population-level exposure with a 12 x 12 km grid-cell spatial framework in the context of modeling both short-term and long-term $PM_{2.5}$ exposure and mortality risk (medium-high and medium, respectively)
    - Shape of the concentration-response relationship for $PM_{2.5}$ exposures and mortality at low ambient PM concentrations (medium-high)

Most of the uncertainties summarized above, and in Appendix C (section C.3), are expected to impact absolute risk estimates similarly across the air quality scenarios evaluated.[72]

[71] As noted in Appendix C, section C.3, the classification of the magnitude of impact for sources of uncertainty included in this qualitative analysis 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).

[72] Exceptions are uncertainties related to the air quality adjustment approaches used, which can differ between model-based adjustments and interpolation/extrapolation-based adjustments.

Thus, while such uncertainties are key considerations when interpreting estimates of absolute risk, they are essentially held constant between air quality scenarios. That is, to the extent particular aspects of the approach tend to bias $PM_{2.5}$ risk estimates either high or low (Appendix C, sections C.3.1 and C.3.2), we expect risk estimates for the various air quality scenarios to be similarly impacted. As a result, compared to estimates of absolute risk, we have greater confidence in the risk reductions estimated between air quality scenarios (i.e. between the current and alternative standards).

3.4 PRELIMINARY CONCLUSIONS ON THE PRIMARY $PM_{2.5}$ STANDARDS

This section describes our preliminary conclusions regarding the adequacy of the current primary $PM_{2.5}$ standards (section 3.4.1) and regarding potential alternatives for consideration (section 3.4.2). As described more fully in section 3.1.2, our approach to reaching preliminary conclusions is based on considering the EPA's assessment of the current scientific evidence for health effects attributable to $PM_{2.5}$ exposures (discussed in detail in U.S. EPA, 2018), quantitative assessments of $PM_{2.5}$-associated health risks, and analyses of $PM_{2.5}$ air quality. In the final PA, such 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 $PM_{2.5}$ standards will reflect the judgments of the Administrator as to what weight to place on the various types of information.

3.4.1 Current Standards

We initially consider the adequacy of the current primary $PM_{2.5}$ standards. These considerations are 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 $PM_{2.5}$ standards?**

In answering this question, we consider the nature of the health effects reported to occur following short- or long-term $PM_{2.5}$ 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 $PM_{2.5}$ exposures shown to cause effects and the ambient concentrations in locations where $PM_{2.5}$ health effect associations have been reported (section 3.2.3); and estimates of $PM_{2.5}$-associated health risks for air quality adjusted to simulate just

meeting the current annual and 24-hour primary $PM_{2.5}$ standards (section 3.3). These considerations, and our preliminary conclusions on the current primary $PM_{2.5}$ standards, are summarized below.

As an initial matter, we note the longstanding body of health evidence supporting relationships between $PM_{2.5}$ exposures (short- and long-term) and mortality or serious morbidity effects. The evidence available in this review (i.e., assessed in U.S. EPA, 2018 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 $PM_{2.5}$ 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, $PM_{2.5}$ health effect associations. Such studies report associations between estimated $PM_{2.5}$ 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 strengthens support for potential biological pathways through which $PM_{2.5}$ exposures could lead to the effects reported in epidemiologic studies. This includes evidence from controlled human exposure and animal toxicological studies reporting cardiovascular effects and animal studies reporting respiratory, nervous system, and lung cancer-related effects.

Epidemiologic studies report $PM_{2.5}$ health effect associations with mortality and/or morbidity in a variety of populations, including in studies examining substantial portions of the U.S. population and studies examining populations and lifestages that may be at comparatively higher risk of experiencing a $PM_{2.5}$-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. These studies indicate that $PM_{2.5}$ health effect associations are robust across approaches to estimating $PM_{2.5}$ exposures, across statistical models (including copollutants models), and across exposure windows. Recent "accountability" studies additionally document that declines in ambient $PM_{2.5}$ 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 $PM_{2.5}$ health effect associations observed 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. Studies that examine the shapes of concentration-response functions over the full distribution of ambient $PM_{2.5}$ concentrations have not identified

a threshold concentration, below which associations no longer exist (U.S. EPA, 2018, section 1.5.3). While such analyses are complicated by the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA, 2018, section 1.5.3), several studies additionally report positive and statistically significant associations in analyses restricted to annual average $PM_{2.5}$ exposures below 12 $\mu g/m^3$ (Di et al., 2017b) and 10 $\mu g/m^3$ (Shi et al., 2016), or to daily exposures below 25 $\mu g/m^3$ (Di et al., 2017a) and 30 $\mu g/m^3$ (Shi et al., 2016).

These and other recent studies provide support for health effect associations at lower ambient $PM_{2.5}$ 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 $PM_{2.5}$ air quality distributions with overall mean concentrations at or above 12.8 $\mu g/m^3$ (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 $PM_{2.5}$ concentrations (i.e., Figure 3-7 and Figure 3-8). These key studies indicate such associations consistently for distributions with long-term mean $PM_{2.5}$ concentrations at or above 8.1 $\mu g/m^3$ (8.2 $\mu g/m^3$ based on studies that use monitors alone to estimate $PM_{2.5}$ exposures), with the large majority (and all but one key U.S. study) reporting overall mean $PM_{2.5}$ concentrations at or above 9.6 $\mu g/m^3$ (10.7 $\mu g/m^3$ based on studies that use monitors alone). Air quality distributions with such low mean concentrations are likely to be allowed by the current $PM_{2.5}$ standards, based on analyses of the relationships between maximum annual $PM_{2.5}$ 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).[73]

We also consider what key epidemiologic studies may indicate for the current standards by calculating values similar to $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations, such "pseudo-design values" also have the advantage of being consistently calculated across key studies, regardless of how the studies themselves estimate $PM_{2.5}$ exposures (e.g., averaging across monitors, predictions from hybrid modeling approaches).

For some key studies that report positive and statistically significant $PM_{2.5}$ health effect associations, substantial portions of study area populations (e.g., > 50% or 75%) lived in

[73] Given that the annual standard is the controlling standard across much of the U.S. (e.g., see section 3.3), the $PM_{2.5}$ air quality distributions that occur in most locations meeting the current annual $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposure estimates (e.g., below 12 $\mu g/m^3$ in Di et al., 2017b; below 10 $\mu g/m^3$ in Shi et al., 2016), particularly given that the excluded exposure estimates account for about half of the deaths in the entire cohort.[74] Thus, analyses of $PM_{2.5}$ pseudo-design values support the occurrence of positive and statistically significant $PM_{2.5}$ 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 $PM_{2.5}$ standards. The risk assessment estimates that the current primary $PM_{2.5}$ standards could allow a substantial number of 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 up to about 50,000 total $PM_{2.5}$-related deaths, including almost 20,000 ischemic heart disease deaths, in a single year. While the absolute numbers of estimated $PM_{2.5}$-associated deaths vary across endpoints, populations, and concentration-response functions, the general magnitude of risk estimates supports the potential for impacts of public health concern in locations meeting the current primary $PM_{2.5}$ standards. This is particularly the case given that the large majority of $PM_{2.5}$-associated deaths for air quality just meeting the current standards are estimated at annual average $PM_{2.5}$ concentrations from about 10 to 12 $\mu g/m^3$. These annual average $PM_{2.5}$ 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 $PM_{2.5}$ health effect associations.

Based on the information summarized above, and discussed in more detail in sections 3.2 and 3.3 of this draft PA, we particularly note the following in reaching preliminary conclusions on the current primary $PM_{2.5}$ standards:

- There is a long-standing body of strong health evidence demonstrating relationships between long- or short-term $PM_{2.5}$ exposures and a variety of outcomes, including mortality and serious morbidity effects. Studies published since the last review have reduced key

[74] $PM_{2.5}$ effect estimates in these restricted analyses are slightly larger than in those based on the entire cohort.

uncertainties and broadened our understanding of the health effects that can result from exposures to $PM_{2.5}$.

- Recent U.S. and Canadian epidemiologic studies provide support for generally positive and statistically significant health effect associations across a broad range of ambient $PM_{2.5}$ 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 $PM_{2.5}$ standards.
- Analyses of $PM_{2.5}$ 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 $PM_{2.5}$ standards could allow a substantial number of $PM_{2.5}$-associated deaths in the U.S. The large majority of these estimated deaths are associated with the annual average $PM_{2.5}$ 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 $PM_{2.5}$ standards.

In contrast to this preliminary conclusion, a conclusion that the current primary $PM_{2.5}$ 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 $PM_{2.5}$ air quality distributions likely to have been allowed by the current primary standards, or on the $PM_{2.5}$ 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 $PM_{2.5}$ exposures could cause serious health effects increases as the ambient concentrations being considered fall farther below the $PM_{2.5}$ exposure concentrations shown to cause effects in experimental studies. In the current review, such studies generally examine the occurrence of $PM_{2.5}$-attributable effects following exposures to $PM_{2.5}$ concentrations well-above those likely to occur in the ambient air in areas meeting the current primary $PM_{2.5}$ 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 accountability studies that document improving health with declining $PM_{2.5}$. In the current review, such studies evaluate air quality improvements with "starting" mean $PM_{2.5}$ concentrations (i.e., prior to the reductions being evaluated) from about 13 to > 20 $\mu g/m^3$ (i.e., Table 3-3).[75]

[75] As noted above, these retrospective studies tend to include data from earlier time periods where ambient $PM_{2.5}$ concentrations in the U.S. were considerably higher than they are at present.

- 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 $PM_{2.5}$-associated mortality risks (section 3.3.2.4).

The considerations and preliminary conclusions discussed above are intended to inform the Administrator's judgments regarding the current primary $PM_{2.5}$ standards. In presenting these considerations and preliminary 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 $PM_{2.5}$ 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 the final PA will seek to provide information on the range of policy options that could be supported by the scientific information, and given our preliminary conclusion (noted above) that the evidence and information can reasonably be viewed as calling into question the adequacy of the current primary $PM_{2.5}$ standards, in the next section we additionally consider support for potential alternative standards.

3.4.2 Potential Alternative Standards

In this section, we consider the potential alternative primary $PM_{2.5}$ 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 $PM_{2.5}$ standards: indicator, averaging time, form, and level. The sections below discuss our consideration of these elements, and our preliminary 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 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 $PM_{2.5}$ standard.

3.4.2.1 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposures, the Agency retained $PM_{2.5}$ mass as the indicator for fine particles and did not supplement the $PM_{2.5}$ 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 $PM_{2.5}$ exposures (U.S. EPA, 2018). 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 $PM_{2.5}$ mass (U.S. EPA, 2018, section 1.5.4). The draft ISA specifically notes that the results of recent studies confirm and further support the conclusion of the 2009 ISA that many $PM_{2.5}$ 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 $PM_{2.5}$ mass (U.S. EPA, 2018, 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 $PM_{2.5}$ mass as a whole. As discussed in the draft 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, 2018, section

1.4.3). Thus, for reasons similar to those discussed in the last review (78 FR 3121 to 3123, January 15, 2013), we reach the preliminary conclusion that the available information continues to support the $PM_{2.5}$ mass-based indicator and remains too limited to support a distinct standard for any specific $PM_{2.5}$ component or group of components, and too limited to support a distinct standard for the ultrafine fraction.

3.4.2.2 Averaging Time

In 1997, the EPA initially set an annual $PM_{2.5}$ standard to protect against health effects associated with both long- and short-term $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ exposure durations. Epidemiologic studies continue to provide strong support for health effects associated with both long- and short-term $PM_{2.5}$ exposures based on annual (or multiyear) and 24-hour $PM_{2.5}$ averaging periods, respectively.

With regard to short-term exposures in particular, a smaller number of epidemiologic studies examine associations between sub-daily $PM_{2.5}$ exposures and respiratory effects, cardiovascular effects, or mortality. Compared to 24-hour $PM_{2.5}$ exposure estimates, associations with sub-daily estimates are less consistent and, in some cases, smaller in magnitude (U.S. EPA, 2018, 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 draft 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, 2018, section 1.5.2.1).

Additionally, while recent controlled human exposure studies provide consistent evidence for cardiovascular effects following $PM_{2.5}$ 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 $PM_{2.5}$ exposures, beyond that provided by the current primary standards.

Drawing from the evidence assessed in the draft ISA, and the observations noted above, we reach the preliminary 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 $PM_{2.5}$ standards with these averaging times, when coupled with appropriate forms and levels, can protect against the range of long- and short-term $PM_{2.5}$ 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 $PM_{2.5}$ exposures.

3.4.2.3 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 preliminary 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 $PM_{2.5}$ standard to protect against health effects associated with both long- and short-term $PM_{2.5}$ exposures and a 24-hour standard to provide supplemental protection, particularly against the short-term exposures to "peak" $PM_{2.5}$ concentrations that can occur in some areas (62 FR 38667 to 38668, July 18, 1997). The EPA established the form of the annual $PM_{2.5}$ 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 $PM_{2.5}$ standard as the 98^{th} 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).[76]

In the last review, the EPA's consideration of the form of the annual $PM_{2.5}$ standard again included a focus on the issue of spatial averaging. An analysis of air quality and population demographic information indicated that the highest $PM_{2.5}$ 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 minority populations and populations with lower SES. The Administrator concluded that public health would not be protected with an adequate margin of

[76] 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).

safety in all locations, as required by law, if disproportionately higher $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard. The Agency recognized that the existing 98th percentile form for the 24-hour standard was originally selected to provide a balance between limiting the occurrence of peak 24-hour $PM_{2.5}$ 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 98th percentile $PM_{2.5}$ concentration, compared to the 99th percentile (U.S. EPA, 2011, Figure 2-2, p. 2-62). Thus, the Administrator concluded that it was appropriate to retain the 98th percentile form for the 24-hour $PM_{2.5}$ 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 $PM_{2.5}$ 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) $PM_{2.5}$ 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 $PM_{2.5}$ exposures around these means of the $PM_{2.5}$ air quality distribution. In addition, controlled human exposure studies provide evidence for health effects following single short-term $PM_{2.5}$ 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 98th percentile form for a 24-hour standard that is meant to provide a balance between limiting the occurrence of such peak 24-hour $PM_{2.5}$ concentrations and identifying a stable target for risk management programs. Thus, when the information summarized above is taken together, we reach the preliminary conclusion that it is appropriate in the current review to consider retaining the forms of the current annual and 24-hour $PM_{2.5}$ standards, in conjunction with a revised level as discussed below.

3.4.2.4 Level

With regard to level, we specifically address the following policy-relevant question:

- **For primary $PM_{2.5}$ 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 $PM_{2.5}$ in ambient air?**

In answering this question, we consider key epidemiologic studies that evaluate associations between $PM_{2.5}$ air quality distributions and mortality or morbidity, controlled human exposure studies examining effects following short-term $PM_{2.5}$ exposures, air quality analyses that help to place these studies into a policy-relevant context, and the risk assessment estimates of $PM_{2.5}$-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 98th 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 $PM_{2.5}$ air quality designed to meet an annual standard would likely result not only in lower short- and long-term $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98th percentile form, would result not only in fewer and lower peak 24-hour $PM_{2.5}$ concentrations, but also in lower average $PM_{2.5}$ 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 98th percentile form) is more directly tied to short-term peak $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that comprise the middle portion of the air quality distribution, affording protection against the daily and annual $PM_{2.5}$ exposures that strongly support associations with the most serious $PM_{2.5}$-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 $PM_{2.5}$ standard as the principle means of providing increased public health protection against the bulk of the distribution of short- and long-term $PM_{2.5}$ 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 98th percentile form, primarily as a means of providing supplemental protection against the short-term exposures to peak $PM_{2.5}$ concentrations that can occur in some areas (e.g., those with strong contributions from local or seasonal sources), even when overall mean $PM_{2.5}$ 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 $PM_{2.5}$ concentrations reported in U.S. or Canadian epidemiologic studies, (2) the relationships between long-term mean $PM_{2.5}$ concentrations and annual design values in U.S. CBSAs, (3) the $PM_{2.5}$ pseudo-design values in study locations, (4) the $PM_{2.5}$ exposures shown to cause effects in controlled human exposure studies, and (5) estimated $PM_{2.5}$-associated risks.

(1) Long-Term Mean $PM_{2.5}$ 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 $PM_{2.5}$ concentrations at and above 8.1 $\mu g/m^3$ (8.2 $\mu g/m^3$ based on studies that use monitors alone to estimate $PM_{2.5}$ exposures), with mean concentrations at or above 9.6 $\mu g/m^3$ in most key studies (10.7 $\mu g/m^3$ based on studies that use monitors alone to estimate $PM_{2.5}$ exposures). The ranges of ambient $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. Specifically, two key studies report positive associations between short-term $PM_{2.5}$ exposures and emergency room visits based on cities in Ontario, Canada (Weichenthal et al., 2016b and c), with overall mean $PM_{2.5}$ concentrations around 7.0 $\mu g/m^3$ (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 $PM_{2.5}$ exposure estimates. This study does not report the overall mean $PM_{2.5}$ concentrations in restricted analyses, though such means are presumably somewhat below those based on the overall cohort (i.e., 8.1 and 8.2 $\mu g/m^3$).

(2) Relationships between long-term mean $PM_{2.5}$ concentrations and annual design values (section 3.2.3.3; Appendix B, section B.7)

- Areas meeting a particular annual $PM_{2.5}$ standard would be expected to have average $PM_{2.5}$ 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 $PM_{2.5}$ design values are often 10% to 20% higher than long-term mean $PM_{2.5}$ concentrations (Appendix B, Figure B-7; Table B-8).

(3) $PM_{2.5}$ Pseudo-Design Values in Study Locations (section 3.2.3.2.2 and Appendix B, Figure B-9)

- For most key epidemiologic studies with $PM_{2.5}$ pseudo-design values available, about 25% or more of study area populations lived in locations likely to have met the current primary $PM_{2.5}$ 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 $\mu g/m^3$ correspond to 25^{th} 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 $\mu g/m^3$ correspond to the 25^{th} 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 $\mu g/m^3$ correspond to 50^{th} 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 $\mu g/m^3$ correspond to 50^{th} 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 $\mu g/m^3$ corresponds to the 75^{th} percentile of the study area population (i.e., 75% of the study area population lives in locations with pseudo-design values < 11.0 $\mu g/m^3$). For the Canadian studies with the lowest ambient $PM_{2.5}$ concentrations, annual pseudo-design values from 8.4 to 8.6 $\mu g/m^3$ correspond to 75^{th} percentiles of the study area populations (or health events).

(4) $PM_{2.5}$ 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 $PM_{2.5}$ exposures (U.S. EPA, 2018, Chapter 6), the $PM_{2.5}$ 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) $PM_{2.5}$-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 $\mu g/m^3$ could reduce $PM_{2.5}$-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 $\mu g/m^3$, 14 to 18% for a level of 10.0 $\mu g/m^3$, and 21 to 27% for a level of 9.0 $\mu g/m^3$. For each of these standards, most of the risk remaining is estimated at annual average $PM_{2.5}$ concentrations that fall somewhat below the standard level.

- Risk reductions estimated for an alternative 24-hour standard with a level of 30 $\mu g/m^3$ 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 $\mu g/m^3$). In those few study areas for which risk reductions are estimated upon just meeting an alternative 24-hour standard with a level of 30 $\mu g/m^3$, 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 $PM_{2.5}$ 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 draft 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 $PM_{2.5}$ 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.

3.4.2.4.1 Alternative Annual Standard Levels

As discussed above, the degree to which particular alternative annual standard levels below 12.0 $\mu g/m^3$ 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 $\mu g/m^3$ could be supported to the extent weight is placed on the following:

- Setting a standard expected to maintain the $PM_{2.5}$ 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 $PM_{2.5}$ with overall mean concentrations at or above 9.6 $\mu g/m^3$, while a few studies reporting such associations examine distributions with overall mean concentrations just above 8.0 $\mu g/m^3$ (section 3.2.3.2.1) and (2) analyses of $PM_{2.5}$ air quality in CBSAs indicate that maximum annual $PM_{2.5}$ design values are often 10% to 20% higher than average $PM_{2.5}$ concentrations (i.e., averaged across space and over several years) suggesting that areas meeting a particular annual $PM_{2.5}$ standard would be expected to have average $PM_{2.5}$ 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^{th} 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

$\mu g/m^3$ would be expected to maintain ambient $PM_{2.5}$ 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 $PM_{2.5}$-associated health risks, such that a substantial portion of the risk reduction is estimated at annual average $PM_{2.5}$ concentrations ≥ about 8 $\mu g/m^3$ 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 $\mu g/m^3$ to < 12.0 $\mu g/m^3$, 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 $\mu g/m^3$ and on the small number of studies with 50^{th} percentile pseudo-design values below 10.0 $\mu g/m^3$, a standard higher than 10 $\mu g/m^3$ (but still below 12.0 $\mu g/m^3$) 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 $PM_{2.5}$ 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 $\mu g/m^3$, much of which is estimated at annual average $PM_{2.5}$ concentrations around 8 $\mu g/m^3$, might focus consideration on a standard level above 10 $\mu g/m^3$, where estimated risk reductions would occur at slightly higher concentrations.

A decision to not consider annual standard levels below 10.0 $\mu g/m^3$ 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 $PM_{2.5}$ air quality distributions with overall mean concentrations below 9.6 $\mu g/m^3$, and areas meeting a standard with a level of 10.0 $\mu g/m^3$ would generally be expected to have lower long-term mean $PM_{2.5}$ concentrations (and potentially around 8.0 $\mu g/m^3$ in some areas) (section 3.2.3.2.1; Appendix B, section B.7).
- There is increasing uncertainty in $PM_{2.5}$ 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 50th percentiles of study area populations (or health events) are ≥ about 10.0 μg/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 $PM_{2.5}$-associated mortality risk for standard levels below 10.0 μg/m^3, given that a substantial proportion of the risk reductions estimated for lower standard levels occur at annual average $PM_{2.5}$ concentrations below 8 μg/m^3, and thus below the lower end of the range of overall mean $PM_{2.5}$ 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 μg/m^3, and potentially as low as 8.0 μg/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 $PM_{2.5}$ concentrations (i.e., beyond those achieved by a standard with a level of 10.0 μg/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 μg/m^3 could be supported to the extent greater weight is placed on the following:

- The two key studies in Canada with overall mean $PM_{2.5}$ concentrations below 8.0 μg/m^3 and the potential for overall mean concentrations below 8.0 μg/m^3 in restricted analyses in a key U.S. study (section 3.2.3.2.1);
- The ambient $PM_{2.5}$ 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 25th percentiles of study area populations or health events for most studies, recognizing that the revised standard would be expected to maintain ambient $PM_{2.5}$ concentrations below the concentrations present during study periods for > ~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 $PM_{2.5}$-associated health risks estimated for a level of 9.0 μg/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 μg/m^3 to < 10.0 μg/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 μg/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 μg/m^3, on the potential for overall mean concentrations below 8.0 μg/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 $\mu g/m^3$, given that such risks, which were not quantified in the risk assessment, are likely to occur at annual average $PM_{2.5}$ concentrations largely below 8 $\mu g/m^3$ (i.e., below the mean concentrations in almost all key epidemiologic studies).

3.4.2.4.2 Alternative 24-Hour Standard Levels

We additionally evaluate the degree to which the evidence supports considering potential alternative levels for the 24-hour $PM_{2.5}$ standard, in conjunction with the current 98th 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations, recognizing that the 24-hour standard, with its 98th 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 98th percentile form) largely within the context of limiting short-term exposures to peak $PM_{2.5}$ concentrations. Compared to the annual standard, we recognize that the 24-hour standard is less likely to appropriately limit the more typical $PM_{2.5}$ 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 $PM_{2.5}$ 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, and the 24-hour standard as a means of providing supplemental protection against the short-term exposures to "peak" $PM_{2.5}$ 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 $PM_{2.5}$ 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 $\mu g/m^3$ would be estimated to lower $PM_{2.5}$-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 $PM_{2.5}$ design values at or below 12.0 $\mu g/m^3$ also have maximum 24-hour design values below 35 $\mu g/m^3$ (and below 30 $\mu g/m^3$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations are not disproportionately responsible for reported health effect associations.

- Controlled human exposure studies do provide evidence for health effects following single, short-term $PM_{2.5}$ exposures to concentrations that typically correspond to upper end of the $PM_{2.5}$ 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 $PM_{2.5}$ concentrations at and above 120 $\mu g/m^3$ (at and above 149 $\mu g/m^3$ for vascular impairment, the effect shown to be most consistent across studies), 2-hour ambient concentrations of $PM_{2.5}$ at monitoring sites meeting the current standards almost never exceed 32 $\mu g/m^3$. In fact, even the extreme upper end of the distribution of 2-hour $PM_{2.5}$ concentrations at sites meeting the current standards remains well-below the $PM_{2.5}$ exposure concentrations consistently shown to elicit effects (i.e., 99.9th percentile of 2-hour concentrations at these sites is 68 $\mu g/m^3$ during the warm season). Thus, available $PM_{2.5}$ controlled human exposure studies do not indicate the need for additional protection against exposures to peak $PM_{2.5}$ 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 preliminary conclusion that, in conjunction with a lower annual standard level intended to increase protection against short- and long-term $PM_{2.5}$ exposures broadly across the U.S., the evidence does not support the need for additional protection against short-term exposures to peak $PM_{2.5}$ concentrations. In particular, while epidemiologic studies do support the need to consider increasing protection against the typical 24-hour and annual $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations, the available evidence supports consideration of retaining the current 24-hour standard with its level of 35 $\mu g/m^3$.

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 $PM_{2.5}$ 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 $\mu g/m^3$ could be supported (either alone or in conjunction with a lower annual standard level). The risk assessment estimates that a level of 30 $\mu g/m^3$ 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 $PM_{2.5}$ pseudo-design values at or below about 30 $\mu g/m^3$ (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 $PM_{2.5}$ standards is adopted, the uncertainty inherent in using the 24-hour standard to increase protection against the broad distribution of $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that are directly targeted by the 24-hour standard (i.e., with its 98th 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 98th percentile form, to increase protection against the broad distribution of $PM_{2.5}$ air quality.

3.5 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 $PM_{2.5}$ standards. Important areas for future research include the following:

- Further elucidating the physiological pathways through which exposures to the $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations.
- Improving our understanding of the $PM_{2.5}$ concentration-response relationships near the lower end of the $PM_{2.5}$ 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 $PM_{2.5}$ exposures in epidemiologic studies, including how those uncertainties may vary across space and time, and over the $PM_{2.5}$ air quality distribution. Approaches to incorporating these uncertainties into quantitative estimates of $PM_{2.5}$ 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 $PM_{2.5}$-related effects than the general population and the potential for $PM_{2.5}$ exposures to contribute to the development of underlying conditions that may then confer higher risk of $PM_{2.5}$-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 $PM_{2.5}$ 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 $PM_{2.5}$ that is not captured by existing ambient monitors.

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4 REVIEW OF THE PRIMARY STANDARD FOR PM_{10}

This chapter presents key policy-relevant considerations and preliminary conclusions regarding the public health protection provided by the current primary PM_{10} standard. These considerations and preliminary 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_{10} standard against health effects associated with exposures to $PM_{10-2.5}$?**

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_{10}.

Section 4.1 summarizes the EPA's approach to reviewing the primary PM_{10} 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 draft ISA. Drawing from that consideration of the evidence, section 4.3 summarizes our preliminary conclusions regarding the adequacy of the current primary PM_{10} standard. Section 4.4 discusses areas for future research and data collection to improve our understanding of potential $PM_{10-2.5}$-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_{10} standard, with its level of 150 $\mu g/m^3$ and its one-expected-exceedance form on average over three years, to continue to provide public health protection against exposures to $PM_{10-2.5}$. 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 $PM_{10-2.5}$ (regardless of composition or source or origin), the extent to which a standard with a PM_{10} indicator can provide protection against exposures to $PM_{10-2.5}$, and the degree of public health protection provided by the existing PM_{10} 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 $PM_{10-2.5}$ and mortality or

morbidity in a large number of cities across North America, Europe, and Asia, encompassing a variety of environments where $PM_{10-2.5}$ 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_{10} indicator for a standard meant to protect against exposures to ambient $PM_{10-2.5}$, the Administrator noted that PM_{10} mass includes both coarse PM ($PM_{10-2.5}$) and fine PM ($PM_{2.5}$). As a result, the concentration of $PM_{10-2.5}$ allowed by a PM_{10} standard set at a single level declines as the concentration of $PM_{2.5}$ increases. Because $PM_{2.5}$ concentrations tend to be higher in urban areas than rural areas (e.g., Chan et al., 2018), the Administrator observed that a PM_{10} standard would generally allow lower $PM_{10-2.5}$ concentrations in urban areas than in rural areas. She judged it appropriate to maintain such a standard given that much of the evidence for $PM_{10-2.5}$ toxicity, particularly at relatively low particle concentrations, came from study locations where thoracic coarse particles were of urban origin, and given the possibility that $PM_{10-2.5}$ 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 $PM_{10-2.5}$ 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_{10} standard, based on the varying levels of $PM_{2.5}$ 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_{10} standard, with its level of 150 $\mu g/m^3$ 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 $PM_{10-2.5}$ than for $PM_{2.5}$. In particular, the strongest evidence for health effects attributable to $PM_{10-2.5}$ 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 $PM_{2.5}$, 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 $PM_{10-2.5}$ evidence and information raised questions as to whether additional public health improvements would be achieved by revising the existing PM_{10} 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 $PM_{2.5}$, was limited. Therefore, the extent to which $PM_{10-2.5}$ 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 $PM_{10-2.5}$ and mortality and morbidity reported in epidemiologic studies.

(3) Limitations in $PM_{10-2.5}$ monitoring data (i.e., limited data available from FRM/FEM sampling methods) and the different approaches used to estimate $PM_{10-2.5}$ concentrations across epidemiologic studies resulted in uncertainty in the ambient $PM_{10-2.5}$ concentrations at which the reported effects occur, increasing uncertainty in estimates of the extent to which changes in ambient $PM_{10-2.5}$ concentrations would likely impact public health.

(4) While $PM_{10-2.5}$ 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_{10} concentrations above those allowed by the current 24-hour PM_{10} standard.

(5) The composition of $PM_{10-2.5}$, and the effects associated with various components, were also key uncertainties in the available evidence. Without more information on the chemical speciation of $PM_{10-2.5}$, 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 $PM_{10-2.5}$ itself, as opposed to one or more co-occurring pollutants. This uncertainty reflected the relatively small number of $PM_{10-2.5}$ studies that had evaluated copollutant models, particularly copollutant models that included $PM_{2.5}$, and the very limited body of controlled human exposure evidence supporting the plausibility of $PM_{10-2.5}$-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_{10} standard against exposures to $PM_{10-2.5}$ 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_{10} standard. Thus, the Administrator concluded that the existing 24-hour PM_{10} standard, with its one-expected exceedance form on average over three years and a level of 150 $\mu g/m^3$, was requisite to protect public health with an adequate margin of safety against effects that have been associated with $PM_{10-2.5}$. In light of this conclusion, the EPA retained the existing PM_{10} standard.

4.1.2 Approach in the Current Review

As discussed above for $PM_{2.5}$ (section 3.2.1), in this draft 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, 2018). This approach focuses policy considerations and conclusions on health outcomes for which the evidence is strongest. Unlike for $PM_{2.5}$, the draft ISA does not identify any $PM_{10-2.5}$-related health outcomes for which the evidence supports either a "causal" or a "likely to be causal" relationship. Thus, for $PM_{10-2.5}$ this draft 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 $PM_{10-2.5}$-related health outcomes, compared to the evidence supporting "causal" or "likely to be causal" relationships for $PM_{2.5}$, our approach to evaluating the primary PM_{10} standard in this review is more limited than our approach to evaluating the primary $PM_{2.5}$ standards (discussed in Chapter 3). Specifically, our approach for PM_{10} 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 $PM_{10-2.5}$ health risks. The substantial uncertainty in such analyses, if they were to be conducted based on the currently available $PM_{10-2.5}$ health studies, would limit their utility for informing conclusions on the primary PM_{10} standard. Therefore, as discussed further below, we focus our evaluation of the primary PM_{10} standard on the overall body of evidence for $PM_{10-2.5}$-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_{10} standard will largely be public health policy judgments that will draw upon the scientific evidence for $PM_{10-2.5}$-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 draft ISA (U.S. EPA, 2018) 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 $PM_{10-2.5}$ 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_{10} 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 $PM_{10-2.5}$ exposures.

4.2.1 Nature of Effects

As noted above, for the heath outcome categories and exposure duration combinations evaluated, the draft ISA concludes that the evidence supports causality determinations for $PM_{10-2.5}$ 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, 2018, Table 1-5).

Table 4-1. Key Causality Determinations for $PM_{10-2.5}$ Exposures

Health Outcome	Exposure Duration	2009 PM ISA	2018 draft 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 draft 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, 2018, section 1.4.2, p. 1-40). For example, epidemiologic studies available in the last review relied on various methods to estimate $PM_{10–2.5}$ 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_{10} and $PM_{2.5}$ concentrations at co-located monitors, (2) calculating the difference between county-wide averages of monitored PM_{10} and $PM_{2.5}$ based on monitors that are not necessarily co-located, and (3) direct measurement of $PM_{10-2.5}$ using a dichotomous sampler (U.S. EPA, 2018, section 1.4.2). In the current review, more recent epidemiologic studies continue to use these approaches to estimate $PM_{10-2.5}$ concentrations. Additionally, some recent studies estimate long-term $PM_{10-2.5}$ exposures as the difference between PM_{10} and $PM_{2.5}$ 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 $PM_{10-2.5}$ concentrations have not been systematically evaluated (U.S. EPA, 2018, section 3.3.1.1), contributing to uncertainty regarding the spatial and temporal correlations in $PM_{10-2.5}$ concentrations across methods and in the $PM_{10-2.5}$ exposure estimates used in epidemiologic studies (U.S. EPA, 2018, section 2.5.1.2.3). Given the greater spatial and temporal variability of $PM_{10-2.5}$ and fewer $PM_{10-2.5}$ monitoring sites, compared to $PM_{2.5}$, this uncertainty is particularly important for the coarse size fraction.

Beyond uncertainty associated with $PM_{10-2.5}$ 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 $PM_{10-2.5}$ exposures also continue to contribute broadly to uncertainty in the $PM_{10-2.5}$ health evidence. Uncertainty related to potential confounding stems from the relatively small number of epidemiologic studies that have evaluated $PM_{10-2.5}$ 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 $PM_{10-2.5}$ exposures results from the small number of controlled human exposure and animal toxicology[1] studies that have evaluated the health effects of experimental $PM_{10-2.5}$ inhalation exposures. The evidence supporting the draft ISA's "suggestive" causality determinations for $PM_{10-2.5}$, 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 $PM_{10–2.5}$ 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 $PM_{10–2.5}$ exposure and total (nonaccidental) mortality, though results are inconsistent across studies (U.S. EPA, 2018, Table 11-11). The examination of copollutant models in these studies remains limited and, when included, $PM_{10–2.5}$ effect estimates are often attenuated after adjusting for $PM_{2.5}$ (U.S. EPA, 2018, Table 11-11). Across studies, $PM_{10–2.5}$ exposure concentrations are estimated using a variety of approaches, including direct measurements from dichotomous samplers, calculating the difference between PM_{10} and $PM_{2.5}$ concentrations measured at collocated monitors, and calculating difference of area-wide concentrations of PM_{10} and $PM_{2.5}$. 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, 2018, section 4.2.1.1 and Table 11-11). The draft ISA concludes that this uncertainty "reduces the confidence in the associations observed across studies" (U.S. EPA, 2018, p. 11-121). The draft ISA additionally concludes that the evidence for long-term $PM_{10–2.5}$ exposures and cardiovascular effects, respiratory morbidity, and metabolic disease provide limited biological plausibility for $PM_{10–2.5}$-related mortality (U.S. EPA, 2018, sections 11.4.1 and 11.4). Taken together, the draft ISA concludes that, "this body of evidence is

[1] Compared to humans, smaller fractions of inhaled $PM_{10-2.5}$ penetrate into the thoracic regions of rats and mice (U.S. EPA, 2018, section 4.1.6), contributing to the relatively limited evaluation of $PM_{10-2.5}$ exposures in animal studies.

suggestive, but not sufficient to infer, that a causal relationship exists between long-term $PM_{10-2.5}$ exposure and total mortality" (U.S. EPA, 2018 p. 11-121).

Short-term exposures

The 2009 ISA concluded that the evidence is "suggestive of a causal relationship between short-term exposure to $PM_{10–2.5}$ 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 $PM_{10–2.5}$ exposure and total (nonaccidental) mortality (U.S. EPA, 2018, Table 11-9). Although these studies contribute to increasing confidence in the $PM_{10–2.5}$-mortality relationship, the use of a variety of approaches to estimate $PM_{10-2.5}$ exposures continues to contribute uncertainty to the associations observed. In addition, the draft ISA notes that an analysis by Adar et al. (2014) indicates "possible evidence of publication bias, which was not observed for $PM_{2.5}$" (U.S. EPA, 2018, section 11.3.2, p. 11-102). Recent studies expand the assessment of potential copollutant confounding of the $PM_{10–2.5}$-mortality relationship and provide evidence that $PM_{10-2.5}$ associations generally remain positive in copollutant models, though associations are attenuated in some instances (U.S. EPA, 2018, section 11.3.4.1, Figure 11-28, Table 11-10). The draft ISA concludes that, overall, the assessment of potential copollutant confounding is limited due to the lack of information on the correlation between $PM_{10–2.5}$ 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, 2018, section 11.3.7). The draft ISA concludes that the evidence for $PM_{10-2.5}$-related cardiovascular and respiratory effects provides only limited support for the biological plausibility of a relationship between short-term $PM_{10–2.5}$ exposure and cardiovascular mortality (U.S. EPA, 2018, Section 11.3.7). Based on the overall evidence, the draft ISA concludes that, "this body of evidence is suggestive, but not sufficient to infer, that a causal relationship exists between short-term $PM_{10-2.5}$ exposure and total mortality" (U.S. EPA, 2018, p. 11-116).

4.2.1.2 Cardiovascular Effects

Long-term exposures

In the 2009 PM ISA, the evidence describing the relationship between long-term exposure to $PM_{10-2.5}$ 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 $PM_{10-2.5}$ on the cardiovascular system was lacking (U.S. EPA, 2018, section 6.4, p. 6-251).

The evidence relating long-term $PM_{10-2.5}$ 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 $PM_{10-2.5}$ concentrations (U.S. EPA, 2018, 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, 2018, Figure 6-34); stroke (U.S. EPA, 2018, Figure 6-35); atherosclerosis; venous thromboembolism (VTE); and blood pressure and hypertension (U.S. EPA, 2018, Section 6.4.6). $PM_{10-2.5}$ cardiovascular mortality effect estimates are often attenuated, but remain positive, in copollutants models that adjust for $PM_{2.5}$. For morbidity outcomes, associations are inconsistent in copollutant models that adjust for $PM_{2.5}$, NO_2, and chronic noise pollution (U.S. EPA, 2018, p. 6-272). The lack of toxicological evidence for long-term $PM_{10-2.5}$ exposures represents a substantial data gap (U.S. EPA, 2018, section 6.4.10), resulting in the draft ISA conclusion that "evidence from experimental animal studies is of insufficient quantity to establish biological plausibility" (U.S. EPA, 2018, p. 6-272). 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 $PM_{10-2.5}$ exposure and cardiovascular effects" (U.S. EPA, 2018, p. 6-273).

Short-term exposures

The 2009 ISA found that the available evidence for short-term $PM_{10-2.5}$ exposure and cardiovascular effects was "suggestive of a causal relationship." This conclusion was based on several epidemiologic studies reporting associations between short-term $PM_{10-2.5}$ 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 $PM_{10-2.5}$ 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, 2018, section 6.3.13).

The evidence for short-term $PM_{10-2.5}$ exposure and cardiovascular outcomes has expanded since the last review, though important uncertainties remain. The draft ISA notes that there are a small number of epidemiologic studies reporting positive associations between short-term

exposure to $PM_{10-2.5}$ and cardiovascular-related morbidity outcomes. However, there is limited evidence to suggest that these associations are biologically plausible, or independent of copollutant confounding. The draft ISA also concludes that it remains unclear how the approaches used to estimate $PM_{10-2.5}$ concentrations in epidemiologic studies may impact exposure measurement error. Taken together, the draft ISA concludes that "the evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term $PM_{10-2.5}$ exposures and cardiovascular effects" (U.S. EPA, 2018, p.6-250).

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 $PM_{10–2.5}$ 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 $PM_{10–2.5}$ 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 $PM_{10–2.5}$ exposure.

Recent epidemiologic findings consistently link $PM_{10–2.5}$ 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 $PM_{2.5}$ 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, 2018, Table 5-37). 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 $PM_{10–2.5}$ concentrations and the resulting uncertainty in the spatial and temporal variability in $PM_{10–2.5}$ concentrations compared to $PM_{2.5}$ (U.S. EPA, 2018, sections 2.5.1.2.3 and 3.3.1.1). Taken together, the draft ISA concludes that "the collective evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term $PM_{10-2.5}$ exposure and respiratory effects" (U.S. EPA, 2018, p. 5-266).

4.2.1.4 Cancer

Long-term exposures

In the last review, little information was available from studies of cancer following inhalation exposures to $PM_{10–2.5}$. Thus, the 2009 ISA determined the evidence was "inadequate to assess the relationship between long-term $PM_{10–2.5}$ exposures and cancer" (U.S. EPA, 2009). Since the 2009 ISA, the assessment of long-term $PM_{10–2.5}$ 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 $PM_{10–2.5}$ predictions that have not been validated by monitored $PM_{10–2.5}$ concentrations (U.S. EPA, 2018, sections 3.3.2.3 and 10.3.4). Relatively few experimental studies of $PM_{10–2.5}$ have been conducted, though available studies indicate that $PM_{10–2.5}$ 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, 2018, 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 draft ISA that, **"**the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{10–2.5}$ exposure and cancer**"** (U.S. EPA, 2018, p. 10-85).

4.2.1.5 Metabolic Effects

Long-term exposures

The 2009 ISA did not make a causality determination for $PM_{10-2.5}$-related metabolic effects. Since the last review, one epidemiologic study shows an association between long-term $PM_{10-2.5}$ exposure and incident diabetes, while additional cross-sectional studies report associations with effects on glucose or insulin homeostasis (U.S. EPA, 2018, 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, 2018, Tables 7-15 and 7-16). The evidence base to support the biological plausibility of metabolic effects following $PM_{10-2.5}$ 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, 2018, sections 7.4.1 and 7.4.3). Based on the expanded, though still limited evidence base, the draft ISA concludes that, "[o]verall, the evidence is suggestive of, but not sufficient to infer, a causal relationship between [long]-term $PM_{10–2.5}$ exposure and metabolic effects" (U.S. EPA, 2018, p. 7-61).

4.2.1.6 Nervous system effects

Long-term exposures

The 2009 ISA did not make a causality determination for $PM_{10-2.5}$-related nervous system effects. In the current review, newly available epidemiologic studies report associations between

$PM_{10-2.5}$ and impaired cognition and anxiety in adults in longitudinal analyses (U.S. EPA, 2018, Table 8-25, section 8.4.5). Associations of long-term exposure with neurodevelopmental effects are not consistently reported in children (U.S. EPA, 2018, 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, 2018, section 8.4.5), and for exposure measurement error, given the use of various model-based subtraction methods to estimate $PM_{10-2.5}$ concentrations (U.S. EPA, 2018, Table 8-25). In addition, there is only limited animal toxicological evidence supporting the biological plausibility of nervous system effects (U.S. EPA, 2018, sections 8.4.1 and 8.4.5). Overall, the draft ISA concludes that, "the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{10-2.5}$ exposure and nervous system effects (U.S. EPA, 2018, p. 8-77).

4.2.1.7 Preliminary Conclusions Drawn from the Evidence

Based on the evidence available in the current review, as assessed in the draft ISA (U.S. EPA, 2018) 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 $PM_{10-2.5}$ 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 $PM_{10-2.5}$-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 $PM_{10-2.5}$ 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 $PM_{10-2.5}$-related health effects has expanded, broadening our understanding of the range of health effects linked to $PM_{10-2.5}$ 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 $PM_{10-2.5}$ exposures in epidemiologic studies and the lack of systematic evaluation of these methods, together with the relatively high

spatial and temporal variability in ambient $PM_{10-2.5}$ concentrations and the small number of monitoring sites, results in uncertainty in exposure estimates;

- The limited number of studies that evaluate $PM_{10-2.5}$ 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 $PM_{10-2.5}$ health effect associations from co-occurring pollutants;
- The limited number of controlled human exposure and animal toxicology studies of $PM_{10-2.5}$ inhalation contributes to uncertainty in the biological plausibility of the $PM_{10-2.5}$-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 draft ISA that the evidence for the $PM_{10-2.5}$-related health effects discussed in this section is "suggestive of, but not sufficient to infer" causal relationships.

4.3 PRELIMINARY CONCLUSIONS ON THE ADEQUACY OF THE CURRENT STANDARD

Based on the $PM_{10-2.5}$ health evidence available in the current review, as assessed in the draft ISA and summarized above, 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_{10} standard against health effects associated with exposures to $PM_{10-2.5}$?**

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_{10} standard. We recognize that a final decision on the primary PM_{10} 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_{10} standard in the last review recognized the importance of maintaining some degree of protection against $PM_{10-2.5}$ exposures, given the evidence for $PM_{10-2.5}$-related health effects, but noted uncertainties in the potential public health implications of revising the existing PM_{10} standard. Regarding evidence for $PM_{10-2.5}$-related health effects, the decision noted that epidemiologic studies had reported positive associations between $PM_{10-2.5}$ and mortality or morbidity in cities across North America, Europe, and Asia, encompassing a variety of environments where $PM_{10-2.5}$ sources and composition are expected to vary widely. Although most of these studies examined $PM_{10-2.5}$ 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 $PM_{10-2.5}$, 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_{10} indicator given that the varying concentrations of $PM_{10-2.5}$ permitted in urban versus non-urban areas under a PM_{10} standard, based on the varying levels of $PM_{2.5}$ present (i.e., lower $PM_{10-2.5}$ concentrations allowed in urban areas, where $PM_{2.5}$ concentrations tend to be higher), appropriately reflected differences in the strength of $PM_{10-2.5}$ 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, 2018). 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 draft 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, 2018). Drawing from this information, we reach the preliminary 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 preliminary 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_{10} standard in order to protect against $PM_{10-2.5}$ exposures. Specifically, while the available evidence supports maintaining a PM_{10} standard to provide some measure of protection against $PM_{10-2.5}$ exposures, uncertainties in the evidence lead to questions regarding the potential public health implications of revising the existing PM_{10} standard. Thus, consistent with the approach taken in the last review, we reach the preliminary conclusions that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard and that evidence supports consideration of retaining the current standard in this review. As such, we have not evaluated alternative standards in this draft PA.

4.4 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 $PM_{10-2.5}$ (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 $PM_{10-2.5}$ 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 $PM_{2.5}$ 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 $PM_{10-2.5}$, given its less homogeneous atmospheric distribution compared to fine particles (U.S. EPA, 2018, section 1.2.1.5) and the relatively sparse $PM_{10-2.5}$ 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 $PM_{10-2.5}$ 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 $PM_{10-2.5}$ concentrations, including: (1) difference method with co-located monitors, (2) difference method with area-wide averages of monitored PM_{10} and $PM_{2.5}$, (3) difference method with area-wide averages of modeled PM_{10} and $PM_{2.5}$ or (4) direct measurement of $PM_{10-2.5}$ 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 $PM_{10-2.5}$ ambient concentration data have greatly increased since the 2009 ISA (U.S. EPA, 2018, section 2.5.1.3). Starting in 2011, $PM_{10-2.5}$ 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 $PM_{10-2.5}$ concentrations but have not used direct measurements from NCore or IMPROVE stations to evaluate health effects associations with $PM_{10-2.5}$ 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.

- Additional areas of interest for future research include:
    - Further evaluation of the potential for particular $PM_{10-2.5}$ 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 $PM_{10-2.5}$ concentrations.
    - Identifying novel populations that could be at-risk of $PM_{10-2.5}$-related health effects.
    - Modeling to estimate $PM_{10-2.5}$ 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 preliminary summary conclusions regarding the public welfare protection provide by the current secondary PM standards to protect against PM-related visibility impairment, climate effects, and materials effects. These considerations and preliminary 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 current suite of 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.[1] Preliminary conclusions regarding the public welfare protection provided by the current secondary PM standards are summarized in section 5.3. Section 5.4 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 $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$, and the 24-hour PM_{10} standard, with its level of 150 $\mu g/m^3$ (78 FR 3228, January 15, 2013). The EPA also retained the level, set at 15 $\mu g/m^3$, and averaging time of the annual $PM_{2.5}$ standard, while revising the form. With regard to the form of the annual $PM_{2.5}$ standard, the EPA removed the option for spatial averaging (78 FR

[1] 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.

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[2] 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. For example, the ISA and PA noted the spatial and temporal heterogeneity of PM components

[2] 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, 2018) 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).

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 $PM_{2.5}$ and PM_{10} secondary standards. With regard to the

secondary annual $PM_{2.5}$ standard, the Administrator concluded that it was appropriate to retain a level of 15.0 μg/m^3 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard with its level of 35 μg/m^3. 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.[3] 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 $PM_{2.5}$ 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 Administrator considered the available visibility preference studies that were part of the overall

[3] 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.

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.[4]

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 $PM_{2.5}$ 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 $PM_{2.5}$ in ambient air and PM-related visibility impairment. A $PM_{2.5}$ 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 $PM_{2.5}$ composition and relative humidity. In defining a target level of protection in terms of a $PM_{2.5}$ 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,[5] 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 $PM_{2.5}$ light extinction indicator calculated from $PM_{2.5}$ 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 $PM_{2.5}$, nor did they provide information on the composition of $PM_{2.5}$, 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

[4] 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 Inc., 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.

[5] 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 $PM_{2.5}$ 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 in for calculating the index. These analyses showed good correlation between 24-hour and 4-hour average $PM_{2.5}$ 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^{th} 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^{th}, 95^{th}, and 98^{th} 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^{th} 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^{th} 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)[6] as being a reasonable range of "candidate protection levels" (CPLs).[7] 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 $PM_{2.5}$ 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 $PM_{2.5}$ visibility index with a 24-hour averaging time, a 90^{th} 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 $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$, 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard "provides sufficient protection in all areas against the effects of visibility impairment – i.e., that the existing 24-hour $PM_{2.5}$ 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

[6] 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.

[7] For comparison, 20 dv, 25 dv, and 30 dv are equivalent to 64, 112, and 191 Mm^{-1}, 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 draft ISA and updated air quality-based analyses (Appendix D). Figure 5-1 below illustrates our general approach in developing preliminary 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.

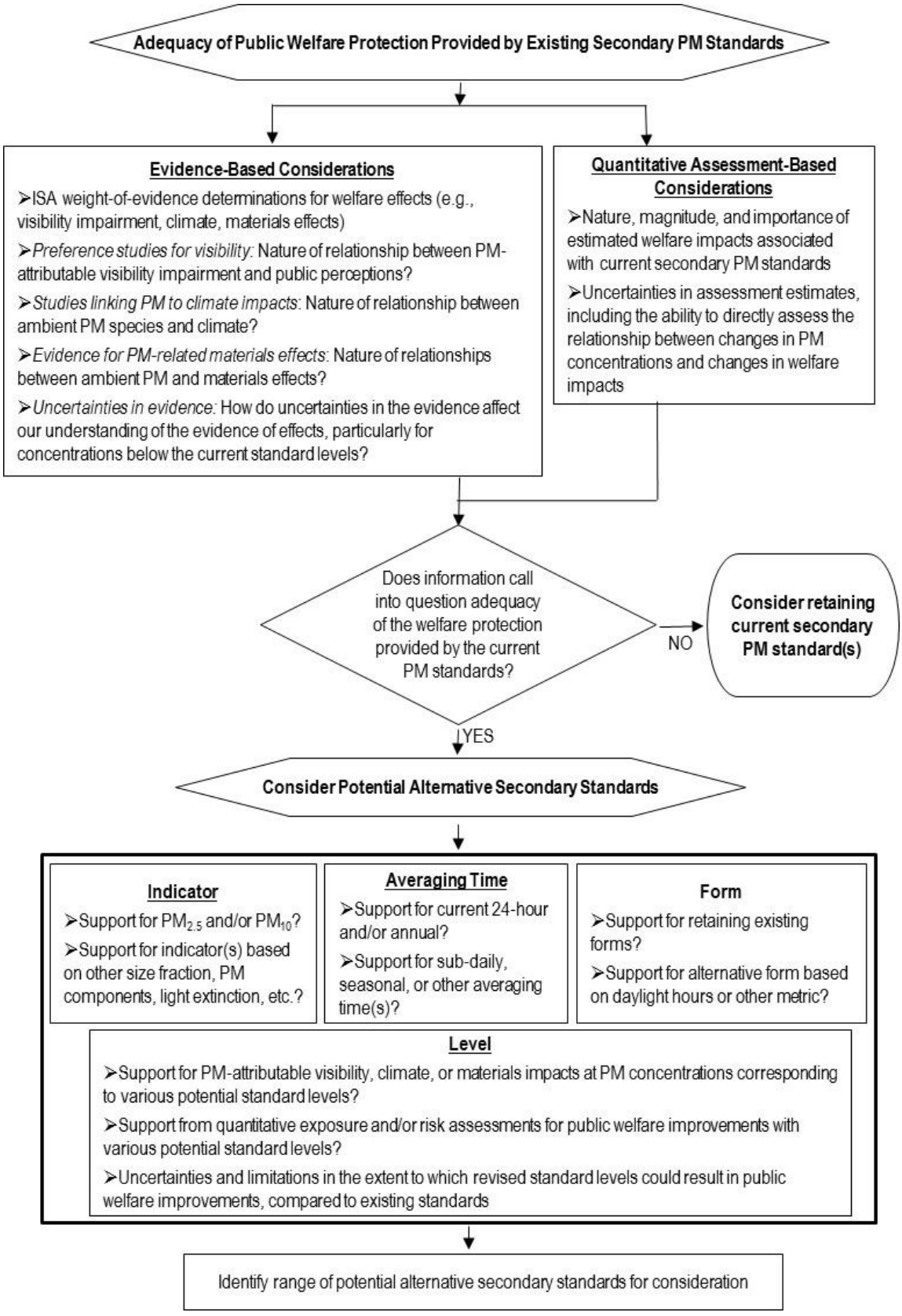

Figure 5-1. Overview of general approach for review of secondary PM standards.

5.2 ADEQUACY OF THE CURRENT SECONDARY PM STANDARDS

In considering the available evidence for welfare effects attributable to PM as presented in the draft 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 draft ISA (U.S. EPA, 2018), 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 draft 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-elated welfare effects.

Effect	2009 PM ISA	2018 draft 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 draft 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 draft 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, 2018, 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 Mm^{-1}). Higher values of light extinction (usually given in terms of Mm^{-1} or 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, 2018, 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, 2018, 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, 2018, 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 draft ISA, no major new developments have been made with these measurement methods since the last review (U.S. EPA, 2018, section 13.2.2.2).

A theoretical relationship between light extinction and PM characteristics has been derived from Mie theory (U.S. EPA, 2018, Equation 13-5) and can be used to estimate light extinction by combining mass scattering efficiencies of particles with particle concentrations (U.S. EPA, 2018, 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,[8] 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, 2018, 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, 2018, 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}.

[8] 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 ot 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).[9]

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.[10] 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, 2018, 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 draft ISA provides a comprehensive discussion of the spatial and temporal patterns of $PM_{2.5}$ composition and its contribution to light extinction from IMPROVE and CSN

[9] 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^{-1} used for Rayleigh scattering in the original and revised equations with a site-specific term based on elevation and mean temperature.

[10] 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.

monitoring sites, which are mostly rural and urban, respectively.[11] The data from these sites for the periods of 2005-2008 and 2011-2014 were used in the draft 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, 2018, 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 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 Mm^{-1}) in the Ohio River Valley,[12] while annual average b_{ext} was below 40 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, 2018, section 13.2.4.1, Figure 13-1, Figure 13-3, Figure 13-5).

Components of $PM_{2.5}$ 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, 2018, 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, 2018, 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 $PM_{10-2.5}$ mass scattering was relatively small in the eastern and

[11] 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, 2018, section 13.2.4.1).

[12] A b_{ext} value of 40 Mm^{-1} corresponds to a visual range of about 100 km.

northwestern U.S., in the Southwest, $PM_{10-2.5}$ mass scattering contributed to more than 20% of light extinction (U.S. EPA, 2018, 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^{-1} in 11 CSN regions, compared to only one IMPROVE region, and was greater than 20 Mm^{-1} 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, 2018, 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, 2018, 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^{-1}. This corresponds to an improvement in average visual range in most Eastern U.S. regions of more than 6 Mm^{-1} (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, 2018, section 13.2.4.1).

In summary, the spatial and temporal analysis of PM monitoring network data in the draft ISA emphasizes that the extent of light extinction by $PM_{2.5}$ depends on $PM_{2.5}$ composition and relative humidity. Regional differences in $PM_{2.5}$ composition greatly influence light extinction. Changes in $PM_{2.5}$ 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,[13] 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

[13] 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 Inc., 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 $PM_{2.5}$ 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).[14] 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 $\mu g/m^3$ of PM) (U.S. EPA, 2010).

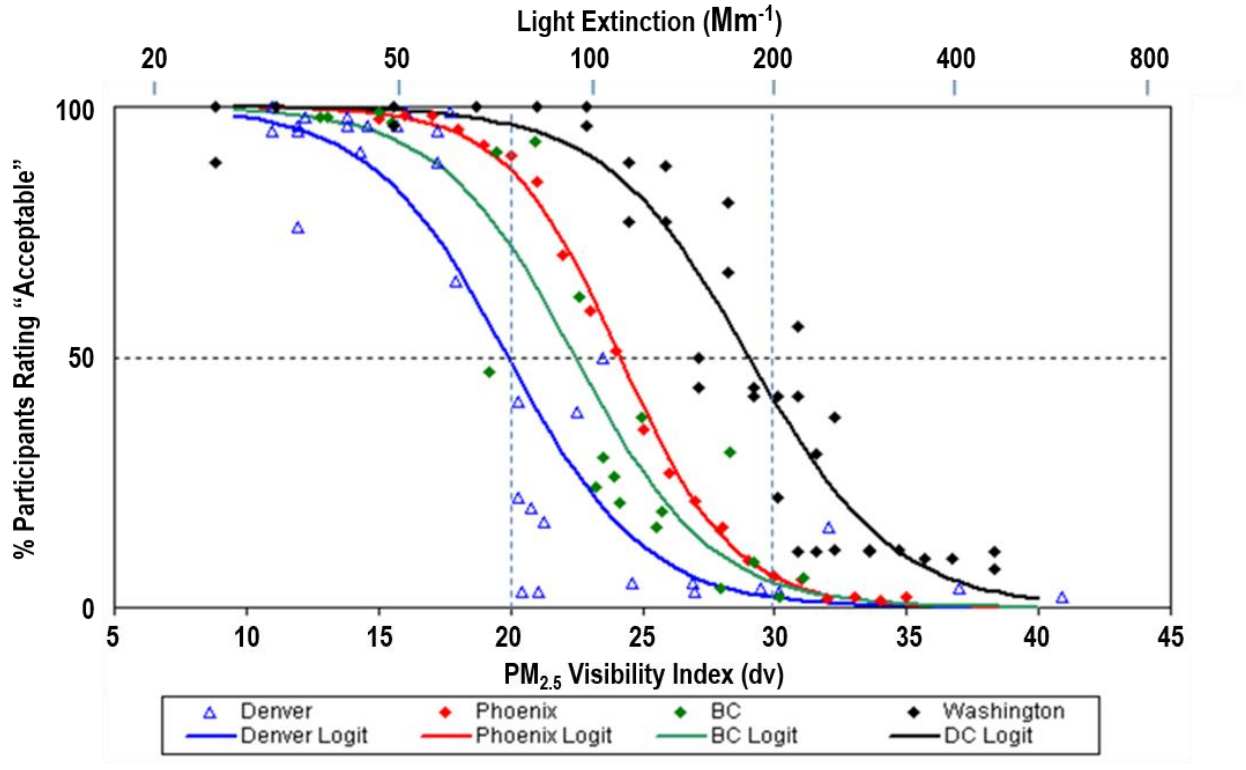

Figure 5-2. Relationship of viewer acceptability ratings to light extinction. (Source: U.S. EPA, 2011, Figure 4-2; U.S. EPA, 2010, Figure 2-16)

[14] 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 $PM_{2.5}$ concentrations in Beijing (with associated visibility impairment) than what is typical in the U.S. (U.S. EPA, 2018, 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 $PM_{2.5}$ 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 visibility, 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[15] for the current secondary standards and a 3-year average visibility metric based on light extinction estimated using IMPROVE equations.[16] 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.

[15] The design value for a 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.

[16] 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ design value for the 2015-2017 period and met strict criteria for PM species for this analysis.[17] 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_{10} 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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[18] at sites meeting the current secondary PM standards, and at

[17] 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 $PM_{2.5}$ mass, sulfate, nitrate, organic carbon, elemental carbon, sea salt (chlorine or chloride), and fine soil (aluminum, silica, calcium, iron, and titanium) measurement.

[18] 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).

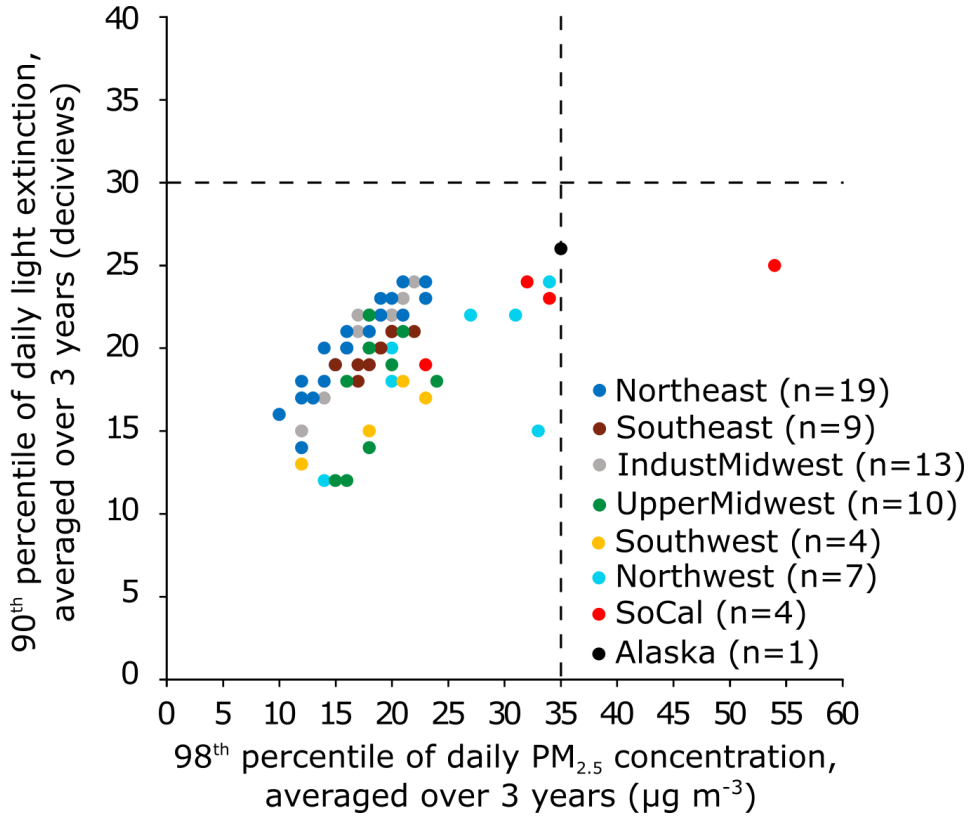

Figure 5-3. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2015-2017 using the original IMPROVE equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ standard (35 µg/m^3) 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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).

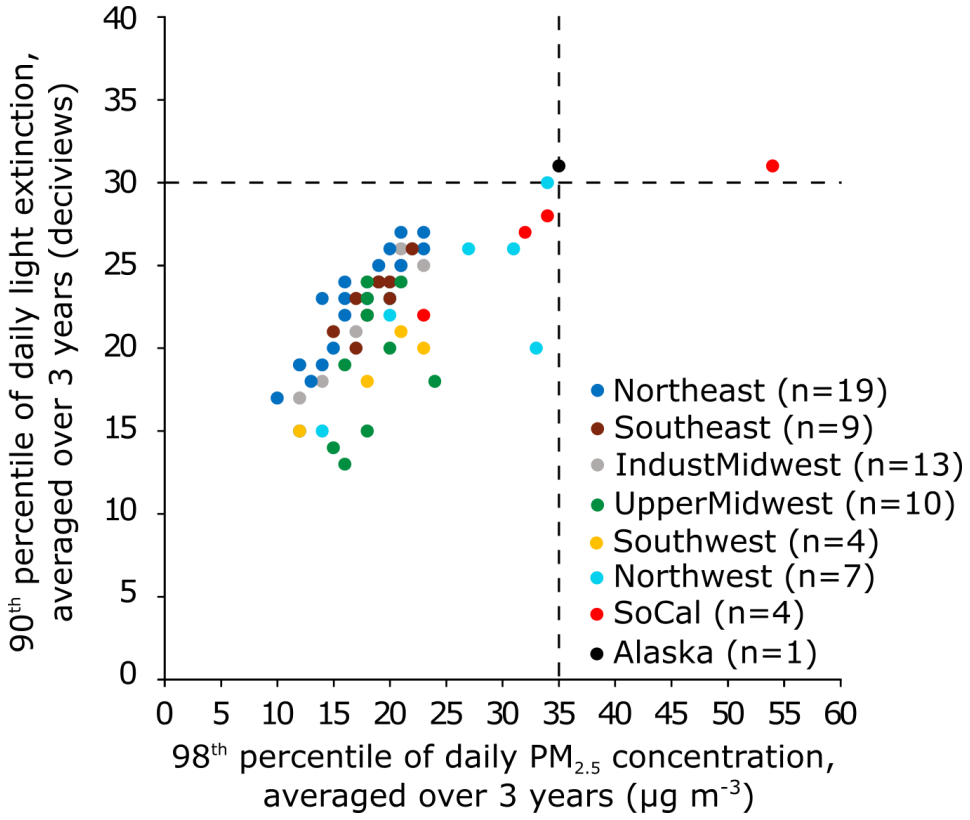

Figure 5-4. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2015-2017 using the Lowenthal and Kumar equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ standard (35 µg/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, $PM_{10\text{-}2.5}$ 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 $PM_{10\text{-}2.5}$ monitors to quantify the contribution of coarse PM to calculated light extinction. Since that time, an expansion in $PM_{10\text{-}2.5}$ monitoring efforts has increased the availability of data for use in estimating light extinction with both $PM_{2.5}$ and $PM_{10\text{-}2.5}$ concentrations included as inputs in the equations. Collocated $PM_{10\text{-}2.5}$ 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_{10} and $PM_{10-2.5}$ 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 draft ISA, which found that mass scattering from $PM_{10–2.5}$ 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, 2018, section 13.2.4.1, p. 13-35).

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 $PM_{2.5}$ 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 draft 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).[19] Since the last review, climate impacts have been extensively studied and the draft ISA concludes that "overall the evidence is sufficient to conclude that a causal relationship exists between PM and climate effects" (U.S. EPA, 2018, 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 draft 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, 2018, 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, 2018, section 13.3.2).

[19] 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 (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, 2018, 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 ± 0.5 watts per square meter (Wm^{-2}) (Boucher, 2013), which is slightly reduced compared to AR4. Estimates of effective radiative forcing[20] 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, 2018, section 13.3.4.1). The IPCC AR5 best estimate for ERFari is -0.45 ± 0.5 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, 2018, 13.3.3.2). IPCC AR5 estimates ERFaci at -0.45 Wm^{-2}, with a 90% confidence interval of -1.2 to 0 Wm^{-2} (U.S. EPA, 2018, section 13.3.4.2).[21] Studies have also calculated the combined effective radiative forcing from aerosol-radiation and aerosol-cloud interactions (ERFari+aci) (U.S. EPA, 2018, section 13.3.4.3). IPCC AR5 reports a best estimate of ERFari+aci of -0.90 (-1.9 to -0.1) 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

[20] 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, 2018, section 13.3.2.2).

[21] While the draft ISA includes estimates of RFaci and ERFaci from a number of studies (U.S. EPA, 2018, section 13.3.4.2, 13.3.4.3, 13.3.3.3), this draft 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, 2018, 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, 2018, section 13.3.3.3). The IPCC AR5 best estimate of RF from the albedo effect is +0.04 Wm^{-2}, with an uncertainty range of +0.02 to +0.09 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 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 H_2O_2 (U.S. EPA, 2018, section 13.3.5.1). The main source of anthropogenic sulfate is from coal-fired power plants, and global trends in the anthropogenic SO_2 emissions are estimated to have increased dramatically during the 20th and early 21st 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, 2018, 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 RFari of -0.4 (-0.6 to -0.2) 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 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 (ERFari+aci) for anthropogenic sulfate has been estimated to be nearly -1.0 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 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, 2018, Lee et al., 2013, section 13.3.5.2; Hauglustaine et al., 2014; 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) 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, 2018, 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) Wm^{-2} and an RFari estimate for SOA from these sources of -0.03 (-0.27 to +0.20) 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 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) Wm^{-2} (Myhre et al., 2013). Biomass burning contributes an additional +0.2 (+0.03 to +0.4) Wm^{-2} to BC RFari, while the albedo effect of BC on snow and ice adds another +0.04 (+0.02 to +0.09) Wm^{-2} (Myhre et al., 2013; U.S. EPA, 2018, 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 ± 0.2 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, 2018, 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 heterogenous distribution across the Earth. As such, patterns of RFari and RFaci 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, 2018, 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., Leibensperger 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, 2018, section 13.3.7). While evidence in this review suggests that PM influenced temperature trends across the southern and eastern U.S. in the 20th 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-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; and,
- 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, 2018, 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,[22] 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

[22] 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, 2018, section 13.4).

Materials damage from PM generally involves one or both of two processes: soiling and corrosion (U.S. EPA, 2018, section 13.4.2). Soiling and corrosion are complex, interdependent processes, typically beginning with deposition of atmospheric PM or SO_2 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, 2018, 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[23] can discolor surfaces by trapping PM more efficiently (U.S. EPA, 2009, p. 9-195; U.S. EPA, 2018, 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_2 to materials such as limestone, granite, and metal. Deposition of SO_2 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, 2018, section 13.4.2). Oxidation of deposited SO_2 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

[23] 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, 2018, 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 (SO_2 and NO_2), while copper and copper alloys were mainly corroded by gaseous pollutants (SO_2 and O_3) and aluminum and aluminum alloy corrosion was mainly attributed to PM and NO_2.

A number of studies have also found materials damage from PM components besides sulfate and black carbon and atmospheric gases besides SO_2. Studies have characterized impacts of nitrates, NO_X, and organic compounds on direct materials damage or on chemical reactions that enhance materials damage (U.S. EPA, 2018, 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, 2018, 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[24] 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,[25] photovoltaic module power transmission was reduced by approximately 3% for every g/m^2 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).

[24] In this discussion of non-visibility welfare effects (section 5.2.2), haze is used as it has been defined n 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.

[25] 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, 2018, 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, 2018, section 13.4.3, Figure 13-31). 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, 2018, 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, 2018, 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, 2018, 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 draft 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, 2018, 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, 2018, 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, 2018, 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 PRELIMINARY CONCLUSIONS ON THE SECONDARY PM STANDARDS

This section discusses preliminary staff conclusions for the Administrator's consideration in judging the adequacy of the current secondary PM standards. These preliminary conclusions are based on consideration of the assessment and integrative synthesis of evidence presented in the draft ISA, as well as our analyses of recent air quality. Taking into consideration the responses to specific questions discussed above, we revisit the overarching policy question for this chapter:

- **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, 2018, section 13.2.3.3; section 5.2.1.1 above). Light extinction by $PM_{2.5}$ is dependent on $PM_{2.5}$ 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 value 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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^{th} 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, 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 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. This final decision will draw upon the available scientific evidence and quantitative analyses on PM-attributable welfare effects, and on judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses.

5.4 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH

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 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 $PM_{2.5}$ 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; and,
  - 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.
- Insufficient evidence is available to relate soiling or damage to specific particulate matter 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. SUPPELMENTAL 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 draft 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 $PM_{2.5}$, PM_{10}, $PM_{10\text{-}2.5}$, AND $PM_{2.5}$ SPECIATION FIGURES

- $PM_{2.5}$ annual average and 98th 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 98th 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_{10} annual average and 98th 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 98th 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*).
- $PM_{10\text{-}2.5}$ annual average and 98th 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 98th 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*).
- $PM_{2.5}$ 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 98th 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 $PM_{2.5}$ DESIGN VALUE TABLE AND INCREMENT FIGURES

- $PM_{2.5}$ 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 $PM_{2.5}$. 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*).
- $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ CONCENTRATION FIGURE

- $PM_{2.5}$ hourly average mass concentrations: calculated from regulatory-quality Federal Equivalent Method monitors. The 2-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 $PM_{2.5}$ mass concentrations above 140 $\mu g/m^3$ at individual sites are illustrated in Figure A-1.

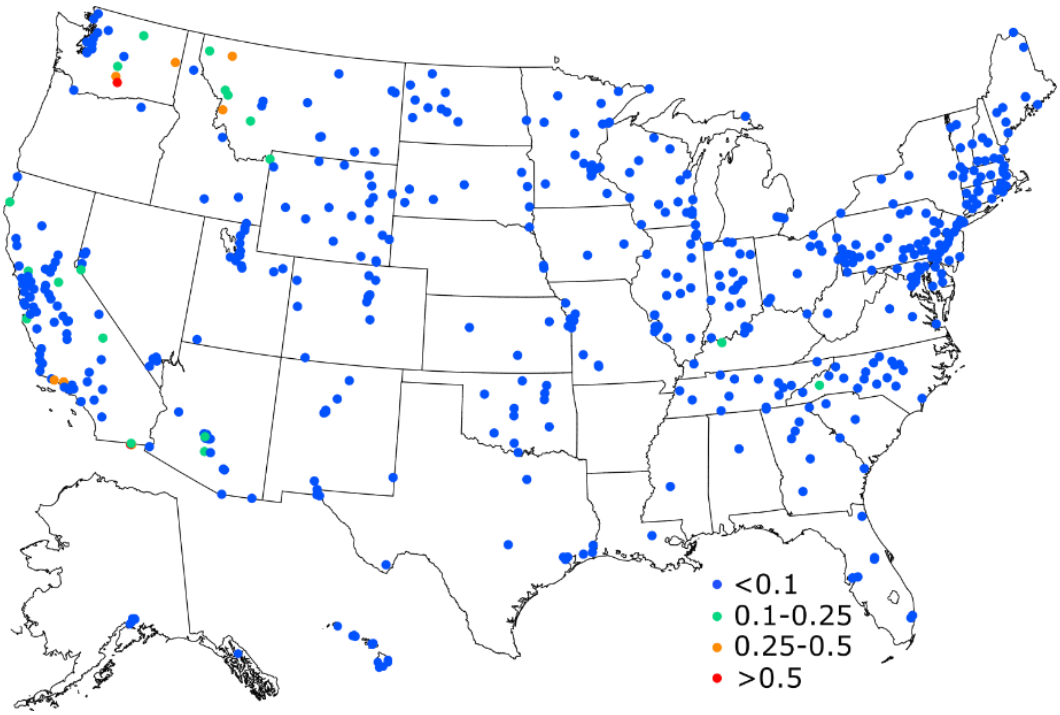


Figure A-1. Percentages of 2015-2017 2-hour average $PM_{2.5}$ mass concentrations above 140 µg/m^3.

A.4 DATA SOURCES FOR ULTRAFINE FRACTION OF $PM_{2.5}$ 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 $PM_{2.5}$ BASED ON HYBRID MODELING APPROACHES

A.5.1 Data Sources for 2011 $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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*.

- 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

- $PM_{2.5}$ 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*; 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*) were averaged annually for each monitor. Corresponding monitor locations are shown in Figure 2-32.

APPENDIX B. DATA INCLUSION CRITERIA AND SENSITIVITY ANALYSES

TABLE OF CONTENTS

This appendix presents supplemental information on the methods used to conduct the analyses discussed in section 3.2.3.2 of this draft 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 $PM_{2.5}$ concentrations presented in Figure 3-7 and Figure 3-8. Sections B.4 to B.6, and sections B.8 and B.9, 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 $PM_{2.5}$ 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 draft ISA U.S. EPA (2018) 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., 2016), 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 $PM_{2.5}$ 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 $PM_{2.5}$ (as opposed to a study mean/median range across study area locations) and based on ambient $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that contributed to reported associations.[1]

Some of the included studies also provide city-specific study mean concentrations and city-specific health events. Hence, $PM_{2.5}$ exposure estimates corresponding to the 10^{th} and 25^{th} percentiles of those events were determined in the following manner. City-specific cases and $PM_{2.5}$ concentrations were input in ascending order by $PM_{2.5}$ 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 $PM_{2.5}$ concentration associated with the cumulative percent closest to the 10^{th} and 25^{th} percentiles were input in Figure 3-7 and the cumulative percent values closest to the associated 10^{th} and 25^{th} percentile inputs are shown in Table B-1[2]. 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. $PM_{2.5}$ concentrations corresponding to the 25^{th} and 10^{th} percentiles of estimated health events.

Citation	10^{th} Percentile $PM_{2.5}$ ($\mu g/m^3$) (Cumulative percent value closest)	25^{th} Percentile $PM_{2.5}$ ($\mu g/m^3$) (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

[1] This is an issue only for some studies of long-term $PM_{2.5}$ exposures. While this approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term $PM_{2.5}$ exposures, under the assumption that spatial patterns in $PM_{2.5}$ 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 $PM_{2.5}$ concentrations that could have contributed to reported health outcomes.

[2] That is, 25% of the total health events occurred in study locations with mean $PM_{2.5}$ concentrations (i.e., averaged over the study period) below the 25^{th} percentiles identified in Figure 3-7 and 10% of the total health events occurred in study locations with mean $PM_{2.5}$ concentrations below the 10^{th} percentiles identified.

B.3 HYBRID MODEL PREDICTED $PM_{2.5}$ 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 draft ISA and that use air quality data to estimate $PM_{2.5}$ 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 $PM_{2.5}$ exposures are estimated in the health study.[3] All studies that met the criteria for inclusion were conducted in the U.S.

Figure 3-8 presents overall means of hybrid model-predicted $PM_{2.5}$ concentrations for key studies, and the concentrations corresponding to the 25^{th} and 10^{th} percentiles of estimated exposures or health events, when available. For Di et al. (2017b), we present 25^{th} and 10^{th} percentiles of annual $PM_{2.5}$ 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^{th} and 10^{th} percentiles of deaths at the zip-code level. These values are illustrated in Figure B-1 and Figure B-2 (Jenkins, 2019b, Jenkins, 2019a).

For other studies, that present 25^{th} percentile exposure estimates in Figure 3-8 [Kloog et al. (2012), Kloog et al. (2014), Shi et al. (2016), Wang et al. (2017)], estimates of 25^{th} percentile exposure estimates were derived from study manuscripts of air quality descriptive statistics and can be found in Table B-2.

[3] For example, due to lack of spatial field availability before 1998, Crouse et al. (2015) use median annual $PM_{2.5}$ concentrations for the 1998-2006 time period (van Donkelaar et al., 2010; van Donkelaar et al., 2015a; van 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 $PM_{2.5}$ monitoring was sparse prior to 1999, with many of the available $PM_{2.5}$ 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 PM2.5 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 $PM_{2.5}$ based on TSP measurements and metropolitan indicator variables because only small number of $PM_{2.5}$ measurements were available, and no measurements were made prior to 1984. Thus, these studies are not included in Figure 3-8.

Percentiles of $PM_{2.5}$ By Zip Code

Threasholds defining percentiles of $PM_{2.5}$ exposure for each zip code.

Percentile of $PM_{2.5}$, Based on ZIP code	$PM_{2.5}$ 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 $PM_{2.5}$ concentrations by zip code corresponding to long-term exposure estimates in Di et al., 2017b.

Percentiles of $PM_{2.5}$ By Zip Code

Threasholds defining percentiles of Daily $PM_{2.5}$ exposure for each zip code.

Percentile of Daily $PM_{2.5}$, Based on ZIP code	$PM_{2.5}$ 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 the 25th and 10th percentiles of deaths at the zip-county level in Di et al., 2017a.

Table B-2. $PM_{2.5}$ concentrations corresponding to the 25th and 10th percentiles of estimated exposures in Figure 3-8.

Citation	10th Percentile $PM_{2.5}$ (µg/m³)	25th Percentile $PM_{2.5}$ (µg/m³)
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[4] was used. For Canadian studies, city-specific population from 2016 Statistics Canada[5] 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.

[4]Available from: https://data.census.gov/cedsci/

[5] 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 $PM_{2.5}$ 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.[6]

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

[6] As discussed below (section B.4.3), not all counties have $PM_{2.5}$ 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).[7] 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[8] 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-1, using a subset of cities reported in Zanobetti et al. (2014). Note, for cities not listed in Table B-1, 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.

[7] 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.

[8] Available from: *http://www.statsamerica.org/Default.aspx*

Table B-3. 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%)
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 $PM_{2.5}$ 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. Air quality data in the U.S. was obtained from the EPA Air Quality System (AQS)[9]. 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 $PM_{2.5}$. For non-regulatory data, only monitors

[9]Available from: https://www.epa.gov/aqs

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.[10] 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 $PM_{2.5}$ 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 at or below each corresponding average max pseudo-DV. Next, the average max pseudo-DV associated with the cumulative population closest to the 5^{th}, 25^{th}, 50^{th}, 75^{th} and 95^{th} percentiles were identified. The actual cumulative percents that are closest to the 5^{th}, 25, 50^{th}, 75^{th}, and 95^{th} percentiles, for all long- and short-term exposure studies and for annual and 24-hr $PM_{2.5}$ 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-4 and Table B-5. Counties that had no air quality monitors or no valid design values did not contribute to the percentile calculation.

[10] Available from: http://maps-cartes.ec.gc.ca/rnspa-naps/data.aspx?lang=en

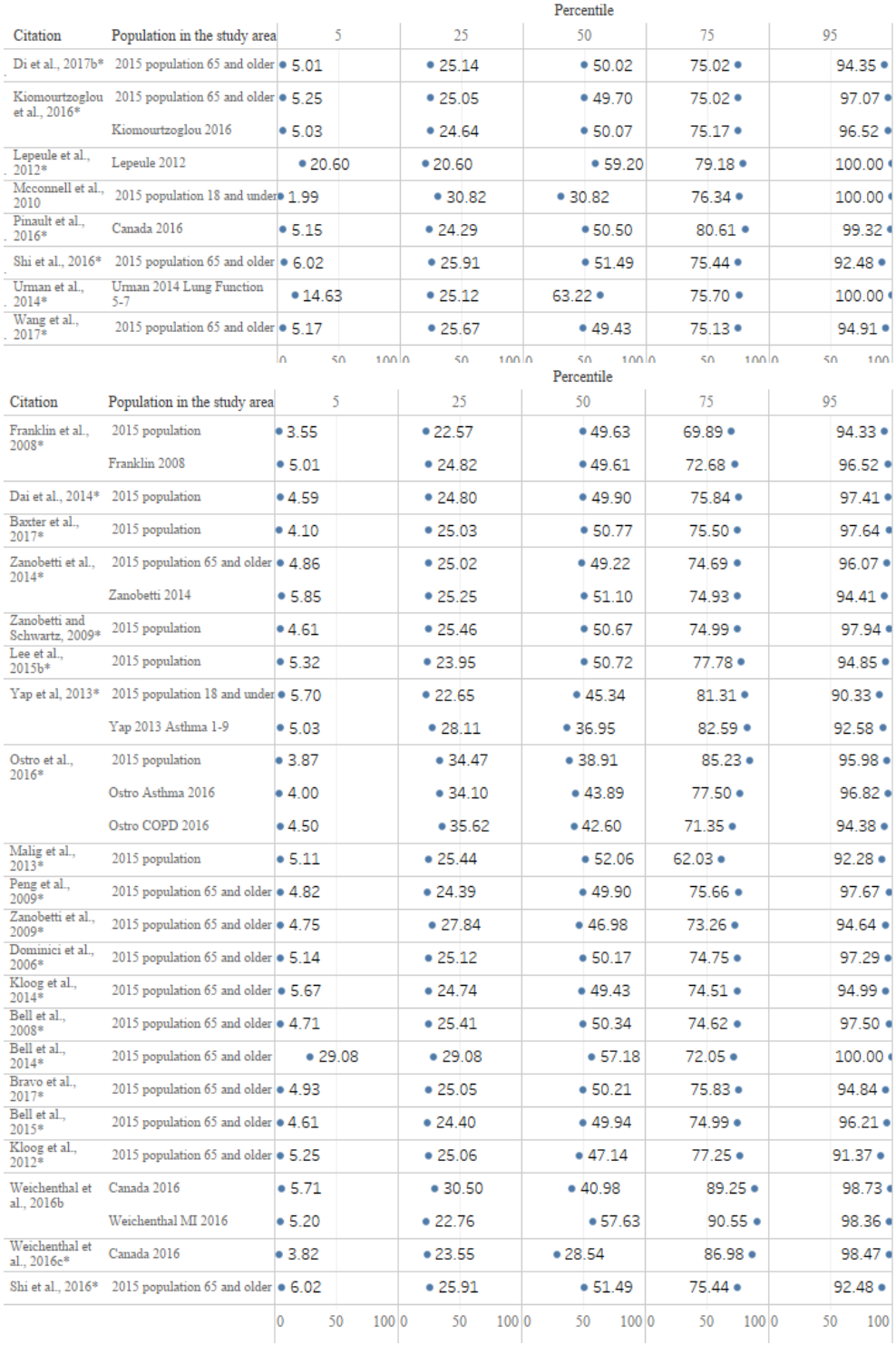
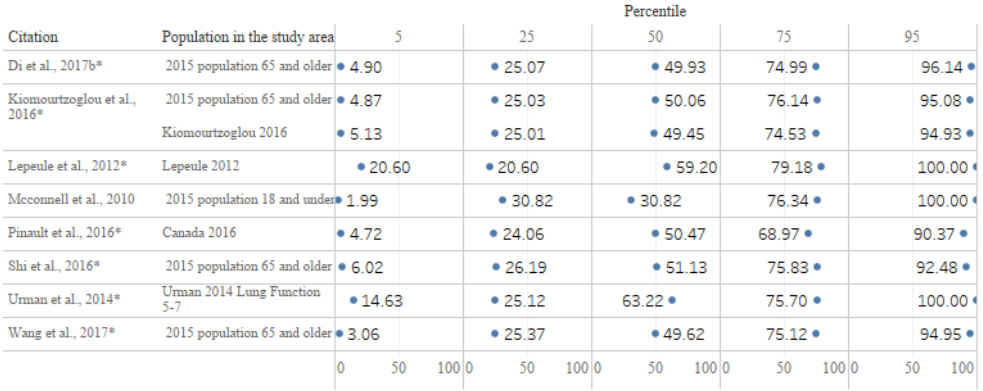


Figure B-3. Cumulative population percentile closest to the 5th, 25th, 50, 75, and 95th percentile: studies of long-term exposure and annual $PM_{2.5}$ concentrations (top panel) and studies of short-term exposure and annual $PM_{2.5}$ concentrations (bottom panel).



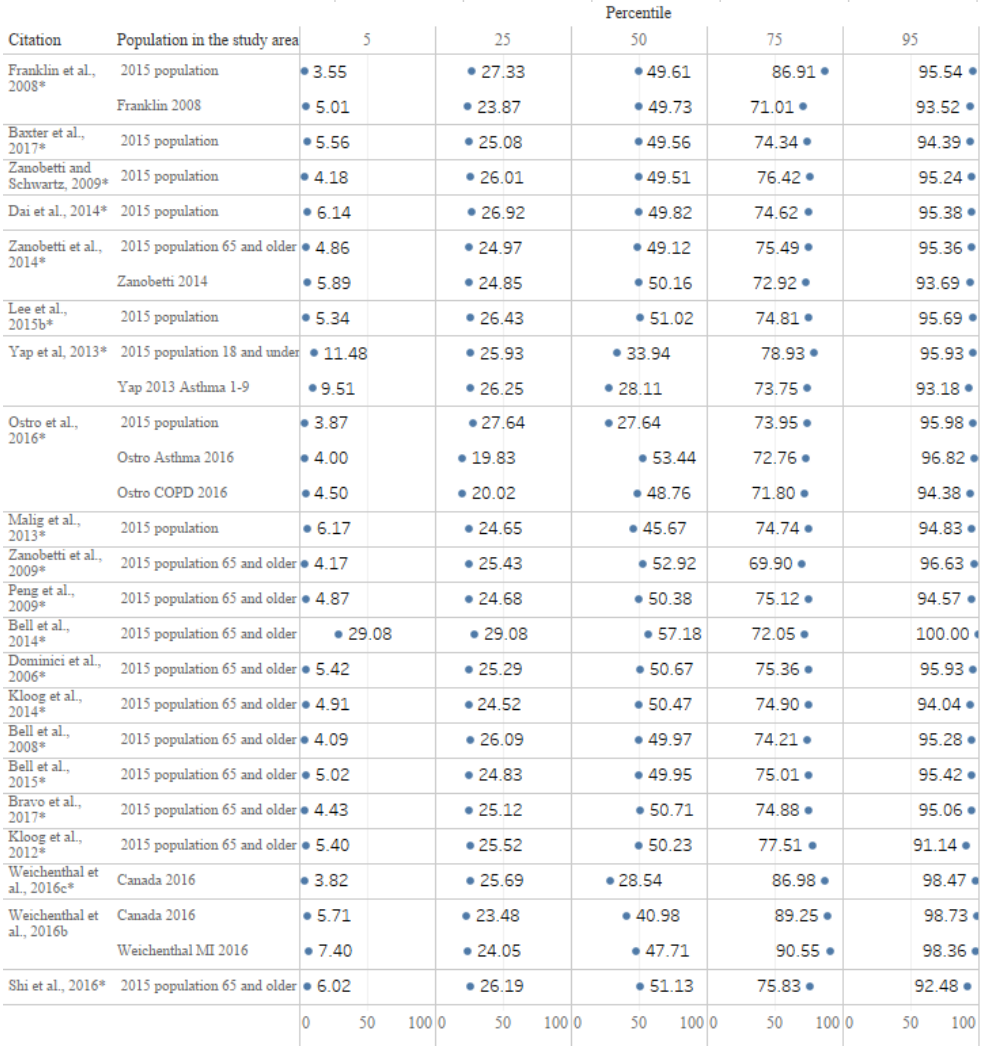


Figure B-4. Cumulative population percentile closest to the 5th, 25th, 50, 75, and 95th percentile: studies of long-term exposure and 24-hr $PM_{2.5}$ concentrations (top panel) and studies of short-term exposure and 24-hr $PM_{2.5}$ concentrations (bottom panel).

Table B-4. Annual average maximum pseudo-DVs corresponding to population or health event percentiles in box-and-whisker plots in Figure 3-9.

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
Kioumourtzoglou 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., 2015	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
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-5. 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., 2015	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.

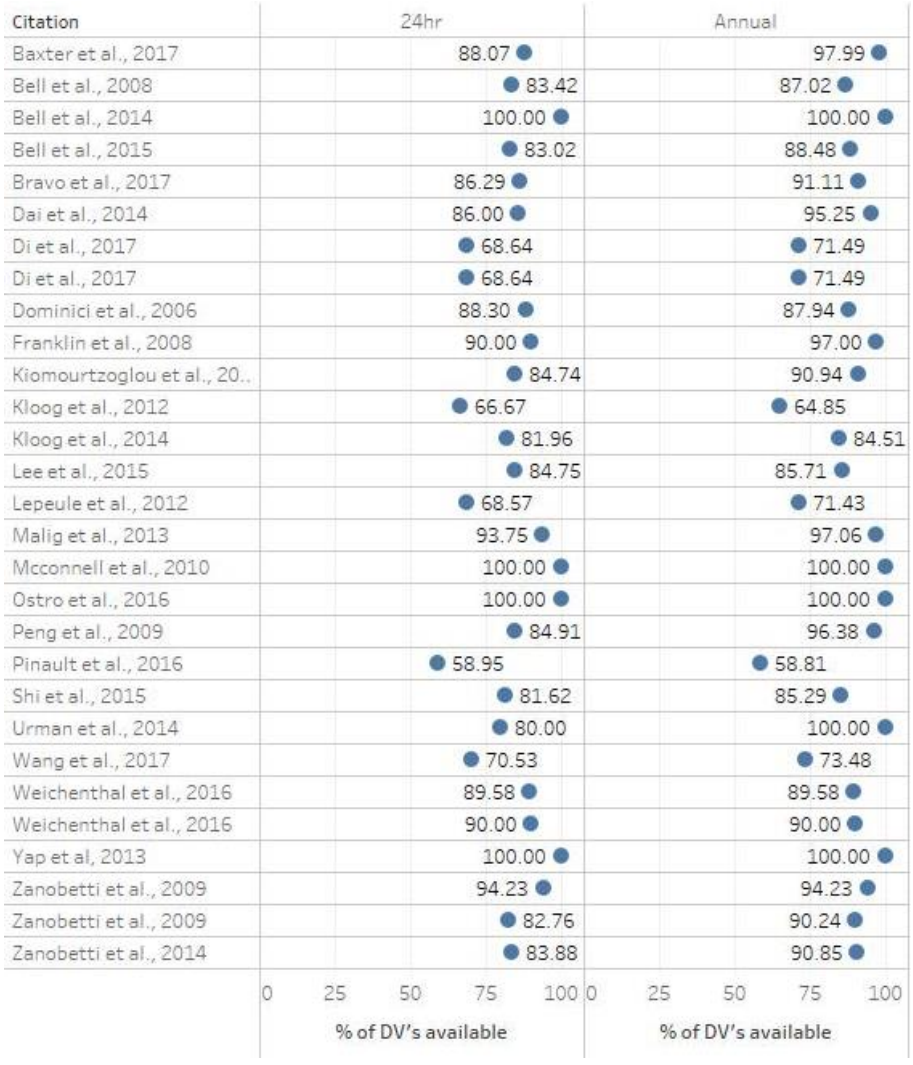


Figure B-5. Studies used in box-and-whisker plots (Figure 3-9 and Figure B-9) and the percent of pseudo-DVs available by study.

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 $PM_{2.5}$ 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 95th 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-6 and Table B-7).

Table B-6. Percent population included in annual pseudo-DV boxplots (Figure 3-9).

Citation	Population Used	Study Area Counties	Total Population	Population with DV	Population with DV (%)
Baxter et al., 2017	US 2015	113	113,053,365	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., 2015	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-7. 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	Kiomourtzoglou 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., 2015	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 $PM_{2.5}$ 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.

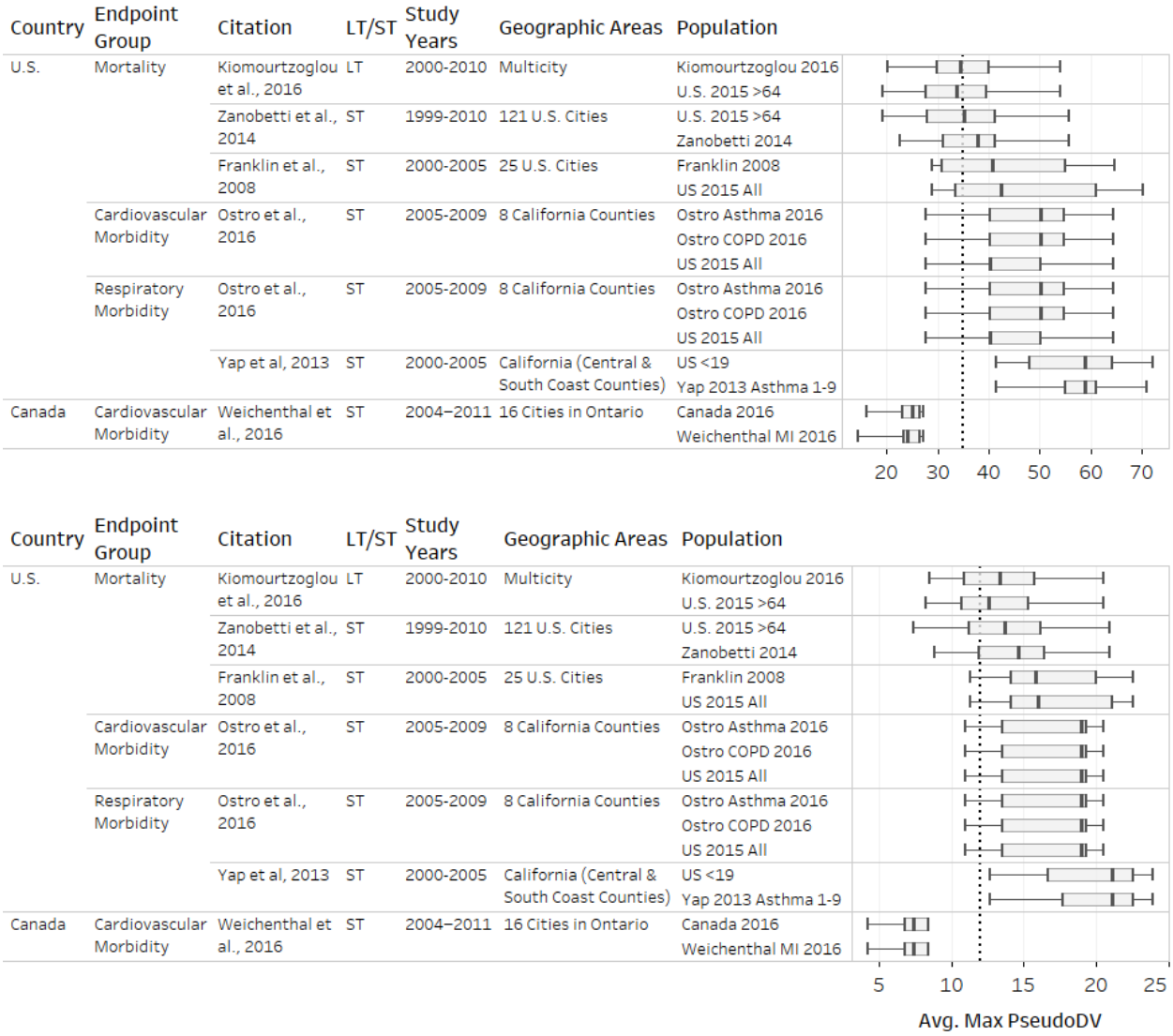


Figure B-6. $PM_{2.5}$ pseudo-design values corresponding to various percentiles of study area populations and health events for studies of 24-hour $PM_{2.5}$ exposures and long-term studies (top panel) and annual $PM_{2.5}$ exposures and long-term studies (bottom panel).

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 $PM_{2.5}$ concentrations (section 2.2.3), areas meeting an annual $PM_{2.5}$ standard with a particular level would be expected to have long-term average $PM_{2.5}$ concentrations (i.e., averaged across space and over time in the area) somewhat below that standard level. Figure B-7 and Table B-8 indicate that, based on recent air quality in U.S. CBSAs, maximum annual $PM_{2.5}$ 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 $PM_{2.5}$ concentrations. Given that higher $PM_{2.5}$ concentrations have been reported at some near-road monitoring sites, relative to the surrounding area (section 2.3.2.2.2), recent requirements for $PM_{2.5}$ 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 $PM_{2.5}$ design values and annual average concentrations.

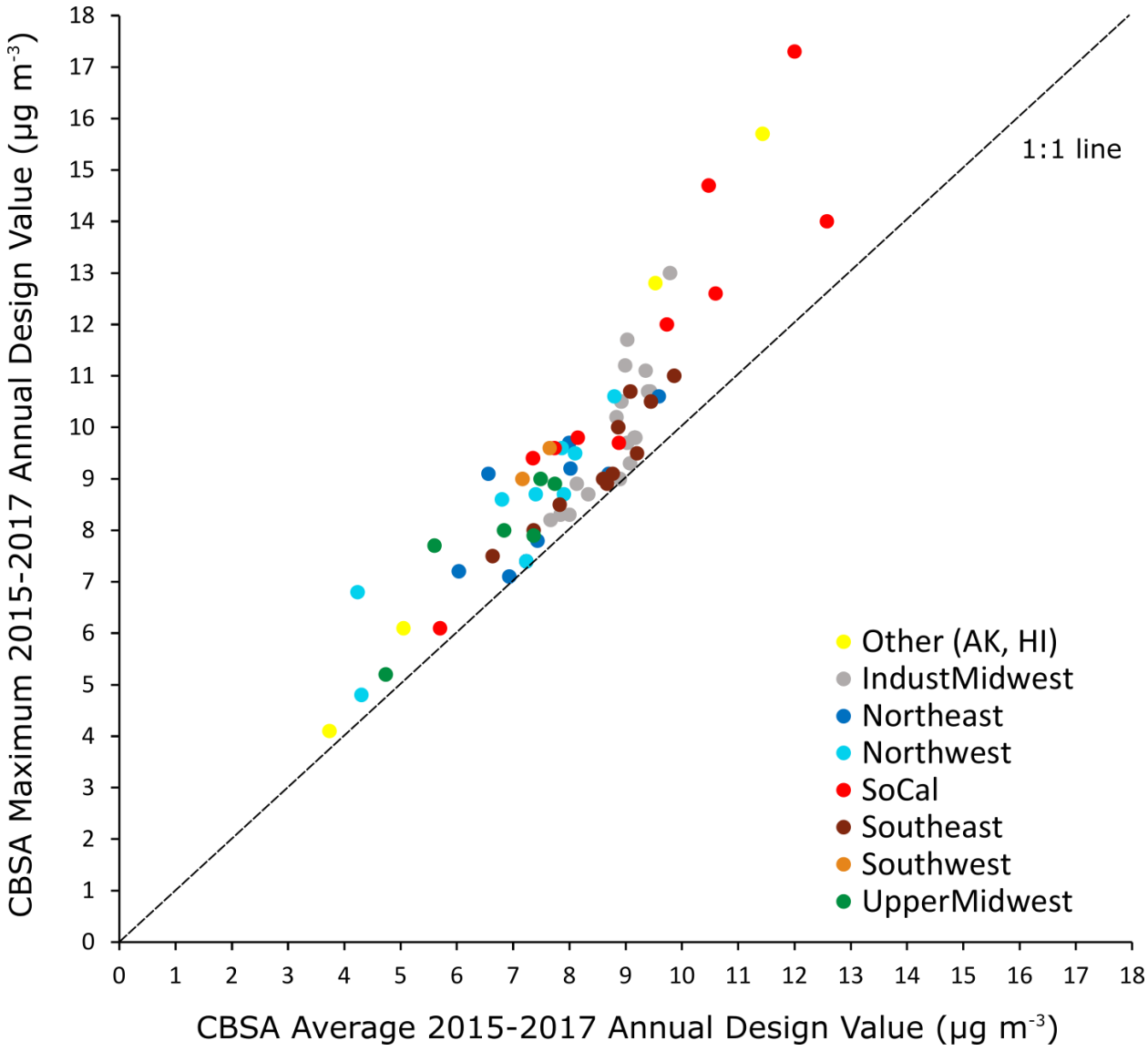


Figure B-7. Comparison of CBSA average annual design values and CBSA maximum annual design values for 2015-2017. (Note: Includes all CBSAs with at least 3 valid annual DVs.)

Table B-8. 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

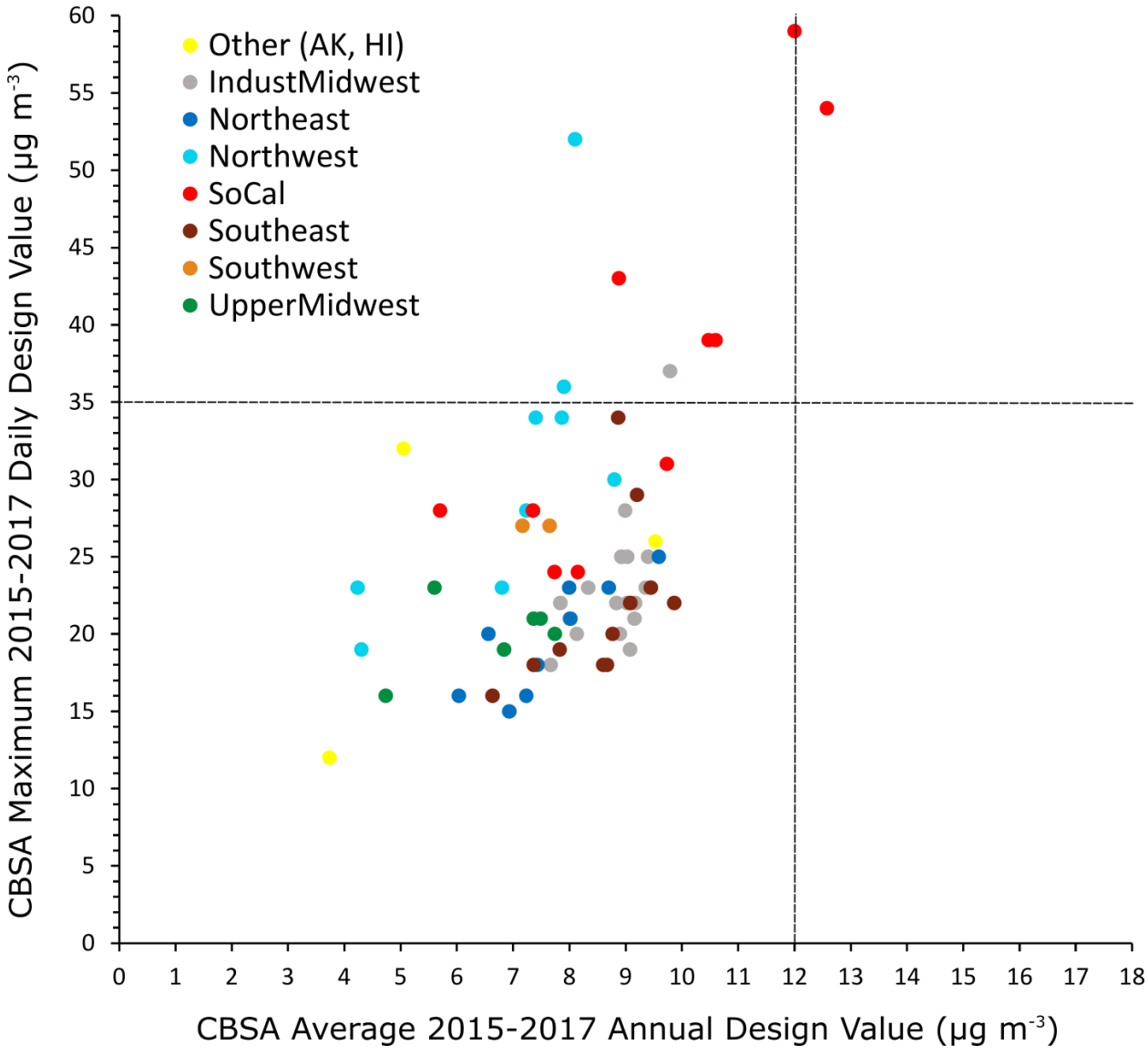


Figure B-8. Comparison of CBSA average annual design values and CBSA maximum daily design values for 2015-2017. (Note: Dashed lines indicate the level of the current 24-hour PM2.5 standard (35 ug m/3) and the current annual PM standard (12 ug m/3). Includes all CBSAs with at least 3 valid daily and 3 valid annual DVs.)[11].

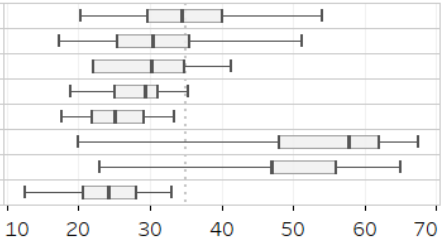
[11] The CBSA maximum 2015-2017 daily design value (y-axis) was cut off at 60 µg/m^3, to improve the visualization of data, but this removed the Fairbanks CBSA from the plot, which had a daily design value of 85 µg/m^3 and an annual design value of 15.7 µg/m^3.

B.8 24-HOUR PSEUDO-DESIGN VALUES AND DISTRIBUTIONS ACROSS STUDY AREAS

As described in section 3.2.3.2.2 of the draft PA, and section B.4 of this appendix, for locations evaluated in key epidemiologic studies we identify annual and 24-hour $PM_{2.5}$ pseudo-design values and the number of people (or health events). Figure 3-9 in the draft 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

Country	Endpoint Group	Citation	Study Years	Geographic Areas
U.S.	Mortality	Kiomourtzoglou et al., 2016*	2000-2010	207 U.S. Cities
		Di et al., 2017b*	2000-2012	U.S. Nationwide
		Lepeule et al., 2012*	2001-2009	6 U.S. Cities
		Shi et al., 2016*	2003-2008	6 NE U.S. States
		Wang et al., 2017*	2000-2013	7 SE U.S. States
	Morbidity	Urman et al., 2014*	2002-2007	8 CA Counties
		Mcconnell et al., 2010	2003-2005	13 CA Communities
Canada	Mortality	Pinault et al., 2016*	2000-2011	Multicity



Short-term exposure studies

Country	Endpoint Group	Citation	Study Years	Geographic Areas
U.S.	Mortality	Franklin et al., 2008*	2000-2005	25 U.S. Cities
		Baxter et al., 2017*	2001-2005	77 U.S. Cities
		Dai et al., 2014*	2000-2006	75 U.S. Cities
		Zanobetti et al., 2014*	1999-2010	121 U.S. Cities
		Zanobetti and Schwartz, 200..	1999-2005	112 U.S. Cities
		Di et al., 2017a*	2000-2012	U.S. Nationwide
		Shi et al., 2016*	2003-2008	6 NE U.S. States
		Lee et al., 2015b*	2007-2011	3 SE U.S. States
	Morbidity	Yap et al, 2013*	2000-2005	CA (Central & Southern Counties)
		Ostro et al., 2016*	2005-2009	8 CA Counties
		Malig et al., 2013*	2005-2008	35 CA Counties
		Zanobetti et al., 2009*	2000-2003	26 U.S. Cities
		Peng et al., 2009*	2000-2006	119 U.S. Urban Counties
		Bell et al., 2014*	2000-2004	4 U.S. Counties, MA & CT
		Dominici et al., 2006*	1999-2002	204 U.S. Urban Counties
		Kloog et al., 2014*	2000-2006	7 U.S. Mid-Atlantic States & D.C.
		Bell et al., 2008*	1999-2005	202 U.S. Urban Counties
		Bell et al., 2015*	1999-2010	213 U.S. Urban Counties
		Bravo et al., 2017*	2002-2006	708 U.S. Counties
		Kloog et al., 2012*	2000-2006	6 NE U.S. States
Canada	Morbidity	Weichenthal et al., 2016c*	2004-2011	15 Ontario Cities
		Weichenthal et al., 2016b	2004–2011	16 Ontario Cities

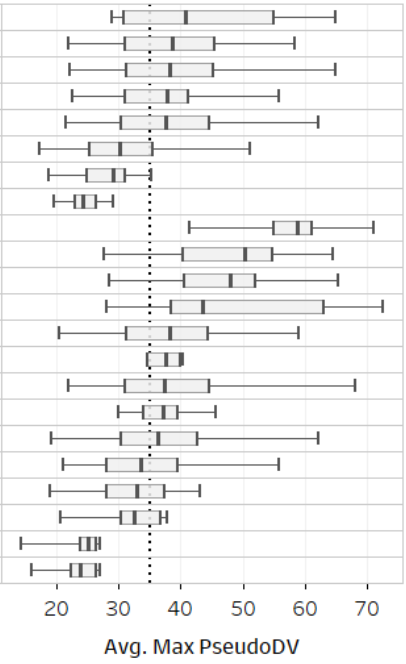


Figure B-9. $PM_{2.5}$ 24-hour pseudo-design values corresponding to various percentiles[1] of study area populations or health events for studies of long-term and short-term $PM_{2.5}$ exposures.[2]

[1] Whiskers reflect $PM_{2.5}$ pseudo-design values corresponding to 5th and 95th percentiles of study area populations (or health events), boxes correspond to the 25th and 75th percentiles, and the vertical lines inside the boxes correspond to 50th percentiles. Asterisks next to study citations denote statistically significant effect estimates.

[2] For most of the studies included in Figure B-9Figure 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 µG/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 Table B-9 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 µg/m^3 (i.e., lower than the current annual standard). Of the counties that have study-period average pseudo-DV's ≤ 12.04 µg/m^3, 89.3% of individual 3-year pseudo-DVs are ≤ 12.04 µg/m^3 (i.e., 3,410 of 3,820 3-year pseudo-DVs).

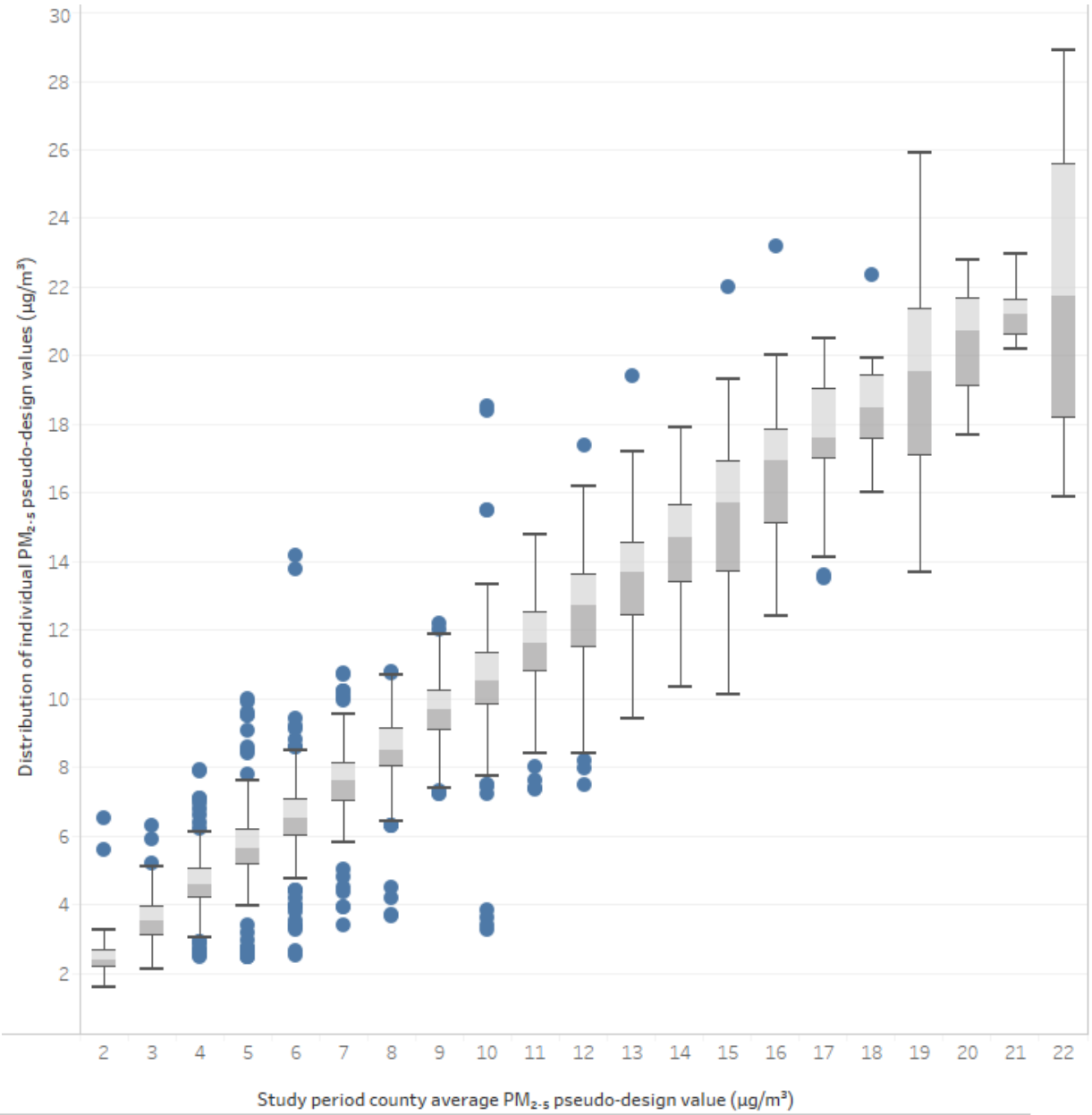


Figure B-10. County average pseudo-DV by 1 µg/m^3 and distribution of individual county pseudo-DVs within each 1 µg/m^3 interval for study counties in Di et al., 2017b. Note: 1 µg/m^3 intervals correspond to pseudo-DVs greater than 2.04 and less than or equal to 3.04, etc. Therefore, values distributions 11 and less correspond to county average pseudo-DVs that meet the annual standard of 12.04 µg/m^3.

Table B-9. County average pseudo-DV by 1 µg/m^3 and distribution of county pseudo-DVs within each 1 µg/m^3 interval for study counties in Di et al., 2017b

County average pseudo-DV $PM_{2.5}$ concentration (µg/m^3) interval	Count (percent) of pseudo-DV's ≤ 12.04 µg/m^3	Count (percent) of pseudo-DV's > 12.04 µg/m^3
$2.04 < PM_{2.5} \leq 3.04$	93 (100.00)	0 (0.00)
$3.04 < PM_{2.5} \leq 4.04$	117 (100.00)	0 (0.00)
$4.04 < PM_{2.5} \leq 5.04$	198 (100.00)	0 (0.00)
$5.04 < PM_{2.5} \leq 6.04$	235 (100.00)	0 (0.00)
$6.04 < PM_{2.5} \leq 7.04$	293 (99.35)	2 (0.68)
$7.04 < PM_{2.5} \leq 8.04$	283 (100.00)	0 (0.00)
$8.04 < PM_{2.5} \leq 9.04$	501 (100.00)	0 (0.00)
$9.04 < PM_{2.5} \leq 10.04$	533 (99.84)	1 (0.19)
$10.04 < PM_{2.5} \leq 11.04$	619 (92.23)	61 (8.97)
$11.04 < PM_{2.5} \leq 12.04$	538 (66.03)	346 (39.14)
$12.04 < PM_{2.5} \leq 13.04$	332 (30.46)	635 (65.67)
$13.04 < PM_{2.5} \leq 14.04$	128 (13.19)	525 (80.40)
$14.04 < PM_{2.5} \leq 15.04$	38 (5.14)	433 (91.93)
$15.04 < PM_{2.5} \leq 16.04$	7 (1.27)	228 (97.02)
$16.04 < PM_{2.5} \leq 17.04$	0 (0.47)	70 (100.00)
$17.04 < PM_{2.5} \leq 18.04$	0 (0.00)	21 (100.00)
$18.04 < PM_{2.5} \leq 19.04$	0 (0.00)	11 (100.00)
$19.04 < PM_{2.5} \leq 20.04$	0 (0.00)	33 (100.00)
$20.04 < PM_{2.5} \leq 21.04$	0 (0.00)	12 (100.00)
$21.04 < PM_{2.5} \leq 22.04$	0 (0.00)	11 (100.00)
$22.04 < PM_{2.5} \leq 23.04$	0 (0.00)	11 (100.00)
Total	3,915 (62.0)	2,400 (38.0)

Figure B-11 and Table B-10 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 µg/m^3. Of the counties that have study-period average pseudo-DV's ≤ 12.04 µg/m^3 98.1% of individual 3-year pseudo-DVs are ≤ 12.04 µg/m^3 (i.e., 102 of 104 3-year pseudo-DVs).

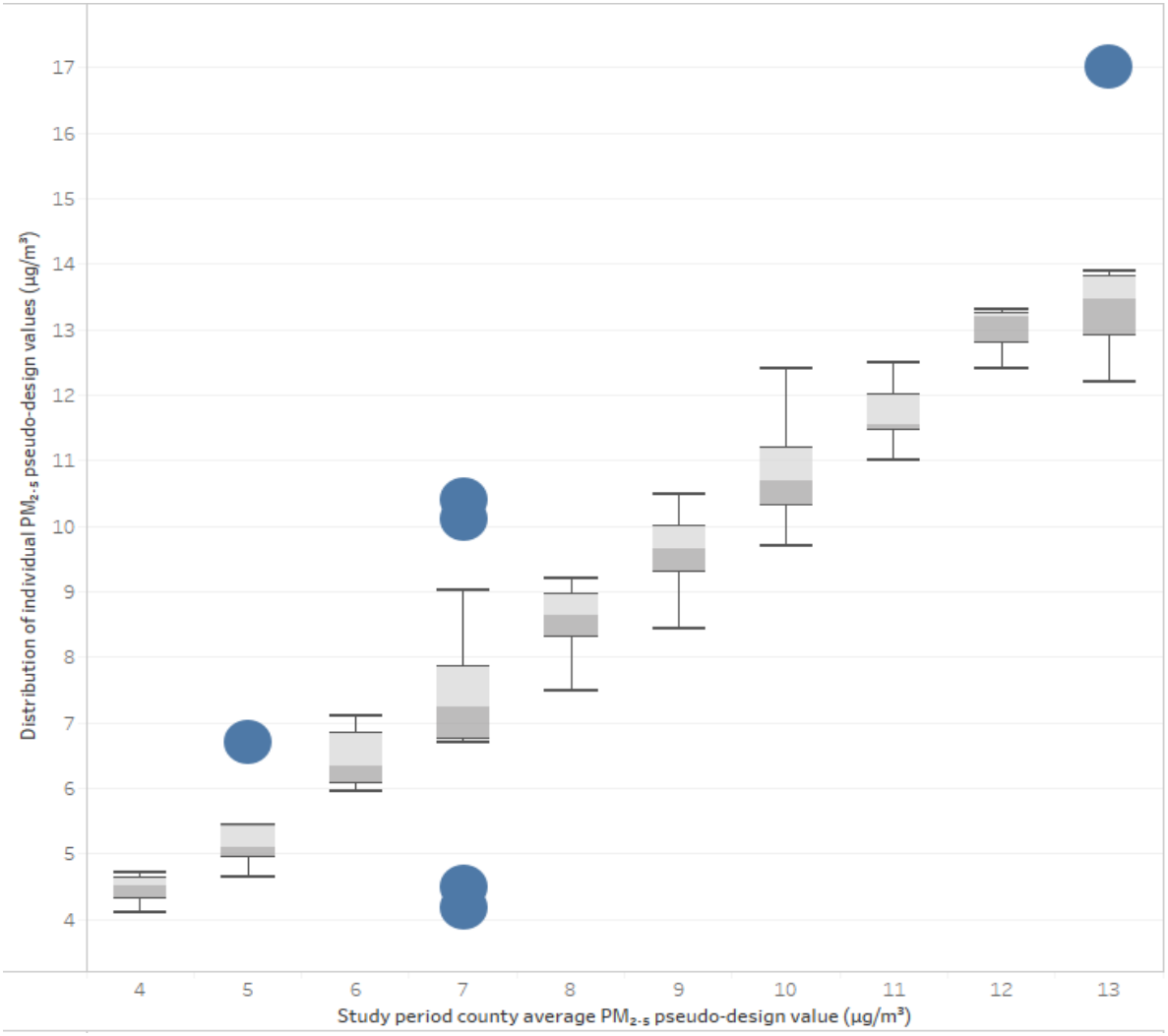


Figure B-11. County average pseudo-DV by 1 µg/m^3 and distribution of individual county pseudo-DVs within each 1 µg/m^3 interval for study counties in Shi et al., 2016. Note: 1 µg/m^3 intervals correspond to pseudo-DVs greater than 2.04 and less than or equal to 3.04, etc. Therefore, values distributions 11 and less correspond to county average pseudo-DVs that meet the annual standard of 12.04 µg/m^3.

Table B-10. County average pseudo-DVs by 1 µg/m^3 and distribution of county pseudo-DVs within each 1 µg/m^3 interval for study counties in Shi et al., 2016.

County average pseudo-DV $PM_{2.5}$ concentration (µg/m^3) interval	Count (percent) of pseudo-DV's ≤ 12.04 µg/m^3	Count (percent) of pseudo-DV's > 12.04 µg/m^3
$4.04 < PM_{2.5} \leq 5.04$	8 (100.00)	0 (0.00)
$5.04 < PM_{2.5} \leq 6.04$	5 (100.00)	0 (0.00)
$6.04 < PM_{2.5} \leq 7.04$	7 (100.00)	0 (0.00)
$7.04 < PM_{2.5} \leq 8.04$	16 (100.00)	0 (0.00)
$8.04 < PM_{2.5} \leq 9.04$	12 (100.00)	0 (0.00)
$9.04 < PM_{2.5} \leq 10.04$	26 (100.00)	0 (0.00)
$10.04 < PM_{2.5} \leq 11.04$	21 (95.45)	1 (0.00)
$11.04 < PM_{2.5} \leq 12.04$	7 (87.50)	1 (0.00)
$12.04 < PM_{2.5} \leq 13.04$	0 (0.00)	4 (0.00)
$13.04 < PM_{2.5} \leq 14.04$	0 (0.00)	8 (0.00)
Total	102 (88.0)	14 (12.0)

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APPENDIX C. SUPPLEMENTAL INFORMATION RELATED TO THE HUMAN HEALTH RISK ASSESSMENT

TABLE OF CONTENTS

This appendix provides supplemental information related to the risk assessment described in section 3.3 of draft 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 draft PM PA, our general approach to estimating $PM_{2.5}$-associated human health risks in this review utilizes concentration-response (CR) functions obtained from epidemiology studies to link ambient $PM_{2.5}$ 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 $PM_{2.5}$-attributable risk is well documented both in previous PM NAAQS-related risk assessments (section 3.1.2 of U.S. EPA, 2010) and in Appendix C of the BenMAP-CE User Manual detail and the reader is referred to those sources for additional detail on this subject.[1] 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 $PM_{2.5}$ concentrations corresponding to air quality scenarios of interest (section C.1.5).

[1] *https://www.epa.gov/benmap/benmap-ce-manual-and-appendices*

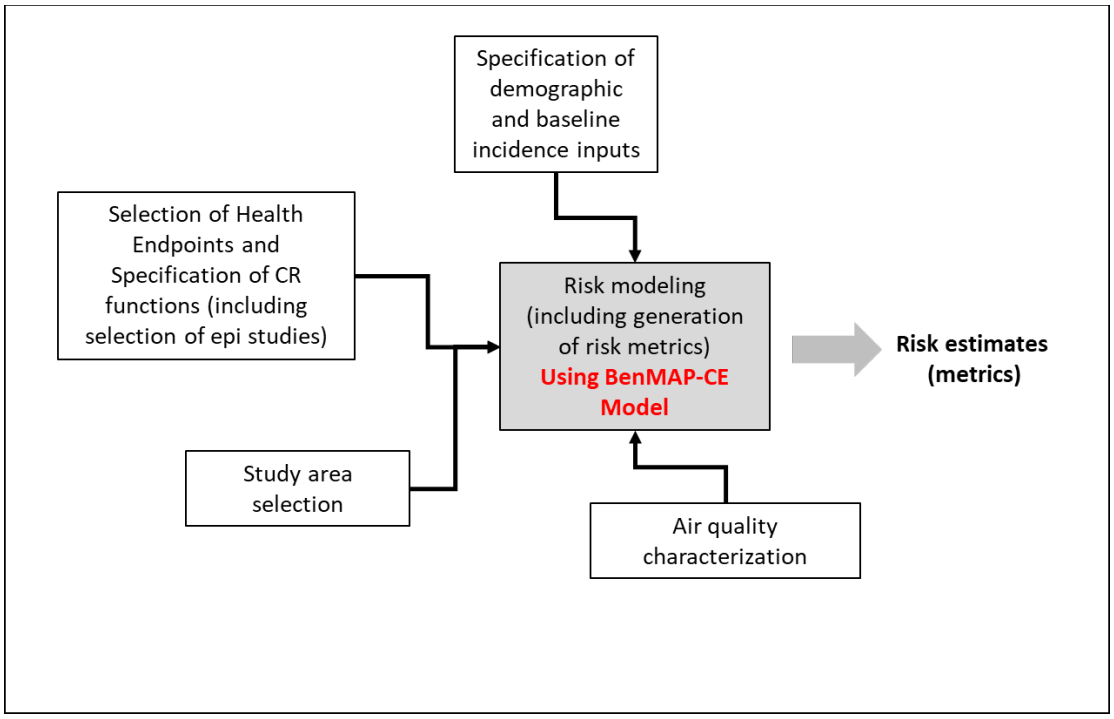

Figure C-1. **Key inputs to the risk assessment**

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 draft PM ISA determines the evidence supports either a "causal" or a "likely to be causal" relationship with short- or long-term $PM_{2.5}$ exposures (U.S. EPA, 2018a). As discussed in Chapter 3 of this draft PA, 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) in the draft ISA Table 3-1 (U.S. EPA, 2018a).

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 draft ISA. These studies are identified in section 3.2.3.2.1 of this draft 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 $PM_{2.5}$-associted 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 $PM_{2.5}$-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 $PM_{2.5}$ exposures based on hybrid modeling approaches:* For long-term $PM_{2.5}$ 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 $PM_{2.5}$ 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.
- *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 $PM_{2.5}$ 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.

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	Epidemiologic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Long-term exposure-related mortality studies										
Di et al., 2017 Air Pollution and Mortality in the Medicare Population	Exploring relationship between air pollution (ozone, $PM_{2.5}$) 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 $\mu g/m^3$ $PM_{2.5}$ 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 $\mu g/m^3$ applicable to only low-ozone days)	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 $PM_{2.5}$ modeled from remote sensing to those for $PM_{2.5}$ 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)	Relative risk (95 percent CI)	IHD	1.15 (1.11-1.19)	1.40E-02	1.78E-03
Pope et al., 2015 Relationships Between Fine Particulate Air Pollution, Cardiometabolic Disorders, and	Evaluates the relationship between long-term exposure to ambient $PM_{2.5}$ and CVD and cardiometabolic disease, including effect modification of the relationships by pre-existing cardiometabolic risk factors	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

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	Epidemiologic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Cardiovascular Mortality	- ACS (30+) (oversampled affluent individuals) - individual-level covariates				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 $PM_{2.5}$ and mortality given recent decline in U.S. ambient PM concentrations. Differentiation of risk for fossil fuel $PM_{2.5}$ versus total $PM_{2.5}$ - 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 $PM_{2.5}$ 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 $PM_{2.5}$ mortality hazard ratios (and 95th percentile CI) per 10 $\mu g/m^3$, 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
Turner et al., 2016 Long-Term Ozone Exposure and Mortality in a Large Prospective Study	Evaluates the relationship between long-term exposure to ambient $PM_{2.5}$ and all-cause and cause-specific mortality. Also, estimated the association between $PM_{2.5}$, regional $PM_{2.5}$, and near-source $PM_{2.5}$ 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 (95th percentile CI) for all-cause and cause-specific mortality in relation to each 10 unit increase in $PM_{2.5}$ 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
	Short-term exposure-related mortality studies									

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	Epidemiologic statistic	Mortality endpoint	Selected effect estimate	Selected beta	Selected beta standard error (SE)
Baxter et al., 2017 Influence of exposure differences in city-to-city heterogeneity in $PM_{2.5}$-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 $PM_{2.5}$ 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
Ito et al., 2013 NPACT study 3. Time-series analysis of mortality, hospitalizations, and ambient $PM_{2.5}$ and its components	Use factor analysis to characterize pollution sources, assess the association between $PM_{2.5}$ and $PM_{2.5}$ 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 $PM_{2.5}$ on hospitalizations and mortality in subjects with diabetes and neurological disorders	Estimates the effect of short-term exposure to $PM_{2.5}$ on all-cause mortality. Additionally, assesses the potential for pre-existing diseases to modify the association between $PM_{2.5}$ 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 $\mu g/m^3$ increase in the two days average $PM_{2.5}$: 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

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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[2]. The BenMAP-CE model[3] 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.[4]
- *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.[4]

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 $PM_{2.5}$ 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.
- *$PM_{2.5}$ 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 $PM_{2.5}$ standards. Inclusion of such areas in the risk assessment necessitates an upward adjustment to $PM_{2.5}$ air quality concentrations in order to simulate just meeting the current standards. Given uncertainty in how such increases could

[2] 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 0).

[3] *https://www.epa.gov/benmap*

[4] *https://www.epa.gov/benmap/benmap-ce-manual-and-appendices*

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 $\mu g/m^3$ for the annual standard and 5 $\mu g/m^3$ 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 $\mu g/m^3$, blue indicates areas that only exceed an annual design value of 10 $\mu g/m^3$, and red indicates areas that exceed both cutoffs.

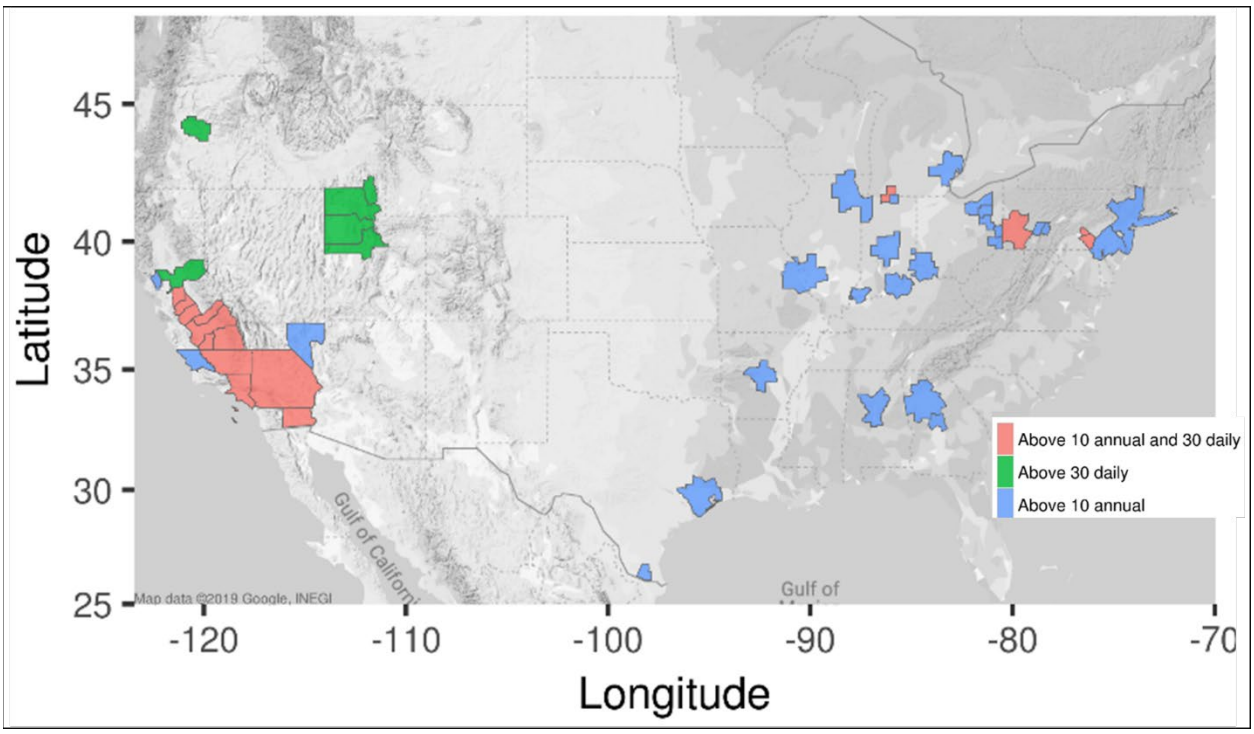


Figure C-2. **Map of the areas modeled in the risk assessment, colored by 2014-2016 $PM_{2.5}$ design values (DV).**

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-third of the total U.S. population above that age. Additional age-specific population information can be found in Table C-2.

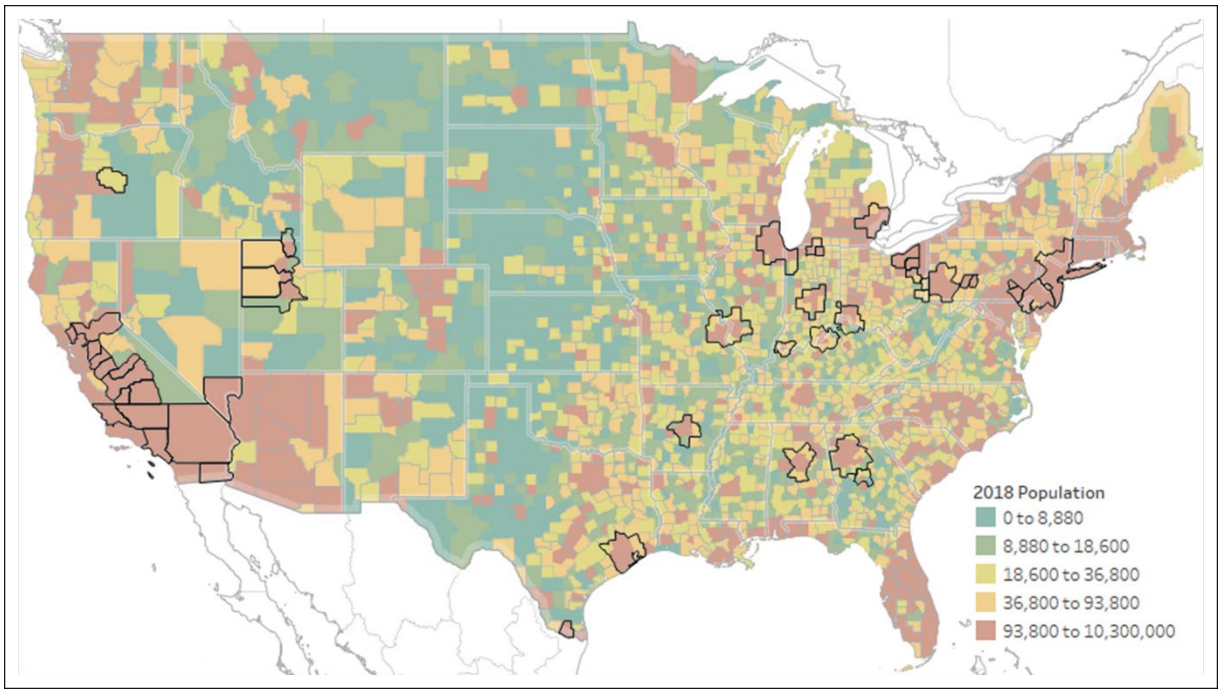


Figure C-3. **Map of the 2018 U.S. population by CBSA, with the selected urban study areas outlined.**

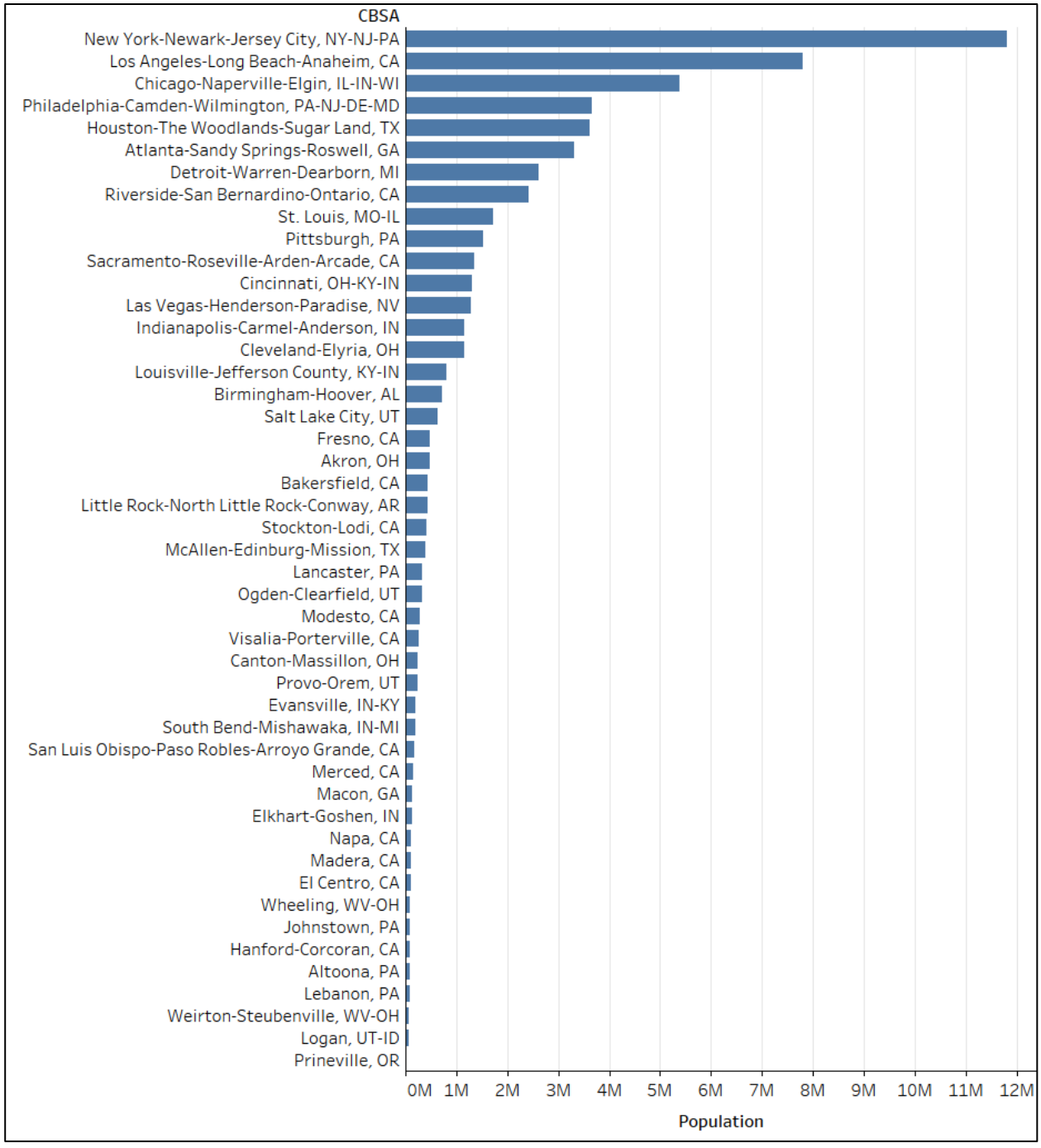
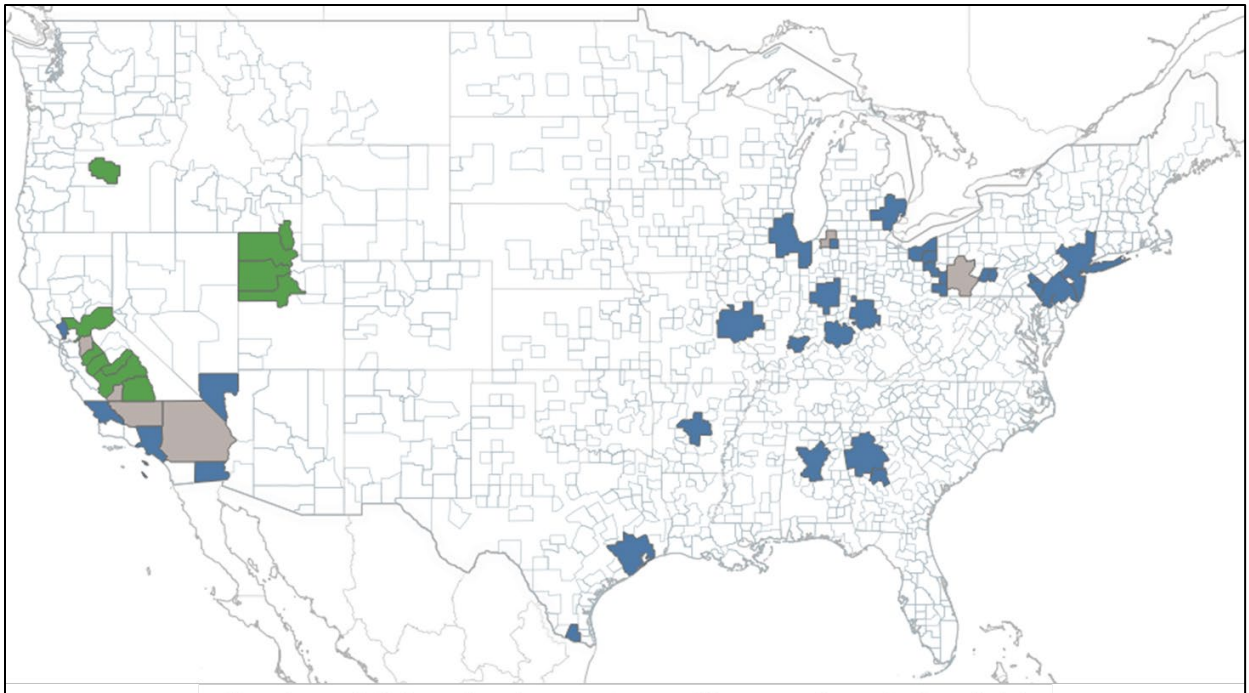


Figure C-4. **Population counts for ages 30 and above from each of the 47 CBSAs included in the risk assessment.**

Table C-2. Population of the 47 urban study areas striated 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 draft 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 $\mu g/m^3$), 11 study areas where just meeting the simulated standards is controlled by the current 24-hr standard (35 $\mu g/m^3$), 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).



Number of Urban Study Areas (CBSAs)	Controlling Standard	Population (≥30 years old)
30	Annual (Blue)	~50M
11	Daily (Green)	~4M
6	Mixed (Grey)	~5M
Total: 47		**~60M**

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 $PM_{2.5}$ in an area will depend on what emissions changes occur and the concentration gradients of $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ 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_2 and NO_X precursor emissions are additionally adjusted to simulate changes in secondarily formed $PM_{2.5}$.
- *Secondary PM-based modeling approach (Sec-PM):* This modeling approach simulates air quality scenarios of interest by preferentially adjusting SO_2 and NO_X precursor emissions to simulate changes in secondarily formed $PM_{2.5}$. In this case, the reductions in $PM_{2.5}$ 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 $PM_{2.5}$ 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 $\mu g/m^3$, respectively (section 3.3.1 of the draft 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 ug/m^3 and alternative annual standard of 10.0 ug/m^3 (section 3.3.1 of the draft 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^{th} percentile of 24-hr $PM_{2.5}$ 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 $PM_{2.5}$ 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, 3.2.3.2, and 3.4.2.4 in the draft 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

$PM_{2.5}$ standards. Tables containing $PM_{2.5}$ 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 $PM_{2.5}$ measurements were analyzed to characterize the magnitude and spatial distribution of $PM_{2.5}$ 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 $PM_{2.5}$ NAAQS[5] in specific areas. An overview of the approach is provided in Figure C-6. The process starts by acquiring $PM_{2.5}$ monitoring data from EPA's Air Quality System (AQS)[6] and simulating $PM_{2.5}$ concentrations with the Community Multiscale Air Quality (CMAQ)[7] 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)[8] to develop a baseline spatial field of $PM_{2.5}$ concentrations and relative response factors (RRFs) for projecting $PM_{2.5}$ concentrations, respectively (Figure C-6, Box 2). $PM_{2.5}$ concentrations are projected in two main steps using output from Downscaler and SMAT-CE (Figure C-6, Box 3). First, the $PM_{2.5}$ 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 $PM_{2.5}$ concentrations are projected using the area-specific percent emission change[9] 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.

[5] The phrase, "just meeting the $PM_{2.5}$ 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).

[6] *www.epa.gov/aqs*

[7] *www.epa.gov/cmaq*

[8] *www.epa.gov/scram/photochemical-modeling-tools*

[9] Scenarios based on a statistical projection approach were also developed for certain cases as discussed below.

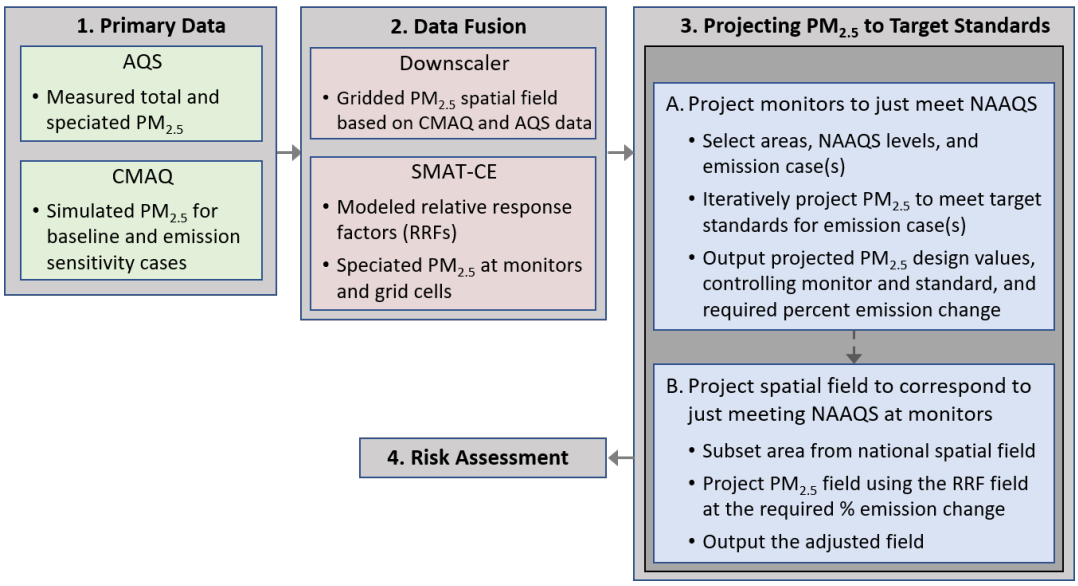


Figure C-6. **Overview of the system for projecting $PM_{2.5}$ concentrations to correspond to just meeting NAAQS.** See section C.1.4.6 and Kelly et al., 2019a for more details.

C.1.4.2 $PM_{2.5}$ Monitoring Data and Area Selection

The 2014-2016 DV period was the most recent period having a complete set of total and speciated $PM_{2.5}$ observations available at the time of the study. $PM_{2.5}$ 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 $PM_{2.5}$ 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[10], DVs for seven areas[11] appeared to meet the threshold due to the influence of wildfires. The influence of 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[12], but the monitor is in a small valley in Oakridge with very local high concentrations of $PM_{2.5}$ 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[13], 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[14]) and provides a precise target for the iterative projection calculations.

[10] "10/30" indicates an annual standard level of 10 μg m^{-3} and a 24-hr standard level of 30 μg m^{-3}

[11] Butte-Silver Bow, MT; Helena, MT; Kalispell, MT; Knoxville, TN; Medford, OR; Missoula, MT; and Yakima, WA

[12] The 410392013 monitor in Oakridge has a 24-hr 2014-2016 DV of 31 μg m^{-3}

[13] The 040213015 monitor in the Phoenix-Mesa-Scottsdale, AZ CBSA has 24-hr 2014-2016 DV of 31 μg m^{-3}

[14] 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

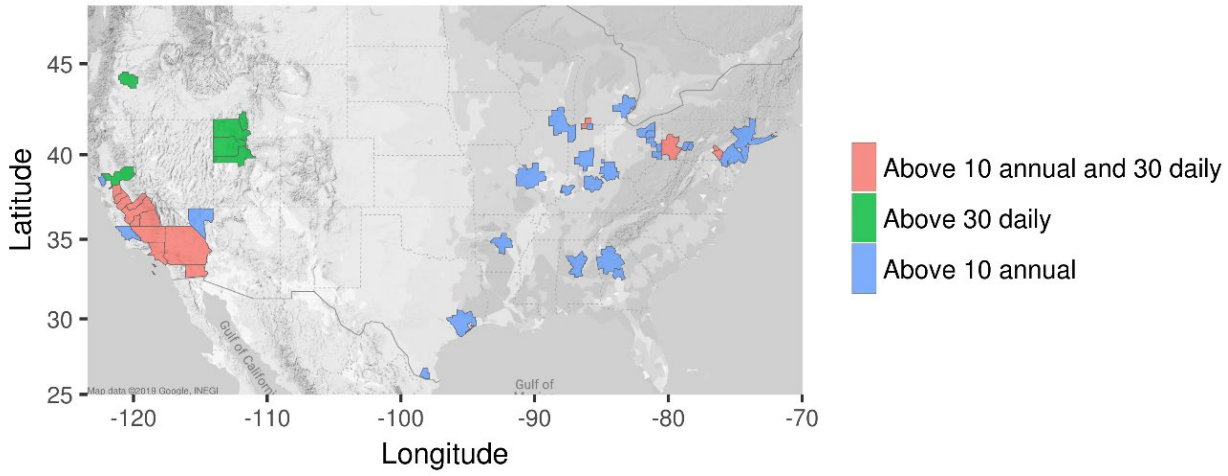


Figure C-7. **CBSAs selected for the risk assessment.** Colors indicate whether the maximum 2014-2016 DVs in the CBSA are above the annual (10 µg/m^3) and/or 24-hr (30 µg/m^3) selection criteria.

Table C-3. Maximum annual and 24-hr $PM_{2.5}$ 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 [a]	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 [a]	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
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

CBSA Name	# of Sites	Annual Max Site	Annual Max 14-16 DV	24-hr Max Site	24-hr Max 14-16 DV
Wheeling, WV-OH	2	540511002	10.24	540511002	22.5
[a] 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

CBSA Name	Associated Counties
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
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

CBSA Name	Associated Counties
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 $PM_{2.5}$ concentrations and estimates of how concentrations would respond to changes in $PM_{2.5}$ and $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ has been described in detail previously (Kelly et al., 2019b. Model performance statistics for $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ in the risk assessment. Model performance statistics for $PM_{2.5}$ by U.S. climate region and season are provided in Table C-6 and statistic definitions can be found in Table C-7.

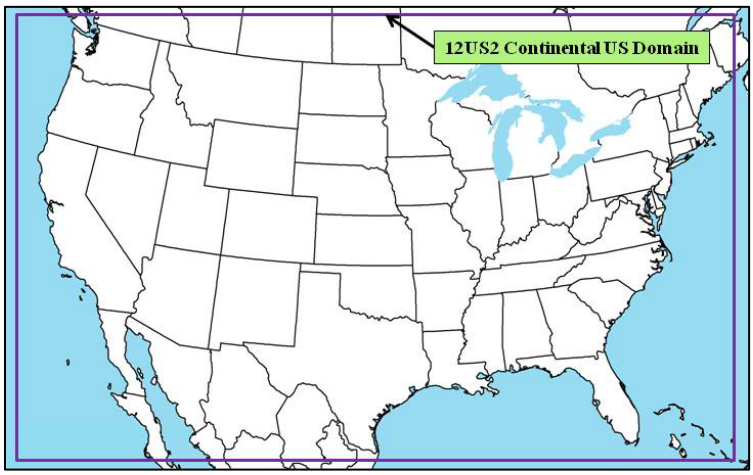


Figure C-9. **CMAQ modeling domain.**

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_3 surface exchange	Bi-directional NH_3 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[15,16] for $PM_{2.5}$ at AQS sites for the 2015 base case.

Region[16]	Season	N	Avg. Obs. ($\mu g\ m^{-3}$)	Avg. Mod. ($\mu g\ m^{-3}$)	MB[15] ($\mu g\ m^{-3}$)	NMB[15] (%)	RMSE[15] ($\mu g\ m^{-3}$)	NME[15] (%)	r[15]
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
	Annual	46157	8.22	8.76	0.54	6.6	4.75	39.1	0.55
Ohio Valley	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

[15] See Table C-7 for definition of statistics.

[16] See Figure C-10 for definition of regions.

Region[16]	Season	N	Avg. Obs. (μg m^{-3})	Avg. Mod. (μg m^{-3})	MB[15] (μg m^{-3})	NMB[15] (%)	RMSE[15] (μg m^{-3})	NME[15] (%)	r[15]
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 (μg 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 (μg m^{-3}) = $\sqrt{\sum_{i=1}^{n}(P_i - O_i)^2/n}$	Root mean-squared error (RMSE)
NMB (%) = $\frac{\sum_i^n(P_i - O_i)}{\sum_i^n O_i} \times 100$	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 (%) = $\frac{\sum_i^n\lvert P_i - O_i\rvert}{\sum_i^n O_i} \times 100$	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 = $\frac{\sum_{i=1}^{n}(P_i - \overline{P})(O_i - \overline{O})}{\sqrt{\sum_{i=1}^{n}(P_i - \bar{P})^2}\sqrt{\sum_{i=1}^{n}(O_i - \bar{O})^2}}$	Pearson correlation coefficient

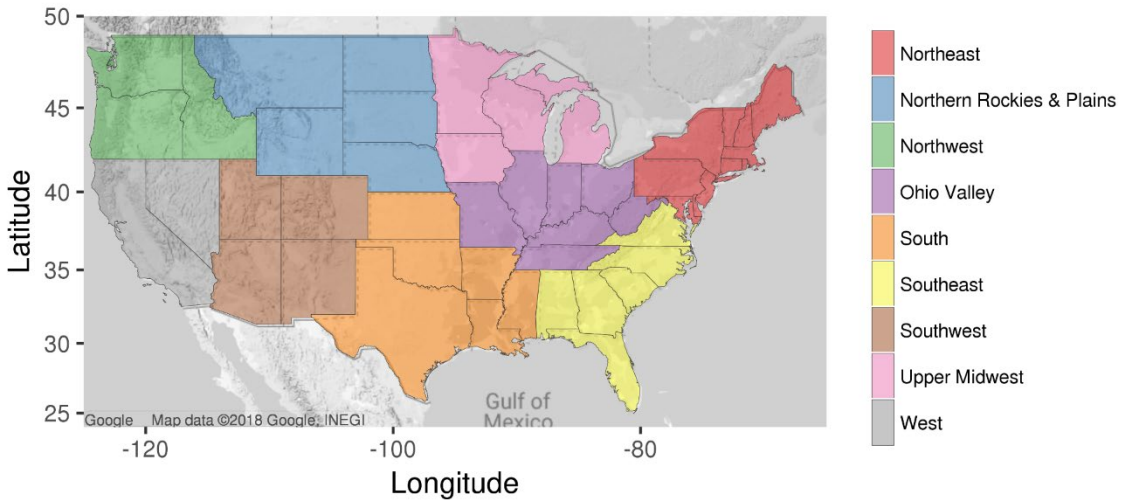


Figure C-10. **U.S. climate regions[17] used in the CMAQ model performance evaluation.**

To inform $PM_{2.5}$ projections, annual CMAQ modeling was conducted using the same configuration and inputs as the 2015 base case simulation but with anthropogenic emissions of primary $PM_{2.5}$ or NOx and SO_2 scaled by fixed percentages. Specifically, seven simulations were conducted with changes in anthropogenic NOx and SO_2 emissions (i.e., combined NOx and SO_2, not separate NOx and SO_2 simulations) of -100%, -75%, -50%, -25%, +25%, +50%, and +75. Two simulations were conducted with changes in anthropogenic $PM_{2.5}$ 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.

The two emission sensitivity scenarios (primary $PM_{2.5}$ and NOx and SO_2) were selected to span a wide range of possible $PM_{2.5}$ spatial response patterns. NOx and SO_2 emission changes influence concentrations of ammonium nitrate and ammonium sulfate, which are secondary pollutants that often have broad spatial distributions. Primary $PM_{2.5}$ emission changes have the greatest influence on $PM_{2.5}$ concentrations close to emission sources. The two distinctly different $PM_{2.5}$ response patterns for primary $PM_{2.5}$ and NOx and SO_2 emission changes enable $PM_{2.5}$ to be projected for a wide range of conditions. Projecting $PM_{2.5}$ for a wide range of conditions is desirable in this study because many $PM_{2.5}$ spatial response patterns can cause $PM_{2.5}$ concentrations to just meet NAAQS.

[17] *https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php*

C.1.4.4 Relative Response Factors for $PM_{2.5}$ Projection

The 2015 base case and sensitivity modeling results were used to develop RRFs for projecting $PM_{2.5}$ concentrations to correspond to just meeting NAAQS (Figure C-6, Box 2, "SMAT-CE"). Baseline $PM_{2.5}$ concentrations are projected by multiplication with RRFs. The RRF for a $PM_{2.5}$ 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 $PM_{2.5}$ species in the sensitivity simulation, and $C_{base,species}$ is the concentration of the $PM_{2.5}$ 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, 2018b). More details on the RRF projection method are provided in EPA's modeling guidance document (U.S. EPA, 2018b) 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 $PM_{2.5}$ were calculated from RRFs for the individual $PM_{2.5}$ species using observation-based estimates of $PM_{2.5}$ species concentrations in SMAT-CE output. Specifically, total $PM_{2.5}$ 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 $PM_{2.5}$ RRFs were used to project base-case $PM_{2.5}$ 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 $PM_{2.5}$ RRFs were generally based on application of the Sulfate, Adjusted Nitrate, Derived Water, Inferred Carbonaceous material balance approacH (SANDWICH) (Frank, 2006) to measurements of $PM_{2.5}$ species concentrations from the Chemical Speciation Network (CSN)[18] and the Interagency Monitoring of Protected Visual Environments (IMPROVE)[19] network. The SANDWICH method corrects for different artifacts in the measurements for $PM_{2.5}$ species and total $PM_{2.5}$. An alternative approach to calculating total $PM_{2.5}$ RRFs was applied for monitors and grid cells in California due to factors including missing data at the Bakersfield speciation monitor[20] throughout 2014

[18] *www.epa.gov/amtic/chemical-speciation-network-csn*

[19] *http://vista.cira.colostate.edu/Improve/*

[20] Site identification number: 060290014

and part of 2015. For projections in California, RRFs were calculated directly from the ratio of CMAQ $PM_{2.5}$ concentration predictions in the sensitivity simulation to the base simulation.

By default, $PM_{2.5}$ 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 $PM_{2.5}$ 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[21], 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[22]. Also, RRFs were set to one[23] in the third quarter (July-September) for select counties in the San Joaquin Vally and Utah[24] to better reflect the seasonal nature of $PM_{2.5}$ in these areas (i.e., $PM_{2.5}$ 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 $PM_{2.5}$ concentrations to the nearest percent emission change. $PM_{2.5}$ RRFs are shown in Figure C-11 and Figure C-12 as a function of changes in anthropogenic primary $PM_{2.5}$ and NOx and SO_2 emissions for monitors in the U.S. during the first and third calendar quarters. Spatial fields of $PM_{2.5}$ RRFs for 50% reductions in anthropogenic primary $PM_{2.5}$ and NOx and SO_2 emissions are shown in Figure C-13.

[21] 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.

[22] This variability is less of an issue in regional modeling applications where emission changes can be targeted to time periods of elevated $PM_{2.5}$ concentrations in the area.

[23] When the RRF is 1, the projected concentration equals the base concentration (equation 3).

[24] 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 $PM_{2.5}$ concentrations occur throughout the year compared with the other SJV counties.

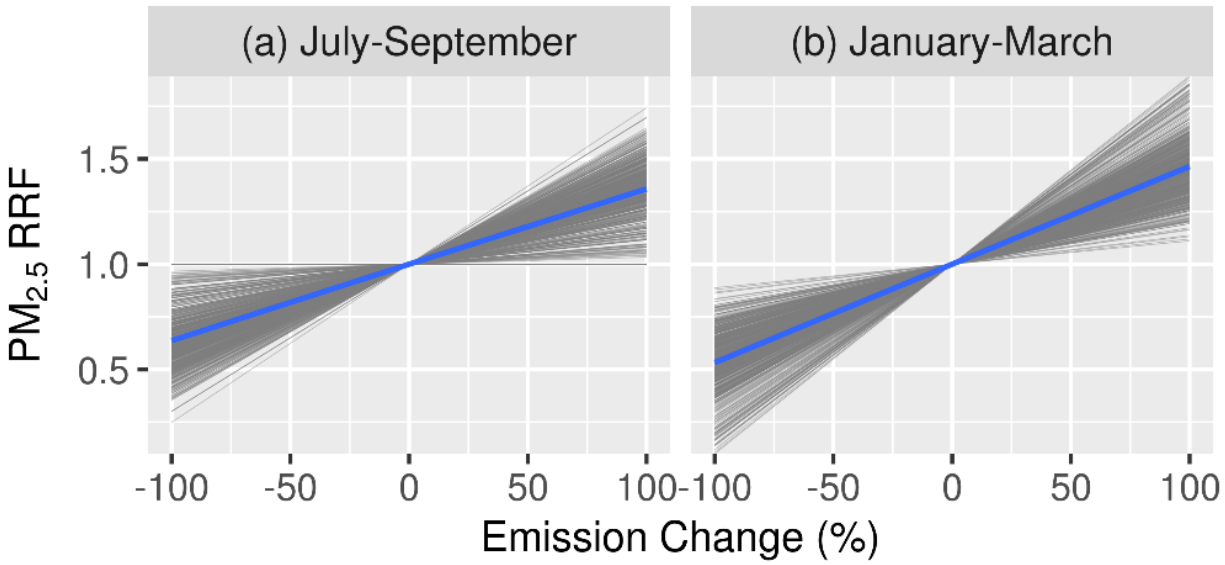


Figure C-11. **Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic primary $PM_{2.5}$ emissions for monitoring sites in the contiguous U.S.**

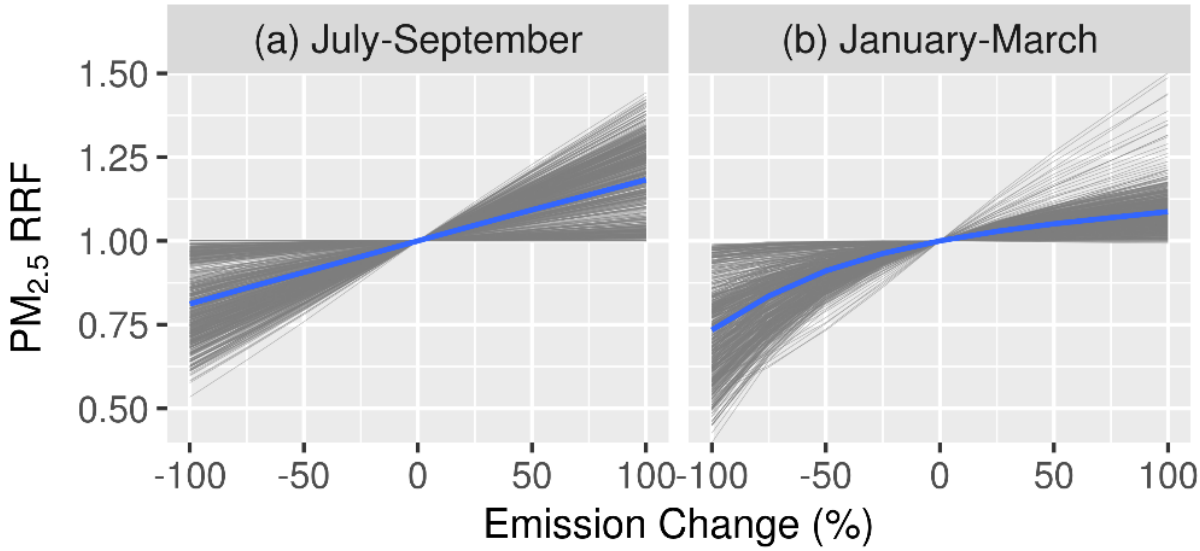


Figure C-12. **Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic NOx and SO_2 emissions for monitoring sites in the contiguous U.S.**

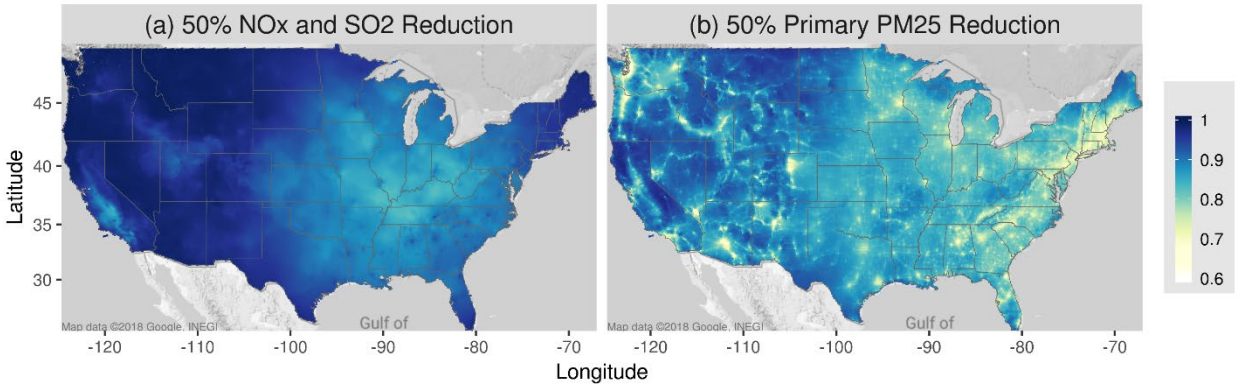


Figure C-13. **Annual average $PM_{2.5}$ RRFs at CMAQ grid-cell centers for 50% reductions in anthropogenic (a) NOx and SO_2 and (b) primary $PM_{2.5}$ emissions.**

C.1.4.5 2015 $PM_{2.5}$ Concentration Fields

To develop a baseline gridded $PM_{2.5}$ concentration field for projection with $PM_{2.5}$ RRFs, a Bayesian statistical model (i.e., Downscaler) was applied (Figure C-6, Box 2, "Downscaler") (Berrocal et al., 2012). Downscaler makes predictions of $PM_{2.5}$ concentrations to a spatial field of receptor points using $PM_{2.5}$ 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 $PM_{2.5}$ concentration over the U.S.

The Downscaler model is routinely applied by U.S. EPA to predict 24-hr average $PM_{2.5}$ concentrations at the centroids of census tracts in the contiguous U.S. (U.S. EPA, 2018c). 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, $PM_{2.5}$ measurements from the IMPROVE monitoring network were used in addition to measurements included in the AQS database. 24-hr average $PM_{2.5}$ concentrations were predicted for the 2015 period, and the 24-hr $PM_{2.5}$ fields were averaged to the quarterly periods of the $PM_{2.5}$ RRFs for use in projection.

Annual average $PM_{2.5}$ concentrations from the monitoring network and CMAQ simulation that were used in model fitting are shown in Figure C-14 along with the resulting Downscaler predictions. Cross-validation statistics are provided in Table C-8 based on comparisons of Downscaler predictions against the 10% of the observations that were randomly withheld from model fitting.

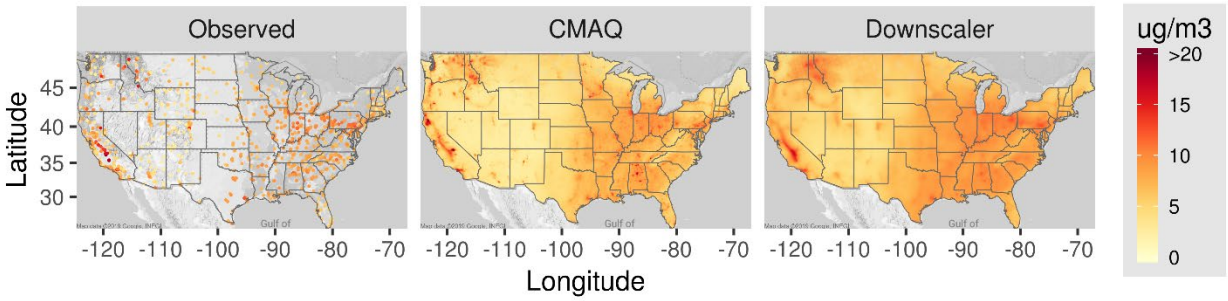


Figure C-14. **Annual average of the 2015 $PM_{2.5}$ observations and CMAQ predictions used in the Downscaler model, and the annual average of the Downscaler $PM_{2.5}$ predictions.**

Table C-8. Cross-validation statistics associated with the 2015 Downscaler predictions.

Number of Monitors	Mean Bias[a] ($\mu g\ m^{-3}$)	Root Mean Squared Error[b] ($\mu g\ m^{-3}$)	Mean Coverage[c]
1101	0.37	3.17	0.95

[a]The mean of all biases across the CV cases, where the bias of each prediction is the downscaler prediction minus the observed value.
[b]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.
[c]A value of 1 is assigned if the measured value lies in the 95th percentile CI of the Downscaler prediction (the Downscaler prediction ± 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 $PM_{2.5}$ to Just Meet the Standards

$PM_{2.5}$ 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)[25] and alternative (10/30)[26] 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 $PM_{2.5}$ RRF. $PM_{2.5}$ 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 (-

[25] Annual standard level of 12 $\mu g\ m^{-3}$ and 24-hr standard level of 35 $\mu g\ m^{-3}$

[26] Annual standard level of 10 $\mu g\ m^{-3}$ and 24-hr standard level of 30 $\mu g\ m^{-3}$

100 to 100%) were performed using bisection iteration until the difference between the 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[27]. In cases where the emission change needed to just meet the target annual or 24-hr standard was outside of the ± 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 ± 100%, then an alternative projection approach was used (discussed below).

Second, 2015 $PM_{2.5}$ 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 $PM_{2.5}$ concentrations based on Downscaler modeling with the gridded spatial fields of quarterly $PM_{2.5}$ RRFs corresponding to the percent emission change required to just meet the controlling standard. The projected fields of quarterly average $PM_{2.5}$ concentrations were then averaged to produce the annual average projected field.

Since $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ emission changes. For three CBSAs[28], standards could not be met using primary $PM_{2.5}$ emission reductions alone. $PM_{2.5}$ concentrations were projected for these areas using a combination of primary $PM_{2.5}$ and NOx and SO_2 emission reductions in the Primary PM scenario[29] (Figure C-15).

[27] 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).

[28] Bakersfield, Hanford-Corcoran, and Visalia-Porterville (all in California)

[29] This approach was applied by using RRFs from the NOx and SO_2 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 $PM_{2.5}$ emission sensitivity simulations to eliminate the remainder of the difference. The fraction of the difference eliminated with NOx and SO_2 emission reductions was as follows: 0.4 for Bakersfield, 0.5 for Visalia-Porterville, and 0.6 for Hanford-Corcoran

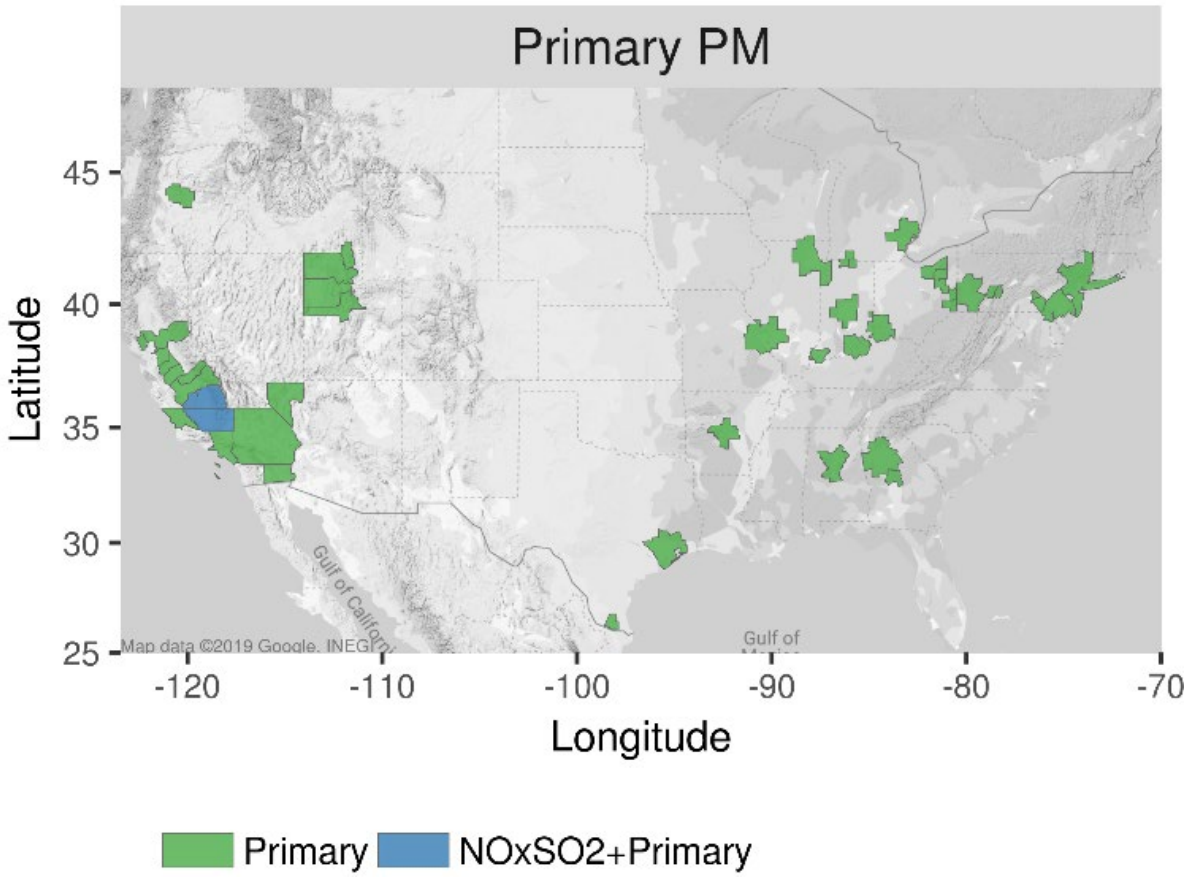


Figure C-15. **Projection method used for each CBSA in the "Primary PM" projection case. See text for details.**

The second scenario is referred to as "Secondary PM" or Sec-PM because projections were largely based on RRFs developed using CMAQ modeling with NOx and SO_2 emission changes, which affect concentrations of secondary PM components such as ammonium nitrate and ammonium sulfate. For 22 CBSAs[30], standards could not be just met using NOx and SO_2 emission changes alone. These areas were projected using the proportional scaling method[31] (Figure C-16). The proportional method was selected to gap-fill the Secondary PM case because

[30] 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

[31] 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 $PM_{2.5}$ 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).

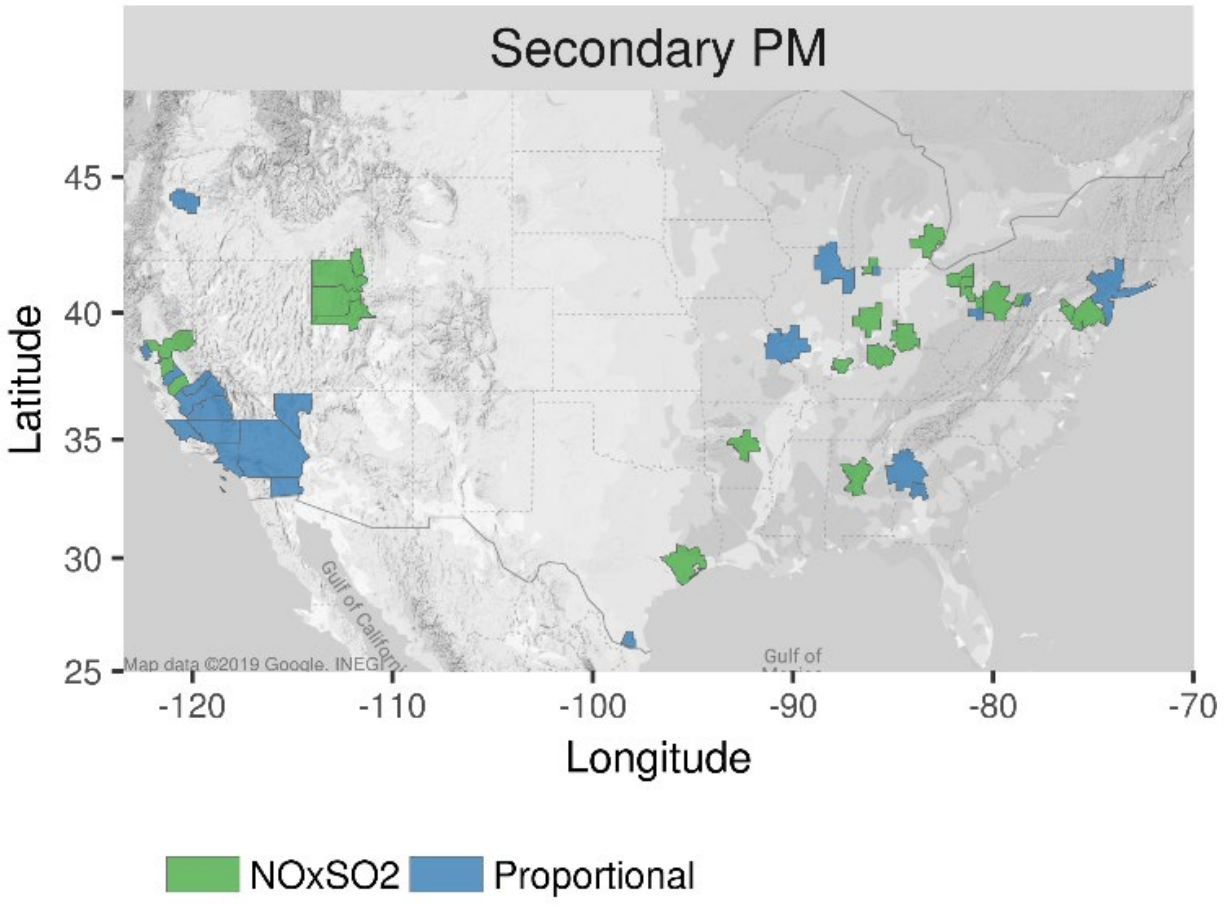


Figure C-16. **Projection method used for each CBSA in the "Secondary PM" projection case.**

The baseline 2015 concentration in the 47 CBSAs is shown in Figure C-17. These concentrations are the same as those in Figure C-14 but are shown only for the CBSAs included in the projections. In Figure C-18, 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-19, the difference between the annual concentration projected for the 10/30 case and the and 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-20. Baseline and projected $PM_{2.5}$ DVs for monitors in the 47 CBSAs are provided in Table C-32, Table C-33, Table C-34, and Table C-35 in section C.4.[32]

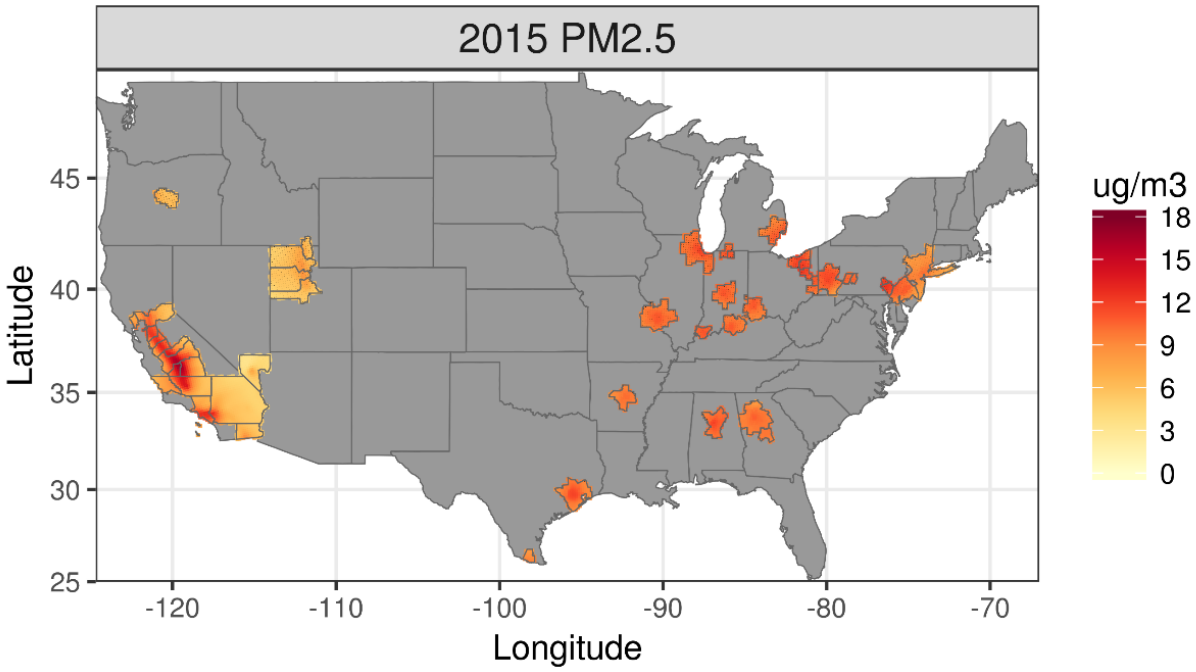


Figure C-17. **Annual average 2015 $PM_{2.5}$ concentrations in the 47 CBSAs based on Downscaler modeling.**

[32] 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 $PM_{2.5}$ 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.

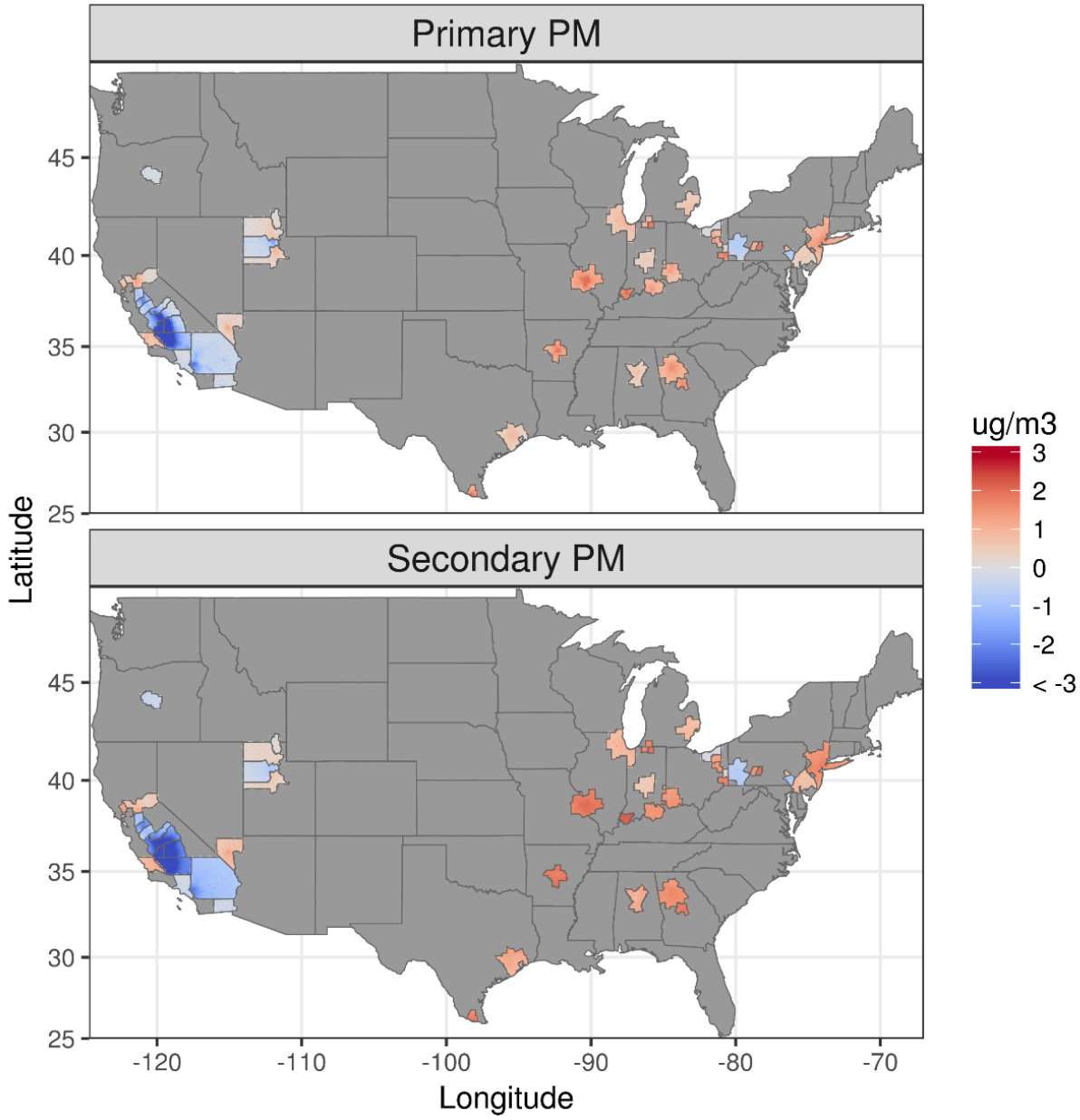


Figure C-18. **Difference between the annual average projected $PM_{2.5}$ concentrations and the 2015 baseline concentrations for the 12/35 projection cases (i.e., 12/35 – baseline).**

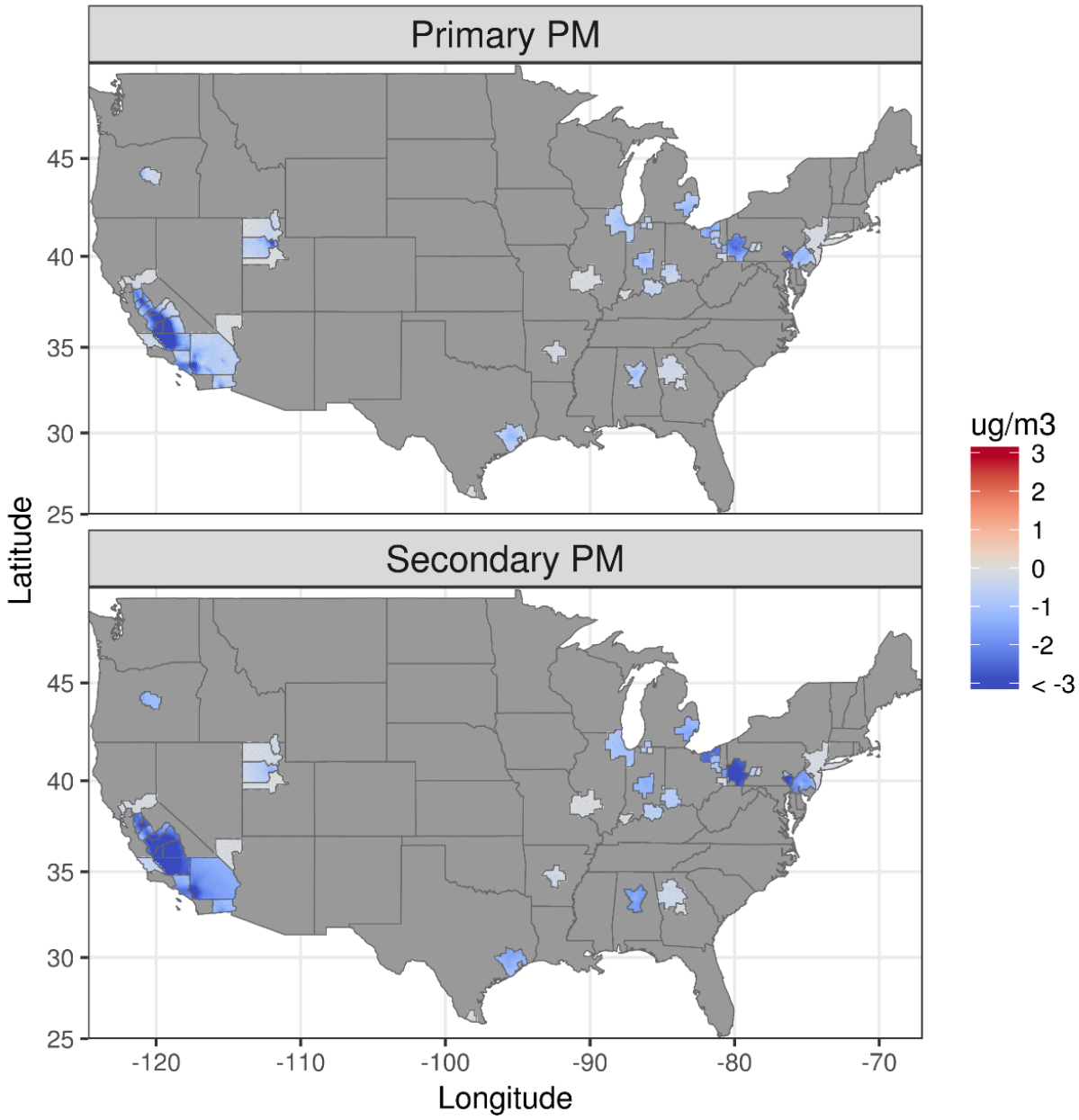


Figure C-19. **Difference between the annual average projected $PM_{2.5}$ concentrations and the 2015 baseline concentrations for the 10/30 projection cases (i.e., 10/30 – baseline).**

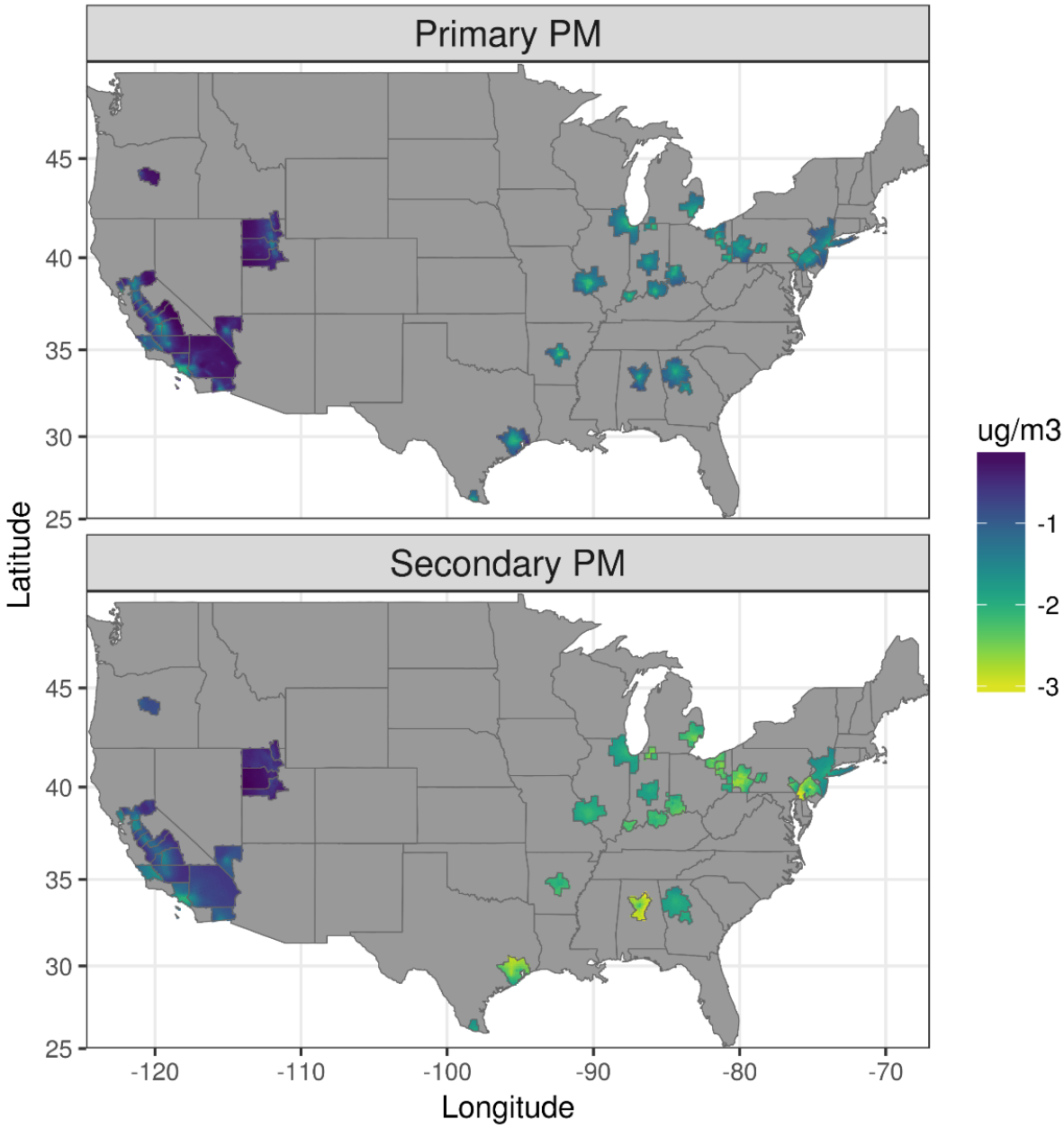


Figure C-20. **Difference between the annual average projected $PM_{2.5}$ concentrations in the 10/30 and 12/35 cases (i.e., 10/30 – 12/35) for the Primary PM and Secondary PM projection cases.**

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 $PM_{2.5}$ or NOx and SO_2 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 $PM_{2.5}$. 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 $PM_{2.5}$. A third limitation arises because many emission cases could be applied to project $PM_{2.5}$ 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.[33] 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-21. 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-

[33] 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 μg/m^3.

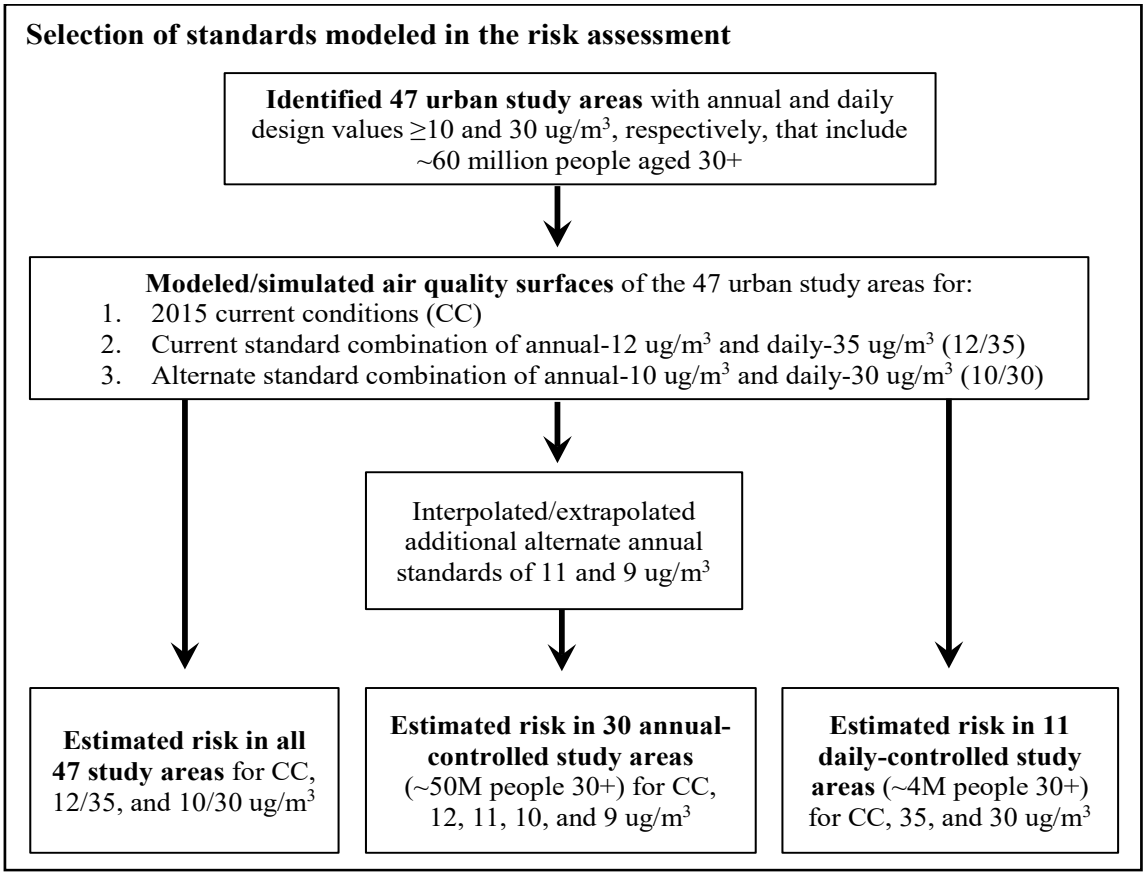


Figure C-21. **Flow diagram of risk assessment technical approach.**

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.2.2 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 $\mu g/m^3$ and 9.0 $\mu g/m^3$. 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.[34] 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

[34] 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-9 and Table C-10). Then we provide tables of individual endpoint- and study- specific CBSA-level risk estimates (Table C-11, Table C-12, Table C-13, Table C-14, Table C-15, Table C-16, Table C-17, Table C-18, and Table C-19).

Table C-9. 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^3)	Alternative 24-hr Standard (30 µg/m^3)
Long-term exposure related mortality						
IHD	Jerrett 2016	15,800	Pri-PM	16,500 (12,600-20,300)	14,400 (11,000-17,700)	16,400 (12,500-20,000)
		(12,100-19,400)	Sec-PM	16,800 (12,800-20,500)	14,200 (10,900-17,500)	16,500 (12,600-20,200)
	Pope 2015	14,900	Pri-PM	15,600 (11,600-19,400)	13,600 (10,100-17,000)	15,400 (11,500-19,200)
		(11,100-18,500)	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	Pri-PM	46,200 (45,000-47,500)	40,300 (39,200-41,400)	45,700 (44,500-47,000)
		(42,900-45,300)	Sec-PM	46,900 (45,600-48,200)	39,700 (38,600-40,800)	46,200 (44,900-47,500)
	Pope 2015	49,000	Pri-PM	51,300 (41,000-61,400)	44,700 (35,700-53,500)	50,700 (40,500-60,700)
		(39,200-58,700)	Sec-PM	52,100 (41,600-62,300)	44,000 (35,100-52,700)	51,300 (41,000-61,400)
	Thurston 2015	12,900	Pri-PM	13,500 (2,360-24,200)	11,700 (2,050-21,100)	13,300 (2,330-24,000)
		(2,250-23,100)	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	Pri-PM	3,890 (1,240-6,360)	3,390 (1,080-5,560)	3,850 (1,230-6,300)
		(1,180-6,060)	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	Pri-PM	2,490 (983-4,000)	2,160 (850-3,460)	2,460 (970-3,950)
		(936-3,810)	Sec-PM	2,530 (998-4,060)	2,120 (837-3,400)	2,490 (982-3,990)
	Ito 2013	1,120	Pri-PM	1,180 (-16-2,370)	1,020 (-14-2,050)	1,160 (-16-2,340)
		(-15-2,260)	Sec-PM	1,200 (-16-2,400)	1,000 (-14-2,020)	1,180 (-16-2,370)
	Zanobetti 2014	3,630	Pri-PM	3,810 (2,530-5,080)	3,300 (2,190-4,400)	3,760 (2,500-5,020)
		(2,410-4,840)	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-10. 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-11. 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	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)	
	(2015)	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
Totals	15,800	16,500	16,800	14,400	14,200	16,400	16,500	2,390	2,870	200	266

* 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 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-Steubenville, 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-13. CBSA level results for the 47 urban study areas using the Di et al., 2017 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	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
Totals	44,100	46,200	46,900	40,300	39,700	45,700	46,200	6,440	7,800	573	772

* 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 Pope et al., 2015 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	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
Totals	49,000	51,300	52,100	44,700	44,000	50,700	51,300	7,100	8,630	644	828

* 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 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
Totals	12,900	13,500	13,700	11,700	11,500	13,300	13,500	1,830	2,230	168	209

* 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 Turner et al., 2016 long-term lung cancer 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	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
Totals	3,700	3,890	3,950	3,390	3,330	3,850	3,890	548	670	42	61

* 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 Baxter et al., 2017 all-cause short-term 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	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
Totals	2,380	2,490	2,530	2,160	2,120	2,460	2,490	335	408	30	39

* 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 Ito et al., 2013 all-cause short-term 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	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
Totals	1,120	1,180	1,200	1,020	1,000	1,160	1,180	158	192	14	18

* 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 Zanobetti et al., 2014 all-cause short-term 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	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
Totals	3,630	3,810	3,870	3,300	3,250	3,760	3,810	513	622	46	62

* 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-20 and Table C-21) where the annual standard was controlling. Then we provide tables of individual endpoint- and study- specific CBSA-level risk estimates (Table C-22, Table C-23, Table C-24, Table C-25, Table C-26, Table C-27, Table C-28, Table C-29, and Table C-30).

Table C-20. 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 (10,200-16,300)	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)
			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 (9,340-15,600)	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)
			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 (36,000-38,000)	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)
			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 (32,800-49,100)	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)
			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 (1,880-19,300)	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)
			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 (1,000-5,160)	Pri-PM	3,400 (1,080-5,550)	3,160 (1,010-5,170)	2,920 (927-4,790)	2,670 (847-4,400)
			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 (784-3,190)	Pri-PM	2,150 (846-3,440)	1,990 (784-3,190)	1,830 (721-2,930)	1,670 (658-2,680)
			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 (-13-1,890)	Pri-PM	1,010 (-14-2,040)	939 (-13-1,880)	864 (-12-1,730)	789 (-11-1,580)
			Sec-PM	1,030 (-14-2,070)	940 (-13-1,890)	847 (-11-1,700)	754 (-10-1,510)
	Zanobetti 2014	3,040 (2,020-4,050)	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)
			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-21. 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-22. 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
Totals	13,300	14,300	14,600	13,300	13,300	12,300	12,100	11,300	10,900	1,140	1,400	2,270	2,770	3,390	4,130

* 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 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
Totals	12,500	13,500	13,700	12,500	12,600	11,600	11,400	10,600	10,200	1,070	1,310	2,130	2,600	3,180	3,880

* 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 Di et al., 2017 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
Totals	37,000	39,800	40,500	36,900	37,000	34,100	33,500	31,200	29,900	3,070	3,800	6,120	7,560	9,150	11,300

* 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 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
Totals	41,000	44,200	45,000	41,000	41,000	37,800	37,100	34,600	33,200	3,390	4,190	6,760	8,350	10,100	12,500

* 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 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
Totals	10,700	11,600	11,800	10,700	10,800	9,900	9,710	9,050	8,650	871	1,080	1,740	2,160	2,610	3,230

* 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 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
Totals	3,150	3,400	3,460	3,160	3,160	2,920	2,860	2,670	2,560	262	327	522	651	780	972

* 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 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
Totals	1,990	2,150	2,190	1,990	1,990	1,830	1,790	1,670	1,600	160	197	319	394	478	592

* 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 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
Totals	940	1,010	1,030	939	940	864	847	789	754	75	93	150	186	226	279

* 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 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
Totals	3,040	3,280	3,340	3,040	3,040	2,790	2,740	2,550	2,440	244	301	487	603	731	904

* 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 $PM_{2.5}$ Levels

The tables of risk results presented in section C.2.1 illustrate the estimated risk of premature death under current and alternative $PM_{2.5}$ 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-22, Figure C-23, Figure C-24, Figure C-25, and Figure C-26) include results for the full set of 47 urban study areas and the second set (Figure C-27 and Figure C-28) 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 $PM_{2.5}$ concentrations (Figure C-22 and Figure C-27). 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 $\mu g/m^3$) 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 $\mu g/m^3$.
- 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-23), (b) simulation of the current standard (12/35 $\mu g/m^3$ – Figure C-24), and (c) simulation of the change (delta) in risk between the current and alternative standards (10/30 $\mu g/m^3$) (Figure C-25). 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 $PM_{2.5}$ concentration (Figure C-26 and Figure C-28). 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 $PM_{2.5}$ 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.[35] 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.

[35] 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 $\mu g/m^3$ 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 $\mu g/m^3$, a single year can have grid cells with annual average concentrations above or below 12.0 $\mu g/m^3$.

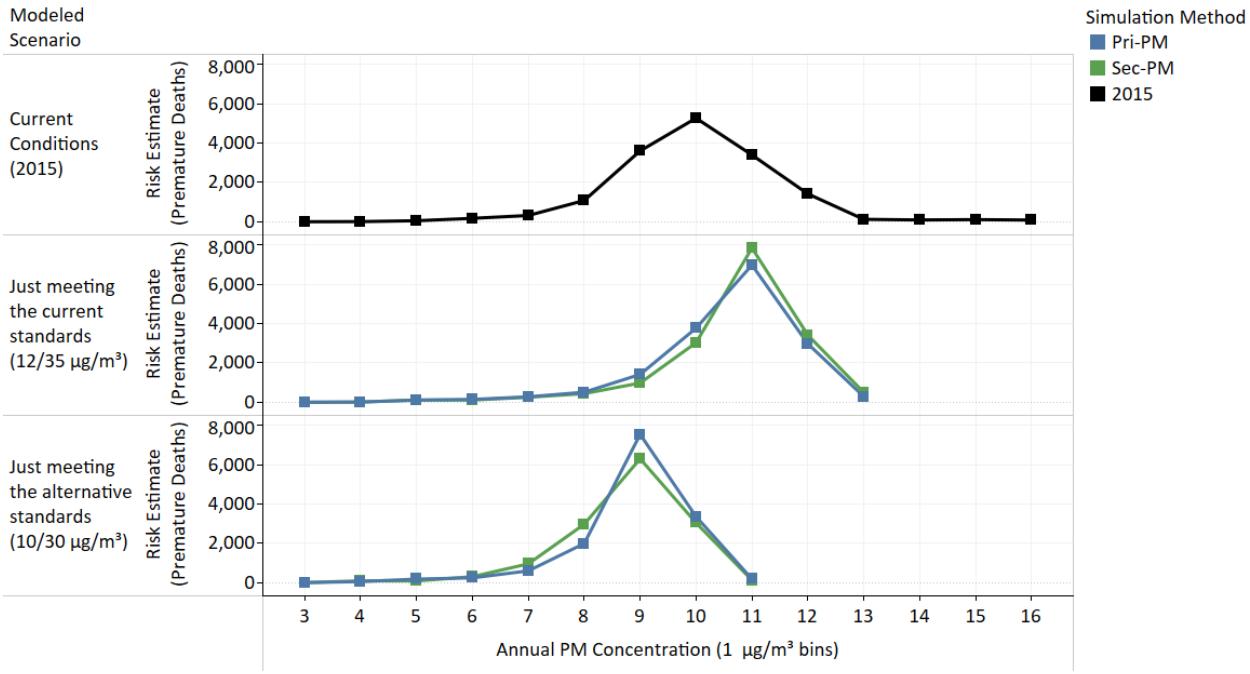


Figure C-22. **Distribution of estimated $PM_{2.5}$-associated mortality for current conditions (2015), current standards (12/35 µg/m^3), and alternative standards (10/30 µg/m^3) simulated for all 47 urban study areas.[36]**

[36] Risk is rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m^3 includes risk occuring at 10.0-10.9 µg/m^3). 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.

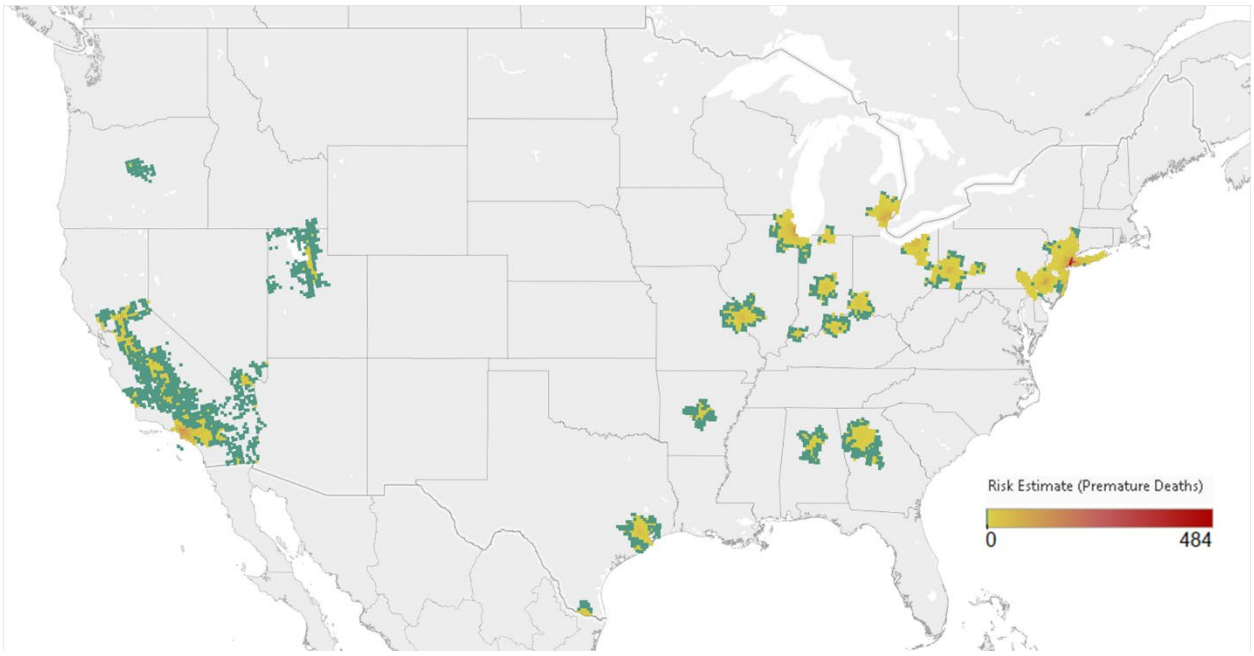


Figure C-23. **Estimated number of premature deaths (by 12 km grid cell) under 2015 current conditions in all 47 study areas.**

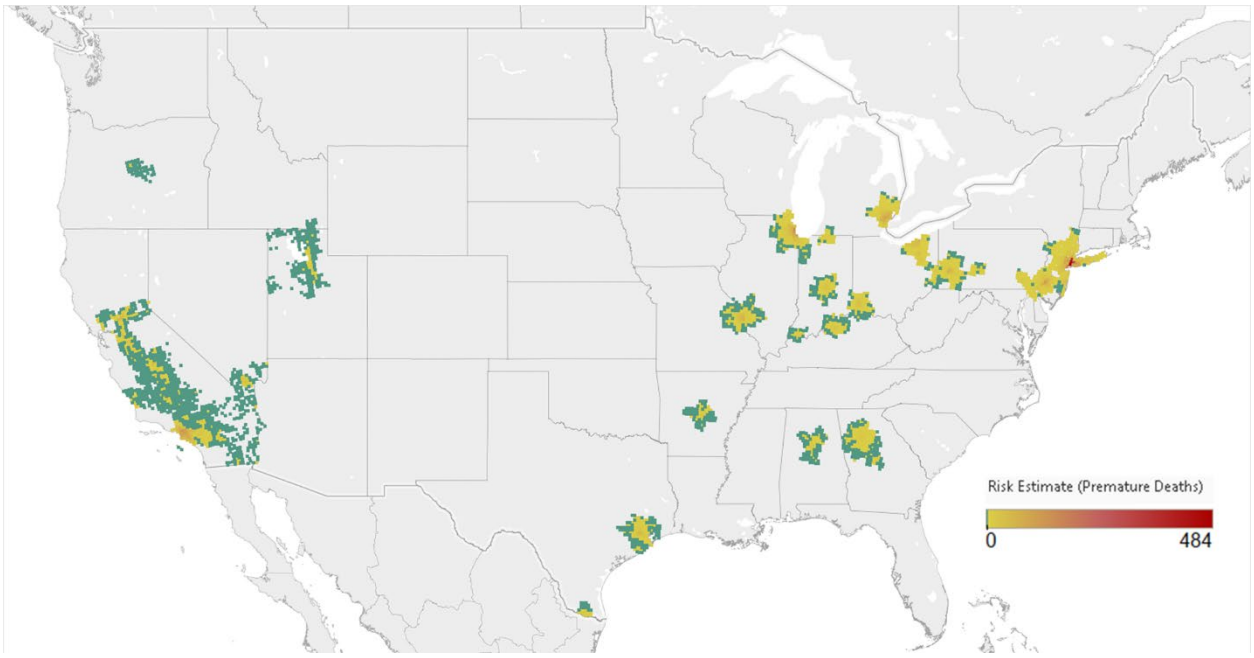


Figure C-24. **Estimated number of premature deaths (by 12 km grid cell) when just meeting the current PM standards (12/35) in all 47 study areas (Pri-PM simulation).**

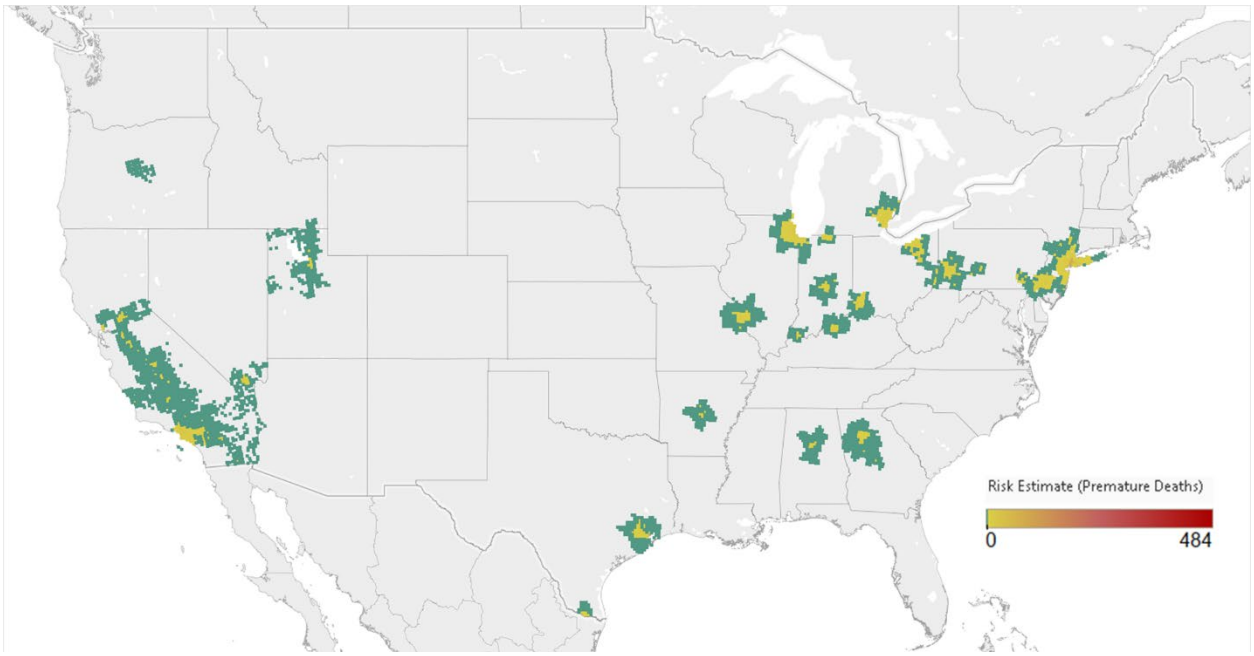


Figure C-25. **Estimated reduction in the number of premature deaths (by 12 km grid cell) when going from just meeting the current standards (12/35) to just meeting the alternative standards (10/30) in all 47 study areas (Pri-PM simulation).**

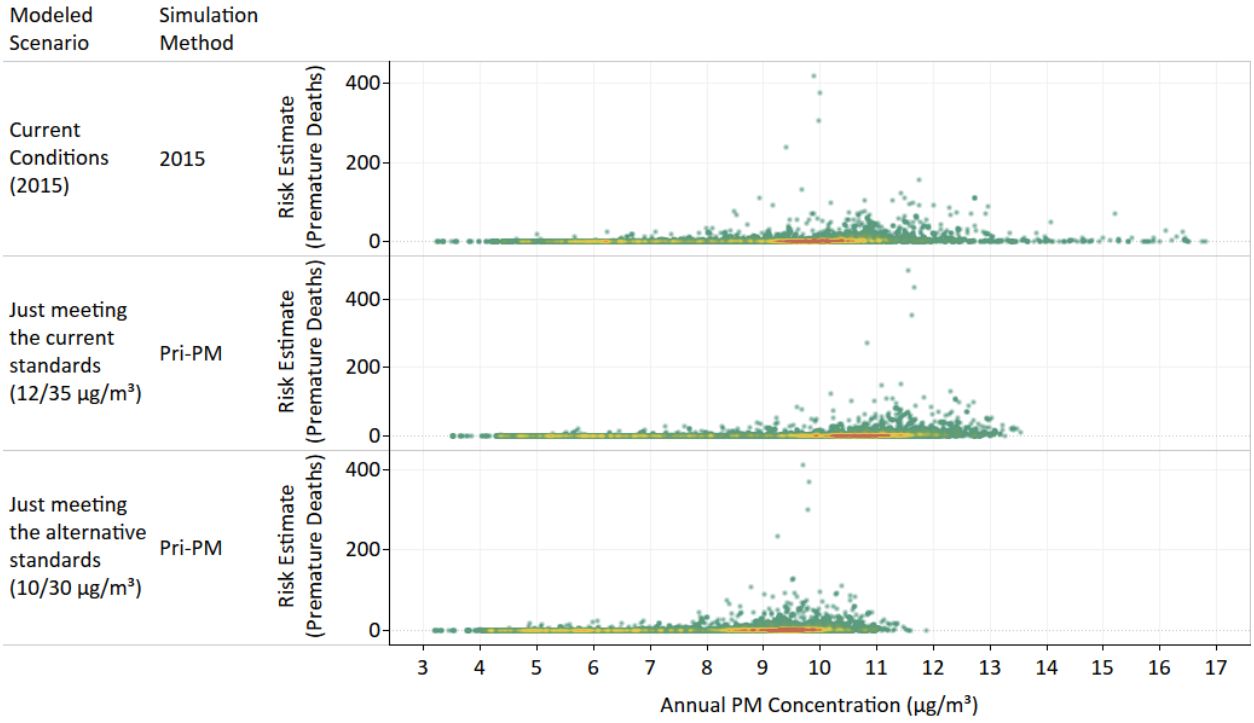


Figure C-26. **Distribution of estimated premature death (by 12 km grid cell) for the current standards (12/35 µg/m^3), alternative standards (10/30 µg/m^3), and current conditions (2015) for all 47 urban study areas (Pri-PM simulation).**

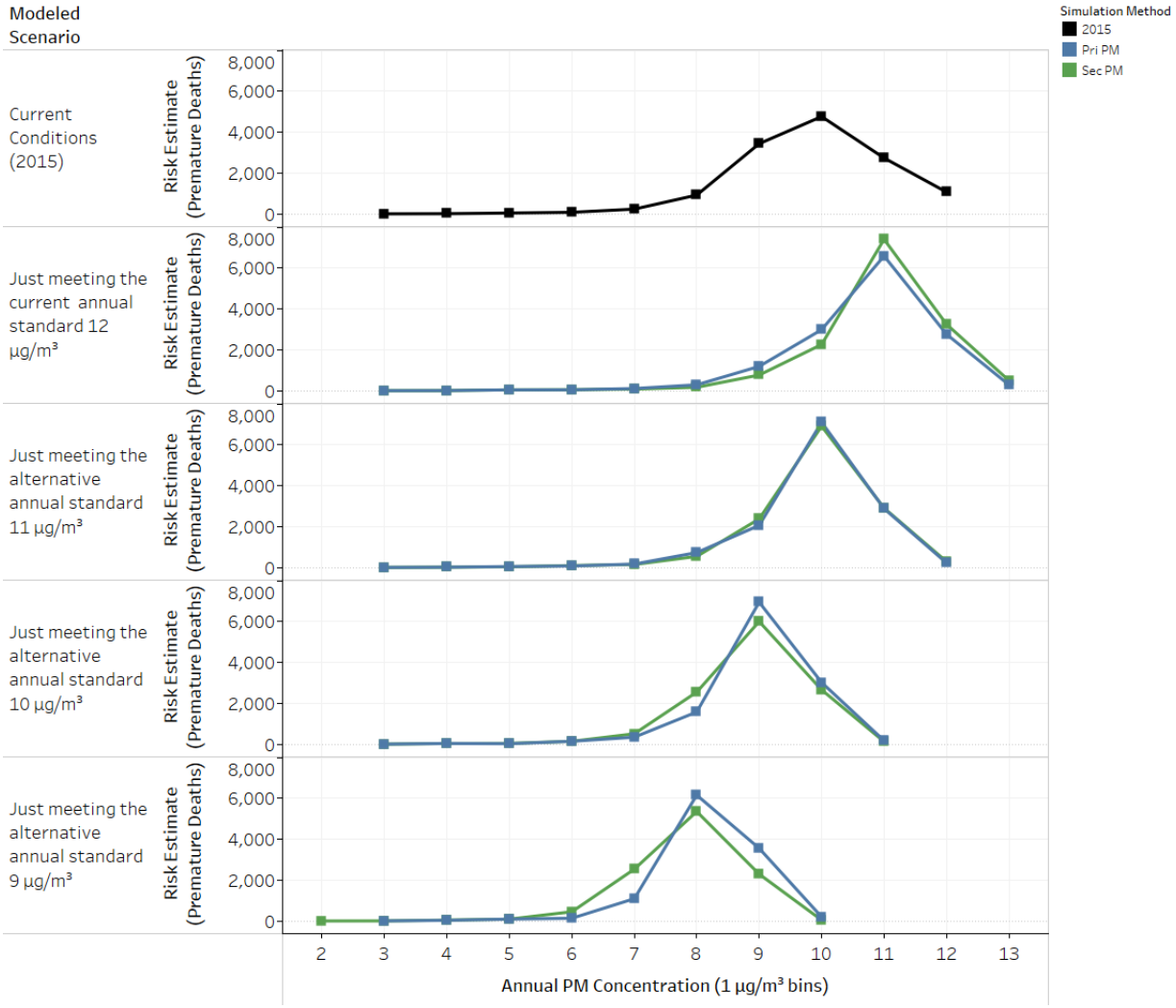


Figure C-27. **Distribution of estimated $PM_{2.5}$-associated mortality for current conditions (2015), the current annual standard (12/35 µg/m³), and alternative standards (9.0, 10.0, and 11.0 µg/m³) simulated for the 30 annual-controlled urban study areas.[37]**

[37] Risk is rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m³ includes risk occuring 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.

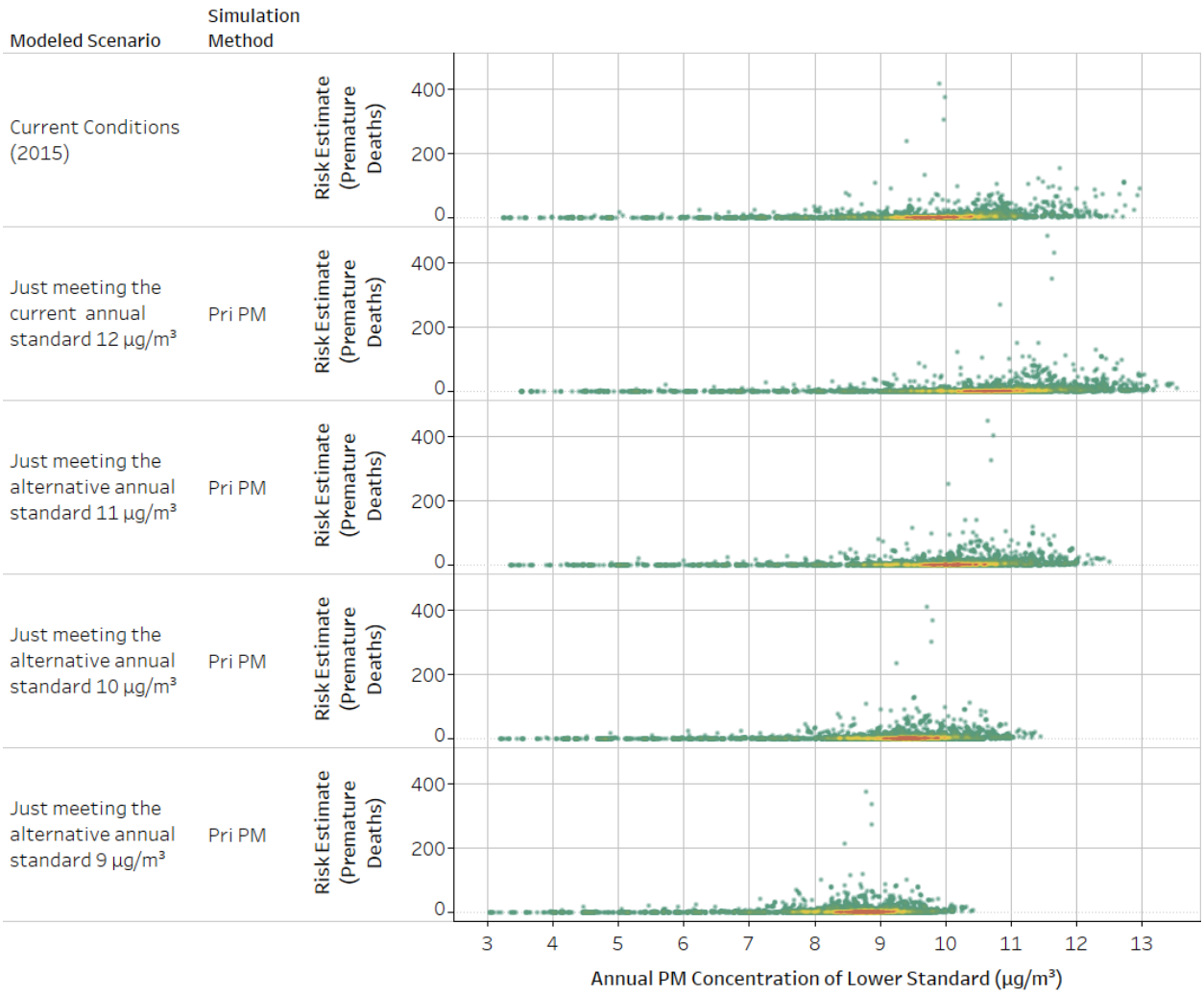


Figure C-28. **Distribution of estimated premature death (by 12 km grid cell) for current conditions (2015), the current annual standard (12.0 µg/m^3), alternative annual standards (9.0, 10.0, 11.0 µg/m^3), and for all 47 urban study areas (Pri-PM simulation).**

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 $PM_{2.5}$ standards, long-term annual mortality ranges up to 52,100 premature deaths (all-cause, based on Pope), including 16,800 IHD-related deaths (based on Jerrett) and 3,950 lung cancer deaths (based on Turner) 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) for the full set of 47 urban study areas (Table C-9).

- In considering the alternative suite of standards (10/30 $\mu g/m^3$) 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-9 and Table C-10).
- Across the full set of alternative annual standards modeled including 11, 10 and 9 $\mu g/m^3$ (each evaluated for the 30 annually-controlled study areas), we see a consistent reduction in mortality (Table C-20 and Table C-21). In addition, we note that these risk reductions are associated with iteratively lower ambient $PM_{2.5}$ concentrations, such that with the lowest annual standard considered (9 $\mu g/m^3$) the majority of remaining risk occurs in grid cells with ambient $PM_{2.5}$ concentrations between 7 and 9 $\mu g/m^3$. In contrast, most of the risk occurring under the current standard occurs in grid cells with ambient concentrations in the range of 10-12 $\mu g/m^3$ (Figure C-26).
- Patterns of risk reduction seen in the summary (aggregated) risk results tables presented both in PA section 3.2 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 $PM_{2.5}$ 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-26 and Figure C-28), we note that risk reductions are estimated in grid cells encompassing a wide range of $PM_{2.5}$ concentrations. This includes grid cells with typical (i.e. frequently occuring) 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 $PM_{2.5}$ risk assessment.

Variability in the risk of $PM_{2.5}$-associated mortality could result from a number of factors. These can include variation in $PM_{2.5}$ 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 $PM_{2.5}$ itself (e.g., concentrations, air quality patterns) and in the ambient pollutants that co-occur with $PM_{2.5}$. In the $PM_{2.5}$ risk assessment discussed in this draft 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 $PM_{2.5}$ health effect associations for either annual or daily $PM_{2.5}$ 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 $PM_{2.5}$ exposures (e.g., hybrid modeling approaches, monitors) and to control for potential confounders. In selecting areas in which to estimate $PM_{2.5}$-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 $PM_{2.5}$ air quality, reflecting the potential for changes in ambient $PM_{2.5}$ concentrations to be influenced by changes in primary $PM_{2.5}$ 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 quantitively 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 $PM_{2.5}$ exposure and all-cause mortality based on effect estimates from Ito et al., 2013 versus long-term $PM_{2.5}$ exposure IHD mortality estimates based on Jerrett et al., 2016, respectively—see tables presenting risk estimates in section 3.3.2 of this draft 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 $PM_{2.5}$ 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, 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., 2017 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. (2017) 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 $PM_{2.5}$ emissions and based on adjusted precursor emissions that contribute to secondary $PM_{2.5}$ formation, the risk assessment provides insight into the potential significance of this source of uncertainty. As discussed in section 3.3.2 of this draft 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-31).

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-31. 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 standard levels using model (Downscaler) based methods	a) The baseline and adjusted 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 would introduce uncertainty into the $PM_{2.5}$ concentration estimates 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 $PM_{2.5}$ or NO_x and SO_2 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. 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 adjusts $PM_{2.5}$ concentrations to just meet standards.	The direction of bias associated with the sources of uncertainty is not obvious	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 $PM_{2.5}$ 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 likely relatively uncertain compared with broad features of the national $PM_{2.5}$ distributions.
b) Simulating just meeting alternative annual standards (9 and 11 ug/m^3) through use of linear extrapolation/ interpolation	The use of interpolation/extrapolation in simulating just meeting the two annual standards introduces uncertainty into the risk assessment since it does not fully capture potential non-linearities associated with just meeting annual standard levels (e.g., formation of secondary $PM_{2.5}$).	both	Medium	We have assigned a medium degree of magnitude to this source of uncertainty but recognize that different elements of this step of extrapolation/interpolation could be subject to varying levels of uncertainty. For example, extrapolation in generating the surface for 9 µg/m^3 is likely subject to greater uncertainty than interpolation for 11 µg/m^3 (since the former extends below the extent of modeled results, while the latter is estimating a surface between two sets of modeled results). Similarly, use of linear extrapolation/interpolation to generate surfaces based on the primary-PM modeled surfaces (for current

Source of Uncertainty	Description	Direction	Magnitude	Comments
				standard and 10 μg/m^3) is likely subject to less uncertainty than using the linear assumption in generating the two surfaces based on the secondary-PM modeled surfaces (for current conditions and 10 μg/m^3) since the latter focuses on secondary formation which could involve a higher degree of non-linearity.
c) 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. There are several potential sources of uncertainty associated with this approach, including (a) degree to which the use of 12 km grid cells to characterizes population-level exposure introduces potential exposure measurement error (e.g., if actual patterns of residential and work-related behavior reflect a spatial scale either larger or smaller than captured by the 12 km grid cell structure) and (b) degree of which the 12 km grid cell framework diverges from the grid framework used in the epidemiology studies providing effect estimates for the risk assessment.	Both	Medium	The degree to which the 12 km grid cell template introduces exposure measurement error will depend on actual patterns of day-to-day mobility, which may well vary across the urban study areas included in the risk assessment. Regarding uncertainty reflecting a potential mismatch between the 12 km grid template used in the risk assessment and the exposure surrogate structure utilized in the actual epidemiology studies, we note that 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. However, generally, this mismatch is not considered to be significant. 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.
d) 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. Similarly, uncertainty can be introduced into the risk assessment if (a) the 12 km grid does not fully capture population-level exposure (which for short-term exposure-related mortality may require more refined patterns of micro-level exposure to be captured) and (b) there is a disconnect between the spatial template used in the underlying short-term epidemiology studies and the 12 km grid template used in the risk assessment.	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 then reflected in the epidemiology studies supplying the effect estimates, which does introduce uncertainty into the analysis.

Source of Uncertainty	Description	Direction	Magnitude	Comments
e) Temporal mismatch between ambient air quality data characterizing exposure and mortality in long-term exposure-related epidemiology studies	Several of the epidemiology studies for long-term exposure-related mortality have a mismatch between the time period associated with ambient $PM_{2.5}$ concentrations used to characterize population-level exposure and mortality data (i.e., the ambient $PM_{2.5}$ data reflects a period following the mortality period specifically for ACS-based assessments – see Jerrett et al., 2016 and Pope et al., 2015)	Both	Low	As long as the overall spatial pattern of ambient $PM_{2.5}$ 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 $PM_{2.5}$ level used in characterizing exposure should not introduce significant uncertainty into the epidemiology studies and associated effect estimates.
f) Shape and corresponding statistical uncertainty around the CR function for long-term and short-term exposure-related mortality (especially at lower ambient PM levels)	Exposure measurement error along with other factors (sample size, control for confounders etc.) can affect the overall confidence associated with fitting specific function to the data. In addition, the degree of confidence in the fit of the function may vary across the range of ambient PM data involved with overall confidence reduced where there is reduced data.	Both	Medium-High	The ISA concludes with regard to long-term exposure-related (nonaccidental) mortality that the majority of evidence supports a linear, no-threshold concentration-response relationship, though some recent evidence indicates the possibility of a nonlinear concentration-response function (U.S. EPA, 2018a). 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 $PM_{2.5}$ concentrations generally below 8 $\mu g/m^3$ because data density is reduced below this concentration (section 11.2.4, p. 11-87). Given that a portion of risk modeling in the risk assessment does involve locations with ambient $PM_{2.5}$ concentrations below 8 ug/m^3 (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^3). With regard to short-term exposure-related mortality, the ISA concludes that, while difficulties remain in assessing the shape of the $PM_{2.5}$-mortality concentration-response relationship, as identified in the 2009 PM ISA, and studies have not conducted systematic evaluations of alternatives to linearity, recent studies continue to provide evidence of a no-threshold linear relationship, with less confidence at concentrations lower than 5 $\mu g/m^3$. Additionally, those studies that conducted analyses focused on examining associations at lower $PM_{2.5}$ concentrations provide initial evidence indicating that associations persist and may be larger in magnitude (i.e., a steeper slope) at lower $PM_{2.5}$ concentrations (section 11.1.10, p. 11-41, USEPA, 2018).

Source of Uncertainty	Description	Direction	Magnitude	Comments
g) Potential co-pollutant confounding on PM risk estimates	The potential for other pollutants (e.g., NO_2, SO_2 and ozone) to confound the $PM_{2.5}$-mortality relationship (related to both short-term and long-term exposure), thereby introducing uncertainty into the analysis.	Both	Low-Medium	With regard to short-term exposure-related mortality, associations with $PM_{2.5}$ were relatively unchanged in copollutant models across the various study locations examined. (ISA section p 1-46, U.S. EPA, 2018a). With regard to long-term exposure-related mortality, overall, associations remained relatively unchanged in copollutant models for total (nonaccidental) mortality, cardiovascular, and respiratory (ISA Figure 11-18, U.S. EPA, 2018a). Studies focusing on copollutant models with NO_2, $PM_{10-2.5}$, SO_2 and benzene were examined in individual studies, and across these studies the $PM_{2.5}$-mortality association was relatively unchanged (ISA Figure 11-19, U.S. EPA, 2018a) .
h) Compositional and source differences in PM	The composition of $PM_{2.5}$ can differ across study areas reflecting underlying differences in primary and secondary $PM_{2.5}$ sources (both natural and anthropogenic). If these compositional differences in fact translate into significant differences in public health impact (per unit concentration in ambient air) for $PM_{2.5}$ then significant uncertainty may be introduced into risk assessments if these compositional differences are not explicitly addressed.	Both	Low	The Integrated Synthesis chapter of the draft ISA (Chapter 1, p. 1-1, U.S. EPA, 2018a) state that, the assessment of PM sources and components confirms and continues to support the conclusion from the 2009 PM ISA: Many $PM_{2.5}$ components and sources are associated with many health effects, and the evidence does not indicate that any one source or component is more strongly related with health effects than $PM_{2.5}$ mass.
i) Lag structure in short-term exposure-related mortality epidemiology studies	It can be difficult to characterize the timing associated with specific $PM_{2.5}$-related health effects and consequently specify the lag-structure that should be used in modeling those health effects. This can introduce uncertainty into the modeling of risk for short-term exposure-related endpoints.	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 $PM_{2.5}$ on cardiovascular morbidity outcomes, specifically those 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). (ISA, section 1.5.2.2, pp. 1-48, 1-49, U.S. EPA, 2018a)

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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 $PM_{2.5}$ 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 (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 $PM_{2.5}$ exposure and risk, and that they reflect consideration of input from experts on PM exposure and risk through CASAC and public reviews.

C.4 $PM_{2.5}$ DESIGN VALUES FOR THE AIR QUALITY PROJECTIONS

Table C-32. $PM_{2.5}$ DVs for the Primary PM projection case and 12/35 standard level.

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
ElCent	060250005	Annual	Yes	0	12	12.63	12.00	33.5	31.3
ElCent	060251003	Annual	No	0	12	7.44	7.01	19.8	18.5
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
LosAng	060590007	Annual	No	0	5	9.63	9.37	-	-
LosAng	060374004	Annual	No	0	5	10.25	9.97	27.3	26.7
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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
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

[a] CBSA names are the first six characters of the full CBSAs names in Table C-3.
[b] Percent reduction in NOx and SO_2 emissions associated with just meeting the standard in this case.
[c] Percent reduction in Primary $PM_{2.5}$ emissions associated with just meeting the standard in this case.

Table C-33. $PM_{2.5}$ DVs for the Secondary PM projection case and 12/35 standard level.

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Chicag	170313301	Annual	No	N/A	N/A	10.37	11.25	23.5	25.5
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Evansv	181630023	Annual	Yes	-89	0	10.11	12.03	21.5	32.5
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
Hanfor	060310004	24-hr	Yes	N/A	N/A	21.98	10.81	72.0	35.4
Hanfor	060311004	24-hr	No	N/A	N/A	16.49	8.11	58.9	29.0
Housto	482011035	Annual	Yes	-91	0	11.19	12.04	22.4	25.2
Housto	482011039	Annual	No	-91	0	9.22	10.16	21.7	24.9
Housto	482010058	Annual	No	-91	0	9.67	10.52	22.3	24.8
Housto	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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
LosAng	060371302	Annual	No	N/A	N/A	11.99	11.66	31.5	30.6
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
OgdenC	490030003	24-hr	No	-53	0	6.35	6.70	-	-
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
SanLui	060792004	Annual	No	N/A	N/A	8.25	9.28	19.8	22.3
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

[a] CBSA names are the first six characters of the full CBSAs names in Table C-3.
[b] Percent reduction in NOx and SO_2 emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.
[c] Percent reduction in Primary $PM_{2.5}$ emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

Table C-34. $PM_{2.5}$ DVs for the Primary PM projection case and 10/30 standard level.

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Chicag	170313301	Annual	No	0	18	10.37	9.38	23.5	21.3
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Evansv	181630023	Annual	Yes	0	2	10.11	10.02	21.5	21.5
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
LosAng	060371302	Annual	No	0	34	11.99	9.64	31.5	27.0
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
OgdenC	490030003	24-hr	No	0	15	6.35	6.02	-	-
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
SanLui	060792004	Annual	No	0	22	8.25	7.76	19.8	19.2
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

[a] CBSA names are the first six characters of the full CBSAs names in Table C-3.
[b] Percent reduction in NOx and SO_2 emissions associated with just meeting the standard in this case.
[c] Percent reduction in Primary $PM_{2.5}$ emissions associated with just meeting the standard in this case.

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Table C-35. $PM_{2.5}$ DVs for the Secondary PM projection case and 10/30 standard level.

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Chicag	170313301	Annual	No	N/A	N/A	10.37	9.38	23.5	21.3
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

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
Evansv	181630023	Annual	Yes	3	0	10.11	10.03	21.5	21.2
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
Hanfor	060310004	24-hr	Yes	N/A	N/A	21.98	9.28	72.0	30.4
Hanfor	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

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LosAng	060371302	Annual	No	N/A	N/A	11.99	9.72	31.5	25.5
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
OgdenC	490030003	24-hr	No	29	0	6.35	6.10	-	-
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	-	-

CBSA [a]	Site	Controlling Standard	Controlling Site?	NOx & SO_2 Reduction (%) [b]	Primary $PM_{2.5}$ Reduction (%) [c]	Base Annual DV ($\mu g\ m^{-3}$)	Projected Annual DV ($\mu g\ m^{-3}$)	Base 24-hr DV ($\mu g\ m^{-3}$)	Projected 24-hr DV ($\mu g\ m^{-3}$)
SanLui	060792004	Annual	No	N/A	N/A	8.25	7.74	19.8	18.6
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

[a] CBSA names are the first six characters of the full CBSAs names in Table C-3.
[b] Percent reduction in NOx and SO_2 emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.
[c] Percent reduction in Primary $PM_{2.5}$ 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 $PM_{2.5}$ 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.[1] 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[2] and sea salt[3] terms in the equation and using a multiplier of 1.6 for converting OC to OM.[4]

[1] 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).

[2] $PM_{2.5}$ 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, $PM_{10-2.5}$ 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 country. Moreover, at the time of the last review, there were few data available from continuous $PM_{10-2.5}$ monitors to quantify the contribution of coarse PM to calculated light extinction.

[3] 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 $PM_{2.5}$ light extinction was generally well below 5% for $PM_{2.5}$ light extinction greater than 24 dv (U.S. EPA, 2010, p. 3-22; U.S. EPA, 2012, p. IV-5).

[4] 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, $PM_{2.5}$ 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,[5] the 2012 analysis explored the relationship between the 3-year design values for the existing 24-hour $PM_{2.5}$ standard and values of the 3-year visibility metric.[6] The analysis indicated that increases in 24-hour $PM_{2.5}$ 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 $PM_{2.5}$ 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 $PM_{2.5}$ standard of 35 $\mu g/m^3$ 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 $PM_{2.5}$ design value[7] 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_{10} 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.

[5] 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).

[6] 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).

[7] 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.

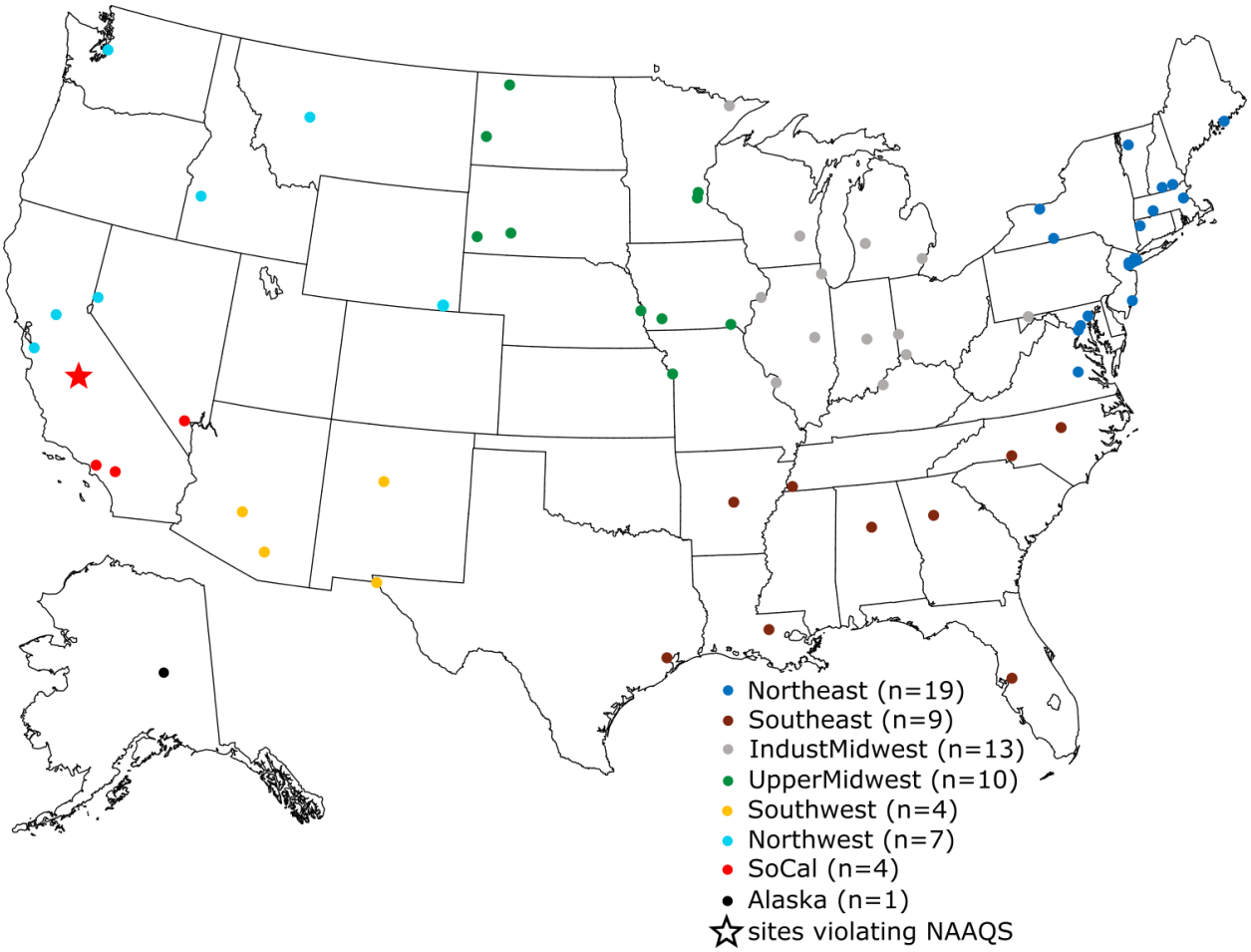

Figure D-1. Locations of monitoring sites with data for 2015-2017 with a valid $PM_{2.5}$ design value and meeting completeness criteria for PM species.

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 $PM_{2.5}$ measurements. The $PM_{2.5}$ and $PM_{2.5}$ 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 $PM_{2.5}$ Concentrations

The raw data for $PM_{2.5}$ site-level daily mass concentrations came from an Air Quality System (AQS)[8] 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[9] or at least 18 valid hourly concentrations measurements.

D.2.1.3 Coarse PM Concentrations

The raw data for $PM_{10-2.5}$ 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 ≥ 11 valid days for each quarter of 2015-2017.

D.2.1.4 $PM_{2.5}$ Component Concentrations

The raw data for $PM_{2.5}$ 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 ≥ 11 valid days for each quarter of 2015-2017.

[8] The Air Quality System is an EPA database of ambient air quality monitoring data (*https://www.epa.gov/aqs*).

[9] 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. $PM_{2.5}$ components from AQS used in IMPROVE equations.

$PM_{2.5}$ Component Drawn from AQS	AQS Parameter Code
Sulfate	88403
Nitrate	88306
OC (TOR[a])	88320, 88370
EC (TOR[a])	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)
[a] 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 $PM_{2.5}$ Design Values

Files for 24-hour $PM_{2.5}$ design values for 2015-2017 are located at *https://www.epa.gov/air-trends/air-quality-design-values*. Data handling of the 2015-2017 $PM_{2.5}$ 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_{10} Design Values

Files for 24-hour PM_{10} 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_{10} design values is described in Appendix K of 40 CFR Part 50.

D.2.1.7 Annual $PM_{2.5}$ Design Values

Files for annual $PM_{2.5}$ design values for 2015-2017 are located at *https://www.epa.gov/air-trends/air-quality-design-values*. Data handling of the 2015-2017 $PM_{2.5}$ 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, $PM_{2.5}$ 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.

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 µg/m^3 of ammonium sulfate,

[AN] is concentration in µg/m^3 of ammonium nitrate,

[OM] is concentration in µg/m^3 of organic matter,

[EC] is concentration in µg/m^3 of elemental carbon,

[FS] is concentration in µg/m^3 of fine soil,

[CM] is concentrations in µg/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 [a]
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 *http://vista.cira.colostate.edu/Improve/the-improve-algorithm/* (Malm et al., 1994).
[a] 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).

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 [ ] ≥ 20	If [ ] <20
Large sulfate	[AS]	[AS]÷20
Small sulfate	0	[AS] - ([AS]÷20)
Large nitrate	[AN]	[AN]÷20
Small nitrate	0	[AN] - ([AN]÷20)
Large OM	[OM]	[OM]÷20
Small OM	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
$f_{SS}(RH)$	1	1	1	1	1	1	1	1	1	1	1	2.3584	2.3799	2.4204	2.4488
$f_S(RH)$	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
$f_L(RH)$	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
$f_{SS}(RH)$	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
$f_S(RH)$	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
$f_L(RH)$	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
$f_{SS}(RH)$	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
$f_S(RH)$	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
$f_L(RH)$	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 [a]
$f_{SS}(RH)$	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
$f_S(RH)$	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
$f_L(RH)$	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 *http://vista.cira.colostate.edu/Improve/the-improve-algorithm/* (Pitchford et al., 2007).

[a] 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[10] with separate mass scattering efficiencies and hygroscopic growth functions for each size (included in the equation as $f_S(RH)$ for small particles, $f_L(RH)$ for large particles, and $f_{SS}(RH)$ for sea salt).

[10] 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 µm and 2.2 for small mode and 0.5 µm and 1.5 for the large mode, respectively.

Lowenthal and Kumar (2016) Equation:

$$b_{ext} \cong 2.2f_S(RH)[small\ sulfate] + 4.8f_L(RH)[large\ sulfate] + 2.4f_S(RH)[small\ nitrate] + 5.1f_L(RH)[large\ nitrate] + 2.8f_S(RH)_{OM}[small\ OM] + 6.1f_L(RH)_{OM}[large\ OM] + 10[EC] + 1[FS] + 1.7f_{SS}(RH)[SS] + 0.6[CM] + 10$$

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[a]	
$f_S(RH)_{OM}$	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).
[a] 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 90th percentile of daily values for each year was calculated, and the three years of values were averaged. The 3-year averages of the 90th percentiles of daily light extinction values were paired with the 2015-2017 $PM_{2.5}$ 24-hour design values for each site having valid data for both statistics.

Table D-6. Derivation of equation variables from AQS $PM_{2.5}$ component concentrations.

Equation Variable	How Calculated from AQS Parameter Values
Ammonium Sulfate	All three equations: 1.375×[Sulfate] [A]
Ammonium Nitrate	All three equations: 1.29×[Nitrate] [B]
Organic Matter	Original IMPROVE equation: 1.6×[OC] [C] Revised IMPROVE equation: 1.6×[OC] [C] Lowenthal and Kumar (2016) equation: 2.1×[OC]
Elemental Carbon	[EC]
Fine Soil	All three equations: [D] 2.2×[Al]+2.49×[Si]+1.63×[Ca]+2.42×[Fe]+1.94×[Ti]
Sea Salt	Revised IMPROVE and Lowenthal and Kumar, 2016 equations:[D] 1.8×[Chloride] 1.8×[Chlorine] (if chloride is missing)

[A] This formula is based on molar molecular weights of ammonium sulfate and sulfate (Malm et al., 1994).
[B] This formula is based on molar molecular weights of ammonium nitrate and nitrate (Malm et al., 1994).
[C] See footnote 4 earlier in this appendix.
[D] 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 $PM_{2.5}$, 24-hour PM_{10} 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 $PM_{2.5}$ and 24-hour PM_{10} 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 $PM_{2.5}$ design values for the 67 monitoring sites in the analyses where light extinction was calculated using the

original IMPROVE equation[11] and the Lowenthal and Kumar IMPROVE equation.[12] Figure D-2 below presents the 3-year visibility metric and the 24-hour $PM_{2.5}$ design values for the 67 monitoring sites with light extinction calculated using the revised IMPROVE equation.[13]

[11] 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.

[12] 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.

[13] 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 $PM_{2.5}$, 24-hour PM_{10}, and annual $PM_{2.5}$ design values, and 3-year visibility metrics at 67 monitoring sites (2015-2017).

Monitor ID	State	Region	24-hour $PM_{2.5}$ Design Value ($\mu g/m^3$) [A]	24-hour PM_{10} Design Value (number of exceedances) [B] [C]	Annual $PM_{2.5}$ Design Value ($\mu g/m^3$) [D]	3-year Visibility Metric (deciviews) [E]		
						Original IMPROVE Equation [F]	Revised IMPROVE Equation [G]	Lowenthal & Kumar IMPROVE Equation [H]
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

[A] The 24-hour $PM_{2.5}$ design value is the 3-year average of the 98th percentile of daily $PM_{2.5}$ mass concentrations. The current 24-hour $PM_{2.5}$ NAAQS is set at a level of 35 µg/m^3.

[B] The 24-hour PM_{10} design value is not to be exceeded more than once per year on average over three years. The current 24-hour PM_{10} NAAQS is set at a level of 150 µg/m3.

[C] For some monitoring locations, PM_{10} design values are not available because of a lack of collocated PM_{10} monitoring at the site or insufficient data after applying completeness criteria for calculating PM_{10} design values.

[D] The annual $PM_{2.5}$ design value is the annual mean, averaged over three years. The current secondary annual $PM_{2.5}$ NAAQS is set at a level of 15.0 µg/m^3.

[E] The 3-year visibility metric is the 3-year average of the 90th percentile of daily light extinction. In the last review, the target level of protection identified for the 3-year visibility metric was 30 deciviews.

[F] 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.

[G] 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.

[H] 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 $PM_{2.5}$, 24-hour PM_{10} and annual $PM_{2.5}$ design values, and 3-year visibility metrics at 20 monitoring sites with collocated $PM_{2.5}$ and PM_{10} monitoring data (2015-2017).

Monitor ID	State	Region	24-hour $PM_{2.5}$ Design Value ($\mu g/m^3$) [A]	24-hour PM_{10} Design Value (number of exceedances) [B C]	Annual $PM_{2.5}$ Design Value ($\mu g/m^3$) [D]	3-year Visibility Metric (deciviews) [E]					
						Original IMPROVE Equation [F]		Revised IMPROVE Equation [G]		Lowenthal & Kumar IMPROVE Equation	
						Without [CM] [H]	With [CM] [I]	Without [CM] [H]	With [CM] [I]	Without [CM] [H]	With [CM] [I]
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

[A] The 24-hour $PM_{2.5}$ design value is the 3-year average of the 98th percentile of daily $PM_{2.5}$ mass concentrations. The current secondary 24-hour $PM_{2.5}$ NAAQS is set at a level of 35 $\mu g/m^3$.

[B] The 24-hour PM_{10} design value is not to be exceeded more than once per year on average over three years. The current secondary 24-hour PM_{10} NAAQS is set at a level of 150 $\mu g/m^3$.

[C] For some monitoring locations, PM_{10} design values are not available because of a lack of collocated PM_{10} monitoring at the site or insufficient data after applying completeness criteria for calculating PM_{10} design values.
[D] The annual $PM_{2.5}$ design value is the annual mean, averaged over three years. The current secondary annual $PM_{2.5}$ NAAQS is set at a level of 15.0 $\mu g/m^3$.
[E] The 3-year visibility metric is the 3-year average of the 90^{th} percentile of daily light extinction. In the last review, the target level of protection identified for the 3-year visibility metric was 30 deciviews.
[F] 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.
[G] 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.
[H] Light extinction was calculated with the coarse mass fraction removed from the equation.
[I] Although the addition of coarse mass increases the daily extinction calculation, it is possible for the 90^{th} percentile value to decrease due to a different set of days having valid measurements of both $PM_{2.5}$ chemical composition and $PM_{10-2.5}$.

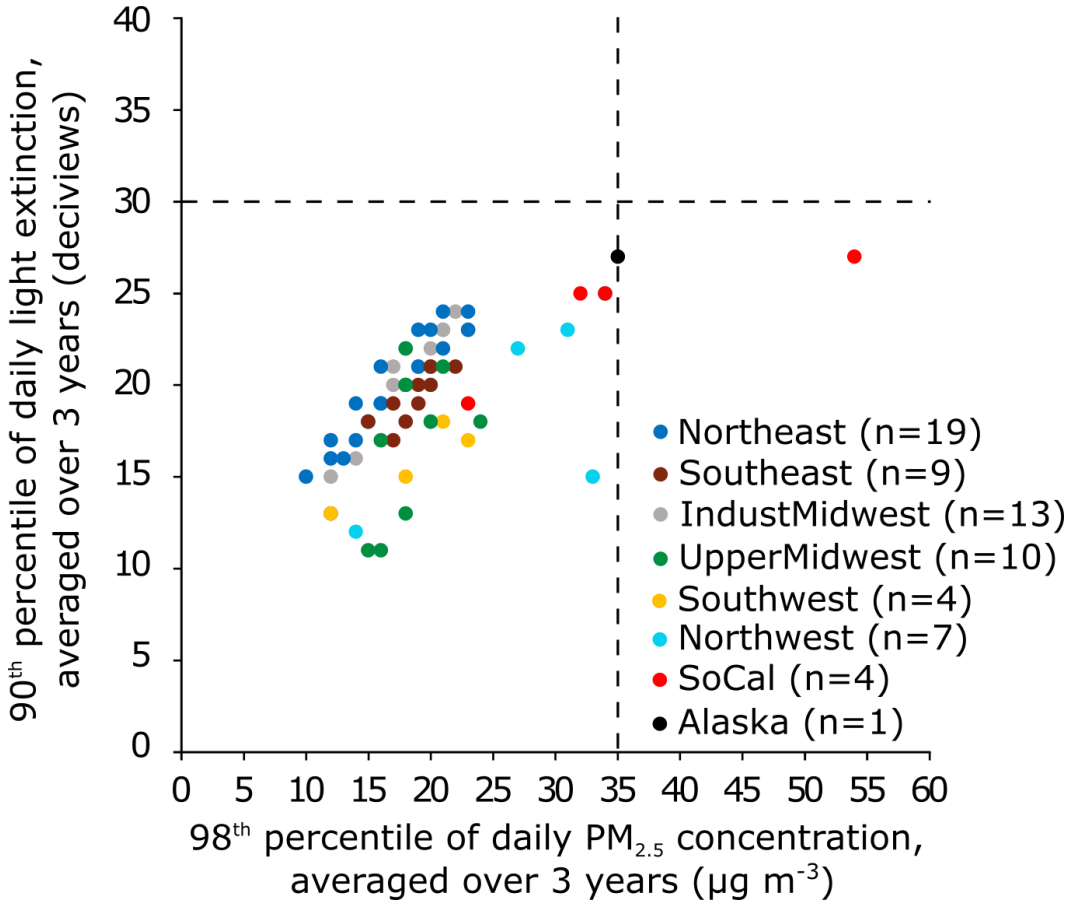

Figure D-2. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2015-2017 using the revised IMPROVE equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ standard (35 µg/m^3) 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 Inc., 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009).[14] Study specific details for these preference studies are shown in Table D-9.

[14] 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 [a]	27 [b]	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 [c]	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	•Rank VAQ (1-7 scale) •Is each slide "acceptable" •"How much haze is too much?"	•Rank VAQ (1-7 scale) •Is each slide "acceptable" •How many days a year would this picture be "acceptable"	•Rank VAQ (1-7 scale) •Is each slide "acceptable"	•Rank VAQ (1-7 scale) •Is each slide "acceptable" •If this hazy, how many hours would it be acceptable (3 slides only) •Valuation question	•Rank VAQ (1-7 scale) •Is each slide "acceptable"
Mean dv found "acceptable"	20.3	23-25	Chilliwack: ~23 Abbotsford: ~19	~20 (range 20-25)	~30

[a] No preference data were collected at a 17th focus group session due ot a slide projector malfunction.
[b] The 27 focus groups were conducted in 6 neighborhood locations in Phoenix, with 3 focus groups held in Spanish.
[C] 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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