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

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

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This draft Policy Assessment has been prepared by staff in the U.S. Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards. Any findings and conclusions are those of the authors and do not necessarily reflect the views of the EPA. Questions or comments related to this document should be addressed to Dr. Lars Perlmutt, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, C539-06, Research Triangle Park, North Carolina 27711 (email: perlmutt.lars@epa.gov).

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1 INTRODUCTION

This document, *Policy Assessment for the Reconsideration of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft* (hereafter referred to as the draft PA), presents the draft policy assessment for the U.S. Environmental Protection Agency's (EPA's) reconsideration of the review of the national ambient air quality standards (NAAQS) for particulate matter (PM) completed in 2020.[1] The overall plan for the 2020 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 the 2020 review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA) and a Policy Assessment (PA). The key considerations presented in this draft PA are intended to provide updates to the policy information to support the reconsideration of the 2020 PM NAAQS final action, which retained the primary and secondary $PM_{2.5}$ and PM_{10} standards without revision (85 FR 82684, December 18, 2020). In reconsidering the 2020 final action, the EPA will consider the scientific and technical analyses on which the December 2020 PM NAAQS final action was based, as well as the newly available scientific information evaluated in the *Supplement to the 2019 Integrated Science Assessment for Particulate Matter (External Review Draft)* (hereafter referred to as the draft ISA Supplement; U.S. EPA, 2021) and the policy implications of the new scientific evidence and updated quantitative analyses presented in this draft PA. Much of the information in this draft PA is drawn directly from information included in the 2019 ISA (U.S. EPA, 2019) and the 2020 PA (U.S. EPA, 2020).

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 reconsideration of the 2020 final decision. Chapter 2 provides an overview of the available information on PM-related emissions, atmospheric chemistry, monitoring and air quality. Chapter 3 focuses on policy-relevant aspects of the currently available health effects evidence as presented in the 2019 ISA and draft ISA Supplement, as well as updated exposure/risk information, and identifies and summarizes the key considerations related to this reconsideration of the primary $PM_{2.5}$ standards. Chapter 4 draws substantially from the information presented in the 2020 PA on the policy-relevant aspects of the

[1] On June 10, 2021, the Agency announced its decision to reconsider the 2020 PM NAAQS final action. The press release for this announcement is available at: *https://www.epa.gov/newsreleases/epa-reexamine-health-standards-harmful-soot-previous-administration-left-unchanged*

health effects evidence presented in the 2019 ISA and identifies and summarizes the key considerations related to this reconsideration of the primary standard PM_{10}. Chapter 5 focuses on policy-relevant aspects of the currently available welfare effects evidence as presented in the 2019 ISA and draft ISA Supplement, as well as updated quantitative analyses for visibility effects, and identifies and summarizes the key considerations related to this reconsideration of the secondary PM standards.[2] More detail about the process for this reconsideration is described in section 1.4.2 below, and the approach for considering the available information for this reconsideration is presented within Chapters 3, 4, and 5 of this draft PA.

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.

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.[3] 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

[2] 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*.

[3] 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.

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[4] take into account the available scientific evidence, as assessed in the *Integrated Science Assessment for Particulate Matter (Final Report)* (2019 ISA [U.S. EPA, 2019]) and in the draft ISA Supplement (U.S. EPA, 2021), as well as additional policy-relevant analyses of air quality and risks. The evaluation and preliminary conclusions presented in this draft PA have been informed by the scientific evidence presented in the 2019 ISA and the draft ISA Supplement, as well as the policy-relevant considerations and conclusions reached in the 2020 PA, along with updated quantitative analyses of air quality, risk, and exposure, where available. Review and comments from the CASAC, as well as public comment, on this draft PA will inform the final evaluation and conclusions in the final PA. The final PA is intended to help the Administrator in considering the 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.

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).

[4] 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).

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."[5] 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."[6]

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 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

[5] 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).

[6] 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."

components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51, *Mississippi v. EPA*, 744 F.3d at 1351, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s), and the kind and degree of uncertainties. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62; *Mississippi v. EPA*, 744 F.3d at 1353.

Section 109(d)(1) of the Act requires a review be completed every five years and, if appropriate, revision of existing air quality criteria to reflect advances in scientific knowledge on the effects of the pollutant on public health and welfare. Under the same provision, the EPA is also to review every five years and, if appropriate, revise the NAAQS, based on the revised air quality criteria.[7]

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 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

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

> 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.[8]

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.

[8] 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).[9] 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.[10] 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

[9] 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 ISA has replaced the AQCD.

[10] 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;[11] and (2) a 24-hour standard with a level of 65 $\mu g/m^3$, 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 levels of NAAQS, the court held that the EPA's approach to establishing the level of the

[11] 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 2012 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 CBSA (78 FR 3236, January 15, 2013).

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).[12] 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). With regard to the primary and secondary standards for fine particles, the EPA revised the level

[12] 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.

of the 24-hour $PM_{2.5}$ standards to 35 $\mu g/m^3$, retained the level of the annual $PM_{2.5}$ standards at 15.0 $\mu g/m^3$, and revised the form of the annual $PM_{2.5}$ standards by narrowing the constraints on the optional use of spatial averaging. With regard to the primary and secondary standards for PM_{10}, the EPA retained the 24-hour standards, with levels at 150 $\mu g/m^3$, and revoked the annual standards.[13] The Administrator judged that the available evidence generally did not suggest a link between long-term exposure to existing ambient levels of coarse particles and health or welfare effects. In addition, a new reference method was added for the measurement of $PM_{10-2.5}$ in the ambient air in order to provide a basis for approving federal equivalent methods (FEMs) and to promote the gathering of scientific data to support future reviews of the PM NAAQS.

Several parties filed petitions for review following promulgation of the revised PM NAAQS in 2006. These petitions addressed the following issues: (1) selecting the level of the primary annual $PM_{2.5}$ standard; (2) retaining PM_{10} as the indicator of a standard for thoracic coarse particles, retaining the level and form of the 24-hour PM_{10} standard, and revoking the PM_{10} annual standard; and (3) setting the secondary $PM_{2.5}$ standards identical to the primary standards. On February 24, 2009, the U.S. Court of Appeals for the District of Columbia Circuit issued its opinion in the case *American Farm Bureau Federation v. EPA*, 559 F. 3d 512 (D.C. Cir. 2009). The court remanded the primary annual $PM_{2.5}$ NAAQS to the EPA because the Agency failed to adequately explain why the standards provided the requisite protection from both short- and long-term exposures to fine particles, including protection for at-risk populations. *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 520-27 (D.C. Cir. 2009). With regard to the standards for PM_{10}, the court upheld the EPA's decisions to retain the 24-hour PM_{10} standard to provide protection from thoracic coarse particle exposures and to revoke the annual PM_{10} standard. *American Farm Bureau Federation,* 559 F. 2d at 533-38. With regard to the secondary $PM_{2.5}$ standards, the court remanded the standards to the EPA because the Agency failed to adequately explain why setting the secondary PM standards identical to the primary standards provided the required protection for public welfare, including protection from visibility impairment. *American Farm Bureau Federation*, 559 F. 2d at 528-32. The EPA responded to the court's remands as part of the next review of the PM NAAQS, which was initiated in 2007 (discussed below).

[13] In the 2006 proposal, the EPA proposed to revise the 24-hour PM_{10} standard in part by establishing a new $PM_{10-2.5}$ indicator for thoracic coarse particles (i.e., particles generally between 2.5 and 10 µm in diameter). The EPA proposed to include any ambient mix of $PM_{10-2.5}$ that was dominated by resuspended dust from high density traffic on paved roads and by PM from industrial sources and construction sources. The EPA proposed to exclude any ambient mix of $PM_{10-2.5}$ that was dominated by rural windblown dust and soils and by PM generated from agricultural and mining sources. In the final decision, the existing PM_{10} standard was retained, in part due to an "inability…to effectively and precisely identify which ambient mixes are included in the [$PM_{10-2.5}$] indicator and which are not" (71 FR 61197, October 17, 2006).

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,[14] 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[15] 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[16] 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).

1.3.5 Review Completed in 2020

In December 2014, the EPA announced the initiation of the 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 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

[14] The history of the NAAQS review process, including revisions to the process, is discussed at *https://www.epa.gov/naaqs/historical-information-naaqs-review-process*.

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

[16] Consistent with the primary standard, the EPA eliminated the option for spatial averaging with the annual standard.

a wide range of external experts as well as EPA staff representing a variety of areas of expertise (e.g., epidemiology, human and animal toxicology, risk/exposure analysis, atmospheric science, visibility impairment, climate effects), were asked to highlight significant new and emerging PM research, and to make recommendations to the Agency regarding the design and scope of this review. This workshop provided for a public discussion of the key science and policy-relevant issues around which the EPA has structured the current review of the PM NAAQS and of the most meaningful new scientific information that would be available in this review to inform our understanding of these issues.

The input received at the workshop guided the EPA staff in developing a draft IRP, which was reviewed by the CASAC Particulate Matter Panel and discussed on public teleconferences held in May 2016 (81 FR 13362, March 14, 2016) and August 2016 (81 FR 39043, June 15, 2016). Advice from the CASAC, supplemented by the Particulate Matter Panel, and input from the public were considered in developing the final IRP for this review (U.S. EPA, 2016). The final IRP discusses the approaches to be taken in developing key scientific, technical, and policy documents in this review and the key policy-relevant issues that will frame the EPA's consideration of whether the current primary and/or secondary NAAQS for PM should be retained or revised.

In May 2018, the Administrator issued a memorandum describing a "back-to-basics" process for reviewing the NAAQS (Pruitt, 2018). This memo announced the Agency's intention to conduct the current review of the PM NAAQS in such a manner as to ensure that any necessary revisions were finalized by December 2020. Following this memo, on October 10, 2018 the Administrator additionally announced that the role of reviewing the key science assessments developed as part of the ongoing review of the PM NAAQS (i.e., drafts of the ISA and PA) would be performed only by the seven-member chartered CASAC (i.e., without the support of the CASAC Particulate Matter Panel that reviewed the draft IRP).[17]

The EPA released the draft ISA in October 2018 (83 FR 53471, October 23, 2018). The draft ISA was reviewed by the chartered CASAC at a public meeting held in Arlington, VA in December 2018 (83 FR 55529, November 6, 2018) and was discussed on a public teleconference in March 2019 (84 FR 8523, March 8, 2019). The CASAC provided its advice on the draft ISA in a letter to the EPA Administrator dated April 11, 2019 (Cox, 2019a). The EPA took steps to address these comments in the final ISA, which was released in December 2019 (U.S. EPA, 2019).

The EPA released the draft PA in September 2019 (84 FR 47944, September 11, 2019). The draft PA was reviewed by the chartered CASAC and discussed in October 2019 at a public

[17] Announcement available at: *https://www.regulations.gov/document/EPA-HQ-OAR-2015-0072-0223*

meeting held in Cary, NC. Public comments were received via a separate public teleconference (84 FR 51555, September 30, 2019). A public meeting to discuss the chartered CASAC letter and response to charge questions on the draft PA was held in Cary, NC in December 2019 (84 FR 58713, November 1, 2019), and the CASAC provided its advice on the draft PA, including its advice on the current primary and secondary PM standards, in a letter to the EPA Administrator dated December 16, 2019 (Cox, 2019b). With regard to the primary standards, the CASAC recommended retaining the current 24-hour $PM_{2.5}$ and PM_{10} standards but did not reach consensus on the adequacy of the current annual $PM_{2.5}$ standard. With regard to the secondary standards, the CASAC recommended retaining the current standards. In response to the CASAC's comments, the 2020 final PA incorporated a number of changes (U.S. EPA, 2020), as described in detail in section I.C.5 of the 2020 proposal (85 FR 24100, April 30, 2020).

On April 14, 2020, the EPA proposed to retain all of the primary and secondary PM standards, without revision. These proposed decisions were published in the Federal Register on April 30, 2020 (85 FR 24094, April 30, 2020). The EPA's final decision on the PM NAAQS was published in the Federal Register on December 18, 2020 (85 FR 82684, December 18, 2020). In the 2020 rulemaking, the EPA retained the primary and secondary $PM_{2.5}$ and PM_{10} standards, without revision. The EPA received three petitions for judicial review (described in more detail in section 1.4.3 below), as well as three petitions for reconsideration of the 2020 final action.

1.4 RECONSIDERATION OF THE 2020 PM NAAQS FINAL ACTION

On January 20, 2021, President Biden issued an "Executive Order on Protecting Public Health and the Environment and Restoring Science to Tackle the Climate Crisis," (Executive Order 13990; 86 FR 7037, January 25, 2021)[18] which directed review of certain agency actions. An accompanying fact sheet provides a non-exclusive list of agency actions that agency heads will review in accordance with that order, including the 2020 Particulate Matter NAAQS Decision.[19]

1.4.1 Decision to Initiate a Reconsideration

On June 10, 2021, the Agency announced its decision to reconsider the 2020 PM NAAQS final action.[20] The EPA is reconsidering the December 2020 decision because the

[18] See *https://www.whitehouse.gov/briefing-room/presidential-actions/2021/01/20/executive-order-protecting-public-health-and-environment-and-restoring-science-to-tackle-climate-crisis/*

[19] See *https://www.whitehouse.gov/briefing-room/statements-releases/2021/01/20/fact-sheet-list-of-agency-actions-for-review/*

[20] The press release for this announcement is available at: *https://www.epa.gov/newsreleases/epa-reexamine-health-standards-harmful-soot-previous-administration-left-unchanged*

available scientific evidence and technical information indicate that the current standards may not be adequate to protect public health and welfare, as required by the Clean Air Act. We note that the 2020 PA concluded that the scientific evidence and information supported revising the level of the primary annual $PM_{2.5}$ standard to below the current level of 12 $\mu g/m^3$ while retaining the primary 24-hour $PM_{2.5}$ standard (U.S. EPA, 2020). The EPA also notes that the 2020 PA concluded that the available scientific evidence and information supported retaining the primary PM_{10} standard and secondary PM standards without revision (U.S. EPA, 2020).

1.4.2 Process for Reconsideration of the 2020 PM NAAQS Decision

In its announcement of the reconsideration of the PM NAAQS, the Agency explained that, in support of the reconsideration, it would develop a supplement to the 2019 ISA and a revised PA. The EPA also explained that the draft ISA Supplement and draft PA would be reviewed at a public meeting by the CASAC, and the public will have opportunities to comment on these documents during the CASAC review process, as well as to provide input during the rulemaking through the public comment process and public hearings on the proposed rulemaking.

On March 31, 2021, the Administrator announced his decision to reestablish the membership of the CASAC to "ensure the agency received the best possible scientific insight to support our work to protect human health and the environment."[21] Consistent with this memorandum, a call for nominations of candidates to the EPA's chartered CASAC was published in the Federal Register (86 FR 17146, April 1, 2021). On June 17, 2021, the Administrator announced his selection of the seven members to serve on the chartered CASAC.[22] [23] Additionally, a call for nominations of candidates to a PM-specific panel was published in the Federal Register (86 FR 33703, June 25, 2021). The members of the PM CASAC panel were announced on August 30, 2021.[24]

The draft ISA Supplement was released in September 2021 (U.S. EPA, 2021). The evidence presented within the 2019 ISA, along with the targeted identification and evaluation of new scientific information in the draft ISA Supplement, provides the scientific basis for the

[21] The press release for this announcement is available at: *https://www.epa.gov/newsreleases/administrator-regan-directs-epa-reset-critical-science-focused-federal-advisory*

[22] The press release for this announcement is available at: *https://www.epa.gov/newsreleases/epa-announces-selections-charter-members-clean-air-scientific-advisory-committee*

[23] The list of members of the chartered CASAC and their biosketches are available at: *https://yosemite.epa.gov/sab/sabpeople.nsf/WebExternalCommitteeRosters?OpenView&committee=CASAC&secondname=Clean%20Air%20Scientific%20Advisory%20Committee%20*

[24] The list of members of the PM CASAC panel and their biosketches are available at: *https://casac.epa.gov/ords/sab/f?p=105:14:9979229564047:::14:P14_COMMITTEEON:2021%20CASAC%20PM%20Panel*

reconsideration of the 2020 PM NAAQS final decision. The draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. In selecting the health effects to evaluate within the draft ISA Supplement, the EPA focused on the strongest causality determinations for health effects categories presented in the 2019 ISA, and the subsequent use of the health effects evidence in the 2020 PA with respect to which were most useful in informing staff conclusions (U.S. EPA, 2021).[25] Specifically, within the draft ISA Supplement, the focus is only on the health effects evidence where the 2019 ISA concluded a "causal relationship" (U.S. EPA, 2021, section 1.2.1). Consistent with the rationale for the health effects, the selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA.[26] Specifically, for welfare effects, the focus within the draft ISA Supplement is on visibility effects. The draft ISA Supplement also considers recent health effects evidence that addresses key scientific topics where the literature has evolved since the 2020 review was completed, specifically since the literature cutoff date for the 2019 ISA.[27]

Building on the rationale presented in section 1.2.1, the draft ISA Supplement considered peer-reviewed studies published from approximately January 2018 through March 2021 that meet the following criteria:

- Health effects:
  - Health effect categories where the 2019 PM ISA concluded a "*causal relationship*" (i.e., short- and long-term $PM_{2.5}$ exposure and cardiovascular effects

[25] As described in section 1.2.1 of the draft ISA Supplement: "In considering the public health protection provided by the current primary $PM_{2.5}$ standards, and the protection that could be provided by alternatives, [the U.S. EPA, within the 2020 PM PA] emphasized health outcomes for which the ISA determined that the evidence supports either a "*causal*" or a "*likely to be causal*" relationship with $PM_{2.5}$ exposures" (U.S. EPA, 2020). Although the 2020 PA initially focused on this broader set of evidence, the basis of the discussion on potential alternative standards primarily focused on health effect categories where the 2019 PM ISA concluded a "*causal relationship*" (i.e., short- and long-term $PM_{2.5}$ exposure and cardiovascular effects and mortality) as reflected in Figures 3-7 and 3-8 of the 2020 PA (U.S. EPA, 2020).

[26] As described in section 1.2.1 of the draft ISA Supplement: The 2019 PM ISA concluded a "*causal relationship*" for each of the welfare effects categories evaluated (i.e., visibility, climate effects and materials effects). While the 2020 PA considered the broader set of evidence for these effects, for climate effects and material effects, it concluded that there remained "substantial uncertainties with regard to the quantitative relationships with PM concentrations and concentration patterns that limit[ed] [the] ability to quantitatively assess the public welfare protection provided by the standards from these effects" (U.S. EPA, 2020).

[27] These key scientific topics include experimental studies conducted at near-ambient concentrations, epidemiologic studies that employed causal modeling methods or conducted accountability analyses, studies that assess the relationship between $PM_{2.5}$ exposure and Coronavirus Disease 2019 (COVID-19) infection and death; and in accordance with recent EPA guidance on addressing environmental justice, studies that examine disparities in $PM_{2.5}$ exposure and the risk of health effects (U.S. EPA, 2021, section 1.2.1).

and mortality). Additionally, for these health effect categories the recent studies evaluated are limited to:

- U.S. and Canadian epidemiologic studies
- Epidemiologic studies that employed causal modeling methods or conducted accountability analyses (i.e., examined the impact of a policy on reducing $PM_{2.5}$ concentrations)

- Welfare Effects:
  - U.S. and Canadian studies that provide new information on public preferences for visibility impairment and/or developed methodologies or conducted quantitative analyses of light extinction
- Key Scientific Topics
  - Experimental studies (i.e., controlled human exposure and animal toxicological) conducted at near-ambient $PM_{2.5}$ concentrations
  - At-Risk Populations
    - U.S. and Canadian-based epidemiologic or exposure studies examining potential disparities in either $PM_{2.5}$ exposures or the risk of health effects by race/ethnicity or socioeconomic status (SES)
  - U.S. and Canadian-based epidemiologic studies that examined the relationship between $PM_{2.5}$ exposures and COVID-19 infection and/or death

Given the narrow scope of the draft ISA Supplement, it is important to recognize that the evaluation does not encompass the full multidisciplinary evaluation presented within the 2019 ISA that would result in weight-of-evidence conclusions on causality (i.e., causality determinations). The draft ISA Supplement critically evaluates and provides key study specific information for those recent studies deemed to be of greatest significance for informing preliminary conclusions on the PM NAAQS in the context of the body of evidence and scientific conclusions presented in the 2019 ISA.

This draft PA considers the scientific evidence presented in the 2019 ISA and draft ISA Supplement. This draft PA additionally considers the quantitative and technical information presented in the 2020 PA, along with updated and newly available analyses since the completion of the 2020 review. For those health and welfare effects for which the draft ISA Supplement evaluated recently available evidence and updated quantitative analyses were supported (i.e., $PM_{2.5}$-related health effects and visibility effects), the draft PA includes consideration of this newly available scientific and technical information in reaching preliminary conclusions. For those health and welfare effects for which newly available scientific and technical information were not evaluated (i.e., $PM_{10-2.5}$-related health effects and non-visibility effects), the preliminary conclusions presented in this draft PA rely heavily on the information that supported the conclusions in the 2020 PA.

1.4.3 Ongoing Litigation

Following publication of the 2020 final action, several parties filed petitions for review of the EPA's final decision in the D.C. Circuit and the Court consolidated the cases. In order to consider whether reconsideration of the 2020 final action was warranted, the EPA moved for two 90-day abeyances in these consolidated cases, which the Court granted. After the EPA announced that is reconsidering the 2020 final decision, the EPA filed a motion with the Court to hold the consolidated cases in abeyance until March 1, 2023. The court has not yet acted on the EPA's motion, which the court granted on October 1, 2021.

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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, 2019b, 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, 2019b, 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, U.S. EPA, 2019b, section 4.1.1).

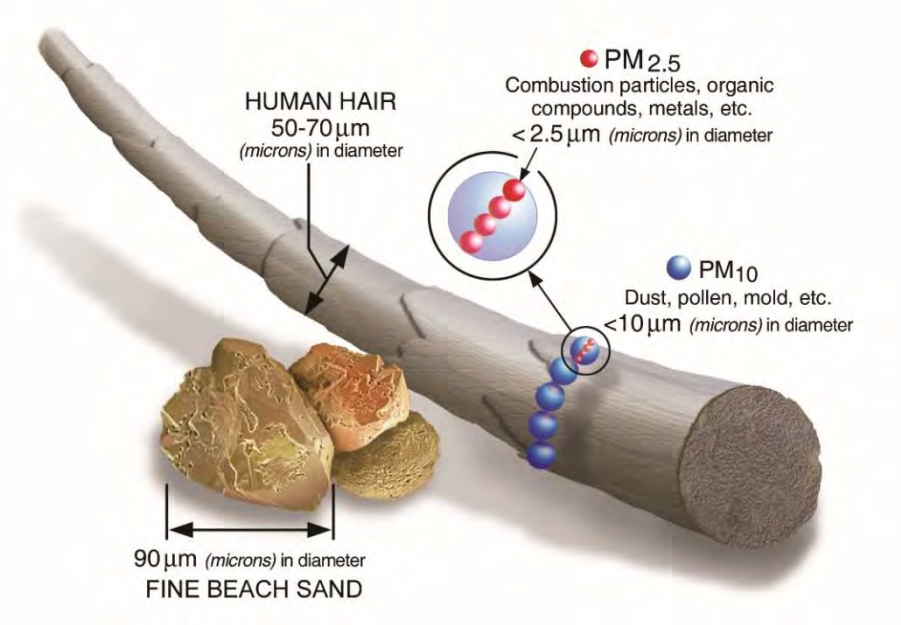

Figure 2-1. Comparisons of $PM_{2.5}$ and PM_{10} diameters to human hair and beach sand.
(Adapted from: *https://www.epa.gov/pm-pollution/particulate-matter-pm-basics*)

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, 2019b, 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, 2019b, section 2.2). $PM_{2.5}$ 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, 2019b, 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, 2019b, 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, 2019b, 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, 2019b, 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, 2019b, 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, 2019b, 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, 2019b, 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 2017 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, with a focus on the 2017 NEI. Section 2.3.1 discusses emission trends and identifies the sectors that have experienced the most change in direct PM and precursor emissions from 1990 to 2017. It should be noted that major decreases have been observed in NO_X and SO_2 emissions over this time, with continued reductions observed from the 2014 NEI to the 2017 NEI. For a more detailed review of the changes in PM and PM precursor emissions from the 2014 NEI to the 2017 NEI, please refer to the 2017 NEI Technical Support Document (U.S. EPA, 2021).

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 pollutant precursors, and hazardous air pollutants from a comprehensive set of air emissions sources, including point sources (e.g., electric generating units, boilers, etc.), nonpoint (or area) sources (e.g., 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 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 2017 NEI, approximately 5.7 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.

[2] These sections do not provide a comprehensive list of all sources, nor do they provide estimates of emission rates or emission factors for all source categories. Individual subsectors of source types were aggregated up to a sector level as used in Figure 2-2 and Figure 2-4. More information about the sectors and subsectors can be found as a part of the 2017 NEI (U.S. EPA, 2021).

EPA, 2019b, section 2.3.1.1). Other lesser-contributing anthropogenic sources of $PM_{2.5}$ emissions nationally include stationary fuel combustion and agriculture sources.

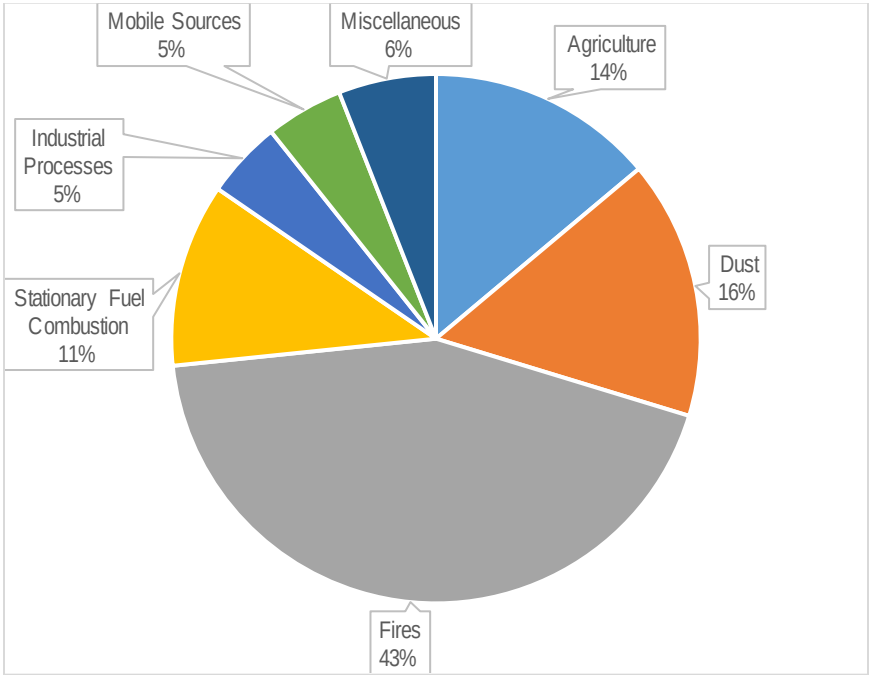

Figure 2-2. Percent contribution of $PM_{2.5}$ national emissions by source sectors. (Source: 2017 NEI)

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 2019 ISA illustrates this variation of primary $PM_{2.5}$ emissions with data from five urban counties in the U.S. (U.S. EPA, 2019b, 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, 2019b, 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 5% to total primary $PM_{2.5}$ emissions nationally as shown in Figure 2-2.

Another way to examine the emissions data shown in Figure 2-2 is by county. Figure 2-3 presents county-based total $PM_{2.5}$ emissions divided by the area of the county to normalize for differences in county size. This "emissions density" map highlights regions of the country with

[3] The five counties included in the 2019 ISA analysis include Queens County, NY, Philadelphia County, PA, Los Angeles County, CA, Sacramento County, CA, and Maricopa County (Phoenix), AZ (U.S. EPA, 2019b, section 2.3.1.2).

the highest total $PM_{2.5}$ emissions by county accounting for county size. While Figure 2-3 shows total $PM_{2.5}$ emissions, different sectors will contribute at different levels across the country.

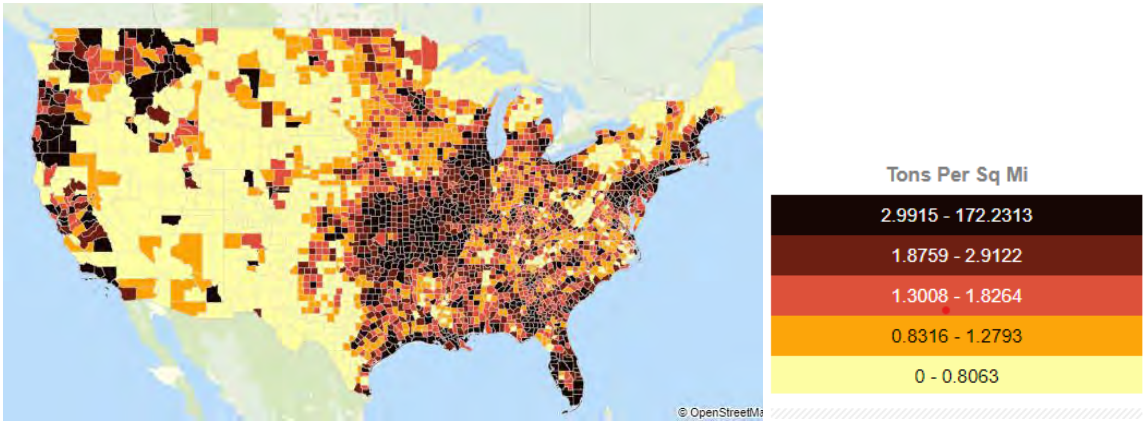

Figure 2-3. 2017 NEI $PM_{2.5}$ Emissions Density Map, tons per square mile

2.1.1.2 Sources Contributing to Primary PM_{10} Emissions

Although the NEI does not estimate emissions of $PM_{10-2.5}$ (coarse PM) 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 2017 NEI, are shown in Figure 2-4. Total PM_{10} emissions are estimated to be about 17 million tons. National emissions of PM_{10} are dominated by dust and agriculture, contributing a combined 70% 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 are considered as a part of the background component of PM as opposed to a primary emission of coarse PM (U.S. EPA, 2019b, section 2.3.3).

As with $PM_{2.5}$, the relative contributions of sources to total PM_{10} emissions varies from location to location (e.g., depending on local climate, geography, degree of urbanization, etc.). However, unlike $PM_{2.5}$, the sectors included in Figure 2-4 are expected to be among the most important contributors to coarse PM emissions 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 2-4 shows the national contributions to

PM_{10} emissions from particular source sectors and Figure 2-5 exhibits the corresponding emissions density map for PM_{10}.

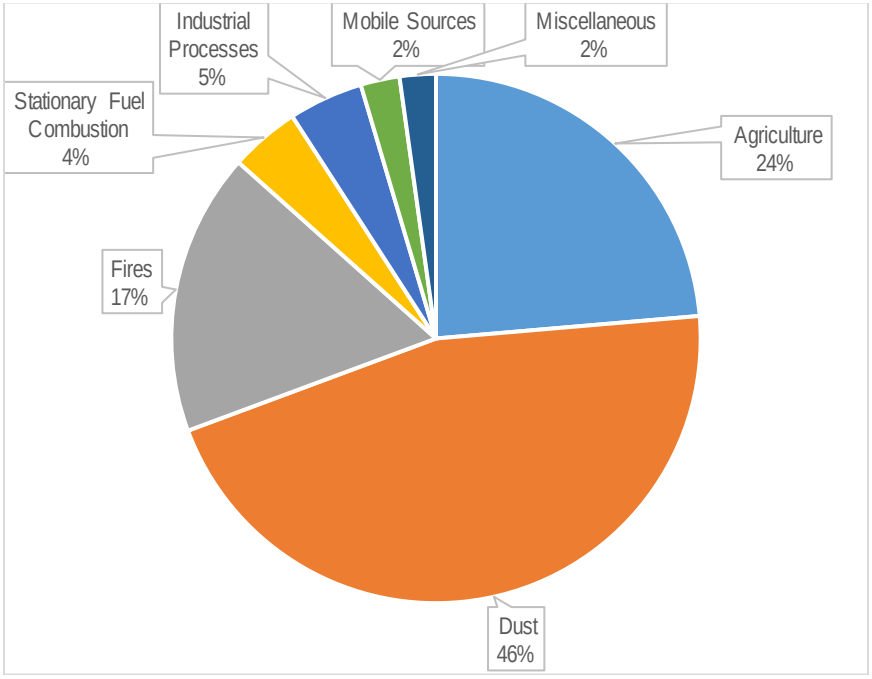

Figure 2-4. Percent contribution of PM_{10} emissions by national source sectors. (Source: 2017 NEI)

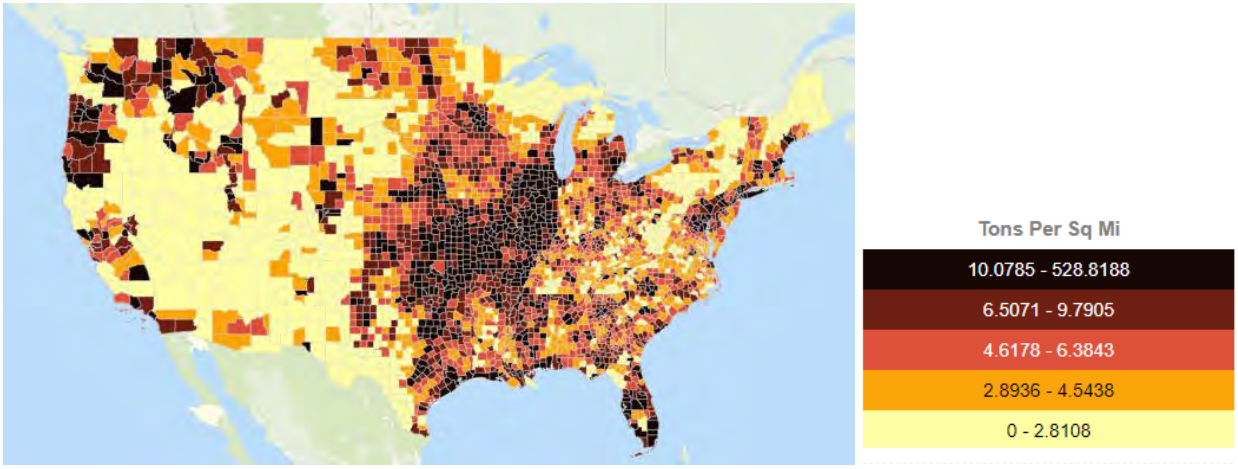

Figure 2-5. 2017 NEI PM_{10} Emissions Density Map, tons per square mile

2.1.1.3 Sources Contributing to Emissions of PM Components and Precursor Gases

Understanding the components of PM is particularly important for providing insight into which sources contribute to PM mass, as well as to better understand 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 2-6 illustrates the sources that contribute to national emissions of elemental and organic carbon based on the 2017 NEI. The top panel of Figure 2-6 shows that fires account for most (i.e., 63%) of the 1.8 million tons of particulate OC emissions estimated in the 2017 NEI, while the bottom panel of Figure 2-6 shows that fires and mobile sources (mostly diesel sources) contribute 71% of the estimated ~ 284,000 tons of particulate EC in the 2017 NEI. It should be noted that the fraction of EC to $PM_{2.5}$ was lower in the 2017 NEI compared to the 2014 NEI, owing to a significantly lower contribution of EC from fires in the 2017 NEI compared to previous NEIs. This change in the EC fraction resulted from an in-house research program to investigate the $PM_{2.5}$ chemical composition of the emissions from fires burning different fuels and in different combustion phases. It should be noted that the OC contributions on a percentage basis increased in accordance with the EC decreases. While these results have not yet been directly published, this information has been acknowledged and used in other EPA analyses (Kelly et al., 2019b, Figure 13).

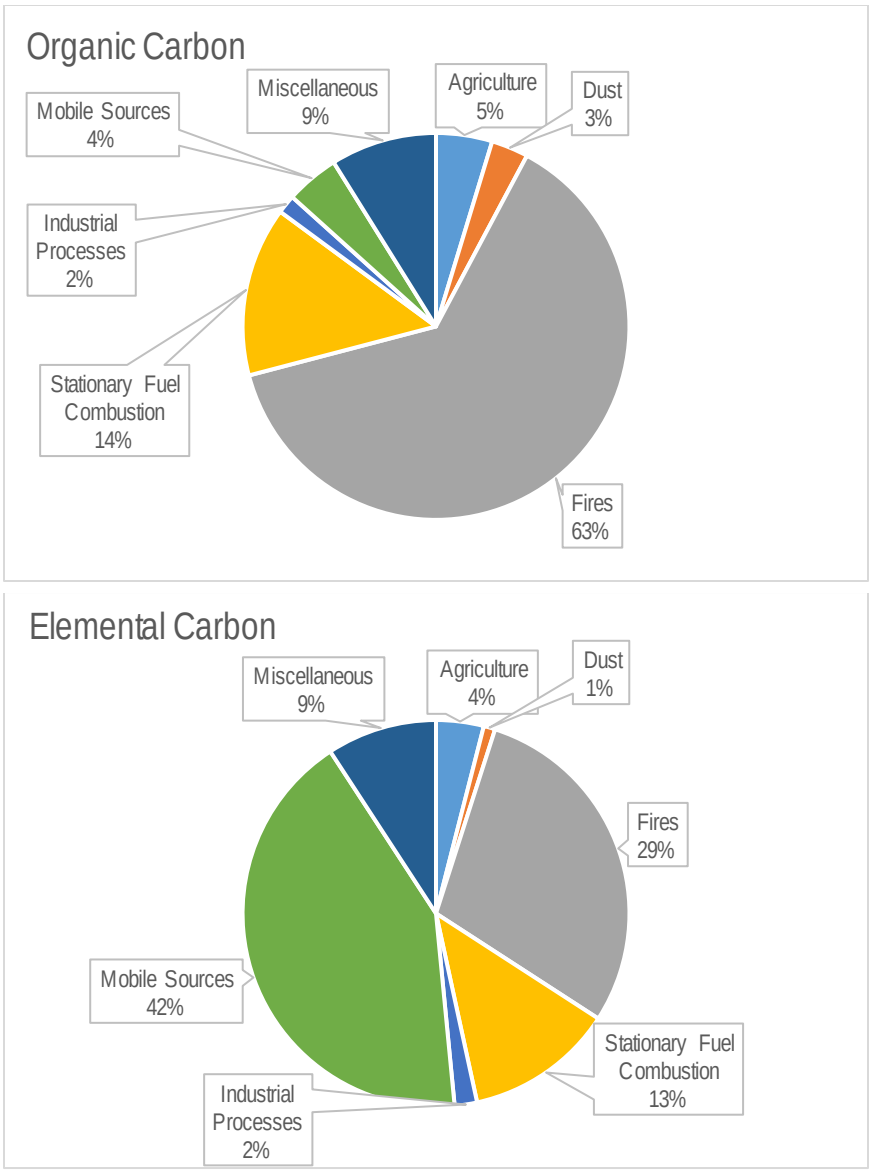

Figure 2-6. Percent contribution to organic carbon (top panel) and elemental carbon (bottom panel) national emissions by source sectors. (Source: 2017 NEI)

Figure 2-7 shows the emissions density map for elemental carbon. This map illustrates that the EC emissions signals are strong in the Southeast U.S, the central region of the U.S. (i.e., Kansas and Oklahoma), and parts of the West and Northwest U.S., where fires make substantial contributions to $PM_{2.5}$. In addition, areas where diesel off-road and on-road sources are a large part of the emissions mix also stand out (urban and highway corridors). The OC density map (not shown) shows the highest emissions density in locations with substantial biomass burning activity, consistent with most of the OC emissions coming from fires (Figure 2-6).

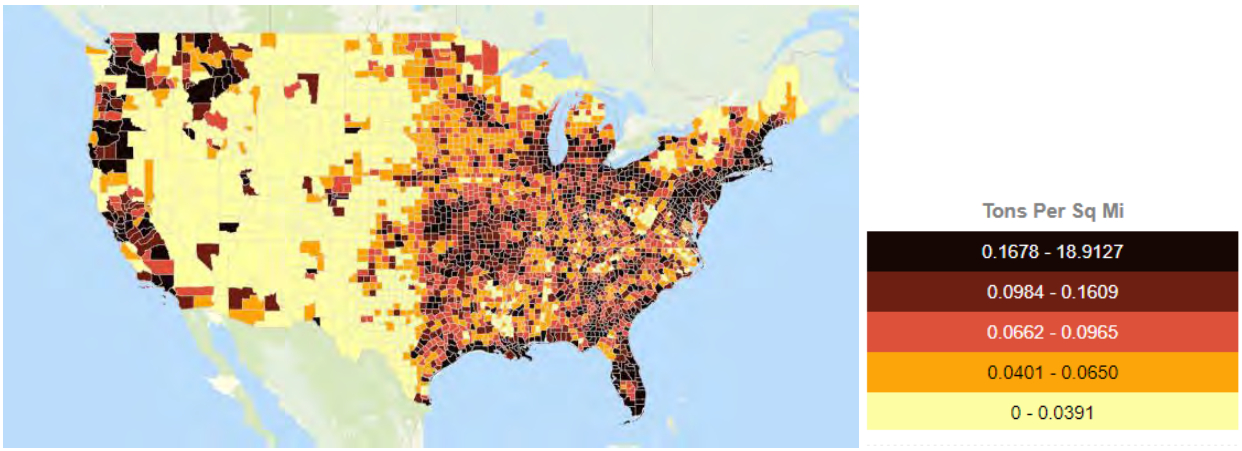

Figure 2-7. 2017 NEI Elemental Carbon Emissions Density Map, tons per square mile.

2.1.1.3.2 Sources Contributing to Emissions of Precursor Gases

As discussed further in the 2019 ISA (U.S. EPA, 2019b, 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, 2019b, 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, 2019b, section 2.3.2).

Emissions of each of the precursor gases noted above are estimated in the NEI and have unique source signatures at the national level. Figure 2-8 illustrates the source contributions at the national level for these $PM_{2.5}$ precursor gases. As shown in Panel A in Figure 2-8, stationary fuel combustion sources contribute nearly 70% of the estimated total of 2.8 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. Anthropogenic NO_X emissions, shown in panel B, are emitted by a range of combustion sources, including mobile sources (59%)

and stationary fuel combustion sources (25%). In the 2017 NEI, there is an estimated total of 10.3 million tons of NO_X emitted. Of the total estimated 4.3 million tons of anthropogenic ammonia (NH_3) emissions shown in panel C of Figure 2-8, 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 (56%) and fertilizer application (25%). In urban areas, on-road mobile sources may also contribute significantly to NH_3 emissions (U.S. EPA, 2019b, Figure 2-3; Sun et al., 2014; U.S. EPA, 2020). Of the estimated 17.2 million tons of VOC emissions from anthropogenic sources, fires (39%) and "miscellaneous" (22%)[4] are the highest contributors, followed by mobile sources (17%) and industrial processes (18%), as shown in Figure 2-8 panel D. It should be noted that as these traditional combustion sources of VOCs are reduced by regulations and controls, new non-combustion sources, such as volatile chemical products (solvents) are emerging as key contributors to anthropogenic VOC totals in some parts of the country, and particularly in urban corridors. In addition, biogenic sources (not shown in Figure 2-8) are significant contributors to both VOC and NOx emissions.

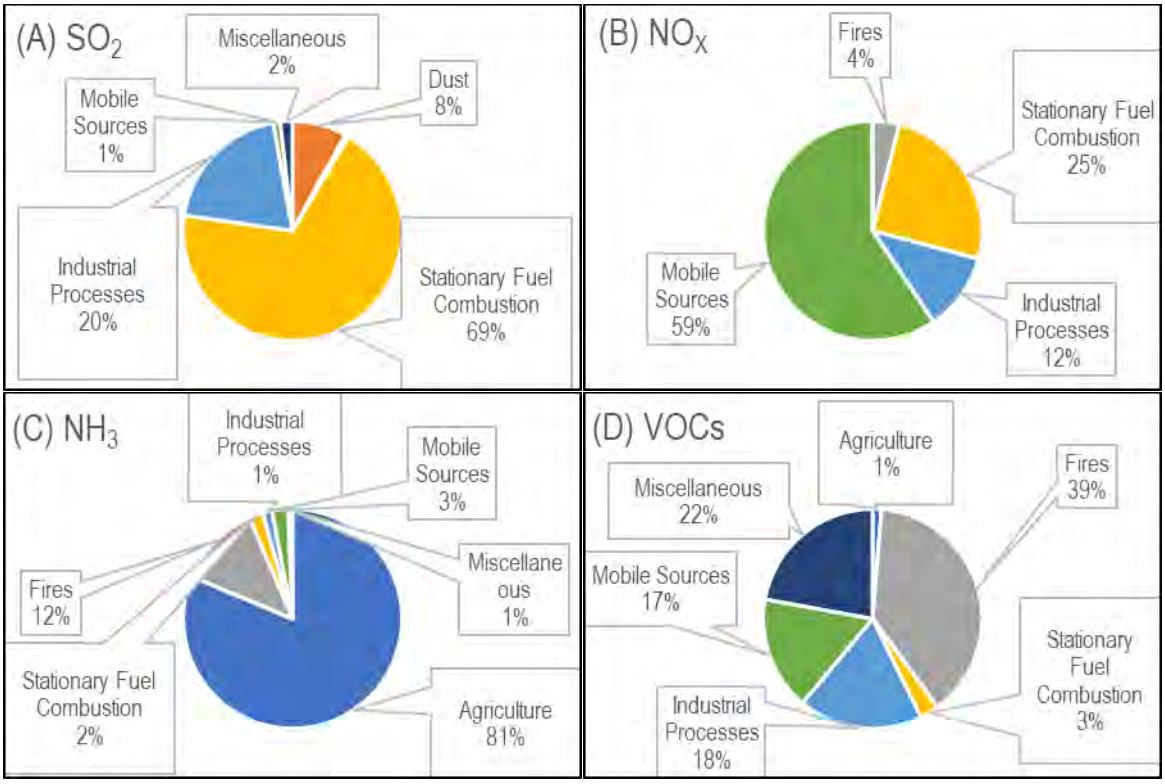

Figure 2-8. Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B), ammonia (panel C), and volatile organic compounds (panel D) national emissions by source sectors. (Source: 2017 NEI). All graphics only show anthropogenic contributions.

[4] The "miscellaneous" category includes such things as solvents, commercial cooking and waste disposal.

Figure 2-9 to Figure 2-12 below show the emissions density maps corresponding to each of the $PM_{2.5}$ precursors included in Figure 2-8.

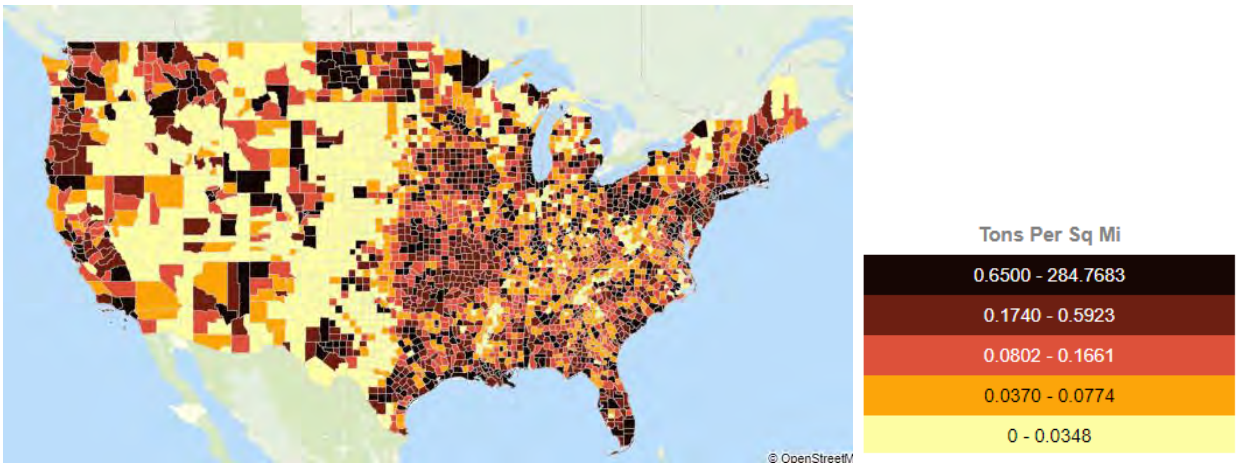

Figure 2-9. SO_2 Emissions Density Map, tons per square mile.

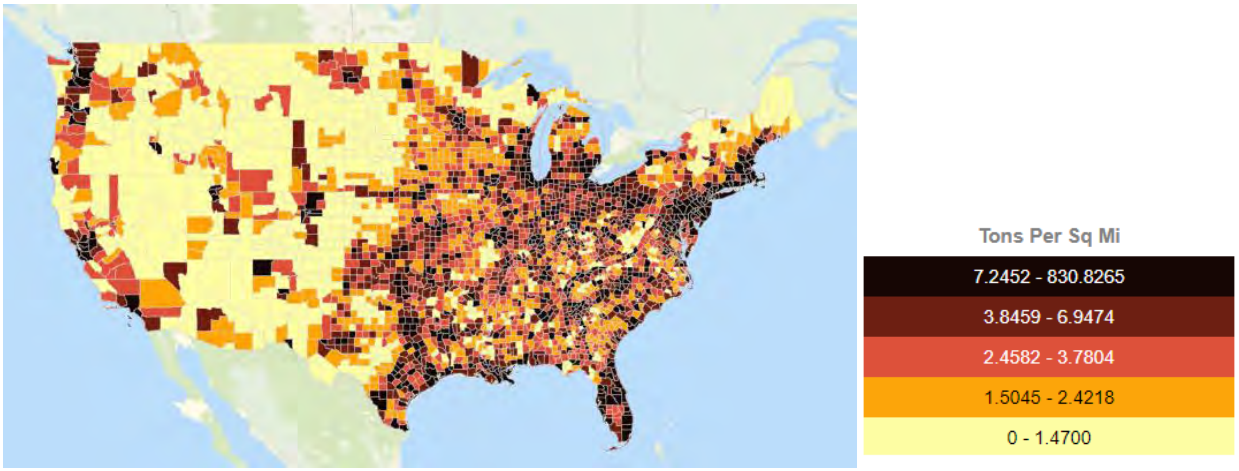

Figure 2-10. NO_X Emissions Density Map, tons per square mile.

Figure 2-11. NH_3 Emissions Density Map, tons per square mile.

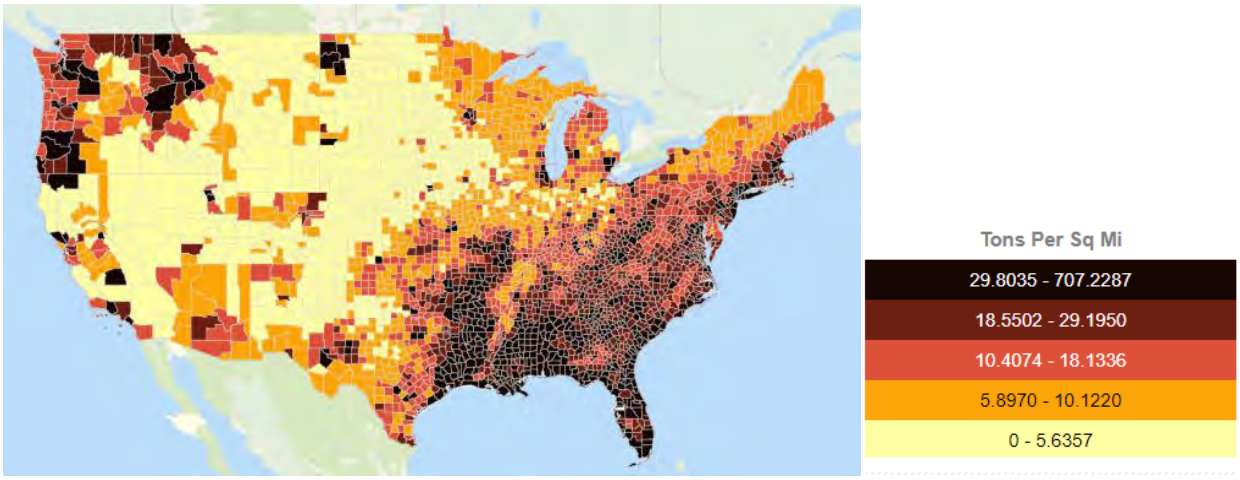

Figure 2-12. Anthropogenic (including wildfires) VOC Emissions Density Map, tons per square mile.

2.1.1.3.3 Uncertainty in Emission Estimates

Accuracy in an emissions inventory reflects the extent to which the inventory represents the actual emissions that occurred. Anthropogenic emissions of air pollutants result from a variety of sources such as power plants, industrial sources, motor vehicles and agriculture. The emissions from any individual source typically vary in both time and space. It is not practically possible to monitor each of the emission sources individually and, therefore, emission inventories necessarily contain assumptions, and must rely too on interpolation and extrapolation from a limited set of sample data.

The NEI process is based on a "bottom up" approach to developing emission estimates. This means that a combination of activity and an appropriate emissions factor is used to estimate emissions for all processes, including accounting for controls as possible. For the thousands of sources that make up the NEI, there is uncertainty in one or all of these factors. For some sources, such as EGUs, direct emission measurements enable the emission factors to be more certain than for sources without such direct measurements. For example, emission factors for residential wood combustion are taken from information available in the literature, regardless of its pedigree and direct applicability to the source in question. Many of these issues related to the analysis of uncertainty in the NEI are discussed by Day et al. (2019).

It is not clear how uncertainties in emission estimates affect air quality modeling, as there are no numerical empirical uncertainty estimates available for the NEI. However, by comparing modeled concentrations to ambient measurements, overall uncertainty in model outputs can be characterized. Some of this uncertainty in model outputs is likely due to uncertainty in emission estimates. The EPA uses information from air quality models and feedback from modelers and other stakeholders to help identify which sectors to prioritize for emissions data methods improvements.

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)[5] 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.

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).[6]

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 $PM_{10-2.5}$ 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

[5] 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).

[6] The AQI is an index for reporting daily air quality and translates air quality data into numbers and colors to help people understand how clean or polluted the air is, and what associated health effects might be a concern, especially for ozone and particle pollution.

composition are discussed below.[7] 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 $PM_{10-2.5}$, 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 2020, unless otherwise noted. Figure 2-13 below illustrates the changes in PM monitoring stations reporting to the EPA's AQS database by size fraction since 1970.

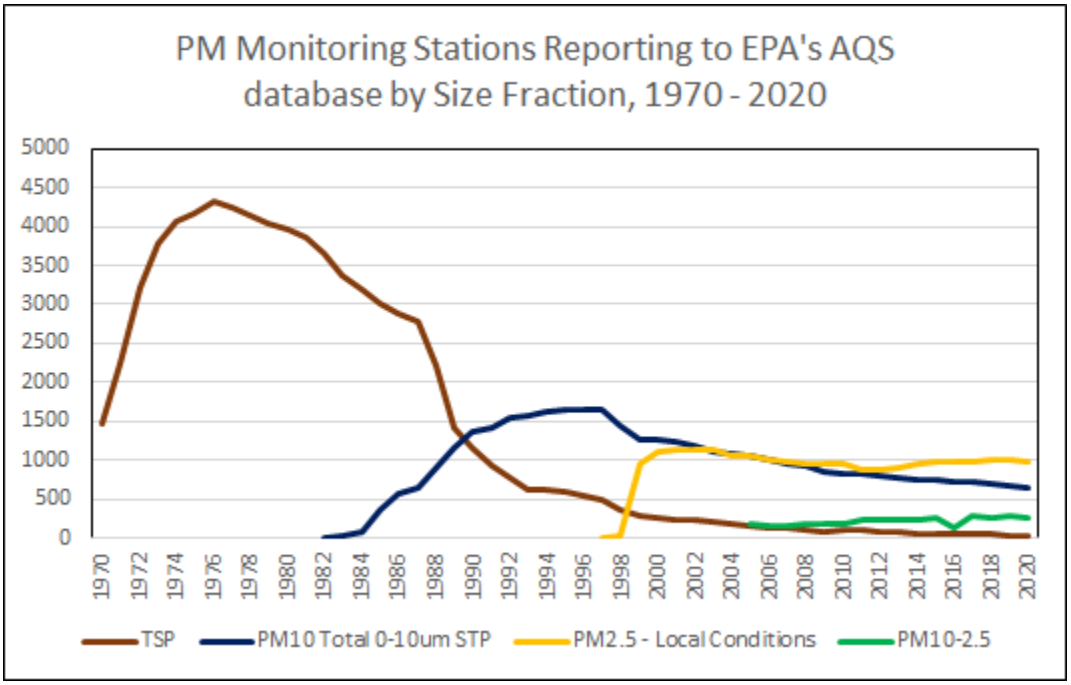

Figure 2-13. PM Monitoring stations reporting to EPA's AQS database by PM size fraction, 1970-2020.

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

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

were in operation. As of 2020, there were 104 TSP samplers still in operation as part of the Pb monitoring program; of these, 25 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[8] and neighborhood[9] scales to effectively characterize the emissions from both mobile and stationary sources, although not ruling out microscale[10] 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 2020, there were 680 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

[8] 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.

[9] 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.

[10] 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.

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, 385 of the stations operate automated continuous mass monitors approved as FEMs and 295 operate FRMs. About 30 of the PM_{10} stations have collocation with both continuous FEMs and FRMs. More than half of the PM_{10} stations with FRMs operate on a sample frequency of one in every sixth day, with about 55 stations operating every third day and another 55 stations 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,[11] 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

[11] 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.

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 for 2020 there were 527 FRM filter-based samplers that provide 24-hour $PM_{2.5}$ mass concentration data. Of these operating FRMs, 68 are providing daily $PM_{2.5}$ data, 340 every third day, and 119 every sixth day.

As of 2020, there are 950 continuous $PM_{2.5}$ mass monitors that provide hourly data on a near real-time basis reporting across the country. A total of 660 of the $PM_{2.5}$ continuous monitors are FEMs and therefore used both for comparison with the NAAQS and to report the AQI. Another 290 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 a third 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.

The quality of the data generated by $PM_{2.5}$ FRMs and automated FEMs were analyzed for years 2018-2020. Data quality terms for measurement uncertainty regularly assessed in the $PM_{2.5}$ monitoring program include precision and bias. Precision is calculated by comparing data from collocated methods of the same make and model operated by the same monitoring organization. Bias is calculated by comparing data from routinely operated FRMs or automated FEMs by the monitoring organization and comparing that to data from reference method audit samplers temporarily collocated and operated independently from the staff in the monitoring organization. Goals for measurement uncertainty are defined in Appendix A to 40 CFR Part 58. They state "Measurement Uncertainty for Automated and Manual $PM_{2.5}$ Methods. The goal for acceptable measurement uncertainty is defined for precision as an upper 90 percent confidence limit for the coefficient of variation (CV) of 10 percent and ±10 percent for total bias." The most recent three-year average estimate of national aggregate $PM_{2.5}$ FRM precision is 7.6% and bias is –7.5%.

Automated $PM_{2.5}$ FEMs include a wide variety of approved methods which can have different measurement principles. Data aggregated across all automated FEMs for years 2018-2020 result in a collocated precision of 12.8%. Bias can be calculated from the reference method audit program and by comparing continuous FEMs to collocated FRMs run by the monitoring agency. The 2018-2020 reference method audit program had a bias of -1.7% with a sample size of 573 audits across all continuous FEMs. Continuous FEMs compared to collocated monitoring agency FRMs were biased higher by 11.5% with a large sample size of 85,539 collocated pairs for 2018-2020 (all cases where both the FRM and continuous FEM are at or above 3.0 $\mu g/m^3$). When evaluating automated FEMs as individual methods, only two of the seven methods with available collocated precision data met the measurement uncertainty goal and six of the eleven methods met the bias goal. However, for collocated precision data and when considering a requirement for approval of candidate FEMs: "Statistical analyses based on the DQO model show that the precision of a candidate method is not, statistically, very important to annual concentration averages used for NAAQS attainment decisions, but would be important for a daily standard" (71 FR 2620, January 17, 2006) In summary, $PM_{2.5}$ automated FEMs tend to have higher collocated precision than FRMs and tend to have a positive bias relative to state and local operated FRMs.

2.2.3.2 Chemical Speciation and IMPROVE Networks

Due to the complex nature of fine particles, the EPA and states implemented the CSN to better understand the components of fine particle mass at selected locations across the country. The CSN was first piloted at 13 sites in 2000, and after the pilot phase, the program continued with deployment of the Speciation Trends Network (STN) later that year. The CSN ultimately grew to 54 trends sites and peaked in operation in 2005 with 252 stations: the 54 trends stations and nearly 200 supplemental stations. The original CSN program had multiple sampler configurations including the Thermo Andersen RAAS, Met One SASS/SuperSASS, and URG 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 2020 $PM_{2.5}$ CSN, long-term measurements are made at about 75 largely urban locations comprised of either the STN or the National Core (NCore) network.[12] NCore is a multipollutant network measuring particles, gases, and basic meteorology that has been in formal operation since January 1, 2011. Particle measurements made at NCore include $PM_{2.5}$ 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 $PM_{10-2.5}$ mass utilizing an FRM, FEM or IMPROVE for some of the rural locations. As of 2020, 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. Six of these stations have collocated sets of CSN samplers where the collocated samplers operate every sixth day to provide precision calculations of each chemical species measured. Another approximately 70 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.[13] 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.[14]

Specific components of fine particles are also measured through the IMPROVE monitoring program,[15] which supports regional haze characterization and tracks changes in

[12] 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.

[13] See https://www.epa.gov/amtic/chemical-speciation-network-csn for more information on the $PM_{2.5}$ speciation monitoring program.

[14] 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.epa.gov/amtic/csn-and-improve-protocol-network-assessment.

[15] 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://vista.cira.colostate.edu/Improve/.

visibility in Class I areas[16] 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 38 locations operated as IMPROVE protocol sites where a monitoring agency has requested participation in the program. These IMPROVE protocol sites operate the same way as the IMPROVE program, but they may serve several monitoring objectives (i.e., the same objectives as the CSN) and are not explicitly tied to the Regional Haze Program. Samplers at IMPROVE stations operate every third day. In January 2016, eight IMPROVE protocol stations stopped operating to ensure a sustainable IMPROVE program moving forward. Details on the process and outcomes of the CSN supplemental and IMPROVE protocol assessments used to identify sites that would no longer be funded are available on a website.[17] 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. CSN and IMPROVE data can also be used to better understand visibility through calculation of light extinction using the IMPROVE algorithm[18] to support reviews of the secondary PM NAAQS.

The quality of the data generated by the $PM_{2.5}$ speciation networks (CSN and IMPROVE) is assessed regularly, using a variety of metrics. Overall network precision, including uncertainties associated with both field operations and laboratory analyses, is assessed using the subset of sites with collocated samplers. Fractional uncertainty is one metric that both speciation networks regularly calculates using collocated data pairs above the MDL and reflects the overall percent uncertainty for the measurements. For CSN data collected between June 2016 and December 2019, the fractional uncertainties range from 5.6% for sulfate to 36.4% for chlorine.[19] For IMPROVE data collected in 2016 and 2017, the fractional uncertainties range from 2% for sulfur and sulfate to 27% for phosphorous.[20] In general, uncertainties are higher for species with

[16] See Regional Haze rule text at 50 CFR Part 51.308(d)(4) and (f)(6) (pasted below) lists SIP requirements, one of which is a "Monitoring Strategy…". This part of the rule doesn't necessarily require IMPROVE, rather it simply assures states that IMPROVE will meet this requirement. Specifically, this text reads: "(6) *Monitoring strategy and other implementation plan requirements.* The State must submit with the implementation plan a monitoring strategy for measuring, characterizing, and reporting of regional haze visibility impairment that is representative of all mandatory Class I Federal areas within the State. Compliance with this requirement may be met through participation in the Interagency Monitoring of Protected Visual Environments network."

[17] See the CSN and IMPROVE Protocol Network Assessment Website at: https://www.epa.gov/amtic/csn-and-improve-protocol-network-assessment

[18] 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.

[19] https://airquality.ucdavis.edu/sites/g/files/dgvnsk1671/files/inline-files/CSN_AnnualReport_2016Data_03.06.2019_FINAL_APPROVED.pdf

[20] http://vista.cira.colostate.edu/improve/wp-content/uploads/2019/11/IMPROVE_QAReport_11.15.2019.pdf

concentrations near the detection limit. Bias for the speciation networks can be assessed using reports from interlaboratory comparisons.[21]

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 being used in situations where the data is not applicable to the NAAQS (78 FR 3241, January 15, 2013).

2.2.4 $PM_{10-2.5}$ Monitoring

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

[21] https://www.epa.gov/amtic/chemical-speciation-network-interlaboratory-performance-evaluation-comparison-results

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 287 stations in 2020 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 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). In 2020, data from 72 monitoring sites across the county were reported from aethalometers and other related commercially available continuous carbon analyzers. While aethalometer and related continuous

carbon data are available at high time resolutions (e.g., 5-minute data), they are 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.[22] 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, 2019a). 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 2020, continuous sulfate is measured at two remaining monitoring sites, one each in Maine and North Carolina. Several other stations have historical data but are no longer monitoring continuous sulfate. Discontinued monitoring efforts for continuous sulfate is likely an outcome of the significantly lower sulfate concentrations throughout the east where these methods were operated. The continuous sulfate analyzer provides hourly data and these data can be readily compared to 24-hour sulfate data which are collected from the ion channel in both the CSN and IMPROVE programs.

In addition, over the last few years, the EPA has investigated the use of several PM sensor technologies as one of several areas of research intended to address the next generation of air measurements. The investigation into air sensors is envisioned to work towards near real-time or continuous measurement options that are smaller, cheaper, and more portable than traditional FRM or FEM methods. These sensor devices have the potential to be used in several applications such as identifying hotspots, informing network design, providing personal exposure monitoring, supporting risk assessments, and providing background concentration data for permitting. The EPA has hosted workshops and published several documents and peer-reviewed articles on this work.[23]

[22] 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.

[23] For more information, see https://www.epa.gov/sciencematters/epas-next-generation-air-measuring-research and https://www.epa.gov/air-sensor-toolbox

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.[24] As illustrated in Figure 2-14,[25] from 1990 to 2017, 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, 2019b, section 2.3.2.1). In more recent years (i.e., 2002 to 2017), 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 2-14 and Table 2-1.[26] 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.

It should be noted that the reductions shown in $PM_{2.5}$ and PM_{10} emissions in Figure 2-14, a Table 2-1, and any subsequent discussions of emission trends are most likely due to changes in the methods used by the EPA to estimate emissions for source sectors over time In all likelihood, emissions from dust and fires have increased over this time, which has been noted earlier in this document and mentioned broadly in the literature as well (Pu and Ginoux, 2017; Li et al., 2021; Liu et al., 2014; Schoennagel et al., 2017). It should also be noted that these data (in Figure 2-14 and Table 2-1) do not include emissions from wildfires, and these emissions can fluctuate greatly from year to year.

[24] More information on these trends, including details on methods and explanations on the noted changes over time is available at https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data.

[25] Emission trends in Figure 2-14 do not include wildfire emissions.

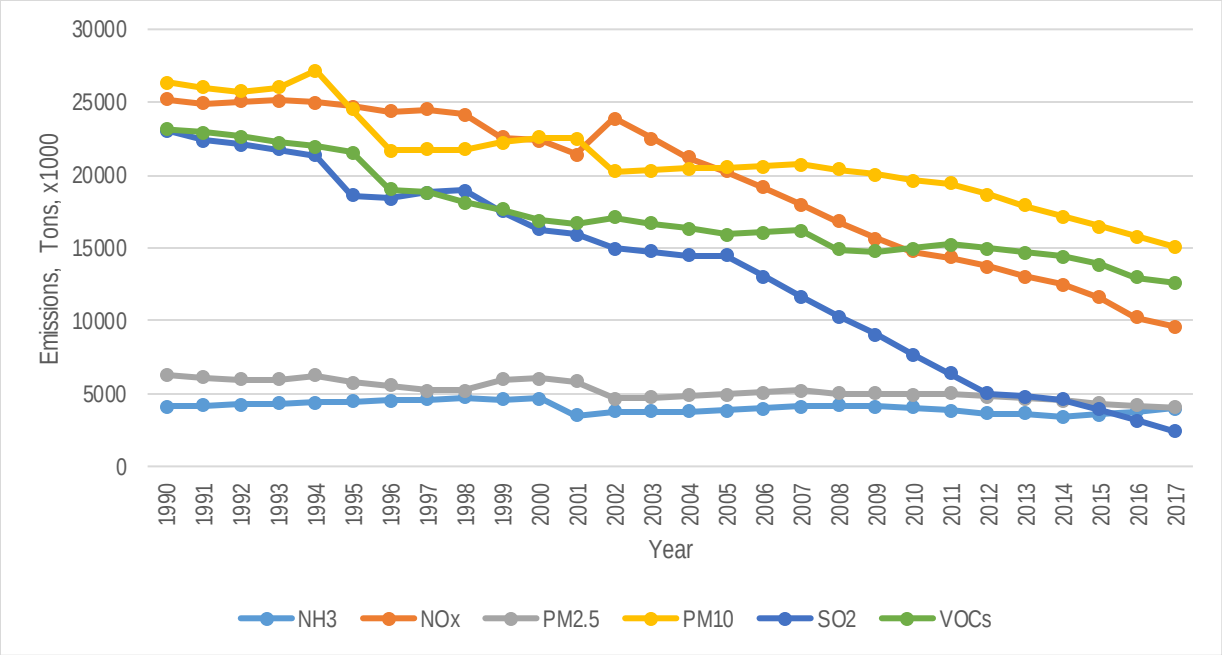

Figure 2-14. National emission trends of $PM_{2.5}$, PM_{10}, and precursor gases from 1990 to 2017.

Table 2-1. Percent Changes in PM and PM precursor emissions in the NEI for the time periods 1990-2017 and 2002-2017.

Pollutant	Percent Change in Emissions: 1990 to 2017	Percent Change in Emissions: 2002 to 2017	Major Sources that contribute to changes over time
NH_3	-3.1%	+5.6%	Agricultural Sources (Fertilizer and Livestock Waste), Fires
NO_X	-62%	-60%	EGUs, Mobile Sources
SO_2	-90%	-84%	EGUs, other Stationary Sources
VOCs	-45%	-26%	Solvents, Fires, Mobile Sources
$PM_{2.5}$	-36%	-14%	Dust, Fires
PM_{10}	-43%	-25%	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 2017 to 2019 averaged 8.0 µg/m^3 (with the 10th and 90th percentiles at 5.9 and 10.0 µg/m^3, respectively) and the 98th percentiles of 24-hour concentrations averaged 21.3 µg/m^3 (with the 10th and 90th percentiles at 14.0 and 29.7 µg/m^3, respectively). Figure 2-15 (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 annual average concentrations generally well below 12.0 $\mu g/m^3$ and 98th percentiles of 24-hour concentrations generally at or below 30 $\mu g/m^3$.

These concentrations are distinct from design values in part because they include days with episodic events like wildfires and dust storms which can have very high $PM_{2.5}$ and/or PM_{10} concentrations. The EPA's Exceptional Events Rule (81 FR 68216, October 3, 2016), most recently updated in 2016, describes the process by which these events can be excluded from the design values used for comparison to the NAAQS. For the remainder of Chapter 2, episodic events are included in the calculations of PM concentrations. When design values are discussed in Chapter 2, regionally-concurred exceptional events (as of June 2021) have been excluded from the analysis.[27]

[27] Regionally-concurred exceptional events are unusual or naturally-occurring events such as wildfires or high wind dust events that have 1) resulted in $PM_{2.5}$ concentrations above the level of the NAAQS, 2) been submitted by tribal, state or local air agencies under the EPA's Exceptional Events Rule to their respective EPA Region, and 3) received concurrence.

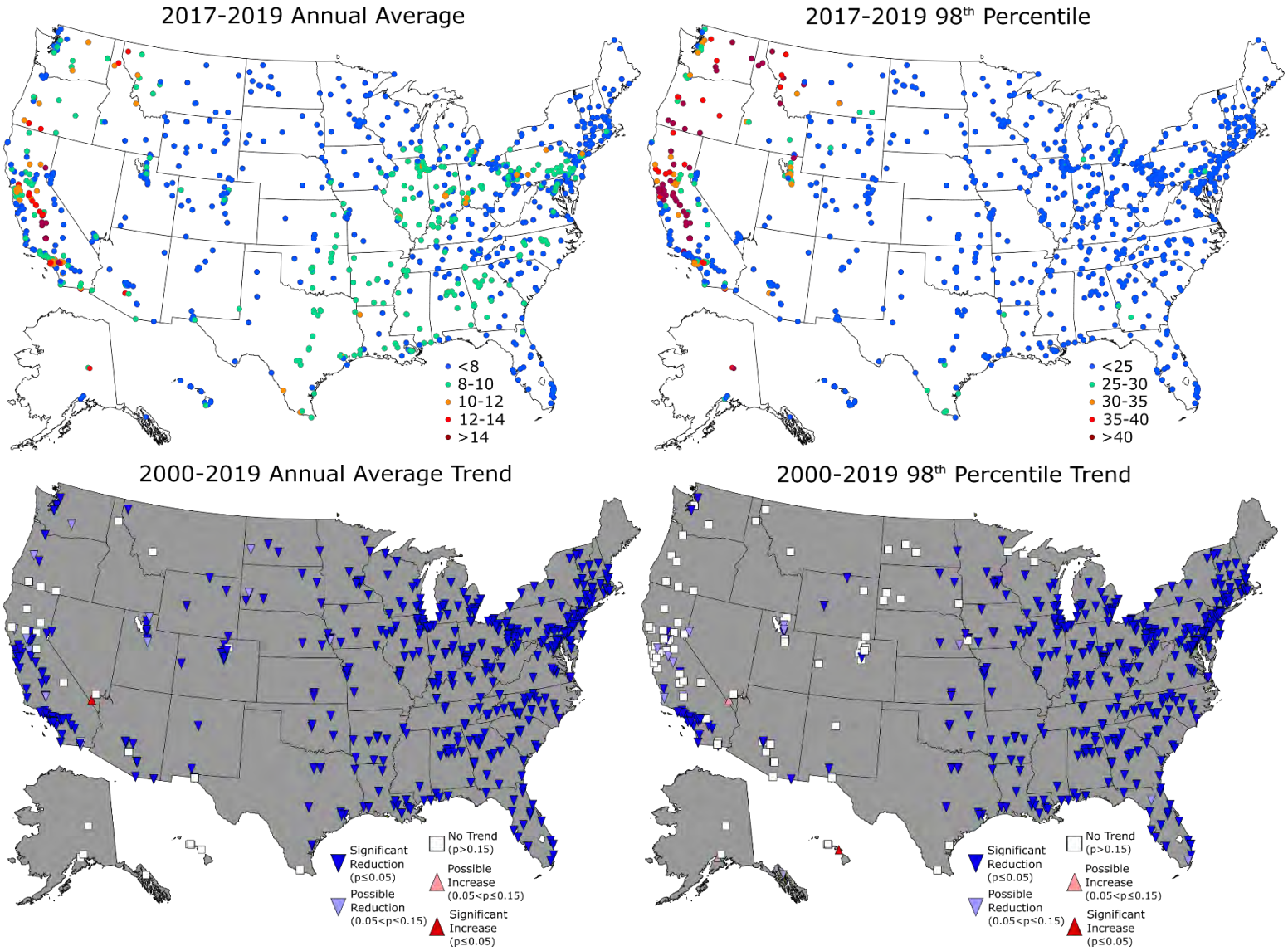

Figure 2-15. Annual average and 98th percentile of 24-hour $PM_{2.5}$ concentrations (in $\mu g/m^3$) from 2017-2019 (top) and linear trends and their associated significance (based on p-values) in $PM_{2.5}$ concentrations from 2000-2019 (bottom).

Analysis of monthly data indicate distinct peaks in national ambient $PM_{2.5}$ concentrations during the summer and the winter (U.S. EPA, 2019b, 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, 2019b, 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, 2019b, 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 2-15 reflect the substantial reductions that have occurred across much of the U.S. over recent years (Figure 2-15, bottom panels and Figure 2-16). From 2000 to 2019, national annual average $PM_{2.5}$ concentrations have declined from 13.5 $\mu g/m^3$ to 7.6 $\mu g/m^3$, a 43% decrease (Figure 2-16).[28] 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) due to the so-called "urban increment" of $PM_{2.5}$ from local sources in an urban area that is additive to the regional and natural background $PM_{2.5}$ concentrations.

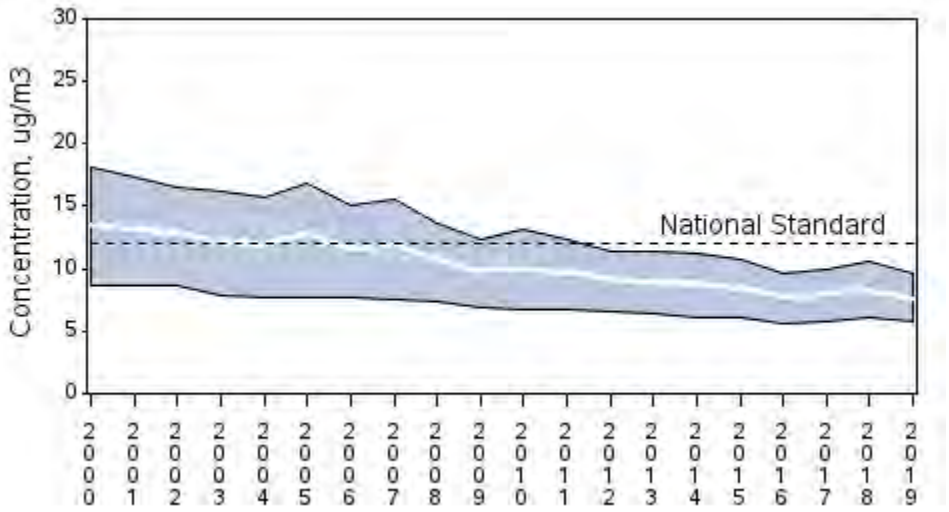

Figure 2-16. Seasonally-weighted annual average $PM_{2.5}$ concentrations in the U.S. from 2000 to 2019 (406 sites). (Note: The white line indicates the mean concentration while the gray shading denotes the 10th and 90th percentile concentrations.)

[28] See https://www.epa.gov/air-trends/particulate-matter-pm25-trends for up-to-date $PM_{2.5}$ trends information.

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 declined significantly (Figure 2-15, 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 2-15, 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 2-17). Such correlations are highest across the eastern U.S. and in coastal California, and are somewhat lower, though still generally positive, at sites in the Central and Western U.S. (i.e., outside of coastal California).

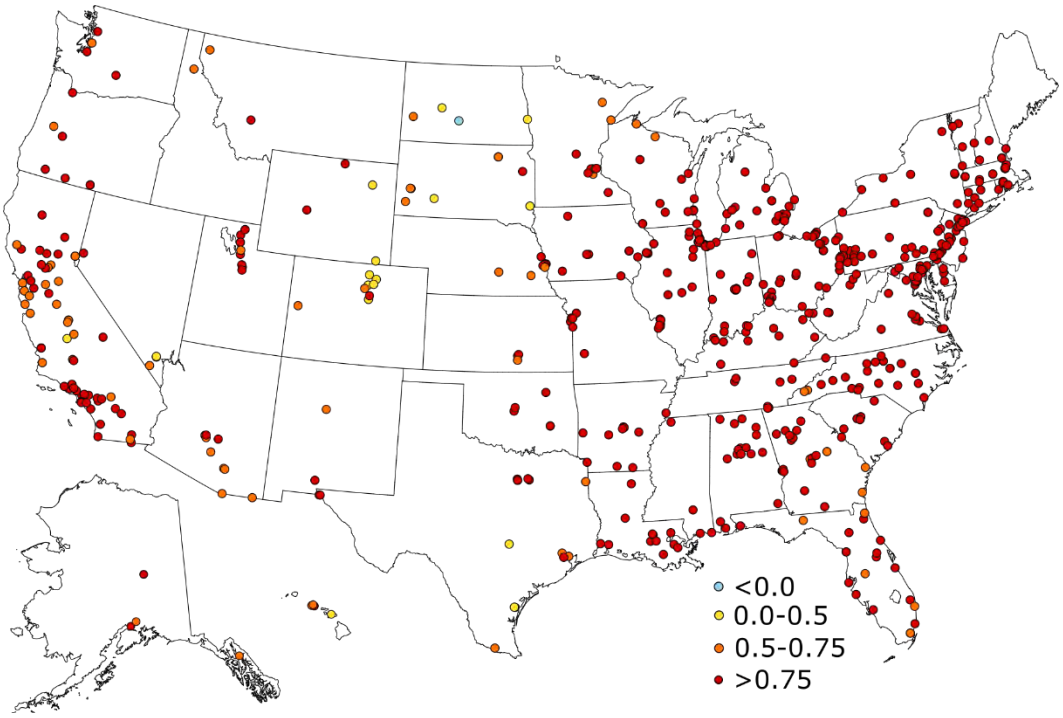

Figure 2-17. Pearson's correlation coefficient between annual average and 98th percentile of 24-hour $PM_{2.5}$ concentrations from 2000-2019.

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 2-18). In the Southeast, Northeast, and Industrial Midwest regions, the annual design values are high relative to the daily design values due in part to the infrequent impacts of episodic events

like wildfire or dust storms. On the other hand, the Northwest region has very high daily design values relative to the annual design values. This is due to episodically high $PM_{2.5}$ concentrations that affect the region, both from wintertime stagnation events and summer/fall wildfire smoke events.[29] 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.

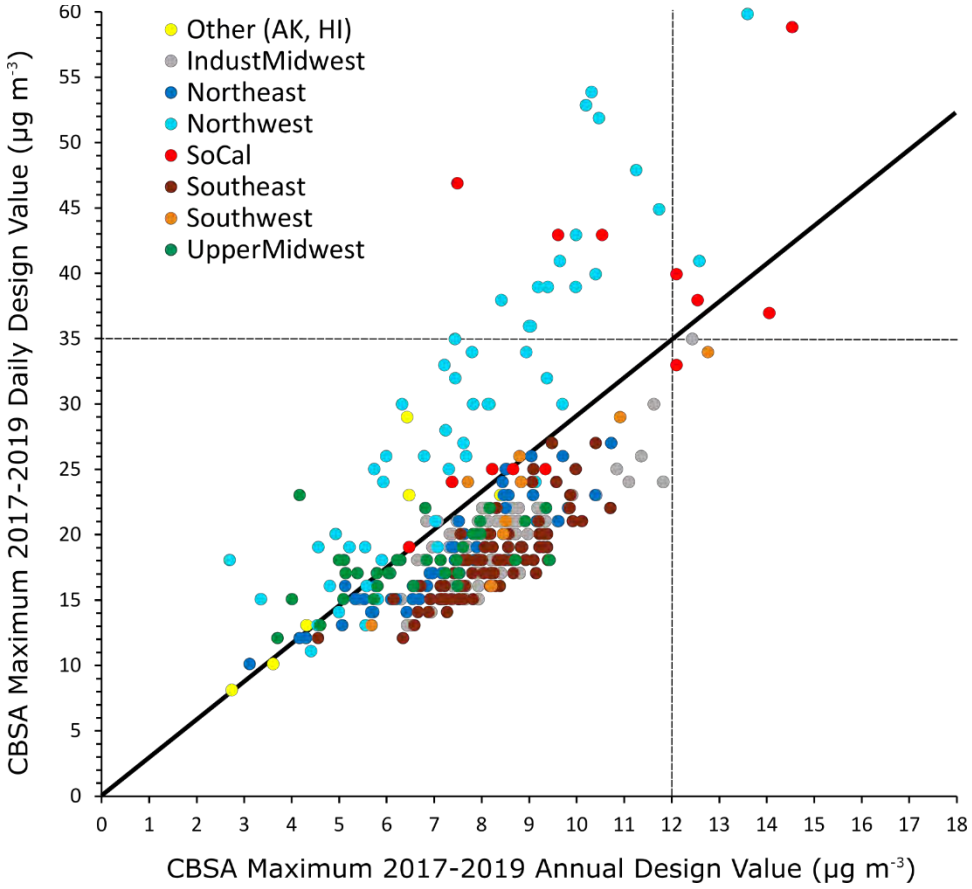

Figure 2-18. Scatterplot of CBSA maximum annual versus daily design values (2017-2019) with the solid black line representing the ratio of daily and annual NAAQS values.

2.3.2.2.2 $PM_{2.5}$ Near Major Roadways

Because of its longer atmospheric lifetime (U.S. EPA, 2019b, 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, 2019b, section 2.5.1.2.1). Analyses in the 2009 ISA for PM indicated that correlations between

[29] Due to the recent time period shown in Figure 2-18, it is likely that some of the annual and daily design values are affected by potential exceptional events associated with wildfire smoke that have yet to be regionally-concurred and removed from the design value calculations. The EPA defines exceptional events as unusual or natural-occurring events that 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 2017-2019 period.

$PM_{2.5}$ monitoring sites up to a distance of 100 km from each other were greater than 0.75 in most 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, 2019b, 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 2012 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 2014-2015. Gantt et al. (2021) found that this near-road increment has a diurnal cycle, with a peak during the morning rush hour. This near-road increment likely is additive to the urban increment of $PM_{2.5}$ from local sources in the CBSA including mobile sources on the numerous non-highway roads that are not monitored by the near-road network. For 2016-2018, Gantt et al. (2021) also reported that 52% and 24% of the time the near-road sites reported the highest annual and 24-hour $PM_{2.5}$ design value in the CBSA, respectively. Of the CBSAs with the highest annual design values at near-road sites reported by Gantt et al. (2021), those design values were, on average, 0.8 $\mu g/m^3$ higher than at the highest measuring non-near-road sites (range is 0.1 to 2.1 $\mu g/m^3$ higher at near-road sites).

Although most near-road monitoring sites do not have sufficient data to evaluate long-term trends in near-road $PM_{2.5}$ concentrations, Gantt et al. (2021) analyzed data at one long-term near-road-like site in Elizabeth, NJ,[30] and found that the annual average increment has generally decreased between 2001 and 2018 from about 2.0 $\mu g/m^3$ to about 1.3 $\mu g/m^3$. The trend in the near-road increment of elemental carbon at the Elizabeth, NJ site has shown a similar reduction, with values of ~1.0 $\mu g/m^3$ in 2001 decreasing to ~0.5 $\mu g/m^3$ in 2018. These data are consistent with the timing of EPA emission standards for motor vehicles.[31] 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. may have experienced similar decreasing trends in near-road $PM_{2.5}$ increments.

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

[30] 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.

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

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 2019 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, 2019b, 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 2-19 below presents the frequency distribution of 2-hour average $PM_{2.5}$ mass concentrations from all FEM $PM_{2.5}$ monitors in the U.S. for 2017-2019.[32] At sites meeting the current primary $PM_{2.5}$ standards, these 2-hour concentrations generally remain below 10 $\mu g/m^3$, and virtually never exceed 30 $\mu g/m^3$. Two-hour concentrations are higher at sites violating the current standards, generally remaining below 16 $\mu g/m^3$ and virtually never exceeding 80 $\mu g/m^3$.

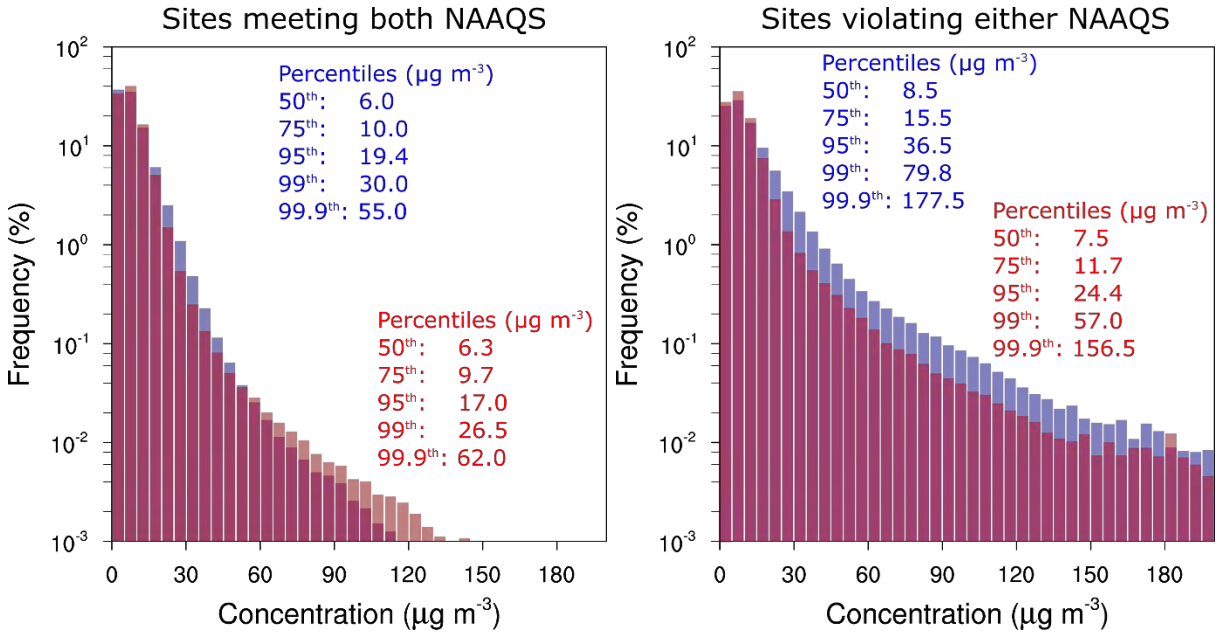

Figure 2-19. Frequency distribution of 2017-2019 2-hour averages for sites meeting both or violating either $PM_{2.5}$ NAAQS for October to March (blue) and April to September (red).

[32] 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. Similar analyses of 4-hour and 5-hour $PM_{2.5}$ concentrations are presented in Appendix A, Figure A-2 and Figure A-3, respectively.

The extreme upper end of the distribution of 2-hour $PM_{2.5}$ concentrations is shifted higher during the warmer months (red in Figure 2-19), 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 2-19), 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 high concentrations of organic carbon are widespread across most of the Continental U.S., as shown in Figure 2-20. 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.

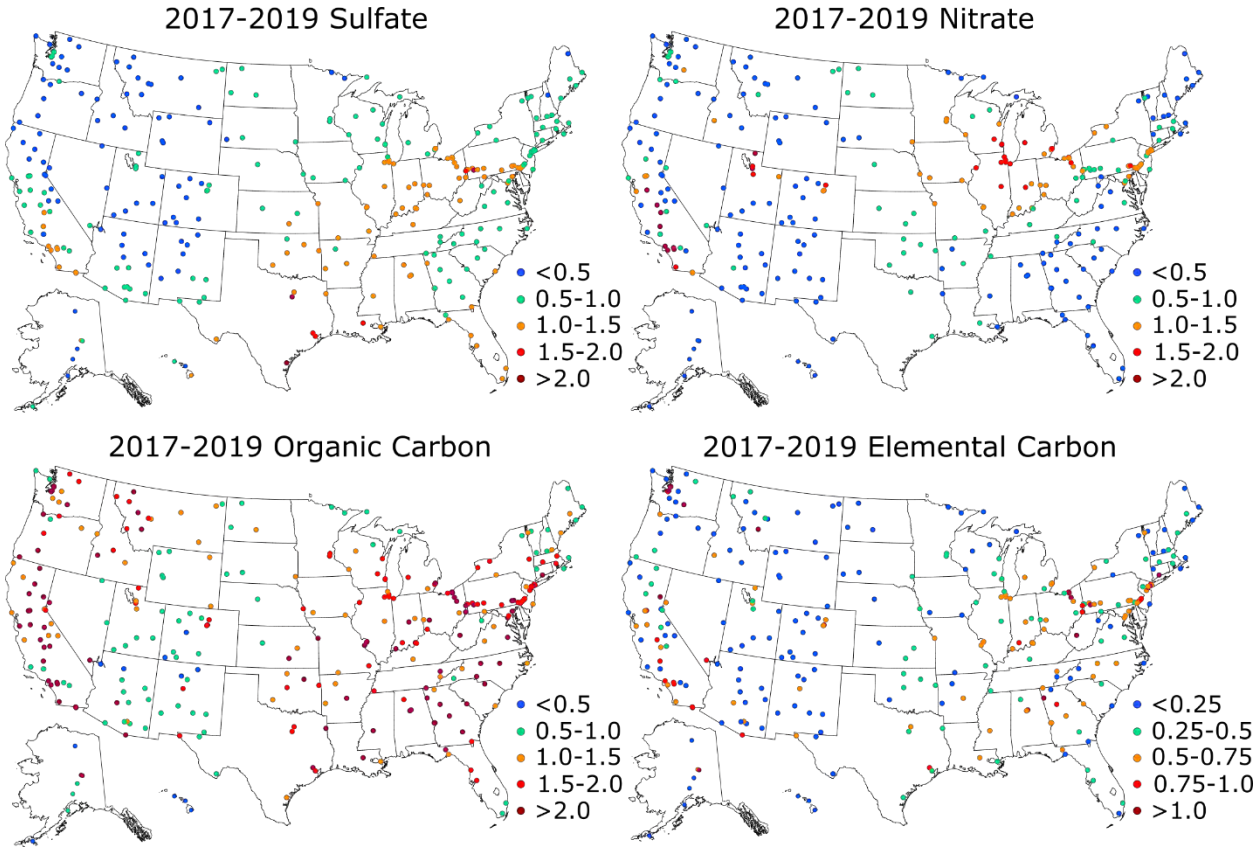

Figure 2-20. Annual average $PM_{2.5}$ sulfate, nitrate, organic carbon, and elemental carbon concentrations (in µg/m^3) from 2017-2019.

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, 2019b, 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, 2019b, 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 2017-2019 average of 2nd highest 24-hour PM_{10} concentration was 68 $\mu g/m^3$ (with the 10th and 90th percentiles at 28 and 124 $\mu g/m^3$, respectively) (Figure 2-21, top panels).[33] 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, 2019b, 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.

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

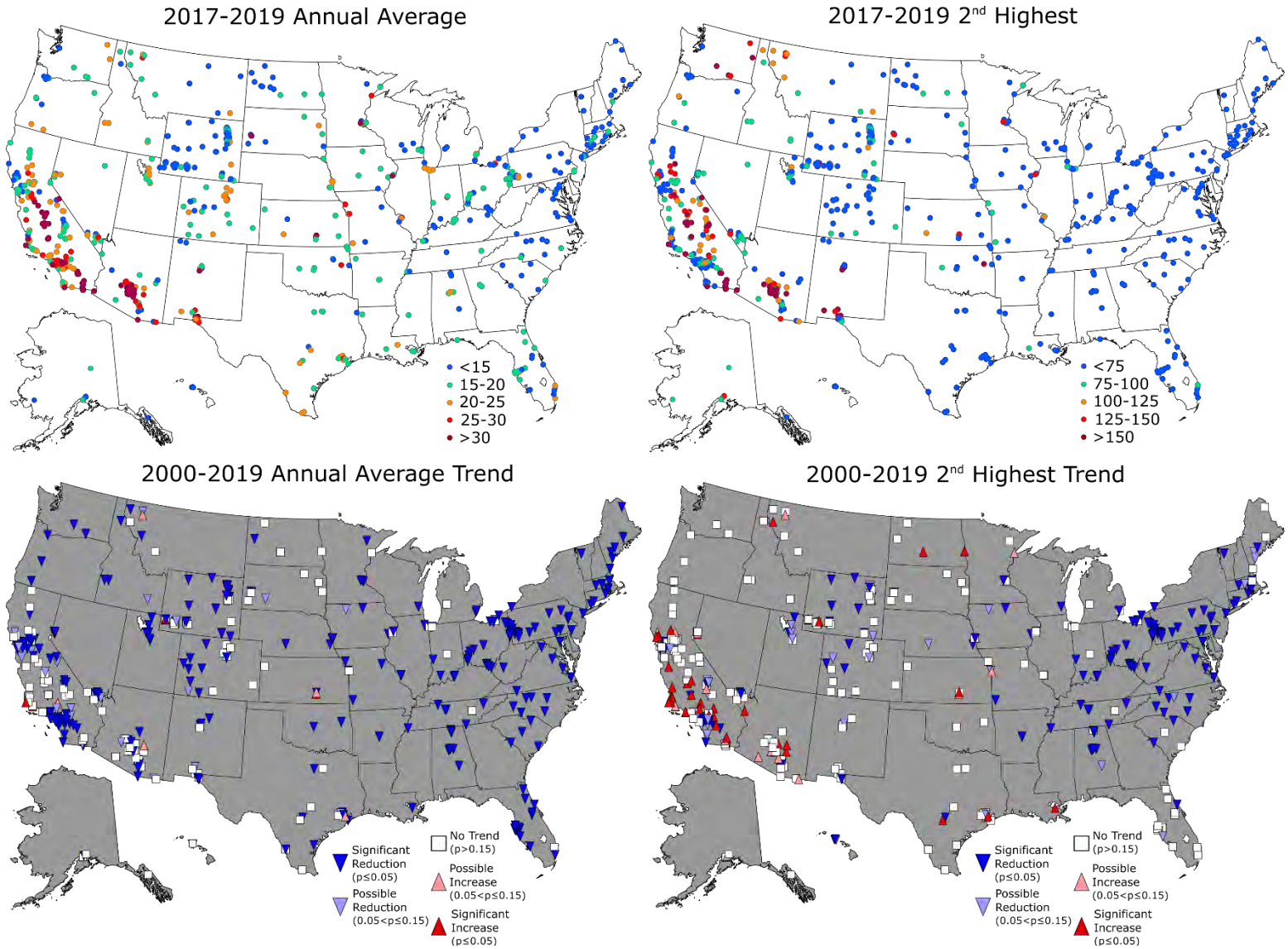

Figure 2-21. Annual average and 2nd highest PM_{10} concentrations (in µg/m^3) from 2017-2019 (top) and linear trends and their associated significance in PM_{10} concentrations from 2000-2019 (bottom).

Recent ambient PM_{10} concentrations reflect reductions that have occurred across much of the U.S. (Figure 2-21, bottom panels). From 2000 to 2019, 2nd highest 24-hour PM_{10} concentrations have declined by about 46% (Figure 2-22).[34] 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 2-21, bottom panels).

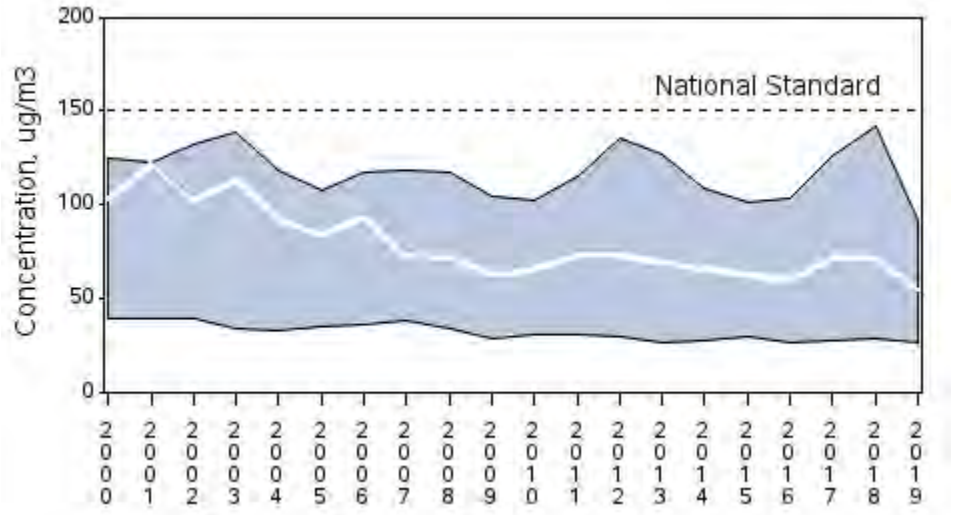

Figure 2-22. National trends in Annual 2nd Highest 24-Hour PM_{10} concentrations from 2000 to 2019 (262 sites). (Note: The white line indicates the mean concentration while the gray shading denotes the 10th and 90th percentile concentrations.)

Compared to previous reviews, data available from the NCore monitoring network in the current reconsideration 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 2-23). At most sites in the eastern U.S., the majority of PM_{10} mass is comprised of $PM_{2.5}$. 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.

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

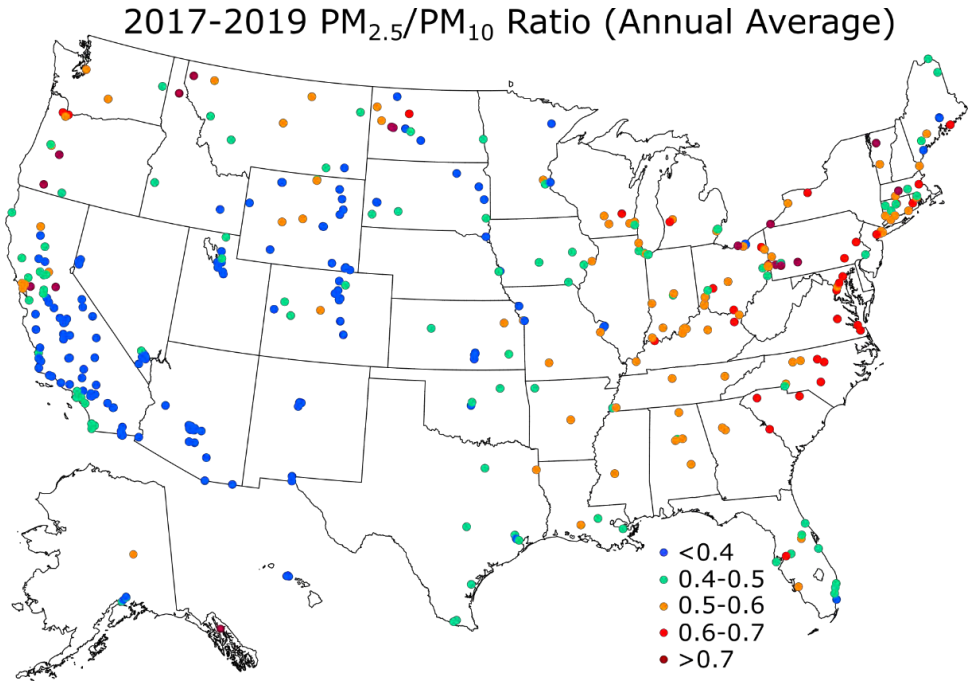

Figure 2-23. Annual average $PM_{2.5}/PM_{10}$ ratio for 2017-2019.

For days with very high PM_{10} concentrations (Figure 2-24), 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 $PM_{2.5}$.

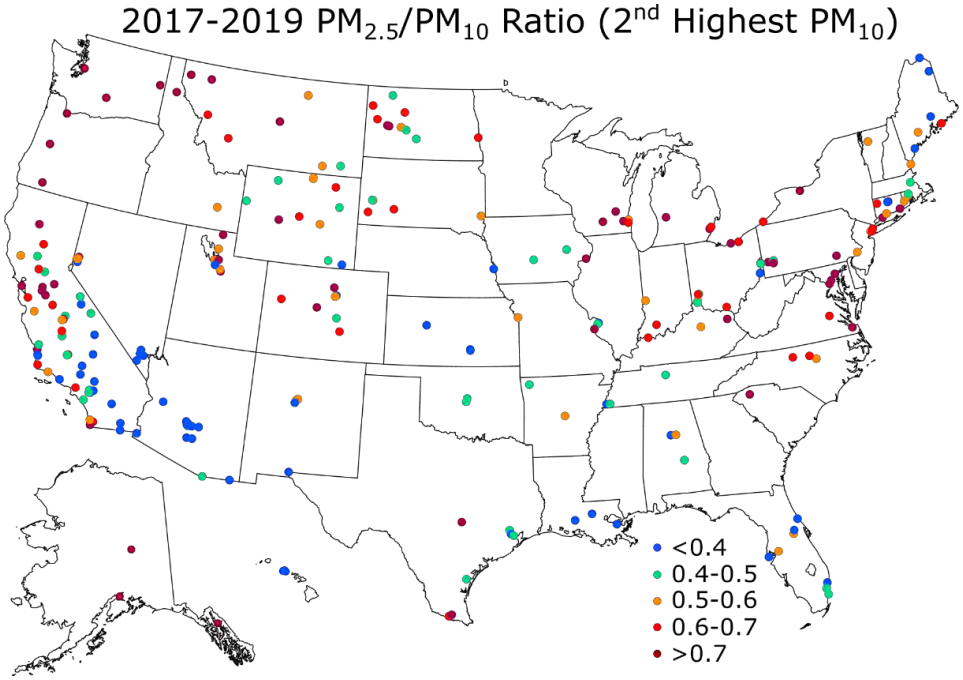

Figure 2-24. $PM_{2.5}/PM_{10}$ ratio on the date of the second highest PM_{10} concentrations for 2017-2019.

2.3.2.5 National Characterization of $PM_{10-2.5}$ Mass

Since the 2012 review, the availability of $PM_{10-2.5}$ ambient concentration data has greatly increased. As illustrated in Figure 2-25[35] (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 2-25, lower panels).

[35] The sites shown in Figure 2-25 have a data completeness of either 75% or ≥182 valid days in each year.

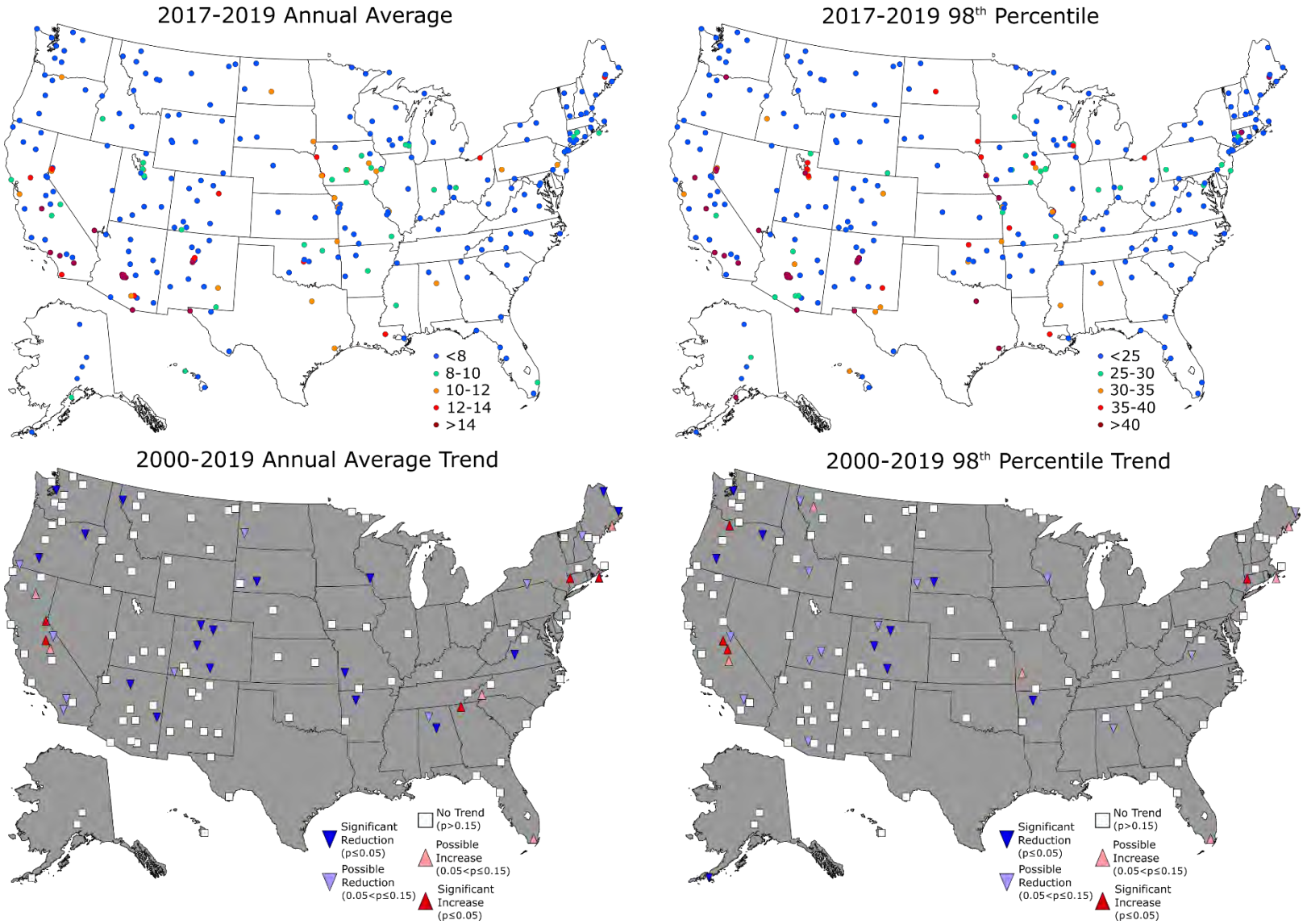

Figure 2-25. Annual average and 98th percentile $PM_{10-2.5}$ concentrations (μg/m³) from 2017-2019 (top) and linear trends and their associated significance in $PM_{10-2.5}$ concentrations from 2000-2019 (bottom).

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, 2019b). 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 2-26; U.S. EPA, 2019b, 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.

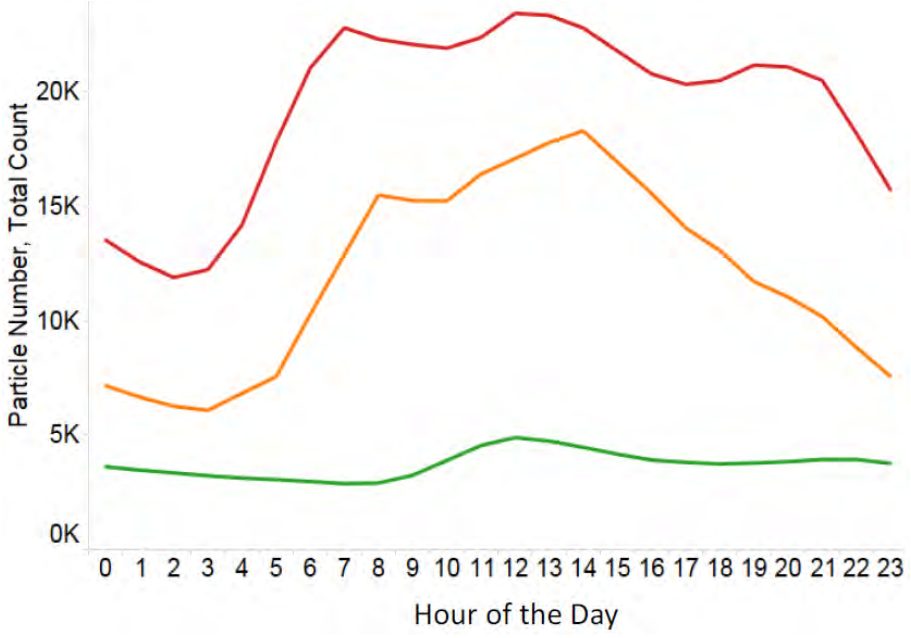

Figure 2-26. Average hourly particle number concentrations from three locations in the State of New York for 2014 to 2015 (green is Steuben County, orange is Buffalo, red is New York City). (Source: Figure 2-18 in U.S. EPA, 2019b).

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 (66 FR 5002, January 18, 2001), a reduction in local industrial activity, and the closure of a nearby coal-fired power plant (Wang et al., 2011; U.S. EPA, 2019b, section 2.5.2.1.4).

In addition, at a site in Illinois the annual average particle number concentration declined between 2000 and 2019, closely matching the reductions in annual $PM_{2.5}$ mass over that same period (Figure 2-27, below). Particle number concentrations at this site are closer to those of the background site in Figure 2-27 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.

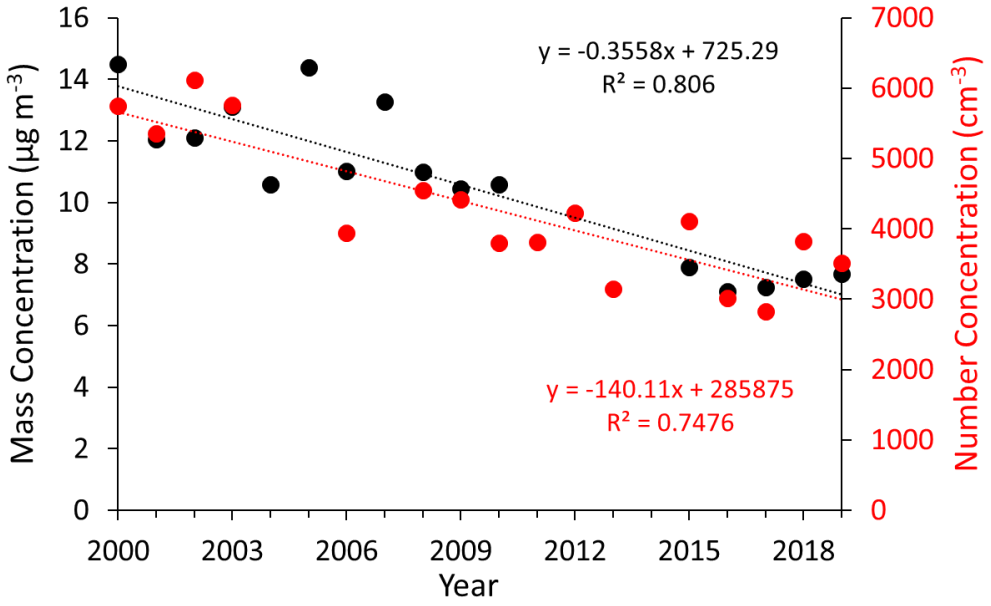

Figure 2-27. Time series of annual average mass and number concentrations (left) and scatterplot of mass vs. number concentration (right) between 2000-2019 in Bondville, IL.

2.3.3 Characterizing Ambient $PM_{2.5}$ Concentrations for Exposure

Epidemiologic studies use various methods to characterize exposure to ambient $PM_{2.5}$. The methods used to estimate $PM_{2.5}$ concentrations can vary from traditional methods using monitoring data from ground-based monitors to those using more complex hybrid modeling approaches. Studies using hybrid modeling approaches aim to broaden the spatial coverage of estimated $PM_{2.5}$ concentrations by expanding beyond just those areas with monitors and providing estimates in areas that do not have ground-based monitors (i.e., areas that are generally less densely populated and tend to have lower $PM_{2.5}$ concentrations). As such, the

hybrid modeling approaches tend to broaden the areas captured in the exposure assessment, and in doing so, the studies that utilize these methods tend to report lower mean $PM_{2.5}$ concentrations than monitor-based approaches. Further, other aspects of the method used to calculate $PM_{2.5}$ concentrations (i.e. population weighting, trim mean) can also have an impact on the predicted exposure and the related study-reported mean concentration.

2.3.3.1 Predicted Ambient $PM_{2.5}$ and Exposure Based on Monitored Data

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, 2019b, chapter 3).

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 2-28 and Figure 2-29 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 2-29 indicates a more variable relationship between maximum 24-hour $PM_{2.5}$ design values and annual average concentrations.

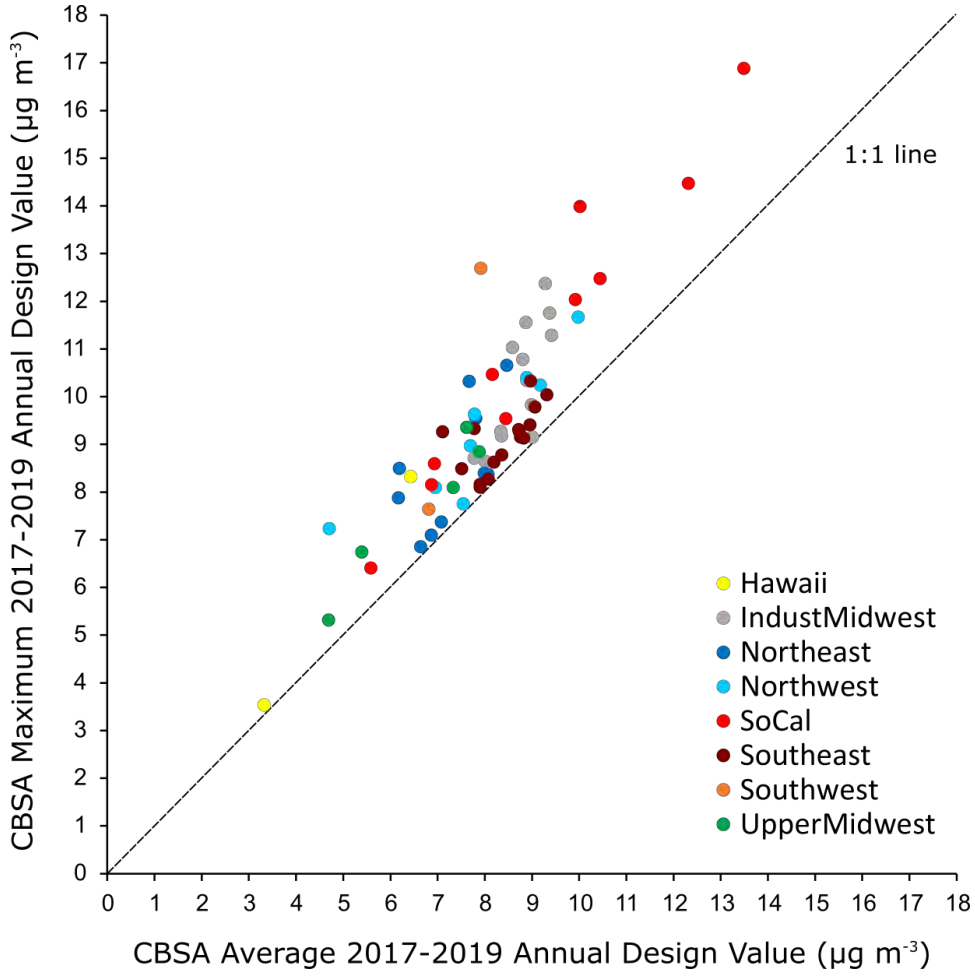

Figure 2-28. Comparison of CBSA average annual design values and CBSA maximum annual design values for 2017-2019. (Note: Includes all CBSAs with at least 3 valid annual DVs.)

Table 2-2. Nationwide averages of ratios of maximum annual $PM_{2.5}$ design values to average composite monitor $PM_{2.5}$ concentrations across CBSAs.

Years of Monitoring Data	Number of Monitors per CBSA	Number of CBSAs	Ratio of Maximum Annual DV to CBSA Average	Ratio of Maximum 24-hour 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
2017-2019	3 or more	67	1.16	1.22
	4 or more	47	1.19	1.25
	5 or more	32	1.21	1.26

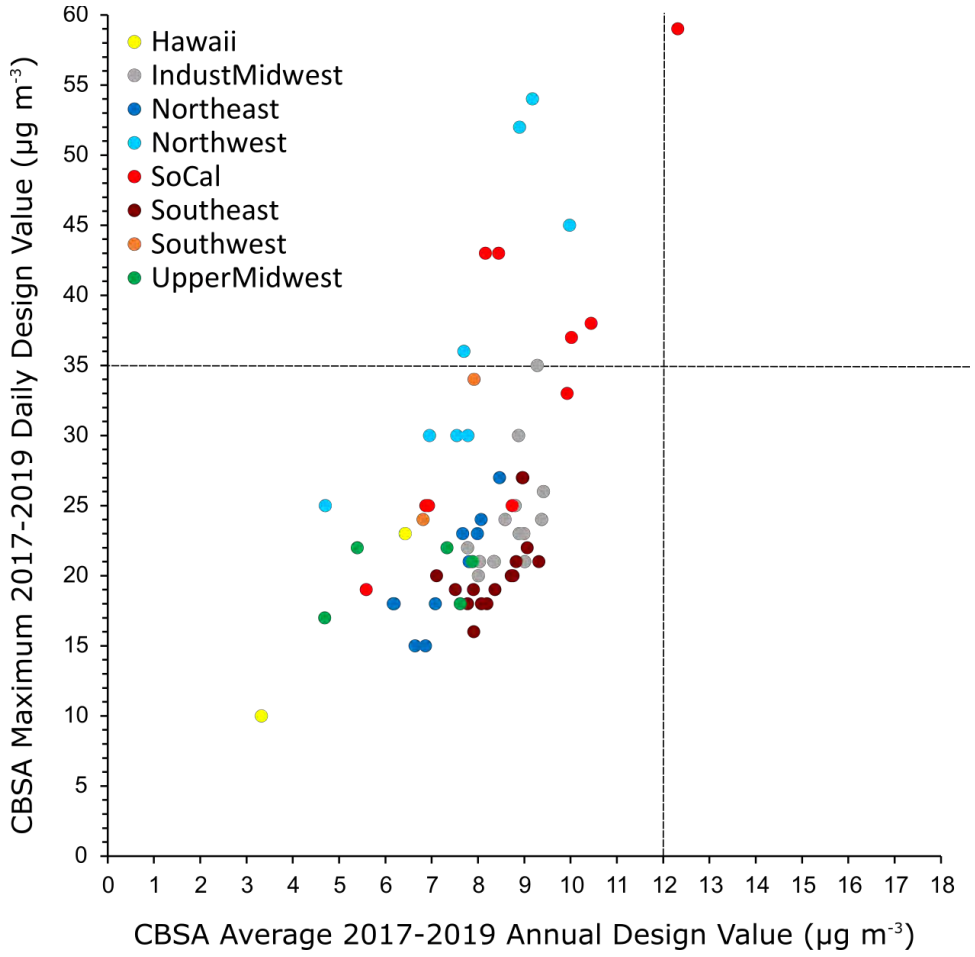

Figure 2-29. Comparison of CBSA average annual design values and CBSA maximum daily design values for 2017-2019. (Note: Dashed lines indicate the level of the current 24-hour $PM_{2.5}$ standard (35 µg/m^3) and the current annual PM standard (12 µg/m^3). Includes all CBSAs with at least 3 valid daily and 3 valid annual DVs.)

2.3.3.2 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, 2019b, 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, 2019b, 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, 2019b, 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.

2.3.3.2.1 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, Berrocal et al., 2020). 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[36] 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. An ensemble model based on random forest, neural network, and gradient boosting methods has also been recently applied to develop daily 1-km $PM_{2.5}$ concentration fields over the U.S. for the 2000-2015 period (Di et al., 2019). A wide range of parametric and non-parametric hybrid $PM_{2.5}$ models have recently been reviewed in Chapter 3 of the 2019 ISA (U.S. EPA, 2019b).

2.3.3.2.2 Performance of the Methods

[36] See http://fizz.phys.dal.ca/~atmos/martin/?page_id=140

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 μg 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 μg 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 μg 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 R^2: 0.66-0.70; Wang et al., 2018b; Kelly et al., 2019a) 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., 2019a ; 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 sharp concentration gradients, 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., 2019a). 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., 2019a; Di et al., 2016; van Donkelaar et al., 2019). This trend could be due in part to increases in the fraction of the $PM_{2.5}$ distribution that is explained by less predictable stochastic variation as $PM_{2.5}$ concentrations decrease (Just et al., 2020). Trends in model performance associated with $PM_{2.5}$ concentration (e.g., Figure 2-30) could also be due 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., 2019a; Berrocal et al., 2020).

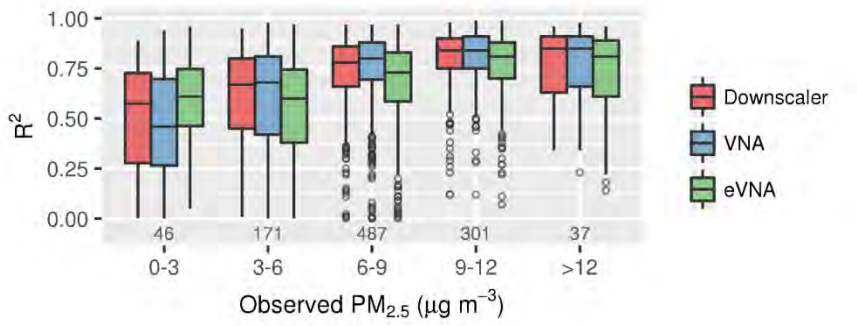

Figure 2-30. R^2 for ten-fold cross-validation of daily $PM_{2.5}$ predictions in 2015 from three methods for individual sites as a function of observed concentration. Text indicates the number of monitors in the $PM_{2.5}$ concentration range. Downscaler: Bayesian downscaler of CMAQ predictions; VNA: Voronoi Neighbor Averaging; eVNA: enhanced-VNA. From Kelly et al., 2019a.

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. Diao et al. (2019) reviewed publicly available $PM_{2.5}$ products and identified inconsistencies in $PM_{2.5}$ predictions from several methods. Kelly et al. (2021) reported broad agreement among model predictions at the national scale but differences in the intra-urban variations in $PM_{2.5}$ concentrations.

2.3.3.2.3 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 2-31, where predictions from the methods were averaged to a common 12-km grid. The fields were developed using a Bayesian downscaler (downscaler, Berrocal et al., 2012), neural network (DI2016, Di et al., 2016), random

forest (HU2017, Hu et al., 2017), and GWR of residuals from satellite-based $PM_{2.5}$ 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.

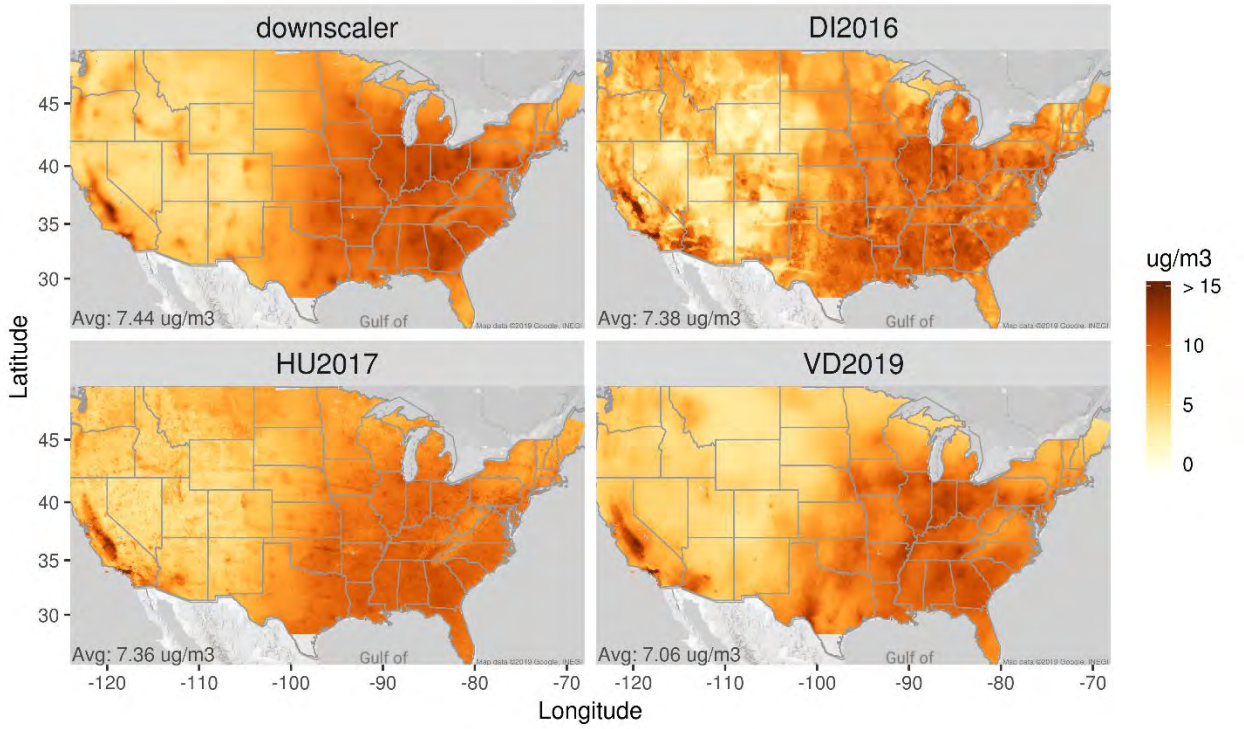

Figure 2-31. Comparison of 2011 annual average $PM_{2.5}$ concentrations from four methods. (Note: These four methods include: downscaler (Berrocal et al., 2012), DI2016 (Di et al., 2016), HU2017 (Hu et al., 2017), and VD2019 (van Donkelaar et al., 2019). Predictions have been averaged to a common 12-km grid for this comparison.)

Table 2-3. Mean 2011 $PM_{2.5}$ concentration by region for predictions in Figure 2-29

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 2-32, $PM_{2.5}$ concentrations predicted by the four methods are shown at their native resolution for regions centered on California, New Jersey, and Arizona. Predictions have sharper spatial gradients and span a wider range of concentrations for the western regions centered on California and Arizona (Figure 2-32, panels a and c) than the eastern region centered on New Jersey (Figure 2-32, panel b). Despite general agreement among predictions for the California and the eastern U.S. areas, the spatial texture of the concentration fields differs among methods. For instance, the 12-km Bayesian downscaler produces the smoothest $PM_{2.5}$ 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 2-32, panel c).

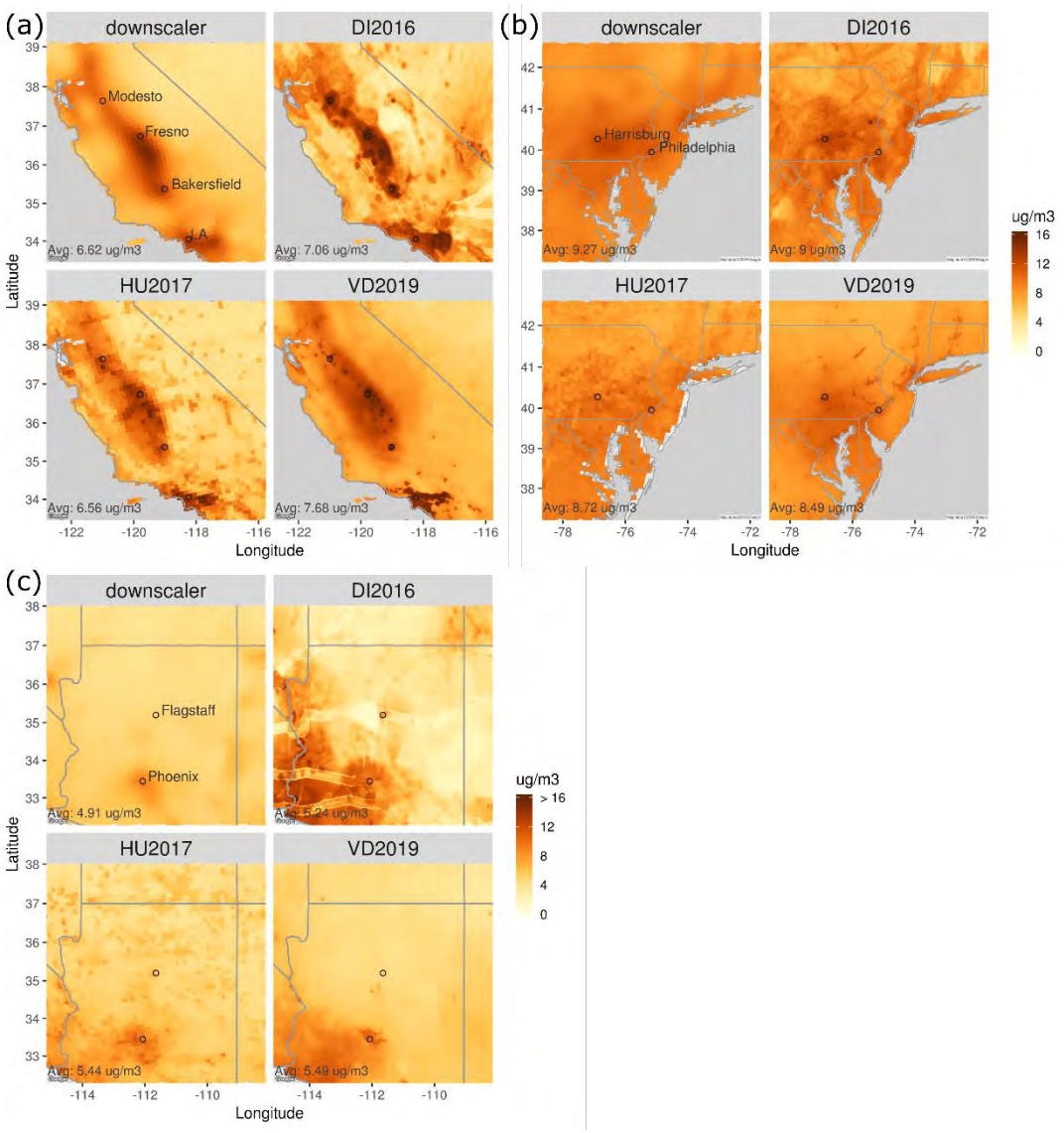

Figure 2-32. Comparison of 2011 annual average $PM_{2.5}$ concentrations from four methods for regions centered on the (a) California (b) New Jersey, and (c) Arizona. Predictions are shown at their native resolution (i.e., about 1-km for DI2016 and VD2019 and 12-km for downscaler and HU2017).

In Figure 2-33, the coefficient of variation (CV; i.e., the standard deviation divided by the mean) among methods is shown in percentage units based on predictions that were averaged to a common 12-km grid. The largest values occur in the western U.S. (Figure 2-33, panel a), where spatial gradients are high, terrain is complex, wildfire is prevalent, monitoring is relatively sparse, and $PM_{2.5}$ concentrations are low on average. The distance from the grid-cell center to the nearest monitor is greater than 100 km for broad areas of the west (Figure 2-34).

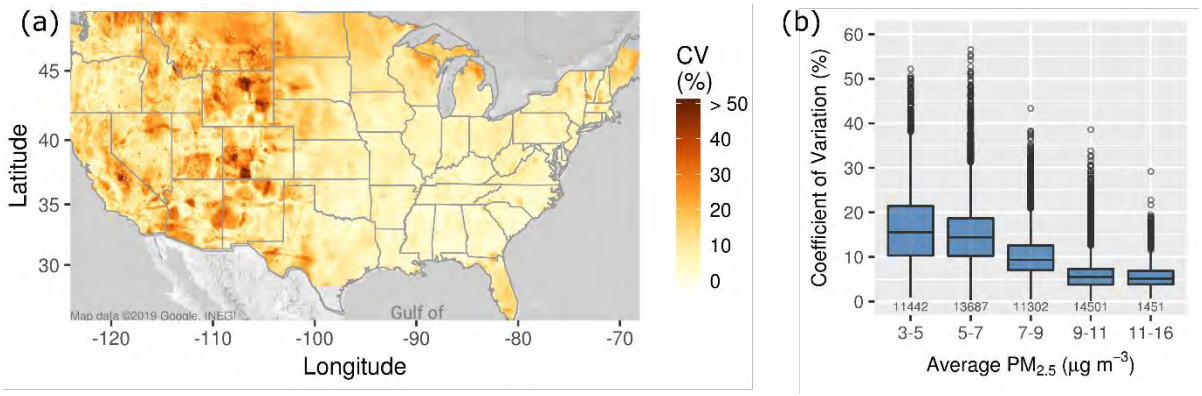

Figure 2-33. (a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in percentage units for the four models in Figure 2-29. (b) Boxplot distributions of CV for grid cells binned by the average $PM_{2.5}$ concentration for the four models. (Note: The box brackets the interquartile 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.)

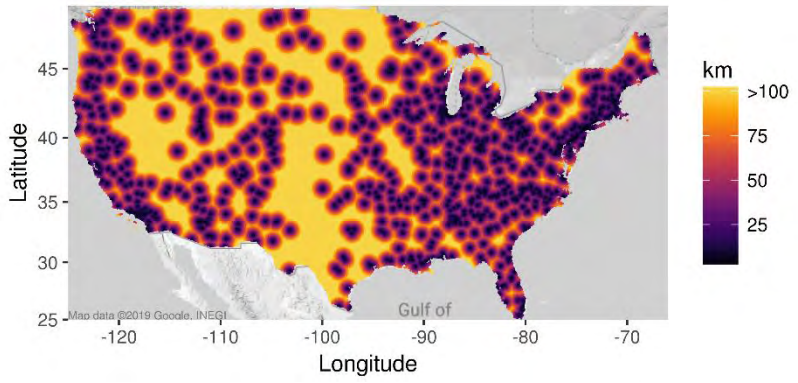

Figure 2-34. Distance from the center of the 12-km grid cells to the nearest $PM_{2.5}$ monitoring site for $PM_{2.5}$ measurements from the AQS database and IMPROVE network.

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 2-35, 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 2-35, second row). The CV among methods increases with decreasing concentration (Figure 2-33 above, panel b), and the median CV is about 15% for grid cells with mean concentrations less than 7 µg/m^3. As illustrated by Figure 2-33 and Figure 2-35, the low-concentration areas with relatively large CVs are in the western U.S. and along the northern and southern border of the eastern U.S.

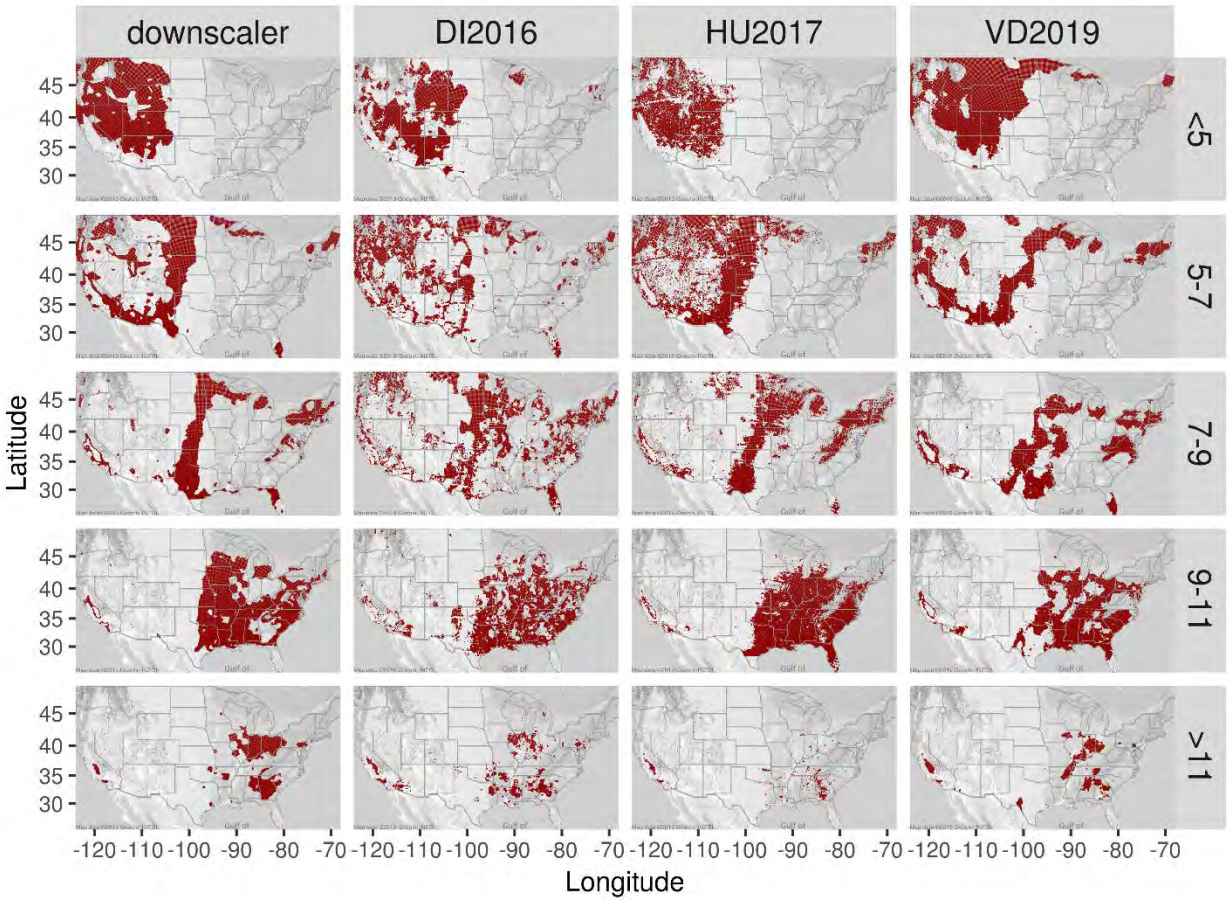

Figure 2-35. Location of $PM_{2.5}$ predictions by range in annual average concentration for the four prediction methods at their native resolution. (Note: Concentration ranges: < 5 $\mu g/m^3$, 5-7 $\mu g/m^3$, 7-9 $\mu g/m^3$, 9-11 $\mu g/m^3$, and >11 $\mu g/m^3$.)

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 2-36. 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 $\mu g/m^3$ in 2016 are limited to California and southwest Arizona.

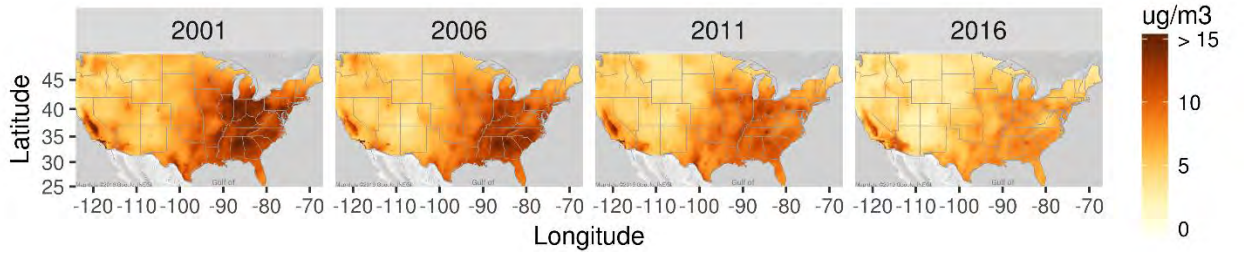

Figure 2-36. Annual mean $PM_{2.5}$ from the VD2019 method (van Donkelaar et al., 2019) for 2001, 2006, 2011, and 2016.

2.3.3.2.4 Comparison of $PM_{2.5}$ Fields in Estimating Exposure and Relative to Design Values

Two types of hybrid approaches that have been utilized in several key $PM_{2.5}$ epidemiologic studies in the 2019 ISA and draft ISA Supplement include neural network approaches and use of GWR of residual $PM_{2.5}$ with land-use and other variables to improve estimates of $PM_{2.5}$ concentration in the US. As such, we further compare these two types of approaches across various scales and taking into account population weighting approaches utilized in epidemiologic studies when estimating $PM_{2.5}$ exposure. Additionally, we assess how average $PM_{2.5}$ concentrations computed using these hybrid surfaces compare to the maximum design values measured at ground-based monitors. For this assessment, we evaluate the DI2019[37] and HA2020[38] surfaces. This analysis may help to inform how the magnitude of the overall study reported mean $PM_{2.5}$ concentrations in epidemiologic studies may be influenced by the approach used to compute that mean and how that value might compare to monitor reported concentrations.

In estimating exposure, some studies focus on estimating concentrations in urban areas, while others examine the entire U.S. or large portions of the country. Figure 2-37 shows the spatial distribution of the annual average $PM_{2.5}$ concentrations for 2015 using the DI2019 surface nationwide (panel A) and for CBSAs only (panel B). As shown in the figure, the geographic coverage is much less when estimating the annual average $PM_{2.5}$ concentrations at the CBSA scale compared to the national scale and tends to be primarily representative of areas that are

[37] This analysis includes an updated version of the surface used in Di et al. (2016). Predictions in Di et al. (2016) were for 2000 to 2012 using a neural network model. The Di et al. (2019) study improved on that effort in several ways. First, a generalized additive model was used that accounted for geographic variations in performance to combine predictions from three models (neural network, random forest, and gradient boosting) to make the final optimal $PM_{2.5}$ predictions. Second, the datasets were updated that were used in model training and included additional variables such as 12-km CMAQ modeling as predictors. Finally, more recent years were included in the Di et al. (2019) study.

[38] The HA2020 field is based on the V4.NA.03 product available at: https://sites.wustl.edu/acag/datasets/surface-pm2-5/. The name "HA2020" comes from the references for this product (Hammer et al., 2020; van Donkelaar et al., 2019).

more urban or densely populated. Further, the areas that are not included in the CBSA-only analysis tend to have lower $PM_{2.5}$ concentrations. These areas tend to be more rural or less densely populated areas, and likely correspond to those locations where monitoring data availability is limited or nonexistent.

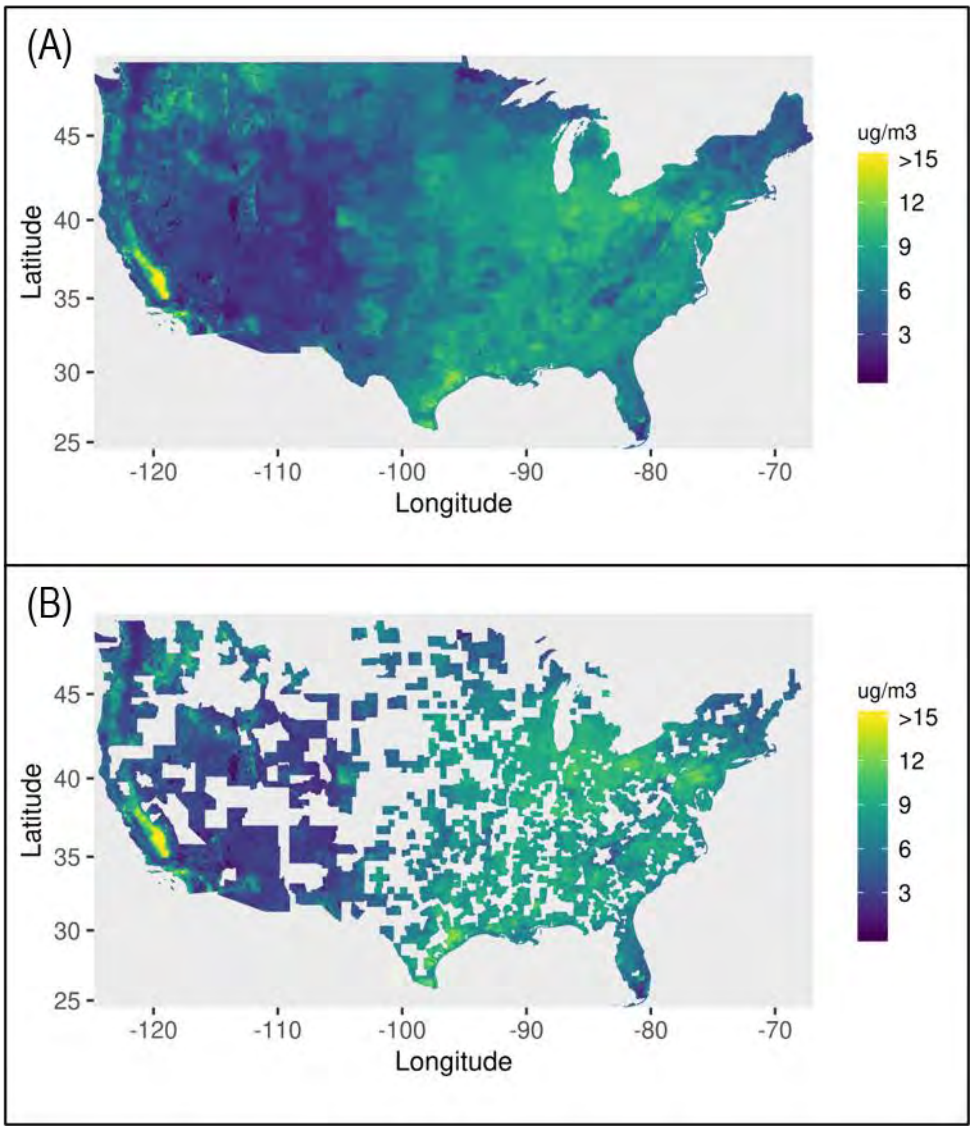

Figure 2-37. Spatial distribution of the annual average $PM_{2.5}$ concentrations for 2015 using the DI2019 surface nationwide (panel A) and for CBSAs only (panel B).

Using the DI2019 and HA2020 surfaces, for each year of available data, the 1 km x 1 km grid cells for each modeled surface within a CBSA were averaged, resulting in an estimated average annual $PM_{2.5}$ concentration at the CBSA spatial resolution. In addition, for each surface, all 1 km x 1 km grid cells were averaged over the conterminous U.S., resulting in an estimated average annual $PM_{2.5}$ concentration at the national scale. These average annual $PM_{2.5}$

concentrations for each year from 2000-2016 for the DI2019 and HA2020 surfaces are shown in Table 2-4. In addition, we also examined the average annual $PM_{2.5}$ concentrations nationwide and in CBSAs in terms of a 3-year average, which is the averaging time of the annual standard. These averages are shown in Table 2-5.

Table 2-4. Average Annual $PM_{2.5}$ Concentration ($\mu g/m^3$) by Year.

Year	DI2019		HA2020	
	Nationwide [a]	CBSAs [b]	Nationwide [a]	CBSAs [b]
2000	8.36	8.96	7.37	7.83
2001	7.88	8.49	7.08	7.61
2002	7.99	8.59	7.37	7.98
2003	8.25	8.72	7.03	7.51
2004	7.62	8.18	6.59	7.13
2005	7.98	8.51	7.34	7.92
2006	7.68	8.13	6.72	7.21
2007	7.90	8.41	7.26	7.69
2008	7.13	7.59	6.51	7.00
2009	6.52	6.94	6.02	6.45
2010	6.71	7.10	6.09	6.47
2011	6.72	7.13	6.31	6.74
2012	6.69	6.95	6.24	6.47
2013	6.15	6.50	5.75	6.14
2014	6.08	6.41	5.61	6.04
2015	6.00	6.25	5.43	5.76
2016	5.29	5.56	4.98	5.36

[a] Nationwide average annual $PM_{2.5}$ concentrations include all 1 km x 1 km grid cells of the modeling surface.
[b] CBSA average annual $PM_{2.5}$ concentrations include only those 1 km x 1 km grid cells that were located within a CBSA.

Table 2-5. Three-Year Average of the Average Annual $PM_{2.5}$ Concentrations ($\mu g/m^3$).

Year	DI2019		HA2020	
	Nationwide [a]	CBSAs [b]	Nationwide [a]	CBSAs [b]
2000-2002	8.08	8.68	7.27	7.81
2001-2003	8.04	8.60	7.16	7.70
2002-2004	7.95	8.50	7.00	7.54
2003-2005	7.95	8.47	6.99	7.52
2004-2006	7.96	8.28	6.88	7.42
2005-2007	7.85	8.35	7.11	7.61
2006-2008	7.57	8.04	6.83	7.30
2007-2009	7.18	7.65	6.60	7.04
2008-2010	6.78	7.21	6.21	6.64
2009-2011	6.65	7.05	6.14	6.55
2010-2012	6.71	7.06	6.21	6.56
2011-2013	6.52	6.86	6.10	6.45
2012-2014	6.31	6.62	5.87	6.22
2013-2015	6.08	6.38	5.60	5.98
2014-2016	5.79	6.07	5.34	5.72

[a] Nationwide average annual $PM_{2.5}$ concentrations include all 1 km x 1 km grid cells of the modeling surface.
[b] CBSA average annual $PM_{2.5}$ concentrations include only those 1 km x 1 km grid cells that were located within a CBSA.

At the national scale, the average annual $PM_{2.5}$ concentrations are slightly higher when using the DI2019 surface compared to the HA2020 surface but are generally similar. The average annual $PM_{2.5}$ concentrations are also slightly lower using the HA2020 surface compared to the DI2019 surface when the analyses are conducted for CBSAs. However, regardless of which surface is used, the average annual $PM_{2.5}$ concentrations for the CBSA-only analyses are somewhat higher than for the nationwide analyses (4-8% higher), likely reflecting the more urban or densely populated areas in the CBSA-only analyses that typically have higher $PM_{2.5}$ in ambient air compared to more rural or less densely populated areas captured in the nationwide analyses.

Similarly, as shown in Table 2-5, for both the DI2019 and HA2020 surfaces, the nationwide average annual $PM_{2.5}$ concentrations, averaged over three years, are lower than the CBSA only average annual $PM_{2.5}$ concentrations, averaged over three years. For the national scale, 3-year averages of the average annual $PM_{2.5}$ concentrations generally range from about 5.3 $\mu g/m^3$ to 8.1 $\mu g/m^3$, compared to the CBSA scale, which ranges from 5.7 $\mu g/m^3$ to 8.7 $\mu g/m^3$.

Overall, these analyses suggest that there are slight differences in the average annual $PM_{2.5}$ concentrations depending on the modeling method employed in a hybrid modeling study.

It is important to recognize that the use of different methods in the hybrid modeling studies to estimate mean $PM_{2.5}$ concentrations may influence the comparability across studies

We next evaluate how the averages of the model surfaces compare to regulatory design values and how population weighting influences the averages. For this analysis, we include CBSAs with three or more valid design values for the 3-year period.[39] The regulatory design values for the CBSAs were calculated for each 3-year period for the CBSAs with 3 or more design values in each of the 3-year periods. Using the maximum design value for each CBSA and by each 3-year period, the ratio of maximum design values to modeled average annual $PM_{2.5}$ concentrations were calculated, for each 3-year period. In addition, we evaluated the influence of population weighting on the average annual $PM_{2.5}$ concentrations using both the DI2019 and HA2020 surfaces for 3-year periods in CBSAs that also have available regulatory design value data. These data are shown in Table 2-6.

Table 2-6. Average Annual $PM_{2.5}$ Concentrations and Ratios to Regulatory Design Values.

Years of Monitoring Data	No. of CBSAs [a]	Average Annual $PM_{2.5}$ Concentration ($\mu g/m^3$) [b]	Population Weighted Average Annual $PM_{2.5}$ Concentration ($\mu g/m^3$) [b]	Average Maximum Annual DVs ($\mu g/m^3$) [b]	Ratio of Average Maximum Annual DVs to Average Annual $PM_{2.5}$ Concentrations	Ratio of Average Maximum Annual DVs to Population Weighted Average Annual $PM_{2.5}$ Concentrations
DI2019 Surface from Di et al. (2019)						
2008-2010	67	8.61	10.17	11.67	1.48	1.15
2011-2013	64	8.10	9.37	10.91	1.47	1.17
2014-2016	61	7.22	8.26	9.57	1.41	1.17
HA2020 Surface from Hammer et al. (2020) and van Donkelaar et al. (2019)						
2008-2010	67	8.25	9.93	11.67	1.50	1.18
2011-2013	64	7.92	9.34	10.91	1.43	1.17
2014-2016	61	6.98	8.19	9.57	1.43	1.18

[a] The number of CBSAs with 3 or more valid design values for the 3-year period
[b] Averaged across CBSAs

As shown in Table 2-6, the results using the DI2019 and HA2020 surfaces are similar for the average annual $PM_{2.5}$ concentrations, by each 3-year period. When population weighting is not applied, the average annual $PM_{2.5}$ concentrations generally range from 7.0 to 8.6 $\mu g/m^3$. When population weighting is applied, the average annual $PM_{2.5}$ concentrations are slightly higher, ranging from 8.2 to 10.2 $\mu g/m^3$. As with CBSAs versus the national comparison above,

[39] More details about the analytical methods used for this analysis are described in section A.7 of Appendix A.

population weighting results in a higher average $PM_{2.5}$ concentration than when population weighting is not applied.

For the CBSAs included in the population weighted analyses, the average maximum annual design values generally range from 9.5 to 11.7 $\mu g/m^3$. As shown in Table 2-6, these analyses show that the results are similar for both the DI2019 and HA2020 surfaces and the maximum annual $PM_{2.5}$ design values are often 40% to 50% higher than average annual $PM_{2.5}$ concentrations when population weighting is not applied. However, when population weighting is applied, the ratio of the maximum annual $PM_{2.5}$ design values to the average annual $PM_{2.5}$ concentrations are lower than when not population weighted, and generally range from 15% to 18%.

2.3.3.2.5 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 is warranted on identifying the most appropriate model performance metrics and comprehensively characterizing model performance to further inform our understanding of the implications of using these fields to estimate $PM_{2.5}$ exposures in health studies.

When additional analyses are done to further compare the DI2019 and HA2020 surfaces, the results suggest the DI2019 and HA2020 surfaces predict similar average annual $PM_{2.5}$

concentrations at the national scale and on average across all CBSAs in the U.S. The spatial scale can affect the magnitude of the average annual $PM_{2.5}$ concentration with somewhat higher concentrations (4-8% higher) resulting from averaging across all CBSAs in the U.S. versus averaging across the entire U.S. Additionally, when average annual $PM_{2.5}$ concentrations from the hybrid modeled surfaces are compared to the average maximum annual design value measured at ground-based monitors in a subset of CBSAs, the average of the maximum annual design values tends to be a 40-50% higher than the average annual $PM_{2.5}$ concentration estimated from the hybrid modeling surfaces. When population weighting is introduced, the average of the maximum annual design values tends to only be 15-18% higher than the average annual $PM_{2.5}$ concentration estimated from the hybrid modeling surfaces. This analysis may help better explain why reported study means from different epidemiologic studies can vary and why these mean values tend to be lower than concentrations reported at ground-based monitors. However, it is important to recognize that these results only reflect two surfaces and two types of approaches and that the use of different hybrid methods to estimate mean $PM_{2.5}$ concentrations may influence the comparability across 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 2012 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. While these exceptional episodes can lead to violations of the daily $PM_{2.5}$ standard (35 $\mu g/m^3$) in some cases (Schweizer et al., 2017), such events are routinely screened for and usually identifiable in the monitoring data. As described further below, contributions to background PM in the U.S. result mainly from sources within North America. Contributions from intercontinental events have also been documented (e.g., transport from dust storms occurring in deserts in North Africa and Asia), but these events are less common and represent a relatively small fraction of background PM in most places.

While the potential sources of background PM discussed above include sources of both fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) 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, 2019b, section 2.3.3).

Biogenic emissions from plants are perhaps the most ubiquitous sources of background PM in the U.S. Certain species of plants and trees can release large amounts of VOCs such as isoprene and monoterpenes that are oxidized in the atmosphere to form organic aerosol. SOA production from biogenic emissions is largest in the southeastern U.S., where conditions are

warm, humid, and sunny for much of the year. Many of the processes involved with biogenic SOA formation are complex and remain highly uncertain. Results from radiocarbon techniques applied to distinguish modern (biogenic or fires) from fossil (anthropogenic) carbon fractions in organic aerosol have suggested comparable contributions from both carbon types in the Southeast where SOA concentrations are high (Schichtel et al., 2008). However, SOA formation from biogenic emission sources can also be facilitated by the presence of anthropogenic precursors (Xu et al., 2015). More work characterizing the interactions of anthropogenic and biogenic emissions is needed to determine the implications of such processes for background PM concentrations.

Soil dust and sea salt have been estimated to account for less than 10% of urban $PM_{2.5}$ 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, 2019b, 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, 2019b, 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 2-38 and Figure 2-39 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[40] image from August 4, 2017 (Figure 2-38) shows smoke from multiple fire detections across southern British Columbia crossing into northern Washington state. Smoke from these fires was also captured at

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

the North Cascades IMPROVE monitor (Figure 2-39), 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.

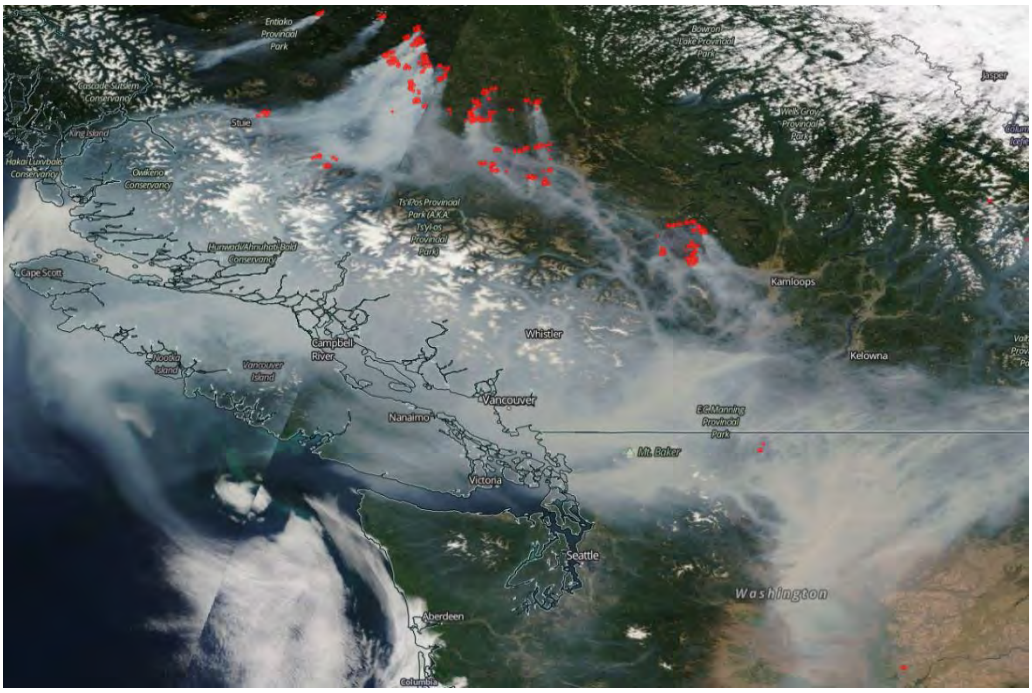

Figure 2-38. Smoke and fire detections observed by the MODIS instrument onboard the Aqua satellite on August 4th, 2017 accessed through NASA Worldview.

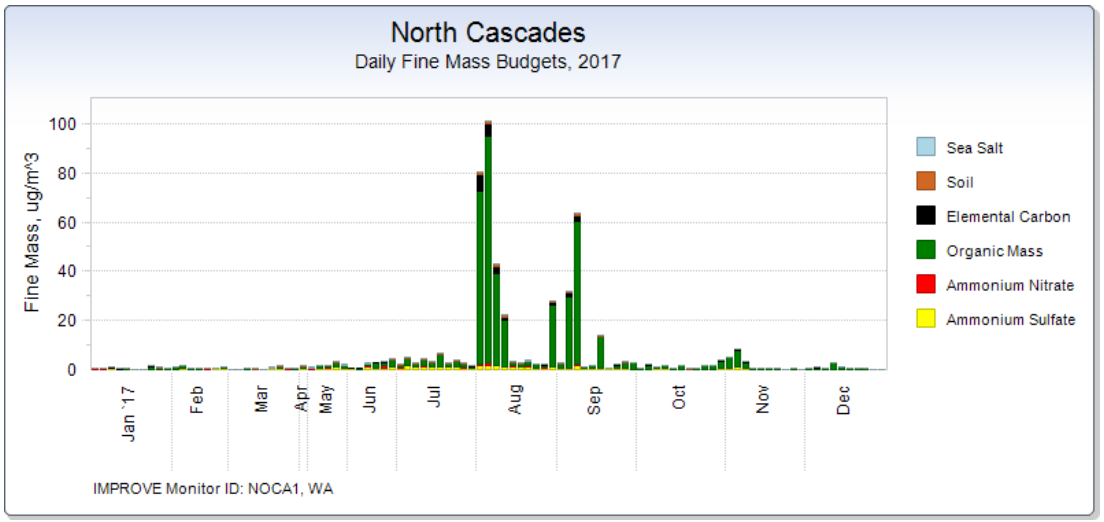

Figure 2-39. Fine PM mass time series during 2017 from the North Cascades IMPROVE site in north central Washington state.[41]

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

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; 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 $\mu g/m^3$ 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 $\mu g/m^3$ 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 $\mu g/m^3$ (Prospero, 1999a; Prospero, 1999b).[42] In June 2020 a large dust transport episode originating in North Africa may have contributed up to 50 $\mu g/m^3$ for several days at multiple sites in the southeastern U.S. (Pu and Jin, 2021).

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

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 at the monitors included in the 2009 ISA 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 2-40 shows observed annual average $PM_{2.5}$ in 2004 and 2016 at the same remote monitors examined in the 2009 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 $\mu g/m^3$ 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 $\mu g/m^3$ 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 2-40 represents 50% or more of total PM in both 2004 and 2016. In addition to the IMPROVE sites from the 2019 ISA, Figure 2-40 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 2-37 will also include the influence of fires for some monitors. The highest organic matter contribution for any of the sites shown in Figure 2-40, 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 reconsideration, 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 2012 PM NAAQS review.

While estimates of total annual background concentrations have generally not changed significantly since the 2012 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.

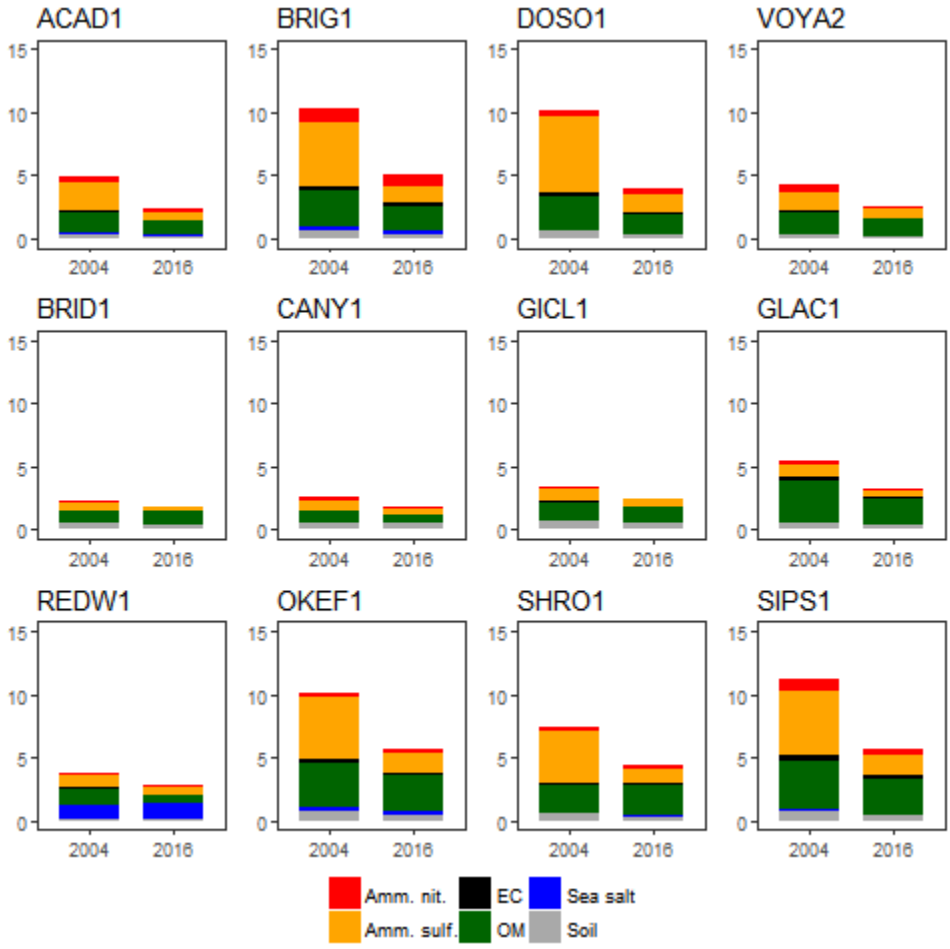

Figure 2-40. Speciated annual average IMPROVE $PM_{2.5}$ in $\mu g/m^3$ at select remote monitors during 2004 and 2016. (Note: Monitor locations are shown in Figure 2-41.)

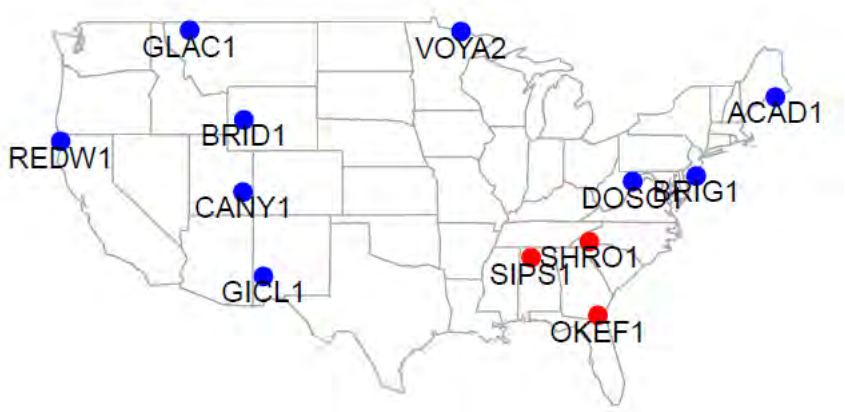

Figure 2-41. Site locations for the IMPROVE monitors in Figure 2-40. (Note: Monitors also assessed in the 2009 ISA are shown in blue. Monitors only examined in this assessment are shown in red.)

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

This chapter presents and evaluates the policy implications of the key aspects of the scientific and technical information pertaining to this reconsideration of the primary $PM_{2.5}$ standards. In so doing, this chapter presents key aspects of the evidence of health effects of $PM_{2.5}$, as documented in the 2019 ISA (U.S. EPA, 2019) and draft ISA Supplement (U.S. EPA, 2021a),[1] with support from the prior ISAs and AQCDs, and associated public health implications. It also presents key aspects of updated quantitative risk analyses conducted for this reconsideration, as detailed in the appendices associated with this chapter. Together this information provides the basis for our evaluation of the scientific information regarding health effects of $PM_{2.5}$ in ambient air and the potential for effects to occur under air quality conditions associated with the existing standard (or any alternatives considered), as well as the associated implications for public health. Our evaluation is focused around key policy-relevant questions derived from the IRP (U.S. EPA, 2016, section 2.1) for the review completed in 2020, and also takes into account conclusions reached in previous reviews. In this way we identify key policy-relevant considerations and summary conclusions regarding the public health protection provided by the current standards for the Administrator's consideration in this reconsideration of the 2020 final decision on the primary $PM_{2.5}$ standards.

Within this chapter, background information on the current standards is summarized in section 3.1. The general approach for considering the available information in this reconsideration, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 3.2. Key aspects of the available health effects evidence and associated public health implications and uncertainties are addressed in section 3.3, and the current air quality and risk information, with associated uncertainties, is addressed in section 3.4. Section 3.5 summarizes the key evidence- and risk-based considerations identified in our evaluation and also presents associated preliminary conclusions on the adequacy of the current standards. Key remaining uncertainties and areas for future research are identified in section 3.6.

[1] As described in detail in section 1.4.2 above and section 3.3 below, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA (U.S. EPA, 2021a). The selection of the health effects to evaluate within the draft ISA Supplement was based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for $PM_{2.5}$-related health effects, the focus within the draft ISA Supplement is on mortality and cardiovascular effects. The draft ISA Supplement does not include an evaluation of studies for other $PM_{2.5}$-related health effects (U.S. EPA, 2021a).

3.1 BACKGROUND ON THE CURRENT STANDARDS

The current primary $PM_{2.5}$ standards were retained in 2020 based on the Administrator's judgments regarding the available scientific evidence, the available risk information regarding the risk that may be allowed by such standards, and the appropriate degree of public health protection provided by the existing standards (85 FR 82718, December 18, 2020). With the 2020 final decision, the EPA retained the primary 24-hour $PM_{2.5}$, with its level of 35 $\mu g/m^3$, and the primary annual $PM_{2.5}$ standard, with its level of 12.0 $\mu g/m^3$. This decision drew upon the scientific evidence assessed in the 2019 ISA, the evidence and quantitative risk information in the 2020 PA, the advice and recommendations of the CASAC, and public comments on the proposed decision (85 FR 24094, April 30, 2020).

The health effects evidence base available in the 2020 review included extensive evidence from previous reviews as well as the evidence that had emerged since the prior review had been completed in 2012. This evidence base, spanning several decades, documents the relationship between short- and long-term $PM_{2.5}$ exposure and mortality or serious morbidity effects. The evidence available in the 2019 ISA reaffirmed, and in some cases strengthened, the conclusions from the 2009 ISA regarding the health effects of $PM_{2.5}$ exposures (U.S. EPA, 2009). Much of the evidence came from epidemiologic studies conducted in North America, Europe, or Asia that demonstrated generally positive, and often statistically significant, $PM_{2.5}$ health effect associations. Such studies reported associations between estimated $PM_{2.5}$ exposures and non-accidental, cardiovascular, or respiratory mortality; cardiovascular or respiratory hospitalizations or emergency department visits; and other mortality/morbidity outcomes (e.g., lung cancer mortality or incidence, asthma development). Experimental evidence, as well as evidence from panel studies, strengthened support for potential biological pathways through which $PM_{2.5}$ exposures could lead to health effects reported in many population-epidemiologic studies, including support for pathways that could lead to cardiovascular, respiratory, nervous system, and cancer-related effects (U.S. EPA, 2019). Based on this evidence, the 2019 ISA concludes there to be a causal relationship between long- and short-term $PM_{2.5}$ exposure and mortality and cardiovascular effects, as well as likely to be causal relationships between long- and short-term $PM_{2.5}$ exposures and respiratory effects, as well as long-term $PM_{2.5}$ exposures and cancer and nervous system effects (U.S. EPA, 2019, section 1.7).

Epidemiologic studies reported $PM_{2.5}$ health effect associations with mortality and/or morbidity across multiple U.S. cities and in diverse populations, including in studies examining populations and lifestages that may be at comparatively higher risk of experiencing a $PM_{2.5}$-related health effect (e.g., older adults, children). The 2019 ISA cited 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, 2019, p. 12-1). In support of the causal and likely to be causal determinations, the 2019 ISA cites substantial evidence for:

- PM-related mortality and cardiovascular effects in older adults (U.S. EPA, 2019, sections 11.1, 11.2, 6.1, and 6.2);
- PM-related cardiovascular effects in people with pre-existing cardiovascular disease (U.S. EPA, 2019, section 6.1);
- PM-related respiratory effects in people with pre-existing respiratory disease, particularly asthma (U.S. EPA, 2019, section 5.1);
- PM-related impairments in lung function growth and asthma development in children (U.S. EPA, 2019, sections 5.1, 5.2, and 12.5.1.1).

The 2019 ISA also noted that stratified analyses (i.e., analyses that allow for comparison of PM-related health effects in subgroups to health effects for full populations) provided strong evidence for racial and ethnic differences in $PM_{2.5}$ exposures and $PM_{2.5}$-related health risk. Such analyses indicated that certain racial and ethnic groups such as Hispanic and non-Hispanic Black populations have higher $PM_{2.5}$ exposures than non-Hispanic White populations, thus contributing to risk of adverse health effects in non-white populations (U.S. EPA, 2019, section 12.5.4). Stratified analyses focused on other groups also suggested 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, 2019, chapter 12).

The risk information available in the 2020 review included risk estimates for air quality conditions just meeting the existing primary $PM_{2.5}$ standards, and also for air quality conditions just meeting potential alternative standards. The general approach to estimating $PM_{2.5}$-associated health risks combined concentration-response functions from epidemiologic studies with model-based $PM_{2.5}$ air quality surfaces, baseline health incidence data, and population demographics for 47 urban areas (U.S. EPA, 2020, section 3.3, Figure 3-10, Appendix C). The risk assessment estimated that the existing primary $PM_{2.5}$ standards could allow a substantial number of $PM_{2.5}$-associated deaths in the U.S. Uncertainty in risk estimates (e.g., in the size of risk estimates) can result from a number of factors, including assumptions about the shape of the concentration-response relationship with mortality at low ambient PM concentrations, the potential for confounding and/or exposure measurement error, and the methods used to adjust $PM_{2.5}$ air quality. In light of the limitations and uncertainties, these risk estimates were given little weight by the Administrator in his decision on the standards (85 FR 82717, December 18. 2020).

Consistent with the general approach routinely employed in NAAQS reviews, the initial consideration in the 2020 review of the primary $PM_{2.5}$ standards was with regard to the adequacy

of protection provided by the then-existing standards. Key aspects of that consideration are summarized in section 3.1.1 below.

3.1.1 Considerations Regarding the Adequacy of the Existing Standards in the 2020 Review

With the 2020 final decision, the EPA retained the primary 24-hour $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$, and the primary annual $PM_{2.5}$ standard, with its level of 12.0 $\mu g/m^3$. The Administrator's conclusions regarding the adequacy of the primary $PM_{2.5}$ standards at the time of the 2020 review was based on consideration of the evidence, analyses and conclusions contained in the 2019 ISA; the quantitative risk assessment in the 2020 PA; advice from the CASAC; and public comments. Key considerations informing the Administrator's decisions that the 2012 standards should be retained are summarized below.

As an initial matter, the Administrator considered the range of scientific evidence evaluating these effects, including studies of at-risk populations, to inform his review of the primary $PM_{2.5}$ standards, placing the greatest weight on evidence of effects for which the 2019 ISA determined there to be a causal or likely to be causal relationship with long- and short-term $PM_{2.5}$ exposures (85 FR 82714-82715, December 18, 2020).

With regard to indicator, the Administrator recognized that, consistent with the evidence available in prior reviews, the scientific evidence in the 2020 review continued to provide strong support for health effects following short- and long-term $PM_{2.5}$ exposures. He noted the 2020 PA conclusions that the information continued to support the $PM_{2.5}$ mass-based indicator and remained 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. Thus, the Administrator concluded that it was appropriate to retain $PM_{2.5}$ as the indicator for the primary standards for fine particulates (85 FR 82715, December 18, 2020).

With respect to averaging time and form, the Administrator noted that the scientific evidence continued to provide strong support for health effects associations with both long-term (e.g., annual or multi-year) and short-term (e.g., mostly 24-hour) exposures to $PM_{2.5}$, consistent with the conclusions in the 2020 PA. In the 2019 ISA, epidemiologic and controlled human exposure studies examined a variety of $PM_{2.5}$ exposure durations. Epidemiologic studies continued to provide strong support for health effects associated with short-term $PM_{2.5}$ exposures based on 24-hour $PM_{2.5}$ averaging periods, and the EPA noted that associations with sub-daily estimates are less consistent and, in some cases, smaller in magnitude (U.S. EPA, 2019, section 1.5.2.1; U.S. EPA, 2020, section 3.5.2.2). In addition, controlled human exposure and panel-based studies of sub-daily exposures typically examined 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 2019 ISA concludes that epidemiologic studies did not indicate that sub-daily averaging periods were more closely associated with health effects than the 24-hour average exposure metric (U.S. EPA, 2019, section 1.5.2.1). Additionally, while controlled human exposure studies provided 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 the studies were well-above the ambient concentrations typically measured in locations meeting the existing standards (U.S. EPA, 2020, section 3.2.3.1). Thus, these studies also did not suggest the need for additional protection against sub-daily $PM_{2.5}$ exposures (U.S. EPA, 2020, section 3.5.2.2). Therefore, the Administrator judged that the 24-hour averaging time remained appropriate (85 FR 82715, December 18, 2020).

With regard to the form of the 24-hour standard (98^{th} percentile, averaged over three years), the Administrator noted that epidemiologic studies continued to provide strong support for health effect associations with short-term (e.g., mostly 24-hour) $PM_{2.5}$ exposures (U.S. EPA, 2020, section 3.5.2.3) and that controlled human exposure studies provided evidence for health effects following single short-term "peak" $PM_{2.5}$ exposures. Thus, the evidence supported retaining a standard focused on providing supplemental protection against short-term peak exposures and supported a 98^{th} percentile form for a 24-hour standard. The Administrator further noted that this form also provided an appropriate balance between limiting the occurrence of peak 24-hour $PM_{2.5}$ concentrations and identifying a stable target for risk management programs (U.S. EPA, 2020, section 3.5.2.3). As such, the Administrator concluded to retain the form and averaging time of the current 24-hour standard (98^{th} percentile, averaged over three years) and annual standard (annual average, averaged over three years) (85 FR 82715, December 18, 2020).

With regard to the level of the standards, in reaching his final decision, the Administrator considered the large body of evidence presented and assessed in the 2019 ISA (U.S. EPA, 2019), the policy-relevant and risk-based conclusions and rationales as presented in the 2020 PA (U.S. EPA, 2020), advice from the CASAC, and public comments. In particular, in considering the 2019 ISA and 2020 PA, he considered key epidemiologic studies that evaluated associations between $PM_{2.5}$ air quality distributions and mortality and morbidity, including key accountability studies; the availability of experimental studies to support biological plausibility; controlled human exposure studies examining effects following short-term $PM_{2.5}$ exposures; air quality analyses; and the important uncertainties and limitations associated with the information (85 FR 82715, December 18, 2020).

As an initial matter, the Administrator considered the protection afforded by both the annual and 24-hour standards together against long- and short-term $PM_{2.5}$ exposures and health effects. The Administrator recognized that the annual standard was most effective in controlling "typical" $PM_{2.5}$ concentrations near the middle of the air quality distribution (i.e., around the

mean of the distribution), but also provided some control over short-term peak $PM_{2.5}$ concentrations. On the other hand, the 24-hour standard, with its 98th percentile form, was most effective at limiting peak 24-hour $PM_{2.5}$ concentrations, but in doing so also had an effect on annual average $PM_{2.5}$ concentrations. Thus, 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 were not expected to be equally effective at limiting both types of exposures. Thus, consistent with previous reviews, the Administrator's consideration of the public health protection provided by the existing primary $PM_{2.5}$ standards was based on his consideration of the combination of the annual and 24-hour standards. Specifically, he recognized that the annual standard was more likely to appropriately limit the "typical" daily and annual exposures that are most strongly associated with the health effects observed in epidemiologic studies. The Administrator concluded that an annual standard (as the arithmetic mean, averaged over three years) remained appropriate for targeting protection against the annual and daily $PM_{2.5}$ exposures around the middle portion of the $PM_{2.5}$ air quality distribution. Further, recognizing that the 24-hour standard (with its 98th percentile form) was more directly tied to short-term peak $PM_{2.5}$ concentrations, and more likely to appropriately limit exposures to such concentrations, the Administrator concluded that the current 24-hour standard (with its 98th percentile form, averaged over three years) remained appropriate 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. However, the Administrator recognized that changes in $PM_{2.5}$ air quality 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, but also in fewer and lower short-term peak $PM_{2.5}$ concentrations. The Administrator further recognized that changes in air quality 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 (85 FR 82715-82716, December 18, 2020).

Thus, in considering the adequacy of the 24-hour standard, the Administrator noted the importance of considering whether additional protection was needed against short-term exposures to peak $PM_{2.5}$ concentrations. In examining the scientific evidence, he noted the limited utility of the animal toxicologic studies in directly informing conclusions on the appropriate level of the standard given the uncertainty in extrapolating from effects in animals to those in human populations. The Administrator noted that controlled human exposure studies provided evidence for health effects following single, short-term $PM_{2.5}$ exposures that corresponded best to exposures that might be experienced in the upper end of the $PM_{2.5}$ air quality distribution in the U.S. (i.e., "peak" concentrations). However, most of these studies examined exposure concentrations considerably higher than are typically measured in areas

meeting the standards (U.S. EPA, 2020, section 3.2.3.1). In particular, controlled human exposure studies often reported 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). To provide insight into what these studies may indicate regarding the primary $PM_{2.5}$ standards, the 2020 PA (U.S. EPA, 2020, p. 3-49) noted that 2-hour ambient concentrations of $PM_{2.5}$ at monitoring sites meeting the current standards almost never exceeded 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 primary $PM_{2.5}$ standards remained well-below the $PM_{2.5}$ exposure concentrations consistently shown in controlled human exposure studies to elicit effects (i.e., 99.9^{th} percentile of 2-hour concentrations at these sites is 68 $\mu g/m^3$ during the warm season). Thus, the available experimental evidence did not indicate the need for additional protection against exposures to peak $PM_{2.5}$ concentrations, beyond the protection provided by the combination of the 24-hour and the annual standards (U.S. EPA, 2020, section 3.2.3.1; 85 FR 82716, December 18, 2020).

With respect to the epidemiologic evidence, the Administrator noted that the studies did not indicate that associations in those studies were strongly influenced by exposures to peak concentrations in the air quality distribution and thus did not indicate the need for additional protection against short-term exposures to peak $PM_{2.5}$ concentrations (U.S. EPA, 2020, section 3.5.1). The Administrator noted that this was consistent with CASAC consensus support for retaining the current 24-hour standard. Thus, the Administrator concluded that the 24-hour standard with its level of 35 $\mu g/m^3$ was adequate to provide supplemental protection (i.e., beyond that provided by the annual standard alone) against short-term exposures to peak $PM_{2.5}$ concentrations (85 FR 82716, December 18, 2020).

With regard to the level of the annual standard, the Administrator recognized that the annual standard, with its form based on the arithmetic mean concentration, was most appropriately meant to limit the "typical" daily and annual exposures that were most strongly associated with the health effects observed in epidemiologic studies. However, the Administrator also noted that while epidemiologic studies examined associations between distributions of $PM_{2.5}$ air quality and health outcomes, they did not identify particular $PM_{2.5}$ exposures that cause effects and thus, they could not alone identify a specific level at which the standard should be set, as such a determination necessarily required the Administrator's judgment. Thus, consistent with the approaches in previous NAAQS reviews, the Administrator recognized that any approach that used epidemiologic information in reaching decisions on what standards are appropriate necessarily required judgments about how to translate the information from the epidemiologic studies into a basis for appropriate standards. This approach included

consideration of the uncertainties in the reported associations between daily or annual average $PM_{2.5}$ exposures and mortality or morbidity in the epidemiologic studies. Such an approach is consistent with setting standards that are neither more nor less stringent than necessary, recognizing that a zero-risk standard is not required by the CAA (85 FR 82716, December 18, 2020).

The Administrator emphasized uncertainties and limitations that were present in epidemiologic studies in previous reviews and persisted in the 2020 review. These uncertainties included exposure measurement error, potential confounding by copollutants, increasing uncertainty of associations at lower $PM_{2.5}$ concentrations, and heterogeneity of effects across different cities or regions (85 FR 82716, December 18, 2020). The Administrator also noted the advice given by the CASAC on this matter. The CASAC members who supported retaining the annual standard expressed their concerns with the epidemiologic studies, asserting that these studies did not provide a sufficient basis for revising the existing standards. They also identified several key concerns regarding the associations reported in epidemiologic studies and concluded that "while the data on associations should certainly be carefully considered, this data should not be interpreted more strongly than warranted based on its methodological limitations" (Cox, 2019, p. 8 consensus responses).

Taking into consideration the views expressed by the CASAC members who supported retaining the annual standard, the Administrator recognized that epidemiologic studies examined associations between distributions of $PM_{2.5}$ air quality and health outcomes, and they did not identify particular $PM_{2.5}$ exposures that cause effects (U.S. EPA, 2020, section 3.1.2). While the Administrator remained concerned about placing too much weight on epidemiologic studies to inform conclusions on the adequacy of the primary standards, he noted the approach to considering such studies in the 2012 review. In the 2012 review, it was noted that the evidence of an association in any epidemiologic study was "strongest at and around the long-term average where the data in the study are most concentrated" (78 FR 3140, January 15, 2013). In considering the characterization of epidemiologic studies, the Administrator viewed that when assessing the mean concentrations of the key short-term and long-term epidemiologic studies in the U.S. that use ground-based monitoring (i.e., those studies where the mean is most directly comparable to the current annual standard), the majority of studies had mean concentrations at or above the level of the existing annual standard, with the mean of the study-reported means or medians equal to 13.5 $\mu g/m^3$, a concentration level above the existing level of the primary annual standard of 12 $\mu g/m^3$. The Administrator further noted his caution in directly comparing the reported study mean values to the standard level given that study-reported mean concentrations, by design, are generally lower than the design value of the highest monitor in an area, which determines compliance. In the 2020 PA, analyses of recent air quality in U.S. CBSAs indicated

that maximum annual $PM_{2.5}$ design values for a given three-year period were often 10% to 20% higher than average monitored concentrations (i.e., averaged across multiple monitors in the same CBSA) (U.S. EPA, 2020, Appendix B, section B.7). He further noted his concern in placing too much weight on any one epidemiologic study but instead judged that it was more appropriate to focus on the body of studies together and therefore noted the calculation of the mean of study-reported means (or medians). Thus, while the Administrator was cautious in placing too much weight on the epidemiologic evidence alone, he noted that: (1) the reported mean concentration in the majority of the key U.S. epidemiologic studies using ground-based monitoring data were above the level of the existing annual standard; (2) the mean of the reported study means (or medians) (i.e., 13.5 $\mu g/m^3$) was above the level of the current standard;[2] (3) air quality analyses showed the study means to be lower than their corresponding design values by 10-20%; and (4) these analyses must be considered in light of uncertainties inherent in the epidemiologic evidence. When taken together, the Administrator judged that, even if it were appropriate to place more weight on the epidemiologic evidence, this information did not call into question the adequacy of the current standards (85 FR 82716-82717, December 18, 2020).

In addition to the evidence, the Administrator also considered the potential implications of the risk assessment. He noted that all risk assessments have limitations and that he remained concerned about the uncertainties in the underlying epidemiologic data used in the risk assessment. The Administrator also noted that in previous reviews, these uncertainties and limitations have often resulted in less weight being placed on quantitative estimates of risk than on the underlying scientific evidence itself (e.g., 78 FR 3086, 3098-99, January 15, 2013). These uncertainties and limitations included uncertainty in the shapes of concentration-response functions, particularly at low concentrations; uncertainties in the methods used to adjust air quality; and uncertainty in estimating risks for populations, locations and air quality distributions different from those examined in the underlying epidemiologic study (U.S. EPA, 2020, section 3.3.2.4). Additionally, the Administrator noted similar concern expressed by some members of the CASAC who support retaining the existing standards; they highlighted similar uncertainties and limitations in the risk assessment (Cox, 2019). In light of all of this, the Administrator judged it appropriate to place little weight on quantitative estimates of $PM_{2.5}$-associated mortality risk in reaching conclusions about the level of the primary $PM_{2.5}$ standards (85 FR 82717, December 18, 2020).

The Administrator additionally considered an emerging body of evidence from accountability studies that examined past reductions in ambient $PM_{2.5}$ and the degree to which

[2] The median of the study-reported mean (or median) $PM_{2.5}$ concentrations is 13.3 $\mu g/m^3$, which was also above the level of the existing standard.

those reductions resulted in public health improvements. While the Administrator agreed with public commenters that well-designed and conducted accountability studies can be informative, he viewed that interpreting such studies in the context of the primary $PM_{2.5}$ standards was complicated by the fact that some of the available studies had not evaluated $PM_{2.5}$ specifically (e.g., as opposed to PM_{10} or total suspended particulates), did not show changes in $PM_{2.5}$ air quality, or had not been able to disentangle health impacts of the interventions from background trends in health (U.S. EPA, 2020, section 3.5.1). He further recognized that the small number of available studies that did report public health improvements following past declines in ambient $PM_{2.5}$ had not examined air quality meeting the existing standards (U.S. EPA, 2020, Table 3-3). This included U.S. studies that reported increased life expectancy, decreased mortality, and decreased respiratory effects following past declines in ambient $PM_{2.5}$ concentrations. Such studies examined "starting" annual average $PM_{2.5}$ concentrations (i.e., prior to the reductions being evaluated) ranging from about 13.2 to > 20 $\mu g/m^3$ (i.e., U.S. EPA, 2020, Table 3-3). Given the lack of available accountability studies reporting public health improvements attributable to reductions in ambient $PM_{2.5}$ in locations meeting the existing standards, together with his broader concerns regarding the lack of experimental studies examining $PM_{2.5}$ exposures typical of areas meeting the existing standards, the Administrator judged that there was considerable uncertainty in the potential for increased public health protection from further reductions in ambient $PM_{2.5}$ concentrations beyond those achieved under the existing primary $PM_{2.5}$ standards (85 FR 82717, December 18, 2020).

When the above considerations were taken together, the Administrator concluded that the scientific evidence assessed in the 2019 ISA, together with the analyses in the 2020 PA based on that evidence and consideration of CASAC advice and public comments, did not call into question the adequacy of the public health protection provided by the existing annual and 24-hour $PM_{2.5}$ standards. In particular, the Administrator judged that there was considerable uncertainty in the potential for additional public health improvements from reducing ambient $PM_{2.5}$ concentrations below the concentrations achieved under the existing primary standards and that, therefore, standards more stringent than the existing standards (e.g., with lower levels) were not supported. That is, he judged that such standards would be more than requisite to protect the public health with an adequate margin of safety. This judgment reflected the Administrator's consideration of the uncertainties in the potential implications of the lower end of the air quality distributions from the epidemiologic studies due in part to the lack of supporting evidence from experimental studies and retrospective accountability studies conducted at $PM_{2.5}$ concentrations meeting the existing standards (85 FR 82717, December 18, 2020).

In reaching this conclusion, the Administrator judged that the existing standards provided an adequate margin of safety. With respect to the annual standard, the level of 12 $\mu g/m^3$ was

below the lowest "starting" concentration (i.e., 13.2 $\mu g/m^3$) in the available accountability studies that showed public health improvements attributable to reductions in ambient $PM_{2.5}$. In addition, while the Administrator placed less weight on the epidemiologic evidence for selecting a standard, he noted that the level of the annual standard was below the reported mean (and median) concentrations in the majority of the key U.S. epidemiologic studies using ground-based monitoring data (noting that these means tend to be 10-20% lower than their corresponding area design values which is the more relevant metric when considering the level of the standard) and below the mean of the reported means (or medians) of these studies (i.e., 13.5 $\mu g/m^3$). In addition, the Administrator recognized that concentrations in areas meeting the existing 24-hour and annual standards remained well-below the $PM_{2.5}$ exposure concentrations consistently shown to elicit effects in human exposure studies (85 FR 82717-82718, December 18, 2020).

In addition, based on the Administrator's review of the science, including controlled human exposure studies examining effects following short-term $PM_{2.5}$ exposures, the epidemiologic studies, and accountability studies conducted at levels just above the existing annual standard, he judged that the degree of public health protection provided by the existing annual standard is not greater than warranted. This judgment, together with the fact that no CASAC member expressed support for a less stringent standard, led the Administrator to conclude that standards less stringent than the existing standards (e.g., with higher levels) were also not supported (85 FR 82718, December 18, 2020).

In reaching his final decision, the Administrator concluded that the scientific evidence and technical information continued to support the existing annual and 24-hour $PM_{2.5}$ standards. This conclusion reflected the Administrator's view that there were important limitations and uncertainties that remained in the evidence. The Administrator concluded that these limitations contributed to considerable uncertainty regarding the potential public health implications of revising the existing primary $PM_{2.5}$ standards. Given this uncertainty, and noting the advice from some CASAC members, he concluded that the primary $PM_{2.5}$ standards, including the indicators ($PM_{2.5}$), averaging times (annual and 24-hour), forms (arithmetic mean and 98^{th} percentile, averaged over three years) and levels (12.0 $\mu g/m^3$, 35 $\mu g/m^3$), when taken together, remained requisite to protect the public health. Therefore, in the 2020 review, the Administrator reached the conclusion that the primary 24-hour and annual $PM_{2.5}$ standards, together, were requisite to protect public health from fine particles with an adequate margin of safety, including the health of at-risk populations, and retained the standards, without revision (85 FR 82718, December 18, 2020).

3.2 GENERAL APPROACH AND KEY ISSUES IN THIS RECONSIDERATION OF THE 2020 FINAL DECISION

As is the case for all such reviews, this reconsideration of the 2020 final decision on the primary $PM_{2.5}$ standards is most fundamentally based on the Agency's assessment of the scientific evidence and associated quantitative analyses to inform the Administrator's judgments regarding primary standards that are requisite to protect public health with an adequate margin of safety. This draft PA is intended to help bridge the gap between the scientific evidence and information assessed in the 2019 ISA and draft ISA Supplement and the judgments required of the Administrator in determining whether it is appropriate to retain or revise the primary $PM_{2.5}$ NAAQS. The approach for this reconsideration builds on the substantial assessments and evaluations performed over the course of the prior reviews (U.S. EPA, 2011; U.S. EPA, 2020), taking into account the more recent scientific information and air quality data now available to inform our understanding of the key policy issues relevant in this reconsideration.

The evaluations in this draft PA of the scientific assessments in the 2019 ISA and the draft ISA Supplement,[3] augmented by the quantitative risk analyses, are intended to inform the Administrator's public health policy judgments and conclusions, including his decisions as to whether to retain or revise the primary $PM_{2.5}$ standards. The draft PA evaluations consider the potential implications of various aspects of the scientific evidence, the risk-based information, and the associated uncertainties and limitations. In so doing, the approach for this draft PA involves evaluating the scientific and technical information to address a series of key policy-relevant questions using both evidence- and risk-based considerations. Together, consideration of the full set of evidence and information available in this reconsideration will inform the answer to the following initial overarching question for the reconsideration:

- **Does the scientific evidence, air quality and quantitative risk information support or call into question the adequacy of the public health protection afforded by the current primary annual and 24-hour $PM_{2.5}$ standards?**

In reflecting on this question, we will consider the body of scientific evidence, assessed in the 2019 ISA and draft ISA Supplement and used as a basis for developing or interpreting risk analyses, including whether it supports or calls into question the scientific conclusions reached in

[3] As described in detail in section 1.4.2, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA (U.S. EPA, 2021a). The selection of the health effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for $PM_{2.5}$-related health effects, the focus within the draft ISA Supplement is on mortality and cardiovascular effects. The draft ISA Supplement does not include an evaluation of studies for other $PM_{2.5}$-related health effects (U.S. EPA, 2021a).

the 2020 review regarding health effects related to exposure to $PM_{2.5}$ in ambient air. Information available in this reconsideration that may be informative to public health judgments regarding significance or adversity of key effects will also be considered. Additionally, the available risk information, whether newly developed for this reconsideration or predominantly developed in the past and interpreted in light of recent information, will be considered, including with regard to the extent to which it may continue to support judgments made in the 2020 review. Further, in considering this question with regard to the primary $PM_{2.5}$ standards, as in all NAAQS reviews, we give particular attention to exposures and health risks to at-risk populations (including at-risk lifestages).[4]

If the information available in this reconsideration suggests that revision of the current primary standards would be appropriate to consider, the draft PA will also evaluate how the standards might be revised based on the scientific information, air quality assessments, and risk information, and also considering what the information indicates as to the health protection expected to be afforded by the current or potential alternative standards. Such an evaluation may consider the effect of revising one or more elements of the standard (indicator, averaging time, level, and form), with the impact evaluated being on the resulting potential standard and all of its elements collectively. Based on such evaluations, the draft PA would then identify potential alternative standards (specified in terms of indicator, averaging time, level, and form) intended to reflect a range of alternative policy judgments as to the degree of protection that is requisite to protect public health with an adequate margin of safety, and options for standards to achieve it. The initial overarching policy-relevant question that frames such an evaluation of what revision of the standard might be appropriate to consider is:

- **What range of potential alternative standards could be supported by the available scientific evidence, air quality and risk information?**

The approach to reaching preliminary conclusions on the current primary $PM_{2.5}$ standards and, as appropriate, on potential alternative standards is summarized in general terms in Figure 3-1.

[4] As used here and similarly throughout this document, the term *population* refers to persons having a quality or characteristic in common, such as a specific pre-existing illness or a specific age or life stage. Identifying at-risk populations involves consideration of *susceptibility* and *vulnerability*. *Susceptibility* refers to innate (e.g., genetic or developmental aspects) or acquired (e.g., disease or smoking status) sensitivity that increases the risk of health effects occurring with exposure to $PM_{2.5}$. *Vulnerability* refers to an increased risk of $PM_{2.5}$-related health effects due to factors such as those related to socioeconomic status, reduced access to health care or exposure.

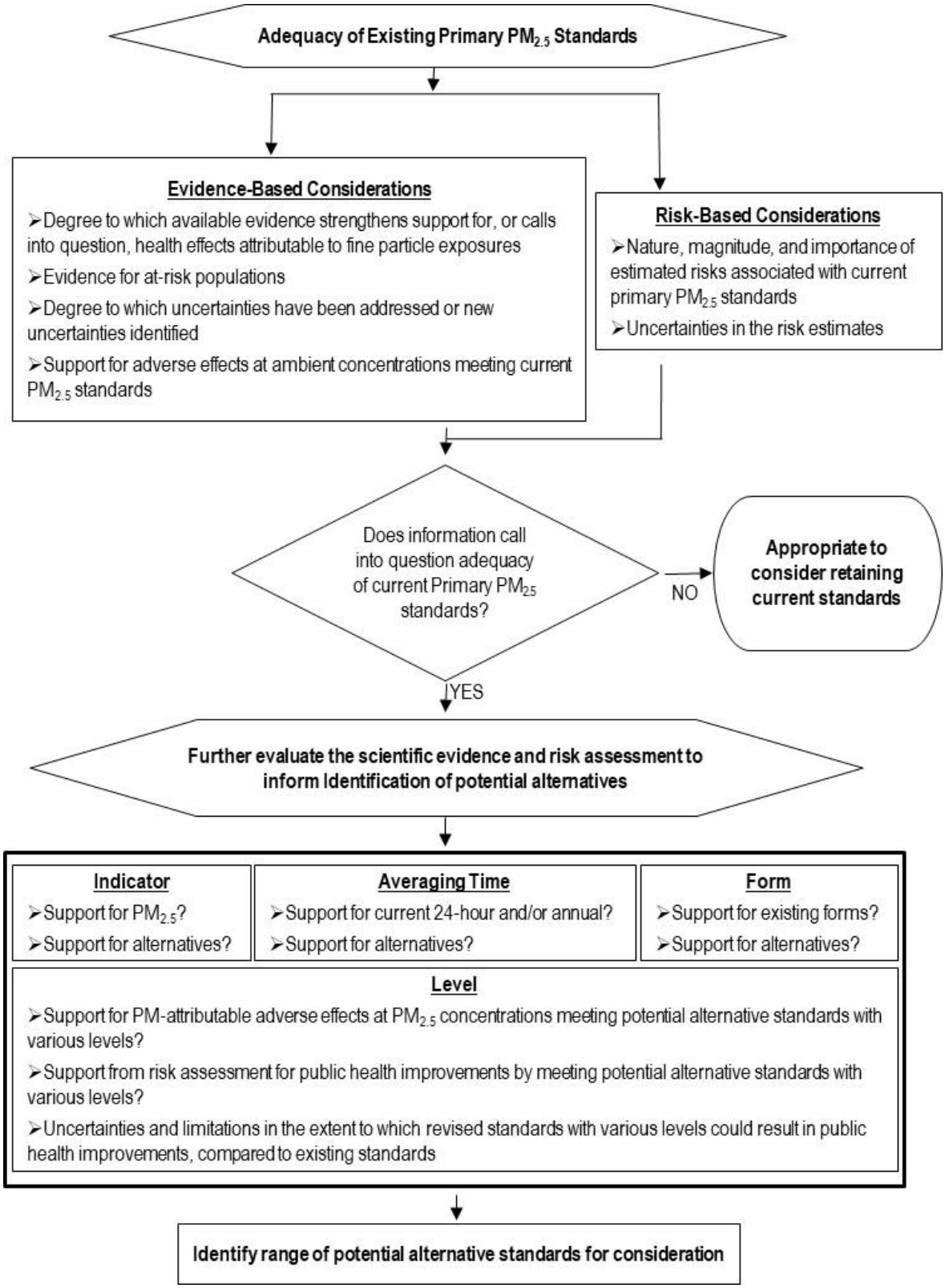


Figure 3-1. Overview of general approach for the reconsideration of the 2020 final decision on the primary $PM_{2.5}$ standards.

The Agency's approach in reconsidering the primary standards is consistent with requirements of the provisions of the CAA related to the review of the NAAQS and with how the EPA and the courts have historically interpreted the CAA. As discussed in section 1.1 above, these provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite (i.e., neither more nor less stringent than necessary) to protect public health with an adequate margin of safety. Consistent with the Agency's approach across all NAAQS reviews, the approach of this draft PA to informing these judgments is based on a recognition that the available health effects evidence generally reflects continuums that include ambient air exposures for which scientists generally agree that health effects are likely to occur through lower levels at which the likelihood and magnitude of response become increasingly uncertain. The CAA does not require the Administrator to establish a primary standard at a zero-risk level or at background concentration levels, but rather at a level that reduces risk sufficiently so as to protect public health, including the health of sensitive groups,[5] with an adequate margin of safety.

The decisions on the adequacy of the current primary $PM_{2.5}$ standards and on any alternative standards considered in a reconsideration are largely public health policy judgments made by the Administrator. The four basic elements of the NAAQS (i.e., indicator, averaging time, form, and level) are generally considered collectively in evaluating the health protection afforded by the current standards, and by any alternatives considered. The Administrator's final decisions draw upon the scientific evidence for health effects, quantitative analyses of population exposures and/or health risks, as available, and judgments about how to consider the uncertainties and limitations that are inherent in the scientific evidence and quantitative analyses.

3.3 HEALTH EFFECTS EVIDENCE

In this section, we draw from the EPA's synthesis and assessment of the scientific evidence presented in the 2019 ISA (U.S. EPA, 2019) and the draft ISA Supplement (U.S. EPA, 2021a) to consider the following policy-relevant question:

[5] More than one population group may be identified as sensitive or at-risk in a NAAQS review. Decisions on NAAQS reflect consideration of the degree to which protection is provided for these sensitive population groups. To the extent that any particular population group is not among the identified sensitive groups, a decision that provides protection for the sensitive groups would be expected to also provide protection for other population groups.

- **To what extent does the currently available scientific evidence, as assessed in the 2019 ISA and the draft ISA Supplement, support or call into question the public health protection afforded by the current suite of $PM_{2.5}$ standards?**

The 2019 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, 2015b, 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 previous reviews, the 2019 ISA adopts 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, 2015b, Preamble Table II). In using the weight-of-evidence approach to inform judgments about the causal nature of relationships between PM exposure and health effects, 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, 2015b, Preamble, section 5.c.). In this draft PA, we consider the full body of health evidence, including evidence from the 2019 ISA and draft ISA Supplement, placing the greatest emphasis on the health effects for which the evidence has been judged in the 2019 ISA to demonstrate a "causal" or a "likely to be causal" relationship with PM exposures. The 2019 ISA defines these causality determinations as follows (U.S. EPA, 2019, p. p-20; U.S. EPA, 2015b):

- 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.

While the 2019 ISA provides the broad scientific foundation for this reconsideration, we recognize that additional literature has become available since the literature cutoff date of the 2019 ISA that expands the body of evidence that can inform the Administrator's judgments on the adequacy of the current primary $PM_{2.5}$ standards. As such, the draft ISA Supplement builds on the information in the 2019 ISA with a targeted identification and evaluation of new scientific information (U.S. EPA, 2021a, section 1.2). The draft ISA Supplement focuses on $PM_{2.5}$ health effects evidence where the 2019 ISA concludes a "causal relationship," because such health effects are given the most weight in an Administrator's decisions in a NAAQS review. The draft

ISA Supplement evaluates newly available evidence related to short- and long-term $PM_{2.5}$ exposure and mortality and cardiovascular effects given the strength of the evidence available in the 2019 ISA and past ISAs and AQCDs, as well as the clear adversity of these endpoints. Specifically, U.S. and Canadian epidemiologic studies for mortality and cardiovascular effects, along with experimental studies related to cardiovascular effects, were considered to be of greatest utility in informing the Administrator's conclusions on the adequacy of the current primary $PM_{2.5}$ standards. While the draft ISA Supplement does not include information for health effects other than mortality and cardiovascular effects, the evidence as it was assessed in the 2019 ISA is considered in this draft PA in reaching preliminary conclusions as a part of the reconsideration of the 2020 final decision.

The draft ISA Supplement also assessed accountability studies because these types of epidemiologic studies were part of the body of evidence that was a focus of the 2020 review. Accountability studies inform our understanding of the potential for public health improvements as ambient $PM_{2.5}$ concentrations have declined over time. Further, the draft ISA Supplement considered studies that employed causal modeling methods, given that such studies were highlighted by the CASAC and identified in public comments in the 2020 review. Since the literature cutoff date for the 2019 ISA, multiple accountability studies and studies that employ causal modeling have become available for consideration in the draft ISA Supplement and in this reconsideration.

The draft ISA Supplement also considered recent health effects evidence that addresses key scientific issues where the literature has expanded since the completion of the 2019 ISA.[6] Given the importance of identifying the populations at increased risk of $PM_{2.5}$-related effects, the draft ISA Supplement also included epidemiologic or exposure studies examining exposure or risk disparities by race/ethnicity or socioeconomic status. The draft ISA Supplement assessed studies that examined the relationship between $PM_{2.5}$ exposures and COVID-19 infection and/or death, as these studies are a new area of research and were raised by a number of public commenters in the 2020 review. These types of studies provide additional information related to factors that may increase risk of $PM_{2.5}$-related health effects and provide additional evidence for consideration by the Administrator in reaching conclusions regarding the adequacy of the current standards.

The evidence presented within the 2019 ISA, along with the targeted identification and evaluation of new scientific information in the draft ISA Supplement, provides the scientific basis for the reconsideration of the 2020 final decision on the primary $PM_{2.5}$ standards. In the

[6] As with the epidemiologic studies for long- and short-term $PM_{2.5}$ exposure and mortality and cardiovascular effects, epidemiologic studies of exposure or risk disparities and COVID-19 infection and/or death were limited to those conducted in the U.S. and Canada.

sections below, we consider the nature of the health effects attributable to long- and short-term fine particle exposures (section 3.3.1), the public health implications and populations potentially at increased risk for PM-related effects (section 3.3.2), and the $PM_{2.5}$ concentrations at which effects have been shown to occur (section 3.3.3).

3.3.1 Nature of Effects

In considering the available evidence for health effects attributable to $PM_{2.5}$ exposures presented in the 2019 ISA and the draft ISA Supplement, this section poses the following policy-relevant questions:

- **To what extent does the currently available scientific evidence strengthen, or otherwise alter, our preliminary conclusions 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 2019 ISA, along with the targeted evaluation of recent evidence in the draft ISA Supplement. In so doing, we place particular emphasis on health outcomes for which the evidence in the 2019 ISA supports either a "causal" or a "likely to be causal" relationship. While the strongest evidence focuses on $PM_{2.5}$, the 2019 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[7] (typically based on physical size, thermal diffusivity or electrical mobility) (U.S. EPA, 2019, Preface, p. 11). Table 3-1 lists causality determinations for all of the health effect categories and exposure durations for both $PM_{2.5}$ and UFP, which we consider within this chapter (adapted from U.S. EPA, 2019, Table 1-4).

[7] Definitions of UFP vary across the scientific literature and, as discussed in sections 3.3.1.5 and 3.3.1.6, 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.

Health Outcome	Size Fraction	Exposure Duration	2009 ISA	2019 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	---	Suggestive of, but not sufficient to infer
		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				

Table 3-1 lists the health outcomes for which the 2019 ISA concludes the evidence supports either a causal, a likely to be causal, or a suggestive relationship. For other health outcomes, the 2019 ISA concludes the evidence is inadequate to infer a causal relationship (U.S. EPA, 2019, Table 1-4).

The 2009 ISA (U.S. EPA, 2009) made causality determinations for the broad category of "Reproductive and Developmental Effects." Causality determinations for 2009 represent this broad category and not specifically for "Male and Female Reproduction and Fertility" and "Pregnancy and Birth Outcomes".

For reproductive and developmental effects, the 2019 ISA's causality determinations reflect the combined evidence for both short- and long-term exposures (U.S. EPA, 2019, Chapter 9).

Sections 3.3.1.1 to 3.3.1.5 summarize the evidence supporting the 2019 ISA's "causal" and "likely to be causal" determinations for $PM_{2.5}$ (italics in Table 3-1) and integrates the recent evidence assessed in the draft ISA Supplement, where available. Section 3.3.1.6 briefly summarizes the evidence supporting the 2019 ISA's "suggestive" determinations, as well as emerging evidence related to COVID-19 infection and death detailed in the draft ISA Supplement. Each of these sections focuses on addressing the policy-relevant questions posed above. Section 3.3.1.7 summarizes the evidence in preceding sections and revisits the policy-relevant questions posed above. Section 3.3.2 describes the public health implications and at-risk populations. In section 3.3.3, we present the $PM_{2.5}$ concentrations in key studies reporting $PM_{2.5}$-related health effects, and section 3.3.4 summarizes the key uncertainties and limitations associated with the health effects evidence.

3.3.1.1 Mortality

Long-term $PM_{2.5}$ exposures

The 2009 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 cohorts, 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).

Cohort studies, assessed in the 2019 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,[8] as well as with specific causes of death, including cardiovascular disease and respiratory disease (U.S. EPA, 2019, section 11.2.2). Many of these 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, 2019, section 11.2.2.1; Figures 11-18 and 11-19). Adding to the 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, 2019, sections

[8] 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, 11.2.5; U.S. EPA, 2021a, Table 11-8). This includes some of the largest cohort studies conducted to date, with analyses of the U.S. Medicare cohort that include nearly 61 million enrollees (Di et al., 2017b) and studies that control for a range of individual and ecological covariates, such as race, age, socioeconomic status, smoking status, body mass index, and annual weather variables (e.g., temperature, humidity).

Many recent North American cohort studies evaluated in the draft ISA Supplement continue to examine the relationship between long-term $PM_{2.5}$ exposure and mortality and report positive and statistically significant associations. Recent studies continue to utilize large and demographically diverse cohorts that are generally representative of the national populations in both the U.S. and Canada, as well as focus on occupation-based specific cohorts. These "studies published since the 2019 ISA support and extend the evidence base that contributed to the conclusion of a *causal relationship* between long-term $PM_{2.5}$ exposure and mortality" (U.S. EPA, 2021a, section 3.2.2.2.1, Figure 3-19, Figure 3-20)

Furthermore, studies in the 2019 ISA and the draft ISA Supplement evaluating cause-specific mortality build on previous research that found consistent, positive associations between cardiovascular and respiratory mortality, as well as other mortality outcomes. For cardiovascular-related mortality, the evidence assessed in the draft ISA Supplement is consistent with the evidence assessed in the 2019 ISA with recent studies reporting positive associations with long-term $PM_{2.5}$ exposure. When evaluating cause-specific cardiovascular mortality, recent studies report positive associations for a number of outcomes including ischemic heart disease (IHD) and stroke mortality (U.S. EPA, 2021a, Figure 3-23). Recent studies also provide some initial evidence that people with pre-existing health issues (such as heart failure and diabetes) are at an increased risk of $PM_{2.5}$-related effects (U.S. EPA, 2021a, section 3.2.2.4) and suggest that these individuals have a higher risk of mortality overall, which was previously only examined in studies that used stratified analyses rather than a cohort of people with an underlying health condition (U.S. EPA, 2021a, section 3.2.2.4). With regard to respiratory mortality, epidemiologic studies assessed in the 2019 ISA and draft ISA Supplement provide continued support for associations between long-term $PM_{2.5}$ exposure and respiratory mortality (U.S. EPA, 2019, section 5.2.10; U.S. EPA, 2021a, Table 3-2).

A series of epidemiologic studies evaluated in the 2019 ISA tested the hypothesis that past reductions in ambient $PM_{2.5}$ concentrations have been associated with increased life expectancy or a decreased mortality rate (U.S. EPA, 2019, section 11.2.2.5). In their original study, Pope et al. (2009) used air quality data in a cross-sectional analysis from 51 metropolitan areas across the U.S., beginning in the 1970s through the early 2000s, to demonstrate that a 10 $\mu g/m^3$ 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, 2019, section 11.2.2.5). Multiple epidemiologic studies that conducted accountability analyses and were published after the literature cutoff date for the 2019 ISA were evaluated in the draft ISA Supplement (U.S. EPA, 2021a, section 3.2.1.3). These studies are consistent with and expand upon the body of evidence from the 2019 ISA. For example, Bennett et al. (2019) reported that $PM_{2.5}$ concentrations above the lowest observed concentration (2.8 $\mu g/m^3$) were associated with a 0.15 year decrease in national life expectancy for women and 0.13 year decrease in national life expectancy for men (U.S. EPA, 2021a, section 3.2.2.2.4, Figure 3-25). Another study compared participants living in areas with $PM_{2.5}$ concentrations >12 $\mu g/m^3$ to participants living in areas with $PM_{2.5}$ concentrations < 12 $\mu g/m^3$ and reported that the number of years of life lost due to living in areas with higher $PM_{2.5}$ concentrations was 0.84 years over a 5-year period (Ward-Caviness et al., 2020; U.S. EPA, 2021a, section 3.2.2.2.4).

Since the 2009 ISA there is an emerging group of studies that used causal modeling statistical methods to further assess relationship between long-term $PM_{2.5}$ exposure and mortality (U.S. EPA, 2019, section 11.2.2.4). The goal of causal modeling methods is to "estimate the difference (or ratio) in the expected value of [an] outcome in the population under the exposure they received versus what it would have been had they received an alternative exposure" (Schwartz et al., 2015). Multiple epidemiologic studies that implemented causal modeling methods and were published since the literature cutoff date of the 2019 ISA were evaluated in the draft ISA Supplement (U.S. EPA, 2021a, section 3.2.2.3). These studies use a variety of statistical methods including generalized propensity score (GPS), inverse probability weighting (IPW), and difference-in-difference (DID) to reduce uncertainties related to confounding bias in the association between long-term $PM_{2.5}$ exposure and mortality. Studies that employed these causal modeling methods reported consistent positive associations that further inform the relationship between long-term $PM_{2.5}$ exposure and total mortality (U.S. EPA, 2021a, section 3.2.2.3). These studies provide further support of associations seen in cohort studies and referenced just above.

The 2019 ISA and draft ISA Supplement also evaluate 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, including methods to estimate exposure, methods to control for confounding, like copollutant

confounding, and the shape of the concentration-response curve. For example, based on its assessment of the evidence, the 2019 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, 2019, section 11.2.5.1). This includes a study Hart et al. (2015) reporting that correction for bias due to exposure measurement error increases the magnitude of the hazard ratios (confidence intervals widen but the association remains statistically significant), suggesting that failure to correct for exposure measurement error could result in attenuation or underestimation of risk estimates.

The 2019 ISA additionally concludes that positive associations between long-term $PM_{2.5}$ exposures and mortality are robust across statistical models that use different approaches to control for confounders or different sets of confounders (U.S. EPA, 2019, sections 11.2.3 and 11.2.5), across diverse geographic regions and populations, and across a range of temporal periods including the periods of declining PM concentrations (U.S. EPA, 2019, sections 11.2.2.5 and 11.2.5.3). Additional evidence further demonstrates that associations with mortality remain robust in copollutants analyses (U.S. EPA, 2019, section 11.2.3), and that associations persist in analyses restricted to long-term exposures below 12 $\mu g/m^3$ (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). Recent studies further assess potential copollutant confounding as reflected in the studies evaluated in the draft ISA Supplement that indicate while there is some evidence of potential confounding of the $PM_{2.5}$-mortality association by copollutants in the some of the studies (i.e., those studies of the MAPLE cohort), this result is inconsistent with other recent studies evaluated in the 2019 ISA that were conducted in the U.S. and Canada that found associations in both single and copollutant models (U.S. EPA, 2019; U.S. EPA, 2021a, section 3.2.2.4 and 3.1.2.2.8). Additionally, a few studies use statistical techniques to reduce uncertainties related to potential confounding in order to further inform conclusions on causality for long-term $PM_{2.5}$ exposure and mortality. For example, studies by Greven et al. (2011), Pun et al. (2017), and Eum et al. (2018) decompose ambient $PM_{2.5}$ into "spatial" and "spatiotemporal" components in order to evaluate the potential for bias due to unmeasured spatial confounding. Eum et al. (2018) and Wu et al. (2020a) also attempted to address long-term trends and meteorological variables as potential confounders and found that not adjusting for temporal trends could overestimate the association, while effect estimates in analyses that excluded meteorological variables remained unchanged compared to main analyses. The results of these

analyses suggest the presence of unmeasured confounding, though they do not indicate the direction or magnitude of the bias that could result.[9]

An additional important consideration in characterizing the public health impacts associated with $PM_{2.5}$ exposure is whether concentration-response relationships are linear across the range of concentrations or if nonlinear relationships exist along any part of this range. Studies evaluated in the 2019 ISA and draft ISA Supplement examine this issue, and continue to provide evidence of linear, no-threshold relationships between long-term $PM_{2.5}$ exposures and all-cause and cause-specific mortality (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section 3.2.2.2.7, Table 3-6). Across the studies evaluated in the 2019 ISA and draft ISA Supplement, a variety of statistical methods have been used to assess whether there is evidence of deviations in linearity (U.S. EPA, 2019, Table 11-7; U.S. EPA, 2021a, section 2.2.3.2). Studies have also conducted cut-point analyses that focus on examining risk at specific ambient $PM_{2.5}$ concentrations. These studies reported results that generally support a linear, no-threshold relationships between long-term $PM_{2.5}$ exposures and total (nonaccidental) mortality, especially at lower ambient $PM_{2.5}$ concentration, with confidence in the linear relationship as low as 5 to 8 $\mu g/m^3$ in some studies (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section 2.2.3.2). There was also some limited evidence indicating that the slope of the concentration-response (C-R) function may be steeper (supralinear) at lower concentrations for cardiovascular mortality (U.S. EPA, 2021a, section 2.2.3.2).

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) when evaluating respiratory and cardiovascular morbidity effects, which are some of the largest contributors to total (nonaccidental) mortality. The 2019 ISA outlines the available evidence for biologically 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 coronary heart disease (CHD), stroke and atherosclerosis (U.S. EPA, 2019, section 6.2.1, Table 11-8), and metabolic effects, including diabetes (U.S. EPA, 2019, section 7.3.1). The 2019 ISA notes "more limited evidence from respiratory morbidity" (U.S. EPA, 2019, p. 11-101) such as development of chronic obstructive pulmonary disease

[9] In public comments on the 2019 draft PA, the authors of the Pun et al. study further note that "the presence of unmeasured confounding…was expected given that we did not control for several potential confounders that may impact $PM_{2.5}$-mortality associations, such as smoking, socio-economic status (SES), gaseous pollutants, $PM_{2.5}$ components, and long-term time trends in $PM_{2.5}$" and that "spatial confounding may bias mortality risks both towards and away from the null" (Docket ID EPA-HQ-OAR-2015-0072-0065; accessible in *https://www.regulations.gov/*)

(COPD) (U.S. EPA, 2019, section 5.2.1) to support the biological plausibility of mortality due to long-term $PM_{2.5}$ exposures (U.S. EPA, 2019, section 11.2.1).

Taken together, recent studies, i.e., those evaluated in the 2019 ISA and in the draft ISA Supplement, reaffirm and further strengthen the body of evidence from the 2009 ISA for the relationship between long-term $PM_{2.5}$ exposure and mortality. Epidemiologic studies evaluated in the 2019 ISA, including recent studies evaluated in the draft ISA Supplement, consistently report positive associations between long-term $PM_{2.5}$ exposure and mortality across different geographic locations, populations, and analytic approaches (U.S. EPA, 2019; U.S. EPA, 2021a, section 3.2.2.4).

As such, these studies reduce key uncertainties identified in the previous review, including those related to potential copollutant confounding, and provide additional information on the shape of the concentration-response curve. As assessed in the 2019 ISA, 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 2019 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, 2019, section 11.2.7; p. 11-102) which is supported and extended by recent evidence evaluated in the draft Supplement (U.S. EPA, 2021a, section 3.2.2.4).

Short-term $PM_{2.5}$ exposures

The 2009 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.

Multicity studies evaluated in the 2019 ISA and draft ISA Supplement 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)[10] at lags of 0 to 1 days in single pollutant-models. Whereas most studies rely on assigning exposures using data from ambient monitors, associations are also reported in studies that employ hybrid modeling approaches using additional $PM_{2.5}$ data (i.e., from satellites, land use information, and air quality modeling, in addition to monitors), allowing for the inclusion of more rural locations in analyses (Kloog et al., 2013, Shi et al., 2016). Consistent with the evidence assessed in previous ISAs, recent studies report more variable results with wider confidence intervals for respiratory mortality (Lavigne et al., 2018; Shin et al., 2021).

Some studies have expanded the examination of potential confounders, including long-term temporal trends, weather, and co-occurring pollutants. Mortality associations were found to remain positive, although in some cases were attenuated, when using different approaches to account for temporal trends or weather covariates (U.S. EPA, 2019, section 11.1.5.1). For example, Sacks et al. (2012) examined the influence of model specification using the approaches for confounder adjustment from models employed in several multicity studies within the context of a common data set (U.S. EPA, 2019, section 11.1.5.1). These models use different approaches to control for long-term temporal trends and the potential confounding effects of weather. The authors report that associations between daily $PM_{2.5}$ and cardiovascular mortality were similar across models, with the percent increase in mortality ranging from 1.5−2.0% (U.S. EPA, 2019, Figure 11-4). Thus, alternative approaches to controlling for long-term temporal trends and for the potential confounding effects of weather may influence the magnitude of the association between $PM_{2.5}$ exposures and mortality but have not been found to influence the direction of the observed association (U.S. EPA, 2019, section 11.1.5.1). Taken together, the 2019 ISA and the draft ISA Supplement conclude that recent multicity studies conducted in the U.S., Canada, Europe, and Asia continue to provide consistent evidence of positive associations between short-term $PM_{2.5}$ exposures and total mortality across studies that use different approaches to control for the potential confounding effects of weather (e.g., temperature) (U.S. EPA, 2019, section 1.4.1.5.1; U.S. EPA, 2021a, section 2.1.1.5.1).

With regard to copollutants, studies evaluated in the 2019 ISA provide additional evidence 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, 2019, 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, 2019, section 11.1.4). Consistent with the studies

[10] As detailed in the Preface to the 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, 2019).

evaluated in the 2019 ISA, studies evaluated in the draft ISA Supplement that used data from more recent years also indicate that associations between short-term $PM_{2.5}$ exposure and mortality remain unchanged in copollutant models. However, the evidence indicates that the association could be larger in magnitude in the presence of some co-occuring pollutants such as oxidant gases (Lavigne et al., 2018; Shin et al., 2021).

The generally positive associations reported with mortality are supported by a small group of studies employing causal modeling methods or quasi-experimental statistical approaches (U.S. EPA, 2019, section 11.1.2.1). For example, two studies by Schwartz et al. (Schwartz et al., 2015; Schwartz et al., 2017) report associations between $PM_{2.5}$ instrumental variables and mortality (U.S. EPA, 2019, Table 11-2), including in an analysis limited to days with 24-hour average $PM_{2.5}$ concentrations <30 $\mu g/m^3$ (Schwartz et al., 2017). In addition to the main analyses, these studies conducted Granger-like causality tests as sensitivity analyses to examine whether there was evidence of an association between mortality and $PM_{2.5}$ after the day of death, which would support the possibility that unmeasured confounders were not accounted for in the statistical model. Neither study reports evidence of an association with $PM_{2.5}$ after death (i.e., they do not indicate unmeasured confounding). A 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. In another study, Schwartz et al. (2018b) utilized three causal methods including instrumental variable analysis, a negative exposure control, and marginal structural models to estimate the association between $PM_{2.5}$ and daily mortality (Schwartz et al., 2018b). Results from this study continue to support a relationship between short-term $PM_{2.5}$ exposure and mortality. Additional epidemiologic studies evaluated in the draft ISA Supplement that employed causal modeling methods to examine the association between short-term $PM_{2.5}$ exposure and mortality also report consistent positive associations in studies that examine effects across multiple cities in the U.S. (U.S. EPA, 2021a).

The positive associations for total mortality reported across the majority of studies evaluated are further supported by analyses reporting generally consistent, positive associations with both cardiovascular and respiratory mortality (U.S. EPA, 2019, section 11.1.3). Recent multicity studies evaluated in the draft ISA Supplement add to the body of evidence indicating a relationship between short-term $PM_{2.5}$ exposure and cause-specific mortality, with more variability in the magnitude and precision of associations for respiratory mortality (U.S. EPA, 2021a; Figure 3-14). For both cardiovascular and respiratory mortality, there has been a 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 events and heart failure, as detailed in the assessment of cardiovascular morbidity (U.S. EPA, 2019, 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 limited biological plausibility for $PM_{2.5}$-related respiratory mortality (U.S. EPA, 2019, Chapter 5).

In the 2009 ISA, one of the main uncertainties identified was the regional and city-to-city heterogeneity in $PM_{2.5}$-mortality associations. Recent studies examine both city-specific as well as regional characteristics to identify the underlying contextual factors that could contribute to this heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Analyses focusing on effect modification of the $PM_{2.5}$-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 observed heterogeneity. Additional studies find that factors related to potential exposure differences, such housing stock and commuting, as well as city-specific factors (e.g., land-use, port volume, and traffic information), may explain some of the observed heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Collectively, studies evaluated in the 2019 ISA and the draft ISA Supplement 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 that could influence exposures (U.S. EPA, 2019, section 11.1.12; U.S. EPA, 2021a, section 3.2.1.2.1)).

A number of 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, 2019, section 11.1.8.1; U.S. EPA, 2021a, section 3.2.1.1). 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.

Multicity studies indicate that positive and statistically significant associations with mortality persist in analyses restricted to short-term $PM_{2.5}$ exposures below 35 $\mu g/m^3$ (Lee et al., 2015),[11] below 30 $\mu g/m^3$ (Shi et al., 2016), and below 25 $\mu g/m^3$ (Di et al., 2017a), indicating that

[11] Lee et al. (2015) also report that positive and statistically significant associations between short-term $PM_{2.5}$ exposures and mortality persist in analyses restricted to areas with long-term concentrations below 12 $\mu g/m^3$.

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 C-R relationship and whether a threshold exists specifically for $PM_{2.5}$ (U.S. EPA, 2019, section 11.1.10). These studies have used various statistical approaches and consistently demonstrate a linear relationship with no evidence of a threshold. Moreover, recent studies evaluated in the draft ISA Supplement provide additional support for a linear, no-threshold C-R relationship between short-term $PM_{2.5}$ exposure and mortality, with confidence in the shape decreasing at concentrations below 5 $\mu g/m^3$ (Liu et al., 2019; Lavigne et al., 2018). 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, 2019). However, given the limited data available at the lower end of the distribution of ambient $PM_{2.5}$ concentrations, the shape of the C-R curve remains uncertain at these low concentrations. Although difficulties remain in assessing the shape of the $PM_{2.5}$-mortality C-R relationship, to date, studies have not conducted systematic evaluations of alternatives to linearity, and recent studies continue to provide evidence of a no-threshold linear relationship, with less confidence at concentrations lower than 5 $\mu g/m^3$.

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 2019 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, 2019, pp. 11-58). Recent evidence evaluated in the draft ISA Supplement provides "additional support to the evidence base that contributed to the conclusion of a causal relationship between short-term $PM_{2.5}$ exposure and mortality" (U.S. EPA, 2021a, section 3.2.1.4, pp 3-69).

3.3.1.2 Cardiovascular Effects

Long-term $PM_{2.5}$ exposures

The scientific evidence reviewed in the 2009 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. and Canadian cohorts that consistently showed positive associations between long-term $PM_{2.5}$ exposure and cardiovascular mortality (Krewski et al., 2009, Miller et al., 2007, et al.,).

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 ISA, the 2019 ISA concludes that recent studies, together with the evidence available in previous reviews, support a causal relationship between long-term exposure to $PM_{2.5}$ and cardiovascular effects. Additionally, recent epidemiologic studies published since the completion of the 2019 ISA and evaluated in the draft ISA Supplement expands the body of evidence and further supports such a conclusion (U.S. EPA, 2021a). As discussed above (section 3.3.1.1), results from U.S. and Canadian cohort studies evaluated in the 2019 ISA consistently report positive associations between long-term $PM_{2.5}$ exposure and cardiovascular mortality (U.S. EPA, 2019, Figure 6-19) in evaluations conducted at varying spatial scales and employing a variety of exposure assessment and statistical methods (U.S. EPA, 2019, section 6.2.10). Positive associations between long-term $PM_{2.5}$ 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, 2019, section 6.2.16; Table 6-52).

The body of literature examining the relationship between long-term $PM_{2.5}$ exposure and cardiovascular morbidity has greatly expanded since the 2009 ISA, with positive associations reported in several cohorts (U.S. EPA, 2019, section 6.2). Though results for cardiovascular morbidity are less consistent than those for cardiovascular mortality (U.S. EPA, 2019, section 6.2), studies in the 2019 ISA and draft ISA Supplement provide some evidence for associations between long-term $PM_{2.5}$ exposures and the progression of cardiovascular disease. Positive associations with cardiovascular morbidity (e.g., coronary heart disease, stroke, arrhythmias, myocardial infarction (MI), and atherosclerosis progression) are observed in several epidemiologic studies (U.S. EPA, 2019, sections 6.2.2. to 6.2.9; U.S. EPA, 2021a, section 3.1.1.4). 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, 2019, 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, 2019, sections 6.2.5 and 6.2.7).

Associations with heart failure are supported by animal toxicological studies demonstrating decreased cardiac contractility and function, and increased coronary artery wall thickness following long-term $PM_{2.5}$ exposure (U.S. EPA, 2019, section 6.2.5.2). Similarly, a limited number of animal toxicological studies demonstrating a relationship between long-term exposure to $PM_{2.5}$ 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. Moreover, a number of studies assessed in the draft ISA Supplement focusing on morbidity outcomes, including those that focused on incidence of MI, atrial fibrillation (AF), stroke, and congestive heart failure (CHF), expand the evidence pertaining to the shape of the C-R relationship between long-term $PM_{2.5}$ exposure and cardiovascular effects. Additionally, studies evaluated in the draft ISA Supplement report positive associations among those with pre-existing conditions, among patients followed after a cardiac event procedure, and among those with a first hospital admission for heart attacks among older adults enrolled in Medicare (U.S. EPA, 2021a, sections 3.1.1 and 3.1.2). A number of these studies use statistical techniques that allow for departures from linearity (U.S. EPA, 2021a, Table 3-3, and generally support the evidence characterized in the 2019 ISA showing linear, no-threshold C-R relationship for most CVD outcomes. However, there is some evidence for a sublinear or supralinear C-R relationship for some outcomes (U.S. EPA, 2021a, section 3.1.2.2.9).[12] Moreover, several recent epidemiologic studies evaluated in the draft ISA Supplement reported that the association between long-term $PM_{2.5}$ exposure with stroke persisted after adjustment for NO_2 but was attenuated in the model with O_3 and oxidant gases represented by the redox weighted average of NO_2 and O_3 (U.S. EPA, 2021a, section 3.1.2.2.8).

Longitudinal epidemiologic analyses also report positive associations with markers of systemic inflammation (U.S. EPA, 2019, section 6.2.11), coagulation (U.S. EPA, 2019, section 6.2.12), and endothelial dysfunction (U.S. EPA, 2019, section 6.2.13). These results are coherent with animal toxicological studies generally reporting increased markers of systemic inflammation, oxidative stress, and endothelial dysfunction (U.S. EPA, 2019, section 6.2.12.2 and 6.2.14).

The 2019 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. Epidemiologic studies in the draft ISA Supplement support and extend the findings characterized in the 2019 ISA, providing additional evidence of positive associations between long-term $PM_{2.5}$ exposure and cardiovascular morbidity (U.S. EPA, 2021a section

[12] As noted above for mortality, uncertainty in the shape of the C-R relationship increases near the upper and lower ends of the distribution due to limited data.

3.1.1.4). 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 (U.S. EPA, 2019, Table 3-2; U.S. EPA, 2021a). 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 2019 ISA conclusion that "a causal relationship exists between long-term exposure to $PM_{2.5}$ and cardiovascular effects" (U.S. EPA, 2019, section 6.2.18). Studies evaluated in the draft ISA Supplement support and extend the evidence that contributed to the conclusion of a causal relationship between long-term $PM_{2.5}$ exposure and cardiovascular effects (U.S. EPA, 2021a, section 3.1.2.4).

Short-term $PM_{2.5}$ exposures

The 2009 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 ISA was from epidemiologic studies of emergency department (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.[13] Key uncertainties from the 2009 ISA 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 ISA identified a growing body of evidence from controlled human exposure and animal toxicological studies, uncertainties remained with respect to biological plausibility.

Recent evidence assessed in the 2019 ISA and the draft ISA Supplement supports 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 multicity epidemiologic studies of emergency department visits and

[13] Some animal studies included in the 2009 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.

hospital admissions for 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, 2019, section 6.1.3.1). Moreover, recent multicity studies, published after the literature cutoff date of the 2019 ISA, are coherent with studies evaluated in the 2019 ISA that report positive association between short-term $PM_{2.5}$ exposure and ED visits and hospital admission for IHD, heart attacks, and HF (U.S. EPA, 2021a, section 3.1). Epidemiologic studies conducted in single cities contribute some support, though associations reported in single-city studies are less consistently positive than in multicity studies, and include a number of studies reporting null associations (U.S. EPA, 2019, sections 6.1.2 and 6.1.3). When considered as a whole; however, the recent body of IHD and HF epidemiologic evidence supports the evidence from previous ISAs reporting mainly positive associations between short-term $PM_{2.5}$ concentrations and emergency department visits and hospital admissions.

Consistent with the evidence assessed in the 2019 ISA, some studies evaluated in the draft ISA Supplement report no evidence of an association with stroke, regardless of stroke subtype. Additionally, as in the 2019 ISA, evidence evaluated in the draft ISA Supplement continues to indicate an immediate effect of $PM_{2.5}$ on cardiovascular-related outcomes primarily within the first few days after exposure, and that associations generally persisted in models adjusted for copollutants (U.S. EPA, 2021a, section 3.1.1.2).

A number of 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, 2019, section 6.1.1). The most consistent evidence from recent controlled human exposure studies is for endothelial dysfunction, as measured by changes in brachial artery diameter or flow mediated dilation. All but one of the available controlled human exposure studies examining the potential for endothelial dysfunction report an effect of $PM_{2.5}$ exposure on measures of blood flow (U.S. EPA, 2019, section 6.1.13.2). These studies report variable results regarding the timing of the effect and the mechanism by which reduced blood flow occurs (i.e., availability vs sensitivity to nitric oxide). Some controlled human exposure studies using CAPs report evidence for small increases in blood pressure (U.S. EPA, 2019, section 6.1.6.3). In addition, although not entirely consistent, there is also some evidence across controlled human exposure studies for conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (HRV) (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). A recent study by Wyatt et al. (2020a) adds to the limited evidence base of controlled human exposure studies conducted at near ambient $PM_{2.5}$

concentrations. The study, completed in healthy young adults subject to intermittent exercise, found some significant cardiovascular effects (e.g., systematic inflammation markers, including C-reactive protein (CRP), and cardiac repolarization).

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 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, 2019, section 6.1.3.3). In addition, and as with controlled human exposure studies, there is generally consistent evidence in animal toxicological studies for indicators of endothelial dysfunction (U.S. EPA, 2019, section 6.1.13.3). Studies in animals also provide evidence for changes in a number of other cardiovascular endpoints following short-term $PM_{2.5}$ exposure. Although not entirely consistent, these studies provide some evidence of conduction abnormalities and arrhythmia (U.S. EPA, 2019, section 6.1.4.4), changes in HRV (U.S. EPA, 2019, section 6.1.10.3), changes in blood pressure (U.S. EPA, 2019, section 6.1.6.4), and evidence for systemic inflammation and oxidative stress (U.S. EPA, 2019, section 6.1.11.3).

In summary, recent evidence evaluated in the 2019 ISA and the draft ISA Supplement 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 2019 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,

2019, p. 6-138), which is further supported by recent studies evaluated in the draft ISA Supplement (U.S. EPA, 2021a section 3.1.1.4).

3.3.1.3 Respiratory Effects

Long-term $PM_{2.5}$ exposures

The 2009 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.

Cohort studies evaluated in the 2019 ISA provided 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, 2019, section 5.2.13). This relationship was further supported by a retrospective study that reports an association between declining $PM_{2.5}$ concentrations and improvements in lung function growth in children (U.S. EPA, 2019, section 5.2.11). Epidemiologic studies also examine asthma development in children (U.S. EPA, 2019, section 5.2.3), with prospective cohort studies reporting generally positive associations, though several are imprecise (i.e., they report wide confidence intervals). Supporting evidence is provided by studies reporting associations with asthma prevalence in children, with childhood wheeze, and with exhaled nitric oxide, a marker of pulmonary inflammation (U.S. EPA, 2019, section 5.2.13). Additionally, animal toxicological study showing the development of an allergic phenotype and an increase in a marker of airway responsiveness provides biological plausibility for allergic asthma (U.S. EPA, 2019, section 5.2.13). Other epidemiologic studies report a $PM_{2.5}$-related acceleration of lung function decline in adults, while improvement in lung function was observed with declining $PM_{2.5}$ concentrations (U.S. EPA, 2019, section 5.2.11). A 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, 2019, section 5.2.11). A common uncertainty across the epidemiologic evidence is the lack of examination of copollutants to assess the potential for confounding. While there is some evidence that associations remain robust in models with gaseous pollutants, a number of these studies examining copollutant confounding were conducted in Asia, and thus have limited generalizability due to high annual pollutant concentrations.

When taken together, the 2019 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 bronchitis symptoms in children with asthma, with an acceleration of lung function decline in adults, and with respiratory mortality and cause-specific respiratory mortality for COPD and respiratory infection" (U.S. EPA, 2019, p. 1-34). In support of the biological plausibility of such associations reported in epidemiologic studies of respiratory health effects, animal toxicological studies continue to provide direct evidence that long-term exposure to $PM_{2.5}$ results in a variety of respiratory effects. Animal studies in the 2019 ISA 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 2019 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, 2019, section 5.2.13).

Short-term $PM_{2.5}$ exposures

The 2009 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 copollutants 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 copollutants 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.

Epidemiologic studies evaluated in the 2019 ISA continue to provide strong evidence for a relationship between short-term $PM_{2.5}$ exposure and several respiratory-related endpoints, including asthma exacerbation (U.S. EPA, 2019, section 5.1.2.1), COPD exacerbation (U.S. EPA, 2019, section 5.1.4.1), and combined respiratory-related diseases (U.S. EPA, 2019, section 5.1.6), particularly from studies examining emergency department visits and hospital admissions. The generally positive associations between short-term $PM_{2.5}$ 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, 2019, sections 5.1.2.2 and 5.4.1.2). The collective body of epidemiologic evidence for asthma exacerbation is more consistent in children than in adults. Additionally, epidemiologic studies examining the relationship between short-term $PM_{2.5}$ exposure and respiratory mortality provide evidence of consistent positive associations, demonstrating a continuum of effects (U.S. EPA, 2019, section 5.1.9).

Building off the studies evaluated in the 2009 and 2019 ISA, 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, 2019, section 5.1.10.1).

In the 2019 ISA, 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, 2019, sections 5.1.2.4.4 and 5.1.4.4.3). The experimental evidence provides biological plausibility for some respiratory-related endpoints, including limited evidence of altered host defense and greater susceptibility to bacterial infection as well as consistent evidence of respiratory irritant effects. Animal toxicological evidence for other respiratory effects is inconsistent. A recent study by Wyatt et al. (2020a) was conducted at near ambient $PM_{2.5}$ concentrations and adds to the limited evidence base of controlled human

exposure studies. The study, completed in healthy young adults subject to intermittent exercise, found some significant respiratory effects (e.g., decrease in lung function).

The 2019 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, 2019, p. 5-155). When taken together, the 2019 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, 2019, p. 5-155).

3.3.1.4 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, 2019, section 10.2.5). Re-analyses 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, 2019, Figure 10-3). Additional support for positive associations with lung cancer mortality is provided by epidemiologic studies using individual-level data to control for smoking status, by studies of people who have never smoked (though such studies generally report wide confidence intervals due to the small number of lung cancer mortality cases within this population), and in analyses of cohorts that relied upon proxy measures to account for smoking status (U.S. EPA, 2019, section 10.2.5.1.1). Although studies that have evaluated lung cancer incidence, including studies of

people who have never smoked, are limited in number, studies in the 2019 ISA generally report positive associations with long-term $PM_{2.5}$ exposures (U.S. EPA, 2019, section 10.2.5.1.2). A subset of the studies focusing on lung cancer incidence also examined histological subtype, providing some evidence of positive associations for adenocarcinomas, the predominate subtype of lung cancer observed in people who have never smoked (U.S. EPA, 2019, section 10.2.5.1.2). Associations between long-term $PM_{2.5}$ 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, 2019, section 10.2.5.1.2).

The 2019 ISA evaluates the degree to which 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, 2019, section 10.2.5.1.3). However, available studies have not systematically evaluated the potential for copollutant confounding by other gaseous pollutants or by other particle size fractions (U.S. EPA, 2019, section 10.2.5.1.3). Compared to total (non-accidental) mortality (U.S. EPA, 2019, section 10.2.4.1.4), fewer studies have examined the shape of the concentration-response curve for cause-specific mortality outcomes, including lung cancer. Several studies of lung cancer mortality and incidence have reported no evidence of deviations from linearity in the shape of the concentration-response relationship (Lepeule et al., 2012; Raaschou-Nielsen et al., 2013; Puett et al., 2014), though authors provided only limited discussions of results (U.S. EPA, 2019, section 10.2.5.1.4).

In support of the biological plausibility of an independent effect of $PM_{2.5}$ on lung cancer, the 2019 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, 2019, 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, 2019, section 10.2.2). Although such effects do not necessarily equate to carcinogenicity, the evidence that PM exposure can damage DNA, and elicit mutations, provides support for the plausibility of epidemiologic associations with lung cancer mortality and incidence. Additional supporting studies indicate the occurrence of micronuclei formation and chromosomal abnormalities (U.S. EPA, 2019, section 10.2.2.3), and differential expression of genes that may be relevant to cancer pathogenesis, following PM exposures. Experimental and

epidemiologic studies that examine epigenetic effects indicate changes in DNA methylation, providing some support for $PM_{2.5}$ exposure contributing to genomic instability (U.S. EPA, 2019, section 10.2.3). Overall, there is limited evidence that long-term $PM_{2.5}$ exposure is associated with cancers in other organ systems, but there is some evidence that $PM_{2.5}$ exposure may reduce survival in individuals with cancer (U.S. EPA, 2019 section 10.2.7; U.S. EPA, 2021a, section 2.1.1.4.1).

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 2019 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, 2019, section 10.2.7).

3.3.1.5 Nervous System Effects

Long-term $PM_{2.5}$ exposures

Reflecting the very limited evidence available in the 2012 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, 2019, section 8.2). Animal toxicology studies assessed in the 2019 ISA 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, 2019, section 8.2.5). Further, while the evidence is limited, the presence of early markers of Alzheimer's disease pathology has been demonstrated in rodents following long-term exposure to $PM_{2.5}$ 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, 2019, section 8.2.6). Additionally, loss of dopaminergic neurons in the substantia nigra, a hallmark of Parkinson disease, has been reported in mice (U.S. EPA, 2019, section 8.2.4), though epidemiologic studies provide only limited support for associations with Parkinson's disease (U.S. EPA, 2019, section 8.2.6). Overall, the lack of consideration of copollutant confounding introduces some uncertainty in the interpretation of epidemiologic studies of nervous system effects, but this uncertainty is partly addressed by the evidence for an independent effect of $PM_{2.5}$ exposures provided by experimental animal studies.

In addition to the findings described above, which are most relevant to older adults, several 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, 2019, section 8.2.7.2), while studies of cognitive function provide little support for an association (U.S. EPA, 2019, section 8.2.5.2). Interpretation of these epidemiologic studies is limited due to the small number of studies, their lack of control for potential confounding by copollutants, and uncertainty regarding the critical exposure windows. Biological plausibility is provided for the ASD findings by a study in mice that found inflammatory and morphologic changes in the corpus collosum and hippocampus, as well as ventriculomegaly (i.e., enlarged lateral ventricles) in young mice following prenatal exposure to $PM_{2.5}$ CAPs.

Taken together, the 2019 ISA concludes that 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, 2019, Table 1-2). Animal toxicology studies provide evidence for a range of nervous system effects in adult animals, including neuroinflammation and oxidative stress, neurodegeneration, and cognitive effects, and effects on neurodevelopment in young animals. The epidemiologic evidence is more limited, but studies generally support associations between long-term $PM_{2.5}$ 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 2019 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, 2019, section 8.2.9).

3.3.1.6 Other Effects

Compared to the health outcomes discussed above, the 2019 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 2019 ISA conclusions for these outcomes for long-term (section 3.3.1.6.1) and short-term (section 3.3.1.6.2) $PM_{2.5}$ and UFP exposures. Section 3.3.1.6.3 summarizes information assessed in the draft ISA Supplement related to the emerging area of COVID-19 infection and death.

3.3.1.6.1 Long-term Exposures

As indicated in Table 3-1 above, the 2019 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, 2019, Preface, p. P-20). 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, 2019, 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 adiposity but the experimental evidence was not entirely consistent. Based on this evidence, the 2019 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, 2019, p. 7-52).

$PM_{2.5}$ – 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, 2019, 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 are 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 2019 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, 2019, p. 9-43).

With regard to pregnancy and birth outcomes, while the collective evidence for many of the outcomes examined is not consistent, there are some animal toxicology and epidemiologic studies that indicate an association between $PM_{2.5}$ 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 2019 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, 2019, p. 9-44).

UFP – Nervous System Effects

The 2009 ISA reported limited animal toxicological evidence of a relationship between long-term exposure to UFP and nervous system effects, with no supporting epidemiologic studies. Animal toxicological studies evaluated in the 2019 ISA substantially add to this evidence base. Multiple toxicological studies of long-term UFP exposure conducted in adult mice provide consistent evidence of brain inflammation and oxidative stress in the whole brain, hippocampus, and cerebral cortex (U.S. EPA, 2019, section 8.6.3). Studies also found morphologic changes, specifically neurodegeneration in specific regions of the hippocampus and pathologic changes characteristic of Alzheimer's disease, and initial evidence of behavioral effects in adult mice (U.S. EPA, 2019, sections 8.6.4 and 8.6.5). Toxicological studies examining pre- and post-natal UFP exposures provide extensive evidence for behavioral effects, altered neurotransmitters, neuroinflammation, and morphologic changes (U.S. EPA, 2019, section 8.6.6.2). Persistent ventriculomegaly was observed in male, but not female, mice exposed postnatally to UFP (U.S. EPA, 2019, section 8.6.6). Epidemiologic evidence is limited to a single study of school children that provides support for the experimental results. This study, which did not consider copollutant

confounding, reports an association between long-term exposure to UFP, which was measured at the school, and decrements on tests of attention and memory. However, uncertainties remain as a result of inadequate assessment of potential copollutant confounding, the spatial variation in UFP concentrations, and exposure measurement error. Based primarily on the animal toxicological evidence of neurotoxicity and altered neurodevelopment, the 2019 ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship" between long-term UFP exposure and nervous system effects (U.S. EPA, 2019, section 8.6.7).

3.3.1.6.2 Short-term Exposures

As indicated in Table 3-1 above, the 2019 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 2019 ISA concludes that the evidence is "suggestive" for short-term UFP exposures and cardiovascular effects, respiratory effects, and nervous system effects. As for the outcomes related to long-term exposures, discussed above, these conclusions reflect evidence that is "generally supportive but not entirely consistent or is limited overall" where "[c]hance, confounding, and other biases cannot be ruled out" (U.S. EPA, 2019, Preface, p.P-20). The basis for these causality determinations is summarized briefly below.

$PM_{2.5}$ – 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, 2019, section 7.1). The 2019 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, 2019, p. 7-11).

$PM_{2.5}$ – 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. Studies assessed in the 2019 ISA provide additional evidence that short-term exposure to $PM_{2.5}$ can affect the nervous system (U.S. EPA, 2019, section 8.1). The strongest evidence is provided by experimental studies in mice that show effects on the brain. These toxicological studies demonstrate changes in neurotransmitters in the hypothalamus that are linked to sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) stress axis activation, as well as upregulation of

inflammation-related genes, changes in cytokine levels, and other changes that are indicative of brain inflammation. In addition, an association of short-term $PM_{2.5}$ exposure with hospital admissions for Parkinson's disease was observed indicating the potential for exacerbation of neurological diseases. The 2019 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, 2019, p. 8-15).

UFP – Cardiovascular effects

In the 2009 ISA, the evidence from toxicological studies, many of which examined exposures to whole diesel exhaust or wood smoke rather than UFP alone, was suggestive of a causal relationship between short-term UFP exposure and cardiovascular effects. Since the 2009 ISA, there have been only a limited number of studies published describing the relationship between short-term UFP exposure and cardiovascular effects. This includes a small number of epidemiologic panel studies that have observed positive associations between short-term exposure to UFPs and measures of HRV (U.S. EPA, 2019, section 6.5.9.1) and markers of coagulation (U.S. EPA, 2019, section 6.5.11.1) although there are also studies that did not report such UFP-related effects. In addition, there is evidence from a single controlled human exposure study indicating decreases in the anticoagulant proteins plasminogen and thrombomodulin in individuals with metabolic syndrome (U.S. EPA, 2019, section 6.5.11.2). There is inconsistent evidence from controlled human exposure and epidemiologic panel studies for endothelial dysfunction, changes in blood pressure, and systemic inflammation following short-term exposure to UFPs. Notably, there is little evidence of an effect when considering short-term UFP exposure on other cardiovascular endpoints as well as cardiovascular-disease emergency department visits or hospital admissions. The assessment of study results across experimental and epidemiologic studies is complicated by differences in the size distributions examined between disciplines and by the nonuniformity in the exposure metrics examined (e.g., particle number concentration, surface area concentration, and mass concentration) (U.S. EPA, 2019, section 1.4.3). When considered as a whole, the 2019 ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term exposure UFP exposure and cardiovascular effects" (U.S. EPA, 2019, p. 6-304).

UFP – Respiratory effects

A limited number of studies examining short-term exposure to UFPs and respiratory effects were reported in the 2009 ISA, which concluded that the relationship between short-term exposure to UFP and respiratory effects is "suggestive of a causal relationship." This conclusion was based on epidemiologic evidence indicating associations with combined respiratory-related diseases, respiratory infection, and asthma exacerbation. In addition, personal exposures to

ambient UFP were associated with lung function decrements in adults with asthma. The few available experimental studies provided limited coherence with epidemiologic findings for asthma exacerbation. Studies assessed in the 2019 ISA add to this evidence base and support epidemiologic evidence for asthma exacerbation and combined respiratory-related diseases but do not rule out chance, confounding, and other biases (U.S. EPA, 2019, section 5.5). For example, associations persist in one epidemiologic study with adjustment for NO_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, 2019, section 5.5). The 2019 ISA concludes that, overall, the evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term UFP exposure and respiratory effects" (U.S. EPA, 2019, p. 5-303).

UFP- Nervous system effects

The 2009 ISA reported limited animal toxicological evidence of a relationship between short-term exposure to UFP and nervous system effects, without supporting epidemiologic studies. Several experimental studies evaluated in the 2019 ISA add to this evidence base. In the 2019 ISA, the strongest evidence for a relationship between short-term UFP exposure and nervous system effects is provided by animal toxicological studies that show inflammation and oxidative stress in multiple brain regions following exposure to UFP. There is a lack of evidence from epidemiologic studies (U.S. EPA, 2019, section 8.5). The 2019 ISA concludes that, overall, the collective evidence is "suggestive of, but not sufficient to infer, a causal relationship between short-term UFP exposure and nervous system effects" (U.S. EPA, 2019, p. 8-86).

3.3.1.6.3 COVID-19 Infection and Death

With the advent of the global COVID-19 pandemic, a number of recent studies evaluated in the draft ISA Supplement examined the role of ambient air pollution, specifically $PM_{2.5}$, on COVID-19 infections and deaths, including a few studies within the U.S. and Canada (U.S. EPA, 2021a; section 3.3.2). While there is no exact corollary within the 2019 ISA for these types of studies, the 2019 ISA presented evidence that evaluates the potential relationship between short- and long-term $PM_{2.5}$ exposure and respiratory infection (U.S. EPA, 2019, section 5.1.5 and 5.2.6). Studies assessed in the 2019 ISA report that some evidence of positive associations

between short-term $PM_{2.5}$ and hospital admissions and emergency department visits for respiratory infections, however the interpretation of these studies is complicated by the variability in the type of respiratory infection outcome examined (U.S. EPA, 2019, Figure 5-7). In the 2019 ISA, studies of long-term $PM_{2.5}$ exposure were limited and while there were some positive associations reported, there was minimal overlap in respiratory infection outcomes examined across studies. Exposure to $PM_{2.5}$ has been shown to impair host defense, specifically altering macrophage function, providing a biological pathway by which $PM_{2.5}$ exposure could lead to respiratory infection (U.S. EPA, 2019, sections 5.1.1 and 5.1.5.) There is some additional evidence that $PM_{2.5}$ exposure can lead to decreases in an individual's immune response, which can subsequently facilitate replication of respiratory viruses (Bourdrel et al., 2021).

As assessed in the draft ISA Supplement, a number of studies examined whether daily changes in $PM_{2.5}$ can influence COVID-19 outcomes (ISA Supplement, section 3.3.2.1). Additionally, several studies assessed in the draft ISA Supplement evaluates whether long-term $PM_{2.5}$ exposure is related to increased susceptibility to COVID-19 outcomes in North America (U.S. EPA, 2021a, section 3.3.2.2). While some of the studies report positive associations, overall, they were subjected to methodological issues that may influence the results, including: (1) the use of ecological study design; (2) some of the studies were conducted during the ongoing pandemic when the etiology of COVID-19 was still not well understood (e.g., specifically, there are important differences in COVID-19 related outcomes by a variety of factors such as race and socioeconomic status); and (3) studies did not account for crucial factors that could influence results (e.g., stay-at-home orders, social distancing, use of masks, and testing capacity) (U.S. EPA, 2021a, chapter 5). Taken together, there is limited evidence at this point in the COVID-19 pandemic to determine if short- or long-term exposure to air pollutants, such as $PM_{2.5}$, influence the spread or susceptibility of COVID-19 in the population.

3.3.1.7 Summary

Based on the evidence assessed in the 2019 ISA and the draft ISA Supplement (U.S. EPA, 2019, U.S. EPA,), and summarized in sections 3.3.1.1 to 3.3.1.6 above, we revisit the policy-relevant questions posed at the beginning of this section:

- **To what extent does the scientific evidence strengthen, or otherwise alter, our preliminary conclusions 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 reviewed in the 2019 ISA and the draft ISA Supplement expand our understanding of the $PM_{2.5}$-related health effects from long- and short- term exposures, as well as reduced important uncertainties identified in prior reviews. Epidemiologic studies

consistently report positive associations between $PM_{2.5}$ exposures and a wide range of health outcomes, including total and cause-specific mortality (e.g., cardiovascular and respiratory mortality), cardiovascular and respiratory morbidity, lung cancer, and nervous system effects. Such associations have been reported in analyses employing 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 U.S. and Canadian epidemiologic studies evaluated in the draft ISA Supplement provide additional support for the conclusions of the 2019 ISA. Overall, these studies support, and in some instances strengthen, the evidence presented in the 2019 ISA of long-term $PM_{2.5}$ exposures and health effects. Cohort studies assessed in the draft ISA Supplement add to the large body of evidence exhibiting consistent, positive associations between long-term $PM_{2.5}$ exposure and mortality detailed in the 2019 ISA. While relatively fewer recent U.S. and Canadian epidemiologic studies examined short-term $PM_{2.5}$ exposure and mortality, these studies continue to provide evidence of positive associations with all-cause and total (nonaccidental) mortality, in addition to cause-specific mortality outcomes. Further, the 2019 ISA and draft ISA Supplement include retrospective studies that demonstrate improvements in health outcomes, including increased life expectancy, decreasing mortality, or decreasing respiratory effects, as a result of decreases in ambient $PM_{2.5}$ concentrations over time. Lastly, 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) when evaluating respiratory and cardiovascular morbidity effects, which are some of the largest contributors to total (nonaccidental) mortality.

Epidemiologic studies (for short-term and long-term exposure) evaluated in the 2019 ISA and the draft ISA Supplement assessed the role potential uncertainties may have on the health-effect associations, and examined various exposure windows, approaches to adjust for confounding variables, and exposure assessment methods that used different sources of data and were conducted at different spatial resolutions. These evaluations increased confidence in the causal relationship between long-term $PM_{2.5}$ exposure and mortality. Moreover, this evidence further informs whether there is evidence of copollutant confounding, and although there were some differences across studies, generally positive associations persisted in copollutant models. Some studies reported that associations persisted in analyses that exclude $PM_{2.5}$ exposures near the upper end of the air quality distribution. Overall, the assessment of the C-R relationship continues to generally support a linear, no-threshold relationship with some recent studies providing evidence for either a sublinear, linear, or supralinear relationship at these lower concentrations.

Building on the evidence presented in the 2019 ISA, the evidence assessed in the draft ISA Supplement provides additional information to address key uncertainties associated with the health effects evidence. The draft ISA Supplement examined an expanded body of evidence related to causal modeling methods, to further evaluate the causal nature of associations between exposure to $PM_{2.5}$ and mortality. Consistent with the 2019 ISA, this expanded body of evidence reduces uncertainties related to confounding and provides robust support for positive and significant associations seen in cohort studies of long-term exposure to $PM_{2.5}$. Although there were fewer more recent multicity studies conducted in the U.S. and Canada examining the relationship between short-term exposure and mortality than for long-term exposure, the studies assessed in the draft ISA Supplement add to the extensive evidence evaluated in the 2019 ISA. Furthermore, these studies report consistent positive associations across studies that are using different exposure assessment methods, statistical models, as well as different methods to control for confounding effects.

Recent U.S. and Canadian epidemiologic studies examining short- and long-term $PM_{2.5}$ exposure and cardiovascular effects provide evidence that is consistent with the evidence evaluated in the 2019 ISA. Studies examining short-term $PM_{2.5}$ exposure report consistent positive associations for cardiovascular-related emergency department visits and hospital admissions, specifically for ischemic heart disease, myocardial infarction, and heart failure. In studies evaluating long-term exposures there remains strong evidence for cardiovascular-related mortality with support from studies of cardiovascular morbidity outcomes, including coronary heart disease, stroke, and atherosclerosis progression, among individuals with preexisting diseases or patients followed after a cardiac event or procedure. In addition, the studies provide evidence of an immediate effect of short-term-related $PM_{2.5}$ exposure on cardiovascular-related outcomes, especially during the first few days following exposure.

With respect to long-term $PM_{2.5}$ exposure, the strongest evidence associated with cardiovascular mortality is exhibited in studies that report positive associations with ischemic heart disease and stroke mortality. Furthermore, recent studies examining association between long-term $PM_{2.5}$ exposure and cardiovascular morbidity, specifically coronary heart disease, stroke, and atherosclerosis progression, most consistently report positive associations when focusing on individuals with pre-existing diseases and patients followed after a cardiac event or procedure, and not the general population as a whole, supporting and extending the evidence presented in 2019 ISA. The 2019 ISA also assessed controlled human exposure studies that were conducted in Europe at near-ambient $PM_{2.5}$ concentrations and provide initial evidence of vascular changes and reductions in heart rate as well as changes in cardiac and lung function as well as inflammation.

The draft ISA Supplement also evaluates epidemiologic studies that examine the relationship between $PM_{2.5}$ exposure and COVID-19 infection and mortality. While these studies report positive associations, there a number of methodological limitations which include: (1) employing an ecological study design, (2) conducting research while COVID-19 etiology was poorly understood, and (3) the lack of accounting for key factors in disease transmission such as use of mask, stay home orders, and testing capacity.

Thus, when taken together, the evidence available in the draft ISA Supplement reaffirms, and in some cases strengthens, the conclusions from the 2019 ISA regarding long- and short-term $PM_{2.5}$ exposures and mortality and cardiovascular effects.

3.3.2 Public Health Implications and At-Risk Populations

The public health implications of the evidence regarding $PM_{2.5}$ health effects, as for other effects, are dependent on the type and severity of the effects, as well as the size of the population affected. Such factors are discussed here in the context of our consideration of the health effects evidence related to $PM_{2.5}$ in ambient air. Additionally, we summarize the information on population groups at risk of the effects of $PM_{2.5}$ in ambient air.

- **Does the evidence alter our understanding of populations that are particularly at risk from $PM_{2.5}$ exposures? What are important uncertainties in that evidence?**

The information available in this reconsideration has not altered our understanding of human populations at risk of health effects from $PM_{2.5}$ exposures. As recognized in the 2020 review, the 2019 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, 2019, p. 12-1). Factors that may contribute to increased risk of $PM_{2.5}$-related health effects include lifestage (children and older adults), pre-existing diseases (cardiovascular disease and respiratory disease), race/ethnicity, and socioeconomic status.[14]

Children make up a substantial fraction of the U.S. population and often have unique factors that contribute to their risk of experiencing a health effect due to exposures to ambient air pollutants because of their continuous growth and development.[15] There is strong evidence that demonstrates $PM_{2.5}$ associated health effects in children, particularly from epidemiologic studies of long-term $PM_{2.5}$ exposure and impaired lung function growth, decrements in lung function, and asthma development. However, there is limited evidence from stratified analyses that children are at increased risk of $PM_{2.5}$-related health effects compared to adults. Additionally,

[14] As described in the 2019 ISA, other factors that have the potential to contribute to increased risk include obesity, diabetes, genetic factors, smoking status, sex, diet, and residential location (U.S. EPA, 2019, chapter 12).

[15] Children, as used throughout this draft PA, generally refers to those younger than 18 years old.

there is some evidence that indicates that children receive higher $PM_{2.5}$ exposures than adults, and dosimetric differences in children compared to adults can contribute to higher doses (U.S. EPA, 2019, section 12.5.1.1).

In the U.S., older adults, often defined as adults 65 years of age and older, represent an increasing portion of the population and often have pre-existing diseases or conditions that may compromise biological function. While there is limited evidence to indicate that older adults have higher exposures than younger adults, older adults may receive higher doses of $PM_{2.5}$ due to dosimetric differences. There is consistent evidence from studies of older adults demonstrating generally consistent, positive associations in studies examining health effects from short- and long-term $PM_{2.5}$ exposure and cardiovascular or respiratory hospital admissions, emergency department visits, or mortality (U.S. EPA, 2019, sections 6.1, 6.2, 11.1, 11.2, 12.5.1.2). Additionally, several animal toxicological, controlled human exposure, and epidemiologic studies did not stratify results by lifestage, but instead focused the analyses on older individuals, and can provide coherence and biological plausibility for the occurrence among this lifestage (U.S. EPA, 2019, section 12.5.1.2).

Individuals with pre-existing disease may be considered at greater risk of an air pollution-related health effect than those without disease because they are likely in a compromised biological state that can vary depending on the disease and severity. With regard to cardiovascular disease, we first note that cardiovascular disease is the leading cause of death in the U.S., accounting for one in four deaths, and approximately 12% of the adult population in the U.S. has a cardiovascular disease (U.S. EPA, 2019, section 12.3.1). Strong evidence demonstrates that there is a causal relationship between cardiovascular effects and long- and short-term exposures to $PM_{2.5}$. Some of the evidence supporting this conclusion is from studies of panels or cohorts with pre-existing cardiovascular disease, which provide supporting evidence but do not directly demonstrate an increase in risk (U.S. EPA, 2019, section 12.3.1). Epidemiologic evidence indicates that individuals with pre-existing cardiovascular disease may be at increased risk for $PM_{2.5}$-associated health effects compared to those without pre-existing cardiovascular disease. While the evidence does not consistently support increased risk for all pre-existing cardiovascular diseases, there is evidence that certain pre-existing cardiovascular diseases (e.g., hypertension) may be a factor that increases $PM_{2.5}$-related risk. Furthermore, there is strong evidence supporting a causal relationship for long- and short-term $PM_{2.5}$ exposure and cardiovascular effects, particularly for IHD (U.S. EPA, 2019, chapter 6, section 12.3.1).

With regard to respiratory disease, we first note that the most chronic respiratory diseases in the U.S. are asthma and COPD. Asthma affects a substantial fraction of the U.S. population and is the leading chronic disease among children. COPD primarily affects older adults and contributes to compromised respiratory function and underlying pulmonary inflammation. The

body of evidence indicates that individuals with pre-existing respiratory diseases, particularly asthma and COPD, may be at increased risk for $PM_{2.5}$-related health effects compared to those without pre-existing respiratory diseases (U.S. EPA, 2019, section 12.3.5). There is strong evidence indicating $PM_{2.5}$-associated respiratory effects among those with asthma, which forms the primary evidence base for the likely to be causal relationship between short-term exposures to $PM_{2.5}$ and respiratory health effects (U.S. EPA, 2019, section 12.3.5). For asthma, epidemiologic evidence demonstrates associations between short-term $PM_{2.5}$ exposures and respiratory effects, particularly evidence for asthma exacerbation, and controlled human exposure and animal toxicological studies demonstrate biological plausibility for asthma exacerbation with $PM_{2.5}$ exposures (U.S. EPA, 2019, section 12.3.5.1). For COPD, epidemiologic studies report positive associations between short-term $PM_{2.5}$ exposures and hospital admissions and emergency department visits for COPD, with supporting evidence from panel studies demonstration COPD exacerbation. Epidemiologic evidence is supported by some experimental evidence of COPD-related effects, which provides support for the biological plausibility for COPD in response to $PM_{2.5}$ exposures (U.S. EPA, 2019, section 12.3.5.2).

There is strong evidence for racial and ethnic disparities in $PM_{2.5}$ exposures and $PM_{2.5}$-related health risk, as assessed in the 2019 ISA and with even more evidence available since the literature cutoff date for the 2019 ISA and evaluated in the draft ISA Supplement. There is strong evidence demonstrating that Black and Hispanic populations, in particular, have higher $PM_{2.5}$ exposures than non-Hispanic White populations (U.S. EPA, 2019, Figure 12-2; U.S. EPA, 2021a, Figure 3-38). Black populations or individuals that live in predominantly Black neighborhoods experience higher $PM_{2.5}$ exposures, in comparison to non-Hispanic White populations. There is also consistent evidence across multiple studies that demonstrate increased risk of $PM_{2.5}$-related health effects, with the strongest evidence for health risk disparities for mortality (U.S. EPA, 2019, section 12.5.4). There is also evidence of health risk disparities for both Hispanic and non-Hispanic Black populations compared to non-Hispanic White populations for cause-specific mortality and incident hypertension (U.S. EPA, 2021a, 3.3.3.2).

Socioeconomic status (SES) is a composite measure that includes metrics such as income, occupation, or education, and can play a role in access to healthy environments as well as access to healthcare. SES may be a factor that contributes to differential risk from $PM_{2.5}$-related health effects. Studies assessed in the 2019 ISA and draft ISA Supplement provide evidence that lower SES communities are exposed to higher concentrations of $PM_{2.5}$ compared to higher SES communities (U.S. EPA, 2019, section 12.5.3; U.S. EPA, 2021a, section 3.3.3.1.1). Studies using composite measures of neighborhood SES consistently demonstrated a disparity in both $PM_{2.5}$ exposure and the risk of $PM_{2.5}$-related health outcomes. There is some evidence that supports associations larger in magnitude between mortality and long-term $PM_{2.5}$ exposures for

those with low income or living in lower income areas compared to those with higher income or living in higher income neighborhoods (U.S. EPA, 2019, section 12.5.3; U.S. EPA, 2021a, section 3.3.3.1.1). Additionally, evidence supports conclusions that lower SES is associated with cause-specific mortality and certain health endpoints (i.e., HI and CHF), but less so for all-cause or total (non-accidental) mortality (U.S. EPA, 2021a, section 3.3.3.1).

- **What does the available information indicate with regard to the size of at-risk populations and their distribution in the U.S.?**

The magnitude and characterization of a public health impact is dependent upon the size and characteristics of the populations affected, as well as the type or severity of the effects. As summarized above, lifestage (children and older adults), race/ethnicity and socioeconomic status are factors that increase the risk of $PM_{2.5}$-related health effects. The American Community Survey (ACS) for 2019 estimates that approximately 22% and 16% of the U.S. population are children (age <18) and older adults (age 65+), respectively. For all ages, non-Hispanic Black and Hispanic populations are approximately 12% and 18% of the overall U.S. population in 2019. Table 3-2 below considers the currently available information that helps to characterize key features of these populations.

Table 3-2. National demographic information, 2019.

Characteristic [1]	Number	Percent of Total
Total	328,239,523	
Child (Age <18)	72,967,785	22.2
Adult (Age 18+)	255,271,738	77.8
All Age Groups		
0-4 years	19,404,835	5.9
5-14 years	41,113,916	12.5
15-19 years	21,353,524	6.5
20-24 years	21,468,680	6.5
25-34 years	45,578,475	13.9
35-64 years	125,246,065	38.1
65+ years	54,074,028	16.4
Race/Ethnicity	328,239,523	
White NH [2]	196,789,401	60
Black NH	40,596,040	12.4
American Indian or Alaska Native NH	2,236,348	0.7
Asian NH	18,427,914	5.6
Hispanic, all	60,481,746	18.4
Other NH	9,708,074	3
Household Income (past 12 months) [3]		
Less than $10,000		5.8

$10,000 to $14,999		4.0
$15,000 to $24,999		8.3
$25,000 to $34,999		8.4
$35,000 to $49,999		11.9
$50,000 to $74,999		17.4
$75,000 to $99,999		12.8
$100,000 to $149,999		15.7
$150,000 to $199,999		7.2
$200,000 or more		8.5
Educational Attainment [4]		
Less than high school	25,618,541	11.4
High school graduate (or equivalent)	60,482,353	26.9
Some college, no degree	44,914,086	20
Associate's degree	19,381,937	8.6
Bachelor's degree	45,730,479	20.3
Graduate or professional degree	28,771,172	12.8

[1] Numbers within selected characteristics may not sum to total due to rounding
[2] NH = non-Hispanic
[3] Household income in the past 12 months in 2019 inflation-adjusted dollars.
[4] Educational attainment for population aged 25 years and older.
Adapted from the 2019 American Community Survey and Housing Survey. Available at:
Demographics: *https://data.census.gov/cedsci/table?q=United%20States&tid=ACSDP1Y2019.DP05*
Income: https://data.census.gov/cedsci/table?q=United%20States&t=Income%20and%20Poverty&tid=ACSST1Y2019.S1901
Education:
https://data.census.gov/cedsci/table?q=United%20States&t=Education%3AEducational%20Attainment&tid=ACSST1Y2019.S1501

As noted above, individuals with pre-existing cardiovascular disease and pre-existing respiratory disease may also be at increased risk of $PM_{2.5}$-related health effects. Table 3-3 below considers the currently available information that helps to characterize key features of populations with cardiovascular or respiratory diseases or conditions. The National Center for Health Statistics data for 2018 indicate that, for adult populations, older adults (e.g., those 65 years and older) have a higher prevalence of cardiovascular diseases compared to younger adults (e.g., those 64 years and younger). For respiratory diseases, older adults also have a higher prevalence of emphysema than younger adults, and adults 44 years or older have a higher prevalence of chronic bronchitis. However, the prevalence for asthma is generally similar across all adult age groups.

With respect to race, American Indians or Alaskan Natives have the highest prevalence of all heart disease and coronary heart disease, while Blacks have the highest prevalence of hypertension and stroke. Hypertension has the highest prevalence across all racial groups compared to other cardiovascular diseases or conditions, ranging from approximately 22% to 32% of each racial group. Overall, the prevalence of cardiovascular diseases or conditions is lowest for Asians compared to Whites, Blacks, and American Indians or Alaskan Natives.

Asthma prevalence is highest among Black and American Indian or Alaska Native populations, while prevalence is generally similar across racial groups for chronic bronchitis and emphysema. Overall, the prevalence for respiratory diseases is lowest for Asians compared to Whites, Blacks, and American Indians or Alaskan Natives. With regard to ethnicity, cardiovascular and respiratory disease prevalence across all diseases or conditions is generally similar between Hispanic and non-Hispanic populations, although non-Hispanics have a slightly higher prevalence compared to Hispanics.

Table 3-3. Prevalence of cardiovascular and respiratory diseases among adults by age, race, and ethnicity in the U.S. in 2018.

	Adults (18+)	Age (%) [1]				Race (%) [2]				Ethnicity (%) [3]	
Chronic Disease or Condition	N (in thousands)	18-44	44-64	65-74	75+	White	Black	American Indian or Alaska Native	Asian	Hispanic	Non-Hispanic
All (N, in thousands)	249,456	115,008	83,038	30,809	20,601	193,454	30,813	2,810	15,960	40,749	208,706
Selected Cardiovascular Diseases/Conditions											
All heart disease	30,252	4.8	11.8	23.6	37.3	11.5	10.0	14.6	7.7	8.2	11.7
Coronary heart disease	15,780	1.0	6.0	15.5	23.9	5.7	5.4	8.6	4.4	5.1	5.7
Hypertension	67,856	8.8	34.4	54.4	61.1	23.9	32.2	27.2	21.9	23.7	25.1
Stroke	7,801	0.6	3.1	6.9	11.8	2.6	3.9	3.0	2.7	2.5	2.9
Selected Respiratory Diseases											
Asthma [4]	19,233	7.2	8.3	8.6	6.7	7.5	9.1	9.5	3.7	6.0	8.1
COPD – chronic bronchitis	9,003	2.2	4.5	5.1	5.6	3.6	3.4	*	1.1	2.7	3.6
COPD – emphysema	3,780	0.2	1.6	4.1	4.5	1.4	1.1	0.4	0.7	1.0	1.4

[1] Percentage of individual adults within each age group with disease, based on N (at the top of each age column).
[2] Percentage of individual adults within each race group with disease, based on N (at the top of each race column).
[3] Percentage of individual adults within each ethnic group with disease, based on N (at the top of each ethnic column).
[4] Asthma prevalence is reported for "still has asthma."
* Estimate does not meet NCHS standards of reliability.
Source: (Insert cites); National Center for Health Statistics, Summary Health Statistics, National Health Interview Survey, 2018; Tables A-1 and A-2.

Taken together, this information indicates that the groups at increased 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.

3.3.3 $PM_{2.5}$ 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 2019 ISA concludes that the evidence supports a "causal" or a "likely to be causal" relationship with $PM_{2.5}$ 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 2019 ISA are informed by studies evaluating a wide range of $PM_{2.5}$ concentrations, this section considers the degree to which the evidence in the 2019 ISA and draft ISA Supplement supports the occurrence of PM-related effects at concentrations relevant to informing conclusions on the primary $PM_{2.5}$ standards. Section 3.3.3.1 considers the exposure concentrations that have been evaluated in experimental studies and section 3.3.3.2 considers the ambient concentrations in locations evaluated by epidemiologic studies.

3.3.3.1 PM Exposure Concentrations Evaluated in Experimental Studies

As stated in the 2019 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, 2015b, 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 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, 2015b, Preamble, p. 11).

In this section, we consider the $PM_{2.5}$ exposure concentrations shown to result 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 or alternative primary standards?**

Controlled Human Exposure Studies

As discussed in detail in the 2019 ISA (U.S. EPA, 2019, 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.[16] The most consistent evidence from these studies is for impaired vascular function (U.S. EPA, 2019, section 6.1.13.2). In addition, although less consistent, the 2019 ISA notes that studies examining $PM_{2.5}$ exposures also provide evidence for increased blood pressure (U.S. EPA, 2019, section 6.1.6.3), conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). The 2019 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, 2019, section 6.1.16). Thus, such studies can provide insight into the potential for specific $PM_{2.5}$ exposures to result in physiological changes that could increase the risk of more serious effects.

Table 3-4 below summarizes information from the 2019 ISA and draft ISA Supplement 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 air.[17]

[16] In contrast, controlled human exposure studies provide little evidence for respiratory effects following short-term $PM_{2.5}$ exposures (U.S. EPA, 2019, section 5.1, Table 5-18). Therefore, this section focuses on cardiovascular effects evaluated in controlled human exposure studies of $PM_{2.5}$ exposure.

[17] Table 3-4 identifies controlled human exposure studies included in the 2019 ISA and draft ISA Supplement that examine the potential for $PM_{2.5}$ exposures to alter markers of cardiovascular function and is ordered by exposure concentration. 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 3-4. 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/m³ $PM_{2.5}$ (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/m³ (unfiltered) vs 3.0 μg/m³ (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
Wyatt et al., 2020a *	Healthy young adults (18-35)	37.8 μg/m³ CAP vs 2.1 μg/m³ (filtered); 4h	Increased blood inflammatory markers; Inconsistent changes in HRV
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	64 μg/m³ CAP (lower exposure); 2 h	No significant change in blood markers of inflammation or oxidative stress
Huang et al., 2012	Healthy adults	90 μg/m³ CAP; 2 h	No significant changes in heart rate variability
Devlin et al., 2003	Healthy older adults	99 μg/m³ CAP [1]; 2 h	Decreased heart rate variability
Hazucha et al., 2013	Adult current and former smokers	109 μg/m³ CAP; 2 h	No significant changes in markers of inflammation or coagulation
Ghio et al., 2000	Healthy young adults	120 μg/m³ CAP; 2 h	Increased fibrinogen (coagulation)
Ghio et al., 2003	Healthy young adults	120 μg/m³ CAP; 2 h	Increased fibrinogen; no significant effect on markers of inflammation
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	140 μg/m³ CAP (higher exposure); 2 h	Increased blood inflammatory markers
Brook et al., 2009	Healthy adults	149 μg/m³ 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/m³ CAP; 2 h	Decreased anti-oxidant/anti-inflammatory capacity when baseline capacity was low
Sivagangabalan et al., 2011	Healthy adults	150 μg/m³ 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

* Study newly assessed in the draft ISA Supplement
[1] 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).

Most of the controlled human exposure studies in Table 3-4 exposed participants to average $PM_{2.5}$ 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 concentrations lower than 120 μg/m^3. Impaired vascular function, the effect

identified in the 2019 ISA as the most consistent across studies (U.S. EPA, 2019, 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 evaluated longer exposure durations (i.e., longer than 2 hours) and lower (i.e., near-ambient) $PM_{2.5}$ concentrations, with significant effects for 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). Wyatt et al. (2020a) adds to this limited evidence base of controlled human exposure studies conducted at near ambient concentrations. This study was a randomized double-blind crossover study in healthy young participants (18-35 years, n=21) who were subject to intermittent moderate exercise and found significant effects for some cardiovascular and (e.g., systematic inflammation markers, cardiac repolarization, and decreased pulmonary function) following 4-hour exposures to 37.8 $\mu g/m^3$. The higher ventilation rate and longer exposure duration in this study compared to most controlled human exposure studies is roughly equivalent to a 2-hour exposure of 75-100 $\mu g/m^3$ of $PM_{2.5}$. Therefore, dosimetric consideration may explain the observed changes in lung function and inflammation in young healthy individuals. While this study provides evidence of some effects at lower $PM_{2.5}$ concentrations, overall there is inconsistent evidence for changes in lung function and inflammation in other controlled human exposure studies evaluated in the 2019 ISA (U.S. EPA, 2019, sections 5.1.7., 5.1.2.3.3, and 6.1.11.2.1; U.S. EPA, 2021a, section 3.3.1).

Taken together, these controlled human exposure studies support biological plausibility for the serious cardiovascular and respiratory effects that have been linked with ambient $PM_{2.5}$ exposures and seen in epidemiologic studies (U.S. EPA, 2019, Chapter 6). However, while these studies are important in establishing biological plausibility, it is unclear how the results alone and the importance of the effects observed in these studies, particularly in studies conducted at near-ambient $PM_{2.5}$ concentrations, should be interpreted with respect to adversity to public health. For example, impaired vascular function, the effect identified as most consistent across studies (U.S. EPA, 2019, section 6.1.13.2), can signal an intermediate effect along the potential biological pathways for cardiovascular effects following short-term exposure to $PM_{2.5}$ and show a role for exposure to $PM_{2.5}$ leading to potential worsening of IHD and heart failure followed potentially by ED visits, hospital admissions, or mortality (U.S. EPA, 2019, section 6.1 and Figure 6-1). However, just observing the occurrence of impaired vascular function alone does not clearly suggest an adverse health outcome. Additionally, associated judgments regarding adversity or health significance of measurable physiological responses to air pollutants have been informed by guidance, criteria or interpretative statements developed within the public health community, including the American Thoracic Society (ATS) and the European Respiratory Society (ERS), which cooperatively updated the ATS 2000 statement *What Constitutes an*

Adverse Health Effect of Air Pollution (ATS, 2000) with new scientific findings, including the evidence related to air pollution and the cardiovascular system (Thurston et al., 2017).[18] With regard to vascular function, the ATS/ERS statement considers the adversity of both chronic and acute reductions in endothelial function. While the ATS/ERS statement concluded that chronic endothelial and vascular dysfunction can be judged to be a biomarker of an adverse health effect from air pollution, they also conclude that "The health relevance of acute reductions in endothelial function induced by air pollution is less certain" (Thurston et al., 2017). This is particularly informative to our consideration of the controlled human exposure studies which are short-term in nature (i.e., ranging from 2- to 5-hours), including those studies that are conducted at near-ambient $PM_{2.5}$ concentrations.

Nonetheless, we note the findings in several of these controlled human exposure studies conducted at near-ambient $PM_{2.5}$ concentrations and the potential of these studies to provide some insight into what these controlled human exposure studies may indicate regarding short-term exposure to peak $PM_{2.5}$ concentrations and how those relate to ambient $PM_{2.5}$ concentrations in areas that meet the primary $PM_{2.5}$ standards. As such, we focus on 2-hour exposures (the exposure window most often utilized) and 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 2-19 (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 2017-2019. At sites meeting the current primary $PM_{2.5}$ standards, most 2-hour concentrations are below 10 $\mu g/m^3$, and almost never exceed 30 $\mu g/m^3$. 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 2-19), 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 62 $\mu g/m^3$ during the warm season). Most of the sites measuring these very

[18] The ATS/ERS described its 2017 statement as one "intended to provide guidance to policymakers, clinicians and public health professionals, as well as others who interpret the scientific evidence on the health effects of air pollution for risk management purposes" and further notes that "considerations as to what constitutes an adverse health effect, in order to provide guidance to researchers and policymakers when new health effects markers or health outcome associations might be reported in future." The most recent policy statement by the ATS, which once again broadens its discussion of effects, responses and biomarkers to reflect the expansion of scientific research in these areas, reiterates that concept, conveying that it does not offer "strict rules or numerical criteria, but rather proposes considerations to be weighed in setting boundaries between adverse and nonadverse health effects," providing a general framework for interpreting evidence that proposes a "set of considerations that can be applied in forming judgments" for this context (Thurston et al., 2017).

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 2-19), the extreme upper end of the distribution is reduced (i.e., 99.9th percentile of 2-hour concentrations is 55 μg/m^3).[19] Given these results, we conclude that $PM_{2.5}$ exposure concentrations evaluated in most of these controlled human exposure studies are well-above the 2-hour ambient $PM_{2.5}$ concentrations typically measured in locations meeting the current primary standards.

Animal Toxicology Studies

The 2019 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 2019 ISA have generally examined short-term exposures to $PM_{2.5}$ concentrations from 100 to >1,000 μg/m^3 and long-term exposures to concentrations from 66 to >400 μg/m^3 (e.g., see U.S. EPA, 2019, Table 1-2). Two exceptions are a study reporting impaired lung development following long-term exposures (i.e., 24 hours per day for several months prenatally and postnatally) to an average $PM_{2.5}$ concentration of 16.8 μg/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 μg/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, 2019, Table 1-2), though still above the ambient concentrations likely to occur in areas meeting the current primary standards. However, noting uncertainty in extrapolating the effects seen in animals, and the $PM_{2.5}$ exposures and doses that cause those effects to human populations, animal toxicology studies are of limited utility in informing decisions on the public health protection provided by the current or alternative primary $PM_{2.5}$ standards. As such, the animal toxicological studies are most useful in providing further evidence to support the biological mechanisms and plausibility of various adverse effects.

[19] Similar analyses of 4-hour and 5-hour $PM_{2.5}$ concentrations are presented in Appendix A, Figure A-2 and Figure A-3, respectively.

3.3.3.2 Ambient PM Concentrations in Locations of Epidemiologic Studies

As summarized in section 3.1.1 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 2019 ISA's "causal" and "likely to be causal" determinations and provide further support for these associations as assessed in the draft ISA Supplement. 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. As noted in section 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 can lead to the reported effects. Rather, health effects can occur over the entire distribution 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, 2019, section 1.5.3). To address these issues, we consider the following question:

- **To what extent does the evidence from epidemiologic studies that have evaluated associations with mortality or morbidity provide support for adverse effects occurring following $PM_{2.5}$ exposures?**

In the absence of discernible thresholds, we consider what information can be provided from epidemiologic studies. In particular, to address the question above, we consider the study-reported ambient $PM_{2.5}$ concentrations reflecting estimated exposure with a focus on the middle portion of the $PM_{2.5}$ air quality distribution, which provides the strongest support for reported health effect associations. The section below discusses the key epidemiologic studies available in this reconsideration and observations from these studies to inform preliminary conclusions on the primary $PM_{2.5}$ standards.

3.3.3.2.1 $PM_{2.5}$ 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. In previous reviews, the decision framework used to judge adequacy of the existing $PM_{2.5}$ standards, and what levels of any potential alternative standards should be considered, placed significant weight on epidemiologic studies that assessed associations between $PM_{2.5}$ exposure and health outcomes that were most strongly supported by the body of scientific evidence. In doing so, the decision framework recognized that while there is no specific point in the air quality distribution of any epidemiologic study that represents a "bright line" at and above which effects have been observed and below which effects have not been observed, there is significantly greater confidence in the magnitude and significance of

observed associations for the part of the air quality distribution corresponding to where the bulk of the health events in each study have been observed, generally at or around the mean concentration. 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 3.3.1.1, 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, 2019, Figure 6-26). For the years 1974 to 2009, Lepeule et al. (2012) report a positive and statistically significant association between estimated long-term $PM_{2.5}$ 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 $\mu g/m^3$). 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).

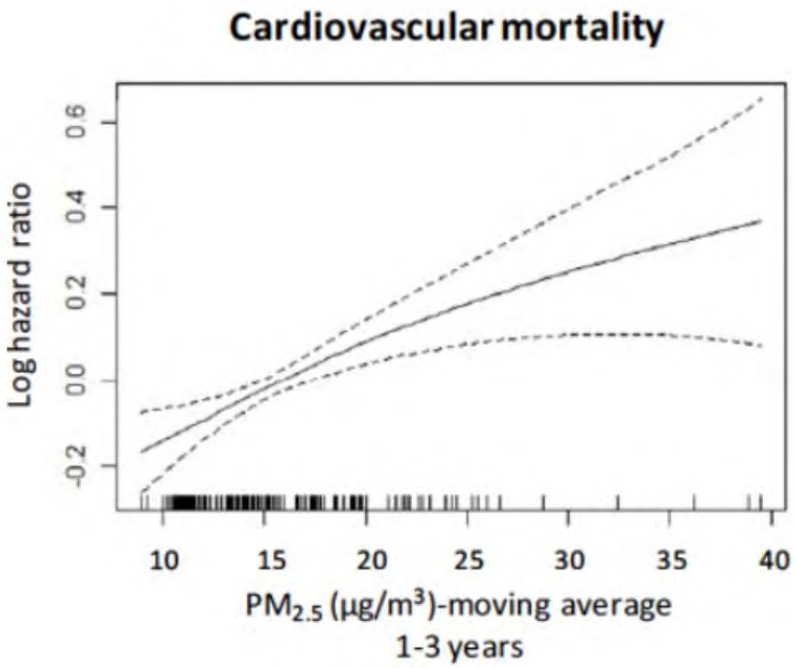


Figure 3-2. Estimated concentration-response function and 95% confidence intervals between $PM_{2.5}$ and cardiovascular mortality in the Six Cities Study (1974-2009) (from Lepeule et al., 2012, supplemental material, figure 1; Figure 6-26 in U.S. EPA, 2019).

Similar to the information presented in Figure 3-2, other studies have also reported that confidence intervals around concentration-response functions are relatively narrow at $PM_{2.5}$ concentrations around the overall mean concentrations reported by those studies, likely reflecting high data density in the middle portions of the distributions (e.g., Crouse et al., 2015; Villeneuve et al., 2015; Shi et al., 2016 as discussed in U.S. EPA, 2019, section 11.2.4). Thus, consistent with the approaches in the 2012 and 2020 reviews (78 FR 3161, January 15, 2013; U.S. EPA, 2011, sections 2.1.3 and 2.3.4.1; 85 FR 82716-82717, December 18, 2020; U.S. EPA, 2020, sections 3.1.2 and 3.2.3), in this reconsideration, we use study-reported means (or medians) of daily and annual average $PM_{2.5}$ concentrations over the entire study period as proxies for the middle portions of the air quality distributions, over which studies generally provide strong support for reported associations. As described further below, when considering the $PM_{2.5}$ air quality distributions in epidemiologic studies in this section, we focus on $PM_{2.5}$ concentrations around these overall means (including concentrations somewhat below the means (e.g., 25^{th} and 10^{th} percentiles)).

In evaluating the overall study-reported means, the focus is on the form, averaging time and level of the current annual $PM_{2.5}$ standard. Consistent with the approaches used in the 2012 and 2020 reviews (78 FR 3161-3162, January 15, 2013; 85 FR 82716-82717, December 18, 2020), this is because the annual standard has been utilized as the primary means of providing

public health protection against the bulk of the distribution of short- and long-term $PM_{2.5}$ exposures. Thus, the evaluation of the study-reported mean concentrations from key epidemiologic studies lends itself best to evaluating the adequacy of the annual $PM_{2.5}$ standard (rather than the 24-hour standard with its 98th percentile form). This is true for the study-reported means from both long-term and short-term epidemiologic studies, recognizing that 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 from short-term studies reflect long-term averages of 24-hour $PM_{2.5}$ exposure estimates. In this way, our examination aims to evaluate the protection provided by the annual $PM_{2.5}$ standard against the exposures that provide strong support for associations with mortality and morbidity in key epidemiologic studies. We note that the protection provided by the annual standard is evaluated in partnership with that provided by the 24-hour standard, with its 98th percentile form, which aims to provide 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.

As in past reviews, application of a decision framework based on assessing means of key epidemiologic studies must also consider how the study means were computed and how these values compare to the annual standard metric (including the level, averaging time and form) and the use of the monitor with the highest $PM_{2.5}$ design value in an area for compliance. In the 2012 review, it was recognized that the key epidemiologic studies computed the study mean using an average across monitor-based $PM_{2.5}$ concentrations. As such, the Agency noted that this decision framework applied an approach of using maximum monitor concentrations to determine compliance with the standard, while selecting the standard level based on consideration of composite monitor concentrations. Further, the Agency included analyses (Hassett-Sipple et al., 2010; Frank, 2012) that examined the differences in these two metrics (i.e., maximum monitor concentrations and composite monitor concentrations) across the U.S. and in areas included in the key epidemiologic studies and found that the maximum design value in an area was generally higher than the monitor average across that area, with that amount varying based on location and concentration. This information was taken into account in the Administrator's final decision in selecting a level for the primary annual $PM_{2.5}$ standard the 2012 review and discussed more specifically in her considerations on adequate margin of safety.

As an initial matter, in this reconsideration, we note that there are a substantial number of different types of studies available since the 2012 review, included in both the 2019 ISA and the draft ISA Supplement. While the key epidemiologic studies in the 2012 review were all monitor-based studies, the newer studies include hybrid modeling approaches which have emerged in the epidemiologic literature as an alternative to approaches that only use ground-based monitors to

estimate exposure. As assessed in the 2019 ISA and draft ISA Supplement, a substantial number of epidemiologic studies used hybrid model-based methods in evaluating associations between $PM_{2.5}$ exposure and health effects. Hybrid model-based studies employ various fusion techniques that combine ground-based monitored data with air quality modeled estimates and/or information from satellites to estimate $PM_{2.5}$ exposures. While these studies provide a broader estimation of $PM_{2.5}$ exposures compared to monitor-based studies (i.e., $PM_{2.5}$ concentrations are estimated in areas without monitors), the hybrid modeling approaches result in study-reported means that are more difficult to relate to the annual standard metric and to the use of maximum monitor design values to assess compliance. In addition, to further complicate the comparison, when looking across these studies, we find variations in how exposure is estimated between such studies, and thus, how the study means are calculated. Two important variations across studies include: (1) variability in spatial scale used (i.e., averages computed across the national (or large portions of the country) versus a focus on only CBSAs) and (2) variability in exposure assignment methods (i.e., averaging across all grid cells, averaging across a scaled up area like a ZIP code, and population weighting). Because of these differences, the application of any decision framework in considering the study-reported mean $PM_{2.5}$ concentrations, given the current state of the science, is more complicated than the approaches used in past reviews. In the sections that follow, we provide detailed analyses of the different air quality and exposure estimation methods in the used in the key epidemiologic studies and consider how those differences translate into comparisons between the mean $PM_{2.5}$ concentrations reported in the studies and the level of the primary annual $PM_{2.5}$ standard.

- **What are the epidemiologic studies assessed in the 2019 ISA and draft ISA Supplement that have the potential to be most informative in reaching preliminary conclusions on the primary $PM_{2.5}$ standards?**

To evaluate the $PM_{2.5}$ air quality distributions in key studies in this draft PA reconsideration, we first identify the epidemiologic studies assessed in the 2019 ISA and draft ISA Supplement that have the potential to be most informative in reaching preliminary conclusions on the primary $PM_{2.5}$ standards. As with 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 2019 ISA. We focus on the health effect associations that are determined in the 2019 ISA and draft ISA Supplement 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/multistate 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. 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.[20] We note that, while we consider studies from Canada in our evaluation of the epidemiologic evidence, there are considerable differences between studies conducted in the U.S. and in Canada, particularly those related to population densities, $PM_{2.5}$ concentration gradients, and source distributions in the two countries. As a result, while we consider the information from studies conducted in Canada, we generally place a greater emphasis on U.S.-based studies.

Figure 3-3 to Figure 3-6 below summarize information from U.S. and Canadian studies that are assessed in the 2019 ISA and draft ISA Supplement and that meet these criteria. For each study, Figure 3-3 to Figure 3-6 present the cohort and/or geographic area examined, the approach used to estimate $PM_{2.5}$ exposures (i.e., monitored or predicted with hybrid modeling methods[21]), 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[22] 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.

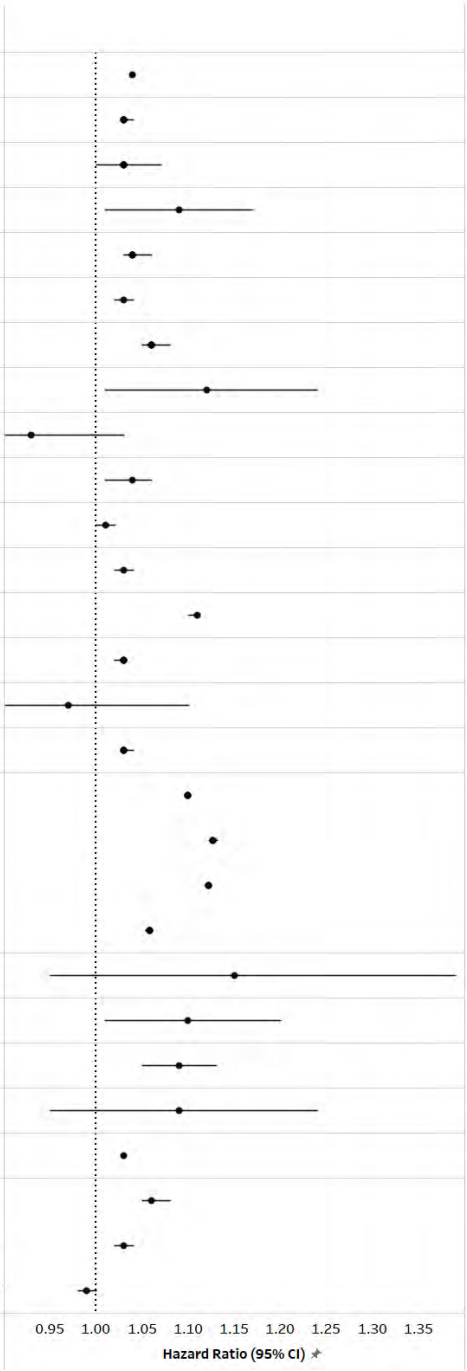
[20] This emphasis on studies conducted in the U.S. or Canada is consistent with the approach in the 2012 and 2020 reviews of the PM NAAQS (U.S. EPA, 2011, section 2.1.3; U.S. EPA, 2020, section 3.2.3.2.1).

[21] 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.

[22] 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).

All-cause mortality (U.S.)

Exposure Proxy	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	Di et al., 2017b	Medicare	2000-2012	2000-2012	11.0 (5th and 95th: 6.21- 15.64)
	Dominici et al., 2019	Medicare	2000-2012	2000-2012	11.0 (NR)
	Elliott et al., 2020	Nurses Health	1988-2008	1988-2007	13.7 (NR)
	Hart et al., 2015	Nurses Health	2000-2006	1999-2006	12.0 (NR)
	Lefler et al., 2019	NHIS	1987-2015	1988-2015	10.7 (NR)
	Pope et al., 2015	ACS CPS-II	1982-2004	1999-2004	12.6 (1.0–28.0)
	Pope et al., 2019	NHIS	1986-2015	1999-2015	10.7 (2.5-19.2)
	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)
	Wang et al., 2020	Medicare	2000-2008	2000-2008	10.3 (NR)
	Weichenthal et al., 2014	Ag Health	1993-2009	2001-2006	Iowa: 8.8; North Carolina: 11.1 (NR)
	Wu et al., 2020	Medicare	2000-2016	2000-2016	9.8 (NR)
Monitor	Eum et al., 2018	Medicare	2000-2012	2000-2012	Overall: 11.7 (NR)
					Central region: 9.9 (NR)
					Eastern region: 12.3 (NR)
					Western region: 11.5 (NR)
	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: 9.0-13.0) (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)



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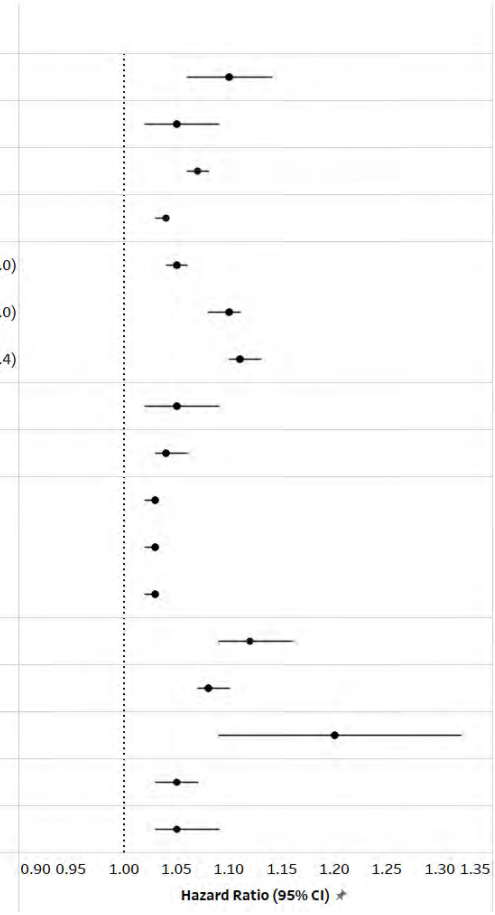
All-cause mortality (Canada)

Exposure Proxy	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)
Modelled	Cakmak et al., 2018	CanCHEC	1991-2011	1984-2011	6.5 (1.2, 24.1)
	Christidis et al., 2019	mCHHS	2000-2016	1998-2015	5.9 (0.4-17.2)
	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)
	Crouse et al., 2019	CanCHEC	2001-2011	1998-2010	7.2 (1-year 1-km mean) (0.0-20.0)
					7.4 (3-year 1-km mean) (0.0-20.0)
					8.0 (8-year 1-km mean) (0.3-18.4)
	Erickson et al., 2020	CanCHEC (Immigrant)	2001-2016	1998-2016	9.3 (Immigrant) (NR)
		CanCHEC (Non-immigrant)	2001-2016	1998-2016	7.5 (Non-Immigrant) (NR)
	Pappin et al., 2019	CanCHEC	1991-2016	1988-2015	7.9 (Year 1991) (0.4-20.0)
					7.2 (Year 1996) (0.4-20.0)
					6.7 (Year 2001) (0.4-18.5)
	Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)
	Pinault et al., 2017	CanCHEC	1991-2011	1998-2011	7.4 (<0.01-20.0)
	Zhang et al., 2021	Ontario Health Study	2009-2017	2000-2016	7.8 (NR)
Monitor	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)



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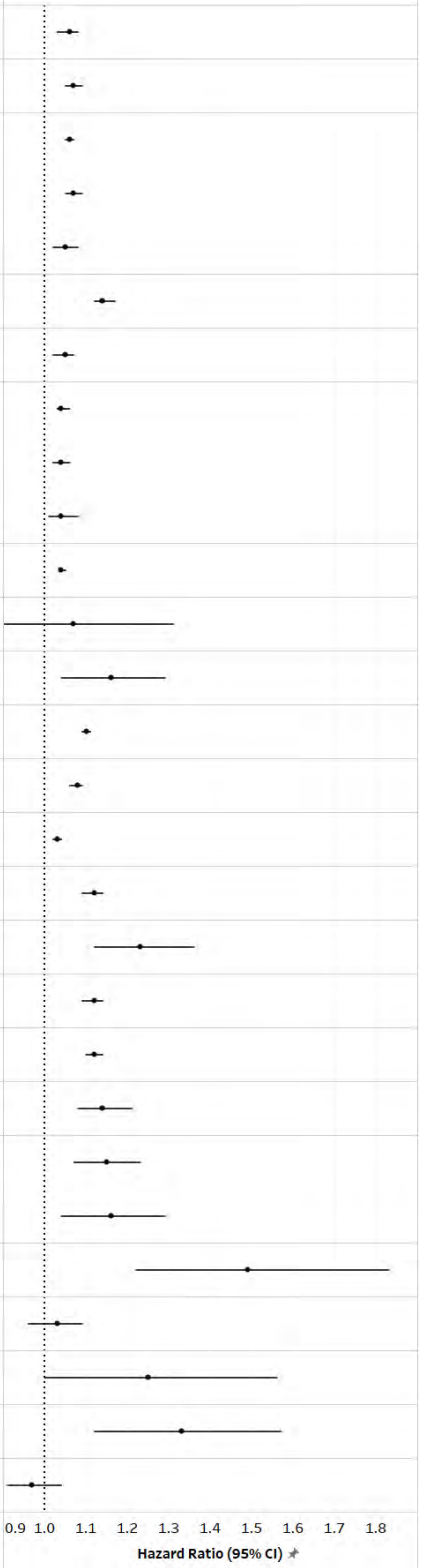
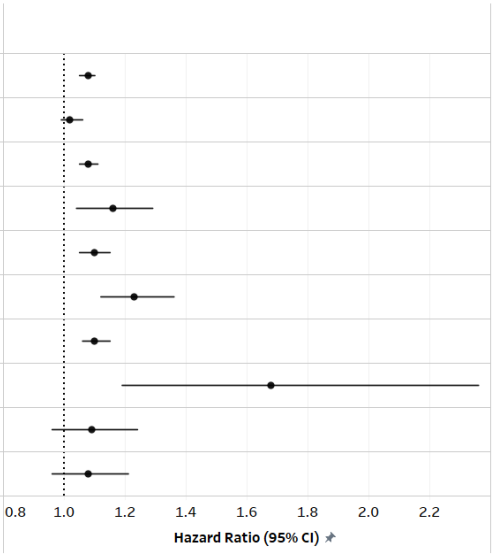
CVD mortality

Exposure Proxy	Country	Citation	Cohort	Health Data	Air Quality Data	Reported PM Mean (Range)(ug/m3)	Health Outcome
Modelled	U.S.	Hayes et al., 2020	NIH-AARP	1995/96-2011	1980-2010	NR (Median: 13.3) (2.9-28.0)	CVD mortality Age 50-71
		Jerrett et al., 2016	ACS CPS-II	1982-2004	2002-2004	12.0 (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+
		Pope et al., 2019	NHIS	1986-2015	1999-2015	10.7 (2.5-19.2)	CVD mortality Age 18-84
		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+
		Wang et al., 2020	Medicare	2000-2008	2000-2008	10.3 (NR)	CVD mortality Age 65-120
		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+
		Chen et al., 2020	ONPHEC	2001-2016	2000-2016	8.6 (NR)	CVD mortality Age 35-85
		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
		Crouse et al., 2019	CanCHEC	2001-2011	1998-2010	7.4 (3-year 1-km mean) (0.0-20.0)	CVD mortality Age 25-89
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)	CVD mortality Age 25-90
		Pinault et al., 2017	CanCHEC	1991-2011	1998-2011	7.4 (<0.01-20.0)	CVD mortality Age 25-89
		Pinault et al., 2018	CanCHEC	2001-2011	1998-2012	7.4 (NR)	CVD mortality Age 25-90
			mCHHS	2001-2008	1998-2013	6.4 (NR)	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
		Zhang et al., 2021	Ontario Health Study	2009-2017	2000-2016	7.8 (NR)	CVD mortality Age 30+
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



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)
		Crouse et al., 2019	CanCHEC	2001-2011	1998-2010	7.4 (3-year 1-km mean) (0.0-20.0)
		Pinault et al., 2016	CCHS	2000-2011	1998-2011	6.3 (1.0-13.0)
		Pinault et al., 2017	CanCHEC	1991-2011	1998-2011	7.4 (<0.01-20.0)
		Zhang et al., 2021	Ontario Health Study	2009-2017	2000-2016	7.8 (NR)
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)

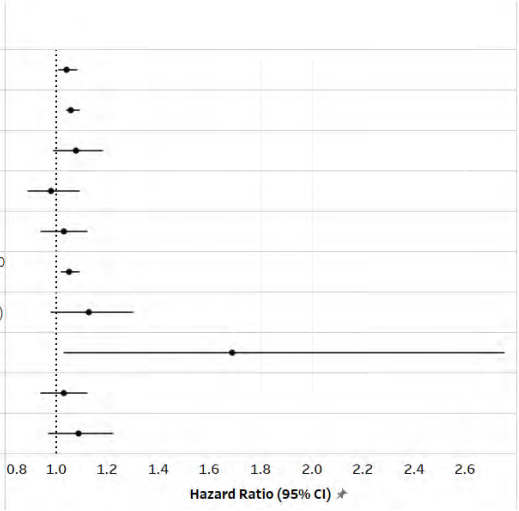
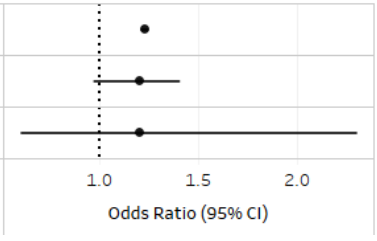


Figure 3-3. Epidemiologic studies examining associations between long-term $PM_{2.5}$ exposures and mortality.

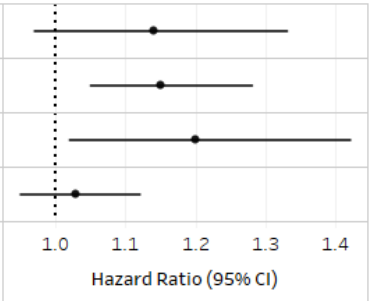
Asthma incidence

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)



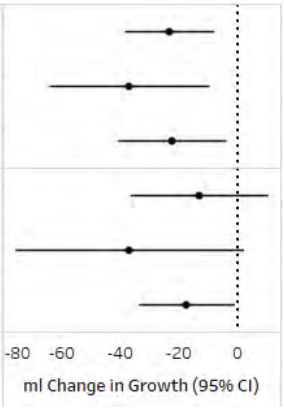
Lung cancer incidence

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

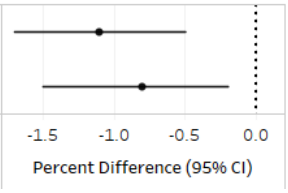
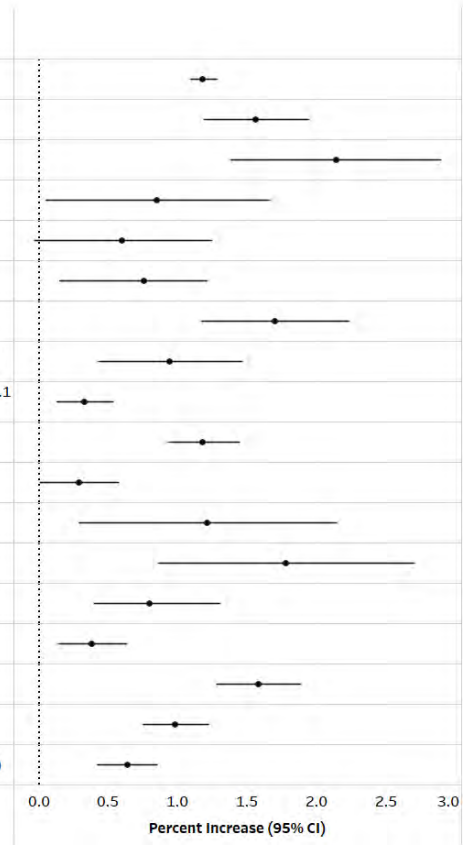


Figure 3-4. Epidemiologic studies examining associations between long-term $PM_{2.5}$ exposures and morbidity.

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 (0.02- 86.2)
		Shi et al., 2016	Medicare	2003-2008	8.21 (0.8-20.22)
Monitor	Canada	Burnett et al., 2003	Statistics Canada	1986-1996	13.3 (NR)
		Burnett et al., 2004	Statistics Canada	1981-1999	12.8 (NR)
		Lavigne et al., 2018	Canadian Mortality Database	1998-2011	8.8 (<1-98.15)
		Liu et al., 2019	MCC	1986-2011	9.3 (NR)
		Shin et al., 2021	National Vital Statistics Database	1984-2012	8.0 (Warm season); 6.0 (Cold season) (NR)
	U.S.	Baxter et al., 2017	NCHS	2001-2005	Cluster Mean Range: 12.2-14.1 (NR)
		Dai et al., 2014	NCHS	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	2000–2005	13.6 (NR)
		Liu et al., 2019	MCC	1987-2006	12.4 (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)



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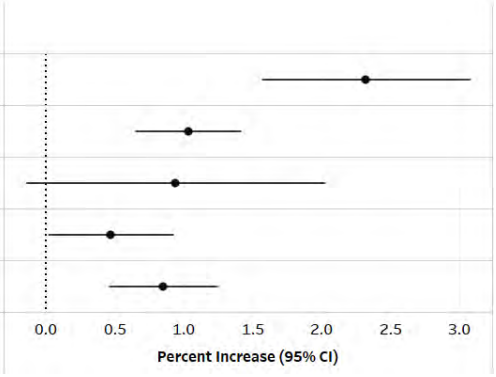
CVD mortality

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 (0.02- 86.2)
Monitor	U.S.	Dai et al., 2014	NCHS	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)



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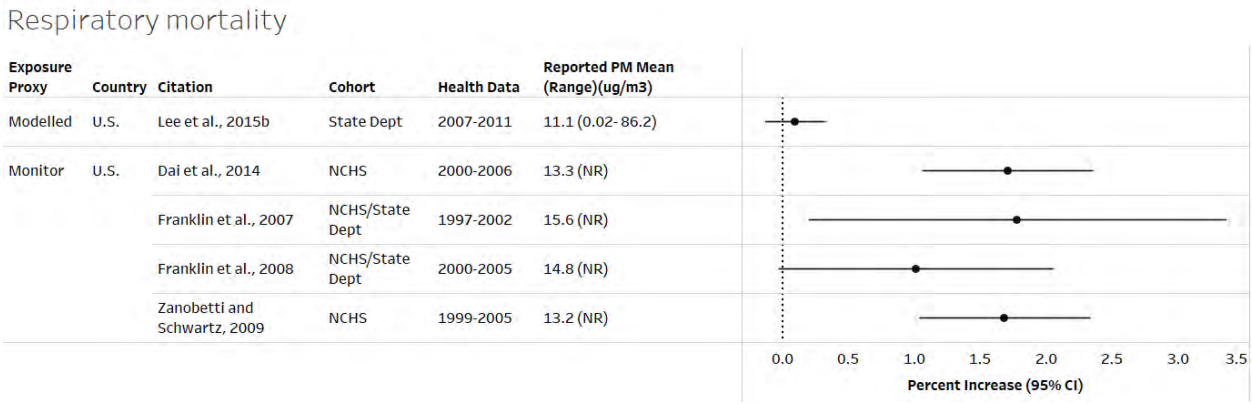
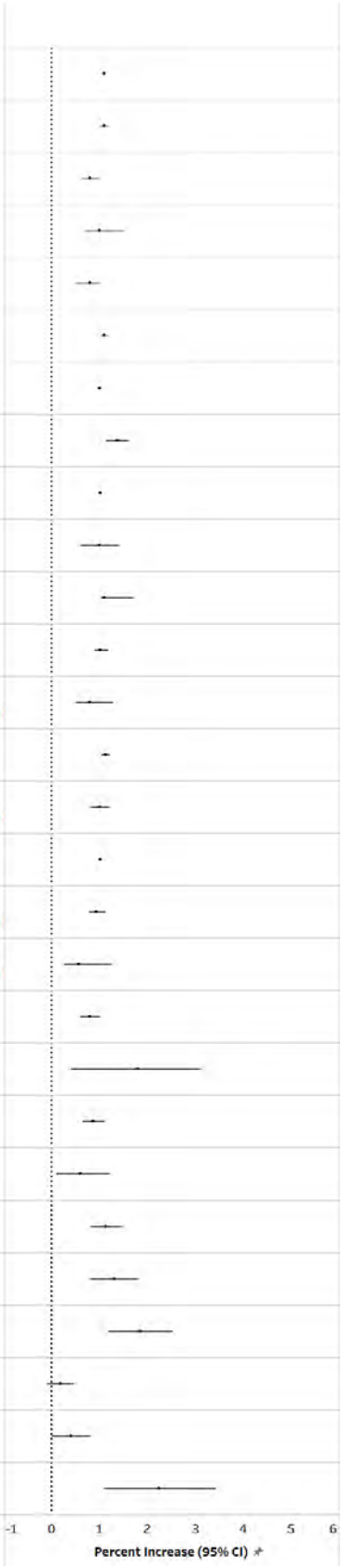


Figure 3-5. Epidemiologic studies examining associations between short-term $PM_{2.5}$ exposures and mortality.[23]

[23] 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 (U.S.)

Exposure Proxy	Endpoint	Health Outcome	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)
Modelled	CVD	All CVD (First HA) Age 18+	deSouza et al., 2021	Medicaid adults	2000-2012	11.5 (NR)
		All CVD Hospital re-admission	Wyatt et al., 2020b	USRDS haemodialysis patients	2008-2014	9.3 (0.05-155.16)
		CVD HA Age 65+	Bravo et al., 2017	Medicare	2002-2006	12.3 (NR)
			Kloog et al., 2012	Medicare	2000-2006	9.6 (0.01-72.59)
			Kloog et al., 2014	Medicare	2000-2006	11.9 (NR)
	CHF	CHF (First HA) Age 18+	deSouza et al., 2021	Medicaid adults	2000-2012	11.5 (NR)
		CHF ED	Krall et al., 2018	ED visit databases	2002-2008	Mean Range: 10.8-15.4 (NR)
		CHF Hospital re-admission	Wyatt et al., 2020b	USRDS haemodialysis patients	2008-2014	9.3 (0.05-155.16)
	IHD	IHD ED	Krall et al., 2018	ED visit databases	2002-2008	Mean Range: 10.8-15.4 (NR)
		IHD HA Age 65+	Kloog et al., 2014	Medicare	2000-2006	11.9 (NR)
	MI	MI (First HA) Age 18+	deSouza et al., 2021	Medicaid adults	2000-2012	11.5 (NR)
	Stroke	HS Incidence Ages 50-79	Sun et al., 2019	WHI (Post-menopausal women)	1993-2012	12.4 (Case day) (NR)
		HS Ages 53-88	Fisher et al., 2019	HPFS	1999-2010	12.9 (IS: 13.1, HS: 11.9, Undertermined: 13.7) (NR)
		IS (First HA) Age 18+	deSouza et al., 2021	Medicaid adults	2000-2012	11.5 (NR)
		IS Ages 53-88	Fisher et al., 2019	HPFS	1999-2010	12.9 (IS: 13.1, HS: 11.9, Undertermined: 13.7) (NR)
		Stroke ED	Krall et al., 2018	ED visit databases	2002-2008	Mean Range: 10.8-15.4 (NR)
		Total Stroke Ages 53-88	Fisher et al., 2019	HPFS	1999-2010	12.9 (IS: 13.1, HS: 11.9, Undertermined: 13.7) (NR)
		Undetermined stroke Ages 53-88	Fisher et al., 2019	HPFS	1999-2010	12.9 (IS: 13.1, HS: 11.9, Undertermined: 13.7) (NR)
Monitor	CVD	CVD HA Age 65+	Bell et al., 2008	Medicare	1999-2005	12.9 (NR)
			Bell et al., 2014	Medicare	2000-2004	14.0 (Median: 11.7) (NR)
			Bravo et al., 2017	Medicare	2002-2006	12.5 (NR)
			Peng et al., 2009	Medicare	2000-2006	NR (Median: 11.8) (NR)
	HF	Heart Failure HA Age 65+	Bell et al., 2015	Medicare	1999-2010	12.3 (6.4- 20.2)
			Dominici et al., 2006	Medicare	1999-2002	13.4 (NR)
			Zanobetti et al., 2009	Medicare	2000-2003	15.3 (NR)
	IHD	IHD HA Age 65+	Bell et al., 2015	Medicare	1999-2010	12.3 (6.4- 20.2)
			Dominici et al., 2006	Medicare	1999-2002	13.4 (NR)
	MI	MI HA Age 65+	Zanobetti et al., 2009	Medicare	2000-2003	15.3 (NR)



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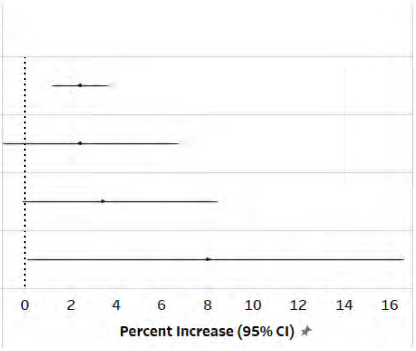
CVD morbidity (Canada)

Exposure Proxy	Endpoint	Health Outcome	Citation	Cohort	Health Data	Reported PM Mean (Range)(ug/m3)
Monitor	Angina	Angina ED	Szyszkowicz et al., 2009	Hospital Database	1992-2003	8.3 (NR)
	Angina/ MI	Angina/MI ED	Stieb et al., 2009	Hospital Database	1992-2003	8.2 (6.7-9.8)
	MI	MI ED	Weichenthal et al., 2016b	NACRS	2004–2011	6.9 (NR)
	HF	Heart Failure ED	Stieb et al., 2009	Hospital Database	1992-2003	8.2 (6.7-9.8)



Respiratory morbidity

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

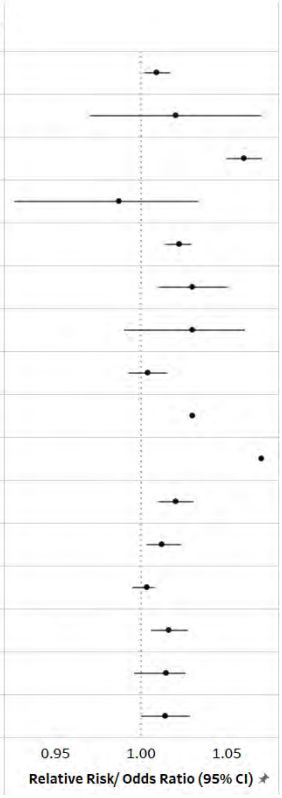


Figure 3-6. Epidemiologic studies examining associations between short-term $PM_{2.5}$ exposures and morbidity.

- **What are the key epidemiologic studies on which the draft PA should focus for informing preliminary conclusions regarding the current and potential alternative primary $PM_{2.5}$ standards? For these key epidemiologic studies, how were the mean $PM_{2.5}$ concentrations calculated?**

Based on the information in Figure 3-3 to Figure 3-6, key epidemiologic studies indicate generally positive and statistically significant associations between estimated $PM_{2.5}$ exposures (short- or long-term) and mortality or morbidity across a range of ambient $PM_{2.5}$ concentrations. Drawing from the multicity studies in Figure 3-3 to Figure 3-6, we identify the key epidemiologic studies most informative to our understanding to evaluate the $PM_{2.5}$ air quality distributions in key studies in this reconsideration. Key epidemiologic studies 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. For some studies of long-term $PM_{2.5}$ exposures, exposure is estimated from air quality data corresponding to only part of the study period, often including only the later years of the health data, and are not likely to reflect the full ranges of ambient $PM_{2.5}$ concentrations that contributed to reported associations.[24] While this approach can be reasonable in the context of an epidemiologic study that is 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. Therefore, we identify studies as key epidemiologic studies when the years of air quality data and health data overlap in their entirety.

Additionally, for studies that estimate $PM_{2.5}$ exposure using hybrid modeling approaches, we also consider the approach used to estimate $PM_{2.5}$ concentrations and the approach used to validate hybrid model predictions when determining those studies that we identify as key epidemiologic studies. Such studies are identified as those that use hybrid modeling approaches for which recent methods and models were used (e.g., recent versions and configurations of the air quality models); studies that are fused with $PM_{2.5}$ data from national monitoring networks (i.e., FRM/FEM data); and studies that reported a thorough model performance evaluation for core years of the study.[25] While numerous approaches to estimating $PM_{2.5}$ concentrations in hybrid modeling studies can be reasonable in the context of an epidemiologic study evaluating health effect associations with $PM_{2.5}$ exposures (e.g., in studies that use satellite data in fused surfaces), our interest is in utilizing the most up to date methods based on surfaces fused with

[24] The following studies do not have an overlap between the years of $PM_{2.5}$ air quality data and the years during which health effects are reported: Miller et al., 2007; Hart et al., 2011; Thurston et al., 2013; Weichenthal et al., 2014;; Pope et al., 2015; Villeneuve et al., 2015; Turner et al., 2016; Weichenthal et al., 2016a; Pinault et al., 2017; Parker et al., 2018; Pope et al., 2019; and Bevan et al., 2021.

[25] The following studies do not meet these criteria: Bravo et al., 2017, Crouse et al., 2015; Puett et al., 2009, Puett et al., 2011, Hystad et al., 2012; Hystad et al., 2013, Hayes et al., 2020; Elliott et al., 2020; Lefler et al., 2019;; Pappin et al., 2019; Cakmak et al., 2018; Fisher et al., 2019; Sun et al., 2019; McClure et al., 2017; Loop et al., 2018 ; and Honda et al., 2017.

monitored $PM_{2.5}$ data in order to inform the consideration of the PM NAAQS, as attainment of the standards is determined based on $PM_{2.5}$ monitoring data.

While all of the key epidemiologic studies in the 2012 review relied on ground-based monitoring information to characterize $PM_{2.5}$ exposure concentrations, as at the time of the 2020 review, a number of the more recent epidemiologic studies in Figure 3-3 to Figure 3-6 utilized various "hybrid modeling" approaches that include fusion techniques that combine ground-based monitored data with air quality modeled estimates and/or information from satellites to estimate $PM_{2.5}$ exposures. Furthermore, some studies use various mathematical approaches (e.g., population weighting, trimmed mean[26]) to compute the study-reported mean from the estimated $PM_{2.5}$ exposure concentrations. The fact that there are more and different techniques utilized to characterize exposure in the key epidemiologic studies in this reconsideration highlights the importance of understanding those techniques and how they compare to each other and to consider how those differences translate into comparisons between the mean $PM_{2.5}$ concentrations reported in the studies and the level of the primary annual $PM_{2.5}$ standard.

As noted above, study-reported mean concentrations in Figure 3-3 to Figure 3-6 were calculated using different methods. This is an important consideration when comparing mean concentrations across studies, as the methods used to estimate $PM_{2.5}$ concentrations can vary from traditional methods using monitoring data from ground-based monitors to those using more complex hybrid modeling approaches. Studies using hybrid modeling approaches aim to broaden the spatial coverage of estimated $PM_{2.5}$ concentrations by bringing in additional information to provide estimates in areas that do not have ground-based monitors (i.e., areas that are generally less densely populated and tend to have lower $PM_{2.5}$ concentrations). As such, the hybrid modeling approaches tend to broaden the areas captured in the exposure assessment, and in doing so, the studies that utilize these methods tend to report lower mean $PM_{2.5}$ concentrations than monitor-based approaches because they include more suburban and rural areas where concentrations are lower. Further, other aspects of the method used to calculate mean $PM_{2.5}$ concentrations can also have an impact on the study-reported mean concentration (i.e., population weighting, trim mean).

In those studies that use ground-based monitors alone to estimate long- or short-term $PM_{2.5}$ concentrations, approaches include: (1) $PM_{2.5}$ concentrations from a single monitor within a city/county; (2) average of $PM_{2.5}$ concentrations across all monitors within a city/county or other defined study area (e.g., CBSA); or (3) population-weighted averages of exposures. Once the study location average $PM_{2.5}$ concentration is calculated, the study-reported long-term

[26] A trimmed mean is a method of averaging that removes a small percentage of the largest and smallest values before calculating the mean.

average is derived by averaging daily/annual $PM_{2.5}$ concentrations across all study locations over the entire study period. Table 3-5 and Table 3-6 list the key U.S. and Canadian epidemiologic studies, respectively, that use ground-based monitors to estimate exposure, gives the reported study mean, and describes the method used to calculate the mean.

Table 3-5. Key U.S. Epidemiologic Studies: Monitor-Based Exposure

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Short-term Exposure Studies					
Bell et al., 2008 *	CVD HA (65+)	202 U.S. Counties (population ≥200,000)	Time-series study (MEDICARE enrollees)	Trimmed mean: 1999-2005 Daily $PM_{2.5}$ concentrations of 202 counties were averaged to calculate overall mean $PM_{2.5}$ exposure for the study location (all and region specific) and study period	12.9 (10^{th}: 9.8, 25^{th}: 11.5)
Bell et al., 2014	CVD, asthma, and COPD HA (65+)	4 Counties in MA and CT	Time-series study (MEDICARE enrollees)	2000-2004 Daily $PM_{2.5}$ concentrations for all four counties (three with single monitor and one with two monitors that used population weighted approach) were used to calculate the overall mean $PM_{2.5}$ for the study location and period	14.0
Bell et al., 2015	HF HA (65+)	213 U.S. Counties	Time-series study (MEDICARE enrollees)	1999-2010 Daily $PM_{2.5}$ concentrations of 213 counties were averaged to calculate overall and region-specific mean $PM_{2.5}$$PM_{2.5}$for the study location and period.	12.3
Bravo et al., 2017	CVD HA (65+)	418 U.S. Counties (population ≥50,000)	Time-series study (MEDICARE enrollees)	2002-2006 Daily $PM_{2.5}$ concentration of 418 counties were averaged to calculate overall mean $PM_{2.5}$ for the study location and period.	12.3
Dai et al., 2014	All-cause, CVD, and respiratory mortality	75 U.S. Cities (available daily mortality data and $PM_{2.5}$ data for at least 400 days 2000-2006)	Time-series study (NCHS)	2000-2006 Daily $PM_{2.5}$ concentration of 75 cities were averaged to calculate overall mean $PM_{2.5}$ for the study location and period	13.3
Dominici et al., 2006 *	HF and COPD HA (65+)	204 Urban U.S. Counties (population >200,000)	Time-series study (MEDICARE enrollees)	Trimmed mean: 1999-2002 Daily $PM_{2.5}$ concentrations for 204 US counties were averaged to calculate overall mean $PM_{2.5}$ concentration for the study regions and period.	13.4

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Franklin et al., 2007 *	All-cause, CVD, and respiratory mortality	27 U.S. Communities in Boston area (with $PM_{2.5}$ monitoring and daily mortality data for at least 2 years of 6-year study period 1997-2000)	Case-crossover study (NCHS)	1997-2000 Daily $PM_{2.5}$ concentrations (from monitors that are highly correlated in the counties and thus representing general population exposure) for 27 communities were averaged to calculate overall mean $PM_{2.5}$ concentration for the study location and period.	15.6 (10^{th}: 10.4, 25^{th}: 12.9)
Franklin et al., 2008 *	All-cause, CVD, and respiratory mortality	25 U.S. Communities for Boston area (with $PM_{2.5}$ monitoring and daily mortality data for at least 4 years of 6-year period 2000-2005)	Case-crossover study (NCHS)	2000-2005 Daily $PM_{2.5}$ concentrations (from monitors that are highly correlated in the counties and thus representing general population exposure) for 25 communities were averaged to calculate overall mean $PM_{2.5}$ concentration for the study location and period.	14.8
Klemm and Mason, 2003 *	All-cause mortality	Harvard Six-City study reanalysis	Time-series study	1979-1988 Daily $PM_{2.5}$ concentration of six cities were used to calculate overall mean $PM_{2.5}$ exposure for the study location (all and by study center) and period.	Median: 14.7: (25^{th}: 9.0)
Krall et al., 2013	All-cause mortality	72 Urban U.S. Communities	Time-series study (NCHS)	2000-2005 Daily $PM_{2.5}$ concentration (including only the source-oriented monitors representative of typical population exposures) of 72 urban communities were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period	13.6
Liu et al., 2019	All-cause and cause-specific mortality	107 U.S. Cities	Time-series study (MCC Collaborative Research Network)	1987-2006 Daily $PM_{2.5}$ concentration averaged across stations within each city was used to calculate an average 2-day moving average $PM_{2.5}$ concentrations for the city. These data were then used to calculate overall mean concentration for the study location and period.	12.4

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Ostro et al., 2016	Asthma and COPD ED	8 Metropolitan Areas/Counties in CA	Case-crossover study	2005-2009 Daily $PM_{2.5}$ concentrations for eight metropolitan counties were used to calculate an overall mean $PM_{2.5}$ concentration for the study location and period.	16.5
Peng et al., 2009b	CVD HA (65+)	119 U.S. Urban Counties>150,000 populations	Time-series study (MEDICARE enrollees)	2000-2006 Daily $PM_{2.5}$ concentrations for 119 counties were used to calculate an overall median $PM_{2.5}$ concentration for the study location and period.	Median: 11.9
Zanobetti et al., 2009	CVD, HF, MI HA (65+)	26 U.S. Cities	Time-series study (MEDICARE enrollees)	2000-2003 Daily average $PM_{2.5}$ data for each county was calculated using an algorithm that accounts for monitor-specific means and variances. Monitors that were not well correlated with other monitors were excluded.	15.3
Zanobetti and Schwartz, 2009 *	All-cause, CVD and respiratory mortality	112 U.S. Cities	Time-series study (NCHS)	1999-2005 Daily $PM_{2.5}$ concentrations (from monitors that are highly correlated in the counties and thus representing general population exposure) for 112 cities were averaged to calculate overall mean $PM_{2.5}$ concentration for the study location and period.	13.2 (10^{th}: 10.3, 25^{th}: 12.5)
Long-term Exposure Studies					
Eum et al., 2018	All-cause mortality	U.S. Geographic regions: "East" of the Mississippi River, "Center" between the Mississippi River and the Sierra Nevada mountain range, and "West" of the Sierra Nevada mountain range	Cohort study (MEDICARE enrollees)	2000-2012 Annual average $PM_{2.5}$ concentrations assigned to individuals living in zip codes with centroids within 6 miles of a valid monitor (monitors with daily measurements for at least 8 calendar years, with each year having 9+ months, and with 4+ daily measurements) were used to calculate overall mean $PM_{2.5}$ concentration for the study location (all and by study region) and study period.	Overall: 11.65 Central: 9.9 Eastern: 12.3 West: 11.5

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Gharibvand et al., 2016	Lung cancer incidence	U.S. Nationwide	Cohort study (AHSMOG-2 study)	2000-2001 Monthly $PM_{2.5}$ concentrations (calculated using at least 75% valid daily data) assigned to study participants based on residential address were used to calculate overall mean $PM_{2.5}$ for the study period.	12.9
Hart et al., 2015	All-cause mortality	U.S. Nationwide	Cohort study (Nurses' Health study)	2000-2012 Monthly $PM_{2.5}$ concentrations assigned to study participants based on the nearest monitor to residence locations were used to calculate overall mean for the study period	12.7
Kioumourtzoglou et al., 2016	All-cause mortality (65+)	207 U.S. cities	Cohort study (MEDICARE enrollees)	2000-2010 Annual $PM_{2.5}$ concentrations for 207 cities were averaged to calculate overall mean $PM_{2.5}$ exposure for the study location (all and region specific) and study period.	12.0
McConnell et al., 2010	Asthma Incidence	13 CA Communities	Cohort study (CHS)	2003-2004 Average annual $PM_{2.5}$ concentrations assigned to study participants based on their community of residence were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.	13.9
Zeger et al., 2008 *	All-cause mortality 65+	668 U.S. Urban Counties	Cohort Study of MEDICARE enrollees (MCAPS)	2000-2005 Average annual $PM_{2.5}$ concentrations of ZIP codes (for zip code centroids within 6 miles of a monitor and with >10 months of data per year) were used to calculate overall mean $PM_{2.5}$ exposure for the study location (all and by region) and the study period.	Central Region median: 10.7 Eastern Region median: 14.0 Western region median: 13.1
* Evaluated in 2012 review					

Table 3-6. Key Canadian Epidemiologic Studies: Monitor-Based Exposure

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Short-term Exposure Studies					
Burnett and Goldberg, 2003 *	All-cause mortality	8 Canadian cities	Time-series study	1986-1996 Daily $PM_{2.5}$ concentrations (day before the death) for 8 Canadian cities were averaged to get overall mean for the study area and period	13.3
Burnett et al., 2004 *	All-cause mortality	12 Canadian cities	Time-series study (data from Statistics Canada)	1981-1999 $PM_{2.5}$Daily $PM_{2.5}$ concentrations for 12 cities (calculated by averaging all monitors within each city) were used along with population information to calculate an overall population weighted $PM_{2.5}$ concentration for the study location and period	12.8
Lavigne et al., 2018	Non-accidental, CVD, and respiratory mortality	24 Canadian cities	Case-crossover study	1998-2011 Daily average $PM_{2.5}$ concentrations assigned to participants based on closest monitor(s) to participant's city of residence. Daily $PM_{2.5}$ concentrations in 24 Canadian cities were used to calculate overall mean $PM_{2.5}$ concentration over the study location and period.	8.8 (Median: 7.1)
Liu et al., 2019	All-cause and cause-specific mortality	25 Canadian cities	Time-series Study (MCC Collaborative Research Network)	1986-2011 $PM_{2.5}$ concentration averaged across stations within each city was used to calculate an average 2-day moving average $PM_{2.5}$ concentrations for the city. These data were then used to calculate overall mean concentration for the study location and period.	9.3
Stieb et al., 2009	Cardiac and respiratory ED visits	7 Canadian cities	Time-series study (Hospital cases)	1992-2003 Daily $PM_{2.5}$ concentrations of the cities (calculated by averaging all monitors within city) were used to calculate the overall mean $PM_{2.5}$ exposure for the study location (by site) and study period.	8.2 (10^{th}: 6.7, 25^{th}: 6.8)
Szyszkowicz, 2009	Angina ED	7 Canadian cities	Time-series study (Hospital cases)	1992-2003 Daily $PM_{2.5}$ concentrations of the cities (calculated by averaging all monitors within city) were used to calculate the overall mean $PM_{2.5}$ exposure for the study location (all and by cities) and study period.	8.3 (10^{th}: 6.4, 25^{th}: 6.5)

Citation	Health Endpoint	Geographic Area	Study Design	Years and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Weichenthal et al., 2016b	MI ED	16 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	2004-2011 Daily $PM_{2.5}$ concentrations in Ontario were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period	6.9
Weichenthal et al., 2016c	Asthma and COPD ED	15 cities in Ontario	Case-crossover design (cases extracted from NACRS database)	2004-2011 Daily $PM_{2.5}$ concentrations in Ontario were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period.	Asthma: 7.1 COPD: 7.1
Long-term Exposure Studies					
Crouse et al., 2012	All-cause mortality	11 Canadian Cities	Cohort study	1987-2001 Annual $PM_{2.5}$ concentrations from monitors and assigned to study participants based on the census division of the residence were used to calculate overall mean $PM_{2.5}$ for the study population and duration.	8.7
* Evaluated in 2012 review					

In the studies that use hybrid modeling approaches to estimate long- or short-term $PM_{2.5}$ concentrations, data can be incorporated from several different sources, including satellites and air quality models, in addition to ground-based monitors, as described in section 2.3.3 above. Compared to ground-based monitors alone, hybrid modeling methods have the potential to improve the characterization of $PM_{2.5}$ concentrations in areas with relatively sparse monitoring networks. These approaches also tend to have lower study-reported mean $PM_{2.5}$ concentrations since they often include estimates of $PM_{2.5}$ concentrations in less populated areas compared to those methods using only ground-based monitored. Studies that use hybrid modeling approaches can estimate $PM_{2.5}$ concentrations at different spatial resolutions, including at 1 km x 1 km grid cells (i.e., Di et al., 2017b and Di et al., 2017a), at 10 km x 10 km grid cells (i.e., Kloog et al., 2014), or at the census tract level (i.e., Bravo et al., 2017). Estimated $PM_{2.5}$ concentrations are then generally averaged up to a larger spatial resolution that corresponds to the spatial resolution for which health data exists (e.g., ZIP code level). These values are then averaged across all study locations at the larger spatial resolution (e.g., averaged across all ZIP codes in the study) over the study period, resulting in the study-reported mean 24-hour average or annual average $PM_{2.5}$ concentration. Table 3-7 and Table 3-8 list the key U.S. and Canadian epidemiologic studies, respectively, that use hybrid modeling approaches to estimate exposure and give the reported study mean and describes the method used to calculate the mean. Studies included in these tables 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. In addition, studies included in Table 3-7 and Table 3-8 are those for which recent methods and models were used (e.g., recent versions and configurations of the air quality models); studies that are fused with $PM_{2.5}$ data from national monitoring networks (i.e., FRM/FEM data); and studies that reported a thorough model performance evaluation for core years of the study.

Table 3-7. Key U.S. Epidemiologic Studies: Model-Based Exposure

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Short-term Exposure Studies					
deSouza et al., 2021	First CVD HA	Continental U.S.	Time-stratified case–crossover design (Medicaid Adults)	2000-2012 Ensemble model (integrating machine learning algorithms) Daily $PM_{2.5}$ estimates of all grid cells averaged at ZIP code were assigned to study participants based on the ZIP code of residence. Daily $PM_{2.5}$ concentration from case days were used to calculate overall case day mean $PM_{2.5}$ concentration for the study location and period.	11.5 (case days mean)
Di et al., 2017a	All-cause mortality (65+)	U.S. Nationwide	Case-crossover study (MEDICARE enrollees)	2000-2012 Artificial Neural Network (Hybrid method) Daily $PM_{2.5}$ concentrations for case and control days assigned to participants based on ZIP code of residence were used to calculate overall mean $PM_{2.5}$ for the study location and period.	11.6 (10^{th}: 4.7, 25^{th}: 6.7)
Kloog et al., 2012	CVD HA (65+)	New England Area with 6 U.S. States	Mixed study design (with time series and cohort components)	2000-2006 Spatiotemporal model Daily $PM_{2.5}$ concentration of all grids within the NE area for acute exposure (0 day lag) were used to calculate overall mean for short-term $PM_{2.5}$ exposure, for the study location and period.	9.6 (25^{th}: 6.4)

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Kloog et al., 2014	CVD and COPD HA (65+)	7 U.S. Mid-Atlantic States and D.C.	Case-crossover design (MEDICARE enrollees)	2000-2006 Spatiotemporal model 2-day moving average of $PM_{2.5}$ concentration of all grids within the mid-Atlantic states were used to calculate overall mean (all area and rural/urban areas) $PM_{2.5}$ exposure for the study location and period.	11.9 (25^{th}: 7.9)
Lee et al., 2015	All-cause, cardiovascular, respiratory mortality	3 U.S. Southeast States	Case-crossover design (Dept. of Pub Health data)	2007-2011 Spatiotemporal model $PM_{2.5}$ Daily $PM_{2.5}$ concentrations for ZIP codes (calculated as averages of all grids within ZIP code or the closest grid cell) within 3 SE states were averaged to calculate overall mean $PM_{2.5}$ concentration (all and by state).	11.1
Qiu et al., 2020	CVD HA	New England (VT, NH, CT, MA, RI and ME)	Case-crossover study applying causal modeling approach (MEDICARE)	2000-2012 Neural network (using machine learning algorithm) Daily $PM_{2.5}$ concentration at grid cells were averaged to estimate exposure at ZIP code level and were assigned to study participants based on ZIP code of residence. Case and control days $PM_{2.5}$ concentration were used to calculate overall mean $PM_{2.5}$ concentration (all, and separately for case and control days) for the study period.	10.0 (AMI: 10.13 CHF: 10.08 IS: 10.10)

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Shi et al., 2016	Total mortality (65+)	New England Area with 6 U.S. States	Open Cohort study (MEDICARE enrollees)	2003-2008 Predicted from 3-stage statistical model Lag01 $PM_{2.5}$ concentrations of all grid cells in the study area were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.	8.2 (25^{th}: 4.6)
Wyatt et al., 2020c	All-cause, CVD, RD 30-day hospital readmissions	530 U.S. counties	Cohort study (USRDS hemodialysis patients)	2008-2014 Spatiotemporal prediction model Daily $PM_{2.5}$ concentrations for grid cells were converted to population-weighted county-level $PM_{2.5}$ estimates using 2010 census tract population estimates. Participants were assigned daily $PM_{2.5}$ based on the county of their last dialysis visit. Daily estimates at county-level were then used to calculate overall $PM_{2.5}$ concentration for the study location and period.	9.29
Long-term Exposure Studies					
Di et al., 2017b	All-cause mortality (65+)	U.S. Nationwide	Cohort study (MEDICARE enrollees)	2000-2012 Artificial Neural Network (Hybrid method) Daily $PM_{2.5}$ concentrations for all ZIP codes were used to calculate overall mean $PM_{2.5}$ for the study location and period.	11.0 (10^{th}: 7.3, 25^{th}: 9.1)
Dominici et al., 2019	All-cause mortality (65+)	U.S. Nationwide	Cohort study (MEDICARE enrollees)	2000-2012 Artificial Neural Network (Hybrid method) Daily $PM_{2.5}$ concentrations for all ZIP codes were used to calculate overall mean $PM_{2.5}$ for the study location and period.	11.0

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Hart et al., 2015	All-cause mortality	U.S. Nationwide	Cohort study (Nurses' Health study)	2000-2012 Spatiotemporal model Monthly $PM_{2.5}$ concentrations assigned to study participants at residence locations were used to calculate overall mean for the study period.	12.0
Kloog et al., 2012	CVD HA (65+)	New England Area with 6 U.S. States	Mixed study design (with time series and cohort components)	2000-2006 Spatiotemporal model Daily $PM_{2.5}$ concentration of all grids within the NE area for chronic exposure (365 day moving average) were used to calculate overall mean for long-term $PM_{2.5}$ exposure, for the study location and period.	9.7 (25^{th}: 9.2)
Shi et al., 2016	Total mortality (65+)	New England Area with 6 U.S. States	Open Cohort study (MEDICARE enrollees)	2003-2008 Predicted from 3-stage statistical model Average annual $PM_{2.5}$ concentrations of all grid cells in the study area were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.	8.1 (25^{th}: 6.2)
Thurston et al., 2016	All-cause, CVD and respiratory mortality	6 U.S. States and 2 MSAs	Cohort study (NIH_AARP cohort)	2000-2008 Spatiotemporal model Average annual $PM_{2.5}$ concentrations of census tract estimates assigned to participants based on the census tract of residence used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.	12.2 Mean range: 2.9-28.0

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Wang et al., 2017	Total mortality (65+)	7 U.S. Southeast States	Cohort study (MEDICARE enrollees)	2000-2013 Three stage Hybrid model $PM_{2.5}$ Average annual $PM_{2.5}$ concentrations of ZIP code tabulation areas were calculated by averaging annual mean $PM_{2.5}$ concentration of all grids in the ZCTA and then used to calculate overall median $PM_{2.5}$ exposure for the study location (overall and by state), and period (overall and by year).	Median: 10.7 Range: 6.0-20.6 (25^{th}: 9.1)
Wang et al., 2020	Non-accidental cause-specific (respiratory, CVD, cancer) mortality	U.S. Nationwide	Cohort study (MEDICARE)	2000-2008 Spatiotemporal prediction model Daily $PM_{2.5}$ concentrations of grids were matched to study participants based on the grid point closest to their residential ZIP code centroid. The estimates were used to calculate overall annual mean $PM_{2.5}$ exposure for the study period.	10.3
Wu et al., 2019	All-cause mortality	New England (VT, NH, CT, MA, RI and ME); 2202 ZIP codes	Causal modeling study (MEDICARE)	2000-2012 Spatiotemporal Prediction model Daily $PM_{2.5}$ exposures determined at grid cells were aggregated using area-weighted average of $PM_{2.5}$ concentrations of all grid cells within the ZIP code and assigned to individuals based on their ZIP code of residence. Annual concentrations were used to calculate overall mean $PM_{2.5}$ concentration over the study period.	9.3 (Trimmed population: 9.4)

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Wu et al., 2020a	All-cause mortality	U.S. Nationwide	Cohort study (MEDICARE)	2000-2016 Ensemble model (integrating machine learning algorithms) Daily $PM_{2.5}$ concentration at grid cells whose centroids were inside the ZIP code boundary were averaged for each year and assigned to participants based on the ZIP code of residence. These data were used to calculate overall mean $PM_{2.5}$ concentration for the study period.$PM_{2.5}PM_{2.5}$	9.8 (<12 ug/m3: 8.4)
[1] None of the studies presented in this table were evaluated in the 2012 review.					

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2 **Table 3-8. Key Canadian Epidemiologic Studies: Model-Based Exposure**

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) µg/m^3
Long-term Exposure Studies					
Bai et al., 2019	CHF and AMI incidence	Ontario	Cohort study (ONPHEC)	1998-2012 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual estimates of $PM_{2.5}$ concentrations assigned to participants based on postal code of residence used to calculate 3-year moving average $PM_{2.5}$ concentration for each year of follow-up in the study. The 3-year moving averages for study participants at the baseline residence location was used to calculate overall mean $PM_{2.5}$ concentration at the beginning of the follow-up period in 2001.	9.6 (25th: 7.9)

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Chen et al., 2020	CVD mortality	Ontario	Cohort study (ONPHEC)	2000-2016 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual estimates of $PM_{2.5}$ concentrations were assigned to participants based on postal code of residence. Annual $PM_{2.5}$ concentrations in the Ontario region were then used to calculate overall mean $PM_{2.5}$ concentration for the study location and period.	8.61
Christidis et al., 2019	Non-accidental mortality	Canada Nationwide	Cohort study (mCHHS)	1998-2015 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 3-year moving average based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period.	5.9 (Median: 5.5; 25^{th}: 4.3)
Crouse et al., 2019	Non-accidental, CVD, respiratory mortality and lung cancer	Canada Nationwide	Cohort study (CanCHEC)	1998-2010 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate moving average at various temporal and spatial scales based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period at various temporal and spatial scales.	1- year in 1 km: Mean: 7.2, 3-year in 1 km: Mean: 7.4, 8-year in 1 km: Mean: 8.0

Citation[1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Erickson et al., 2020	Non-accidental, CVD, and respiratory mortality and cancer	Canada Nationwide	Cohort study (CanCHEC)	1998-2016 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 3-year moving average based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period by immigrant status and duration in Canada.	Non-immigrant: 7.5 Immigrant: 9.3 Pre-1971: 9.1 1971-1980: 9.3 1981-1990: 9.5 1991-2001: 9.7
Erickson et al., 2020	All-cause, CVD, respiratory, and lung cancer mortality	Canada Nationwide	Cohort study (CCHS)	1998-2012 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 3-year moving average based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period.	6.3
Pinault et al., 2018	CVD mortality	Canada Nationwide	Cohort study (CanCHEC, mCHHS)	1998-2012 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 3-year moving average based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period.	CanCHEC: 7.4 mCHHS: 6.4

Citation [1]	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean $PM_{2.5}$ Concentrations	Reported Mean (other percentiles) $\mu g/m^3$
Shin et al., 2019	AF and Stroke (1st HA)	Ontario	Cohort study (ONPHEC)	1998-2012 Fused surface (AOD, GEOS-Chem & geographically weighted regression) Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 5-year moving average based on the location and year of follow-up. The average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the study period.	9.8 (25th: 8)
Zhang et al., 2021	Non-accidental, CVD, and respiratory mortality	Ontario	Cohort study (Ontario Health Study)	Modeled from AOD satellite retrievals 2000-2016 Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year and 5-year moving averages based on the location and year of follow-up. The 5-year average $PM_{2.5}$ concentrations were then used to calculate overall mean $PM_{2.5}$ concentration for the baseline year.	Baseline: 7.8 (Median: 8.0; 25th: 6.7)

[1] None of the studies presented in this table were evaluated in the 2012 review.

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As noted above, the key epidemiologic studies use differing approaches to estimate mean $PM_{2.5}$ concentrations. Approaches differ not only between monitor-based studies and model-based studies, but also between studies using the same types of air quality information. It is important to recognize the differences between the techniques used for estimating mean $PM_{2.5}$ concentrations in epidemiologic studies, in particular when comparing the results across the studies and considering what the study reported means represent and how that information informs our consideration of the form, averaging time and level of the current annual $PM_{2.5}$ standard. To further understand these differences, we seek to answer the following question:

- **How can the approaches used in key epidemiologic studies to estimate exposure affect the study-reported mean $PM_{2.5}$ concentrations? How do these approaches and the resulting means compare to one another?**

In answering this question, we first utilize a simplified example to show differences in the mean concentrations depending on the methods used to estimate exposure. In Figure 3-7 below, we exhibit the state of Georgia and the CBSA of Atlanta-Sandy Springs-Roswell. In this Figure, the gradient of $PM_{2.5}$ concentrations are shown for 1 km x 1 km grid cells using one of the hybrid approaches described in more detail in Chapter 2, referred to as the DI2019[27] hybrid approach, from 2014-2016, as well as the monitor locations within the Atlanta-Sandy Springs-Roswell CBSA and their annual $PM_{2.5}$ design values for 2016. Using these data, several metrics were calculated and shown in Table 3-9 below. For all monitors within the CBSA, the average $PM_{2.5}$ concentration is 9.3 $\mu g/m^3$, while the design value (based on the highest monitored $PM_{2.5}$ concentration in the area) is 10.4 $\mu g/m^3$. This comparison helps to illustrate the fact that composite monitor values tend to be somewhat lower than the highest area monitor values, consistent with the key points made in the 2012 review. This example also communicates how monitors are sited to represent the higher concentrations within the area and that the area's annual design value, which is used for compliance with the standard, is calculated based on the highest monitor in the area.

Next, we evaluate the average estimated $PM_{2.5}$ concentrations from 2014-2016 using the DI2019 hybrid approach and calculate: (1) the average concentration across the entire state; (2) the population weighted average across the entire state; (3) average concentration across the CBSA; and (4) the population weighted average across the CBSA. In doing this, we have focused on using some of the main approaches used in epidemiologic studies to compute study means. At the urban level (e.g., Atlanta-Sandy Springs-Roswell CBSA), the average $PM_{2.5}$ concentration when taking the mean of all grid cells is 9.2 $\mu g/m^3$, whereas the population-

[27] As discussed above in section 2.3.3.2.4, DI2019 refers to estimated $PM_{2.5}$ concentrations from a hybrid modeling approach developed by Di et al. (2019b), which estimates Nationwide $PM_{2.5}$ concentrations from 2000-2016.

weighted mean is 9.6 µg/m^3. Across Georgia, the average $PM_{2.5}$ concentration using the hybrid approach is 8.3 µg/m^3, lower than the population-weighted statewide average of 9.1 µg/m^3. While this is a simple example evaluated in just one state and one CBSA, it suggests that the lowest mean values tend to result from the approaches that use concentrations from all or most grid cells, both urban and rural, across the study area to compute the mean. Higher mean values are observed when the approach focuses on the urban areas alone or when the approach incorporates population weighting. Overall, this example suggests that the means from studies using hybrid modeling approaches are generally lower than the means from monitor-based approaches, and both are lower than the annual design values for the same area. Population-weighting tends to increase the calculated mean, likely because more densely populated areas also tend to have higher $PM_{2.5}$ concentrations. Table 3-9shows how the different approaches affect mean concentration estimates for the example discussed above. Note that while the statewide average using the hybrid approach is quite a bit lower than the mean from either the monitor-based approach or the Atlanta-only hybrid approach, population-weighting the statewide average brings the value closer to the other approaches.

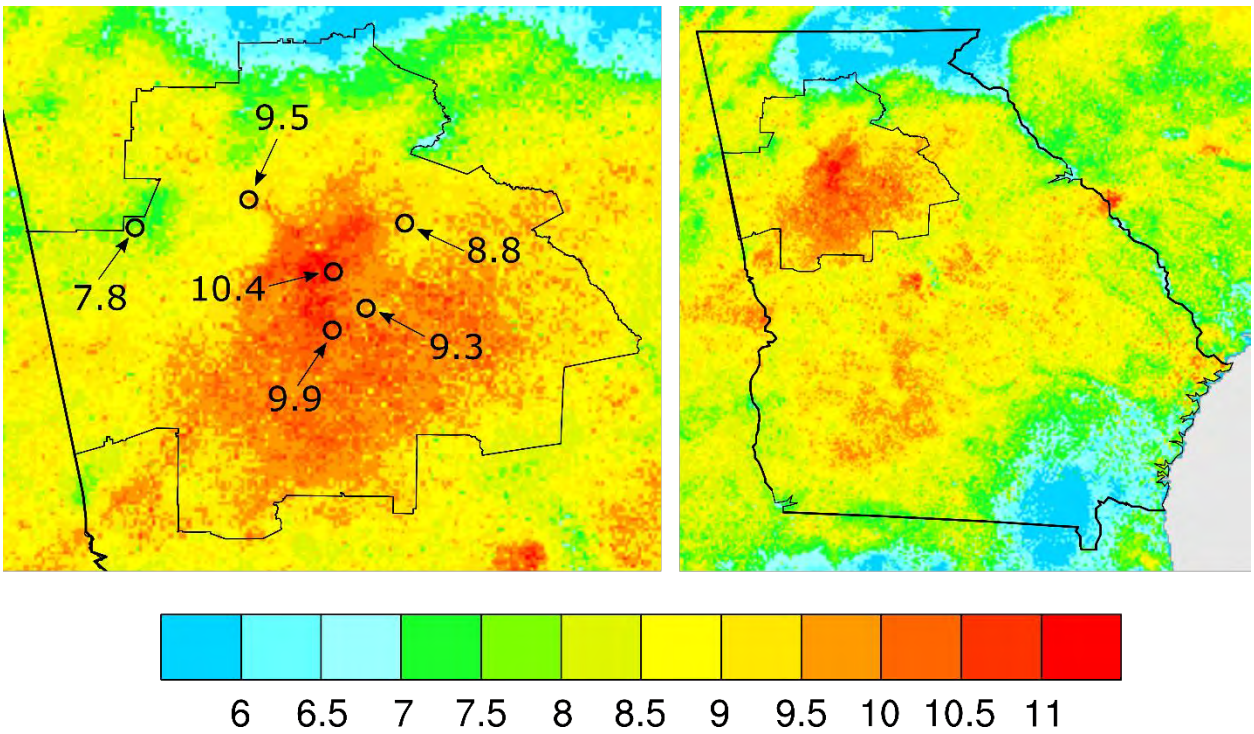


Figure 3-7. Estimated $PM_{2.5}$ concentrations using the DI2019 hybrid approach and monitoring locations and design values for the state of Georgia and the Atlanta-Sandy Springs-Roswell, Georgia CBSA. (Note: Additional information on the DI2019 hybrid approach is described in section 2.3.3.1.4 and in Di et al., 2019a.)

Table 3-9. $PM_{2.5}$ Concentrations Metrics from Monitor and Modeled Data[28]

Description of Metric	$PM_{2.5}$ Concentrations ($\mu g/m^3$)
Atlanta highest monitor	10.4
Atlanta monitored average	9.3
Atlanta spatial average	9.2
Atlanta population-weighted average	9.6
Georgia spatial average	8.3
Georgia population-weighted average	9.1

To expand upon this example in answering our question, we look to the analyses in Chapter 2 which compared area annual design values, composite monitor $PM_{2.5}$ concentrations and mean concentrations from two hybrid approaches. The analyses also included population-weighted mean metrics. In the air quality analyses comparing composite monitored $PM_{2.5}$ concentrations with annual $PM_{2.5}$ design values in U.S. CBSAs, maximum annual $PM_{2.5}$ design values were approximately 10% to 20% higher than annual average concentrations (i.e., averaged across multiple monitors in the same CBSA) (section 2.3.3.1, Figure 2-28 and Table 2-2). The difference between the maximum annual design value and average concentration in an area can be smaller or larger than this range, depending on factors such as the number of monitors, monitor siting characteristics, and the distribution of ambient $PM_{2.5}$ concentrations.[29] 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 2-29 indicates a more variable relationship between maximum 24-hour $PM_{2.5}$ design values and annual average concentrations.

In addition, the air quality analyses in Chapter 2 looked at data from two hybrid modeling approaches. While hybrid modeling approaches are not universal and the various hybrid approaches all have their different nuances, the analysis in Chapter 2 focused on the DI2019 and HA2020 approaches, which have been used in several of the key epidemiologic studies in Table 3-7 and Table 3-8. Section 2.3.3.2.4 details a comparison of $PM_{2.5}$ fields in estimating exposure relative to design values using these two hybrid modeling surfaces. $PM_{2.5}$ concentrations are

[28] "Spatial average" as used in Table 3-9 refers to the average across all grid cells in Atlanta or Georgia using the DI2019 hybrid modeling approach, while "population-weighted average" uses the DI2019 hybrid modeling approach and applies population-weighting to calculate the mean $PM_{2.5}$ concentration.

[29] 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. In the Georgia example above, a near-road monitor was not included in our analysis. The near-road monitor was not added until 2015, and data related to DI2019 ended in 2016. For purposes of developing three-year average concentrations using the most recent data for which we had monitored and modeled data, 2014-2016 data was selected for monitors as well, for which data from 2014-2016 was not available for the near-road monitor.

estimated per year at a 1 km x 1 km spatial resolution. As exhibited in Figure 2-37, the means vary when one estimates $PM_{2.5}$ exposures in urban areas only (CBSAs) versus when the averages used all or most grid cells nationwide. This is likely indicative of the fact that areas included outside of CBSAs tend to be more rural and have lower estimated $PM_{2.5}$ concentrations. This is important to note since, which study area is included in the calculation of the mean (Table 3-7 and Table 3-8 above), and more specifically whether a study is focused on nationwide, regional, or urban areas, will affect the calculation of the study mean based on how many rural areas are included with lower estimated $PM_{2.5}$ concentrations. While the determination of what spatial scale to use to estimate $PM_{2.5}$ concentrations does not inherently affect the quality of the epidemiologic study, the spatial scale can affect the calculation of the long-term mean concentration across the study area and period. As exhibited in Table 2-4, regardless of the hybrid modeling approach assessed, the annual average $PM_{2.5}$ concentrations in CBSA-only analyses are 4-8% higher than for nationwide analyses, likely as a result of higher $PM_{2.5}$ concentrations in more densely populated areas. When evaluating comparisons between surfaces that estimate exposure using population-weighting versus surfaces that do not calculate means using population-weighting, surfaces that calculate long-term mean $PM_{2.5}$ concentrations with population-weighted averages have higher average annual $PM_{2.5}$ concentrations, ranging from 8.2-10.2 $\mu g/m^3$, compared to annual $PM_{2.5}$ concentrations that range from 7.0-8.6 $\mu g/m^3$ in analyses that do not apply population weighting. Average maximum annual design values, on the other hand, exhibit a range from 9.5 to 11.7 $\mu g/m^3$. Analyses exhibit that average maximum annual design values are 40 to 50% higher when compared to annual average $PM_{2.5}$ concentrations estimated without population-weighting and are 15% to 18% higher when compared to average annual $PM_{2.5}$ concentrations with population weighting applied.

The comparisons discussed above show a trend generally observed across the various methods employed to calculate the mean. First, the area annual design values tend to be 10-20% higher than composite monitor values. Additionally, when assessing means from hybrid modeling data, the lowest mean values tend to result from the approaches that use estimated $PM_{2.5}$ concentrations from all or most grid cells, both urban and rural, across the study area to compute the mean. When compared to the area annual design values, these annual design values are higher than means by 40-50%. However, when the approach instead employs methods that population-weight the mean (e.g. average up the grid cells to a ZIP code spatial level), the calculated mean $PM_{2.5}$ concentrations are higher, regardless of the hybrid method employed, and when compared to the area annual design values, design values are only 15-18% higher than means (similar to the differences observed for the composite monitor comparison values for the monitor-based epidemiologic studies). We note that our comparisons used only two hybrid modeling approaches, and while both modeling approaches are popular in the key epidemiologic

studies, they are only just two of the hybrid approaches being used in the literature to estimate $PM_{2.5}$ concentrations. Research groups also continue to develop and improve prediction models to estimate $PM_{2.5}$ concentrations in epidemiologic studies. We also note that different epidemiologic studies use different methods to assign a population weighted average $PM_{2.5}$ concentration to their study population and our comparisons do not assess them all.

Additionally, while these analyses focus on the relationships between study reported means and area annual design values, some studies also provide information on the broader distributions of exposure estimates and/or health events and the $PM_{2.5}$ concentrations corresponding to the lower percentiles of those data (e.g., 25^{th} and/or 10^{th}). We note that this air quality analysis does not provide a similar comparison for these lower percentiles, and that caution should be placed upon any direct comparison of these study reported concentration values corresponding to lower percentiles and annual design values.

In assessing these analyses, we note that these results are most relevant to interpreting U.S. epidemiologic studies. Using information from the U.S.-based analyses for Canadian studies would introduce additional uncertainties, given the differences between U.S. and Canadian studies with respect to population densities, source distributions, and $PM_{2.5}$ concentration gradients. Given these important differences between studies conducted in the two countries and the fact that we lack data and information that would allow us to do similar analyses for Canada, we are unable to provide insight into how the study reported means in the Canadian studies would compare to area design values in the U.S.

To further expand our evaluation of study-reported mean $PM_{2.5}$ 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 lower percentiles of those data (e.g., 25^{th} and/or 10^{th})?**

Figure 3-8 and Figure 3-9 highlight the overall mean (or median) $PM_{2.5}$ concentrations reported in key U.S. and Canadian studies, respectively, that use ground-based monitors alone to estimate long- or short-term $PM_{2.5}$ exposures. For the small subset of studies with available information on the broader distributions of underlying data, Figure 3-8 and Figure 3-9 also identify the study-period mean $PM_{2.5}$ concentrations corresponding to the 25th and 10th percentiles of health events[30] (see Appendix B, Section B.2 for more information).

[30] 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 3-8 and Figure 3-9 and 10% of the total health events occurred in study locations with mean $PM_{2.5}$ concentrations below the 10^{th} percentiles identified.

Figure 3-10 and Figure 3-11 present overall means of predicted $PM_{2.5}$ concentrations for key U.S. and Canadian model-based epidemiologic studies, respectively, and the concentrations corresponding to the 25th and 10th percentiles of estimated exposures or health events[31] when available (see Appendix B, section B.3 for additional information).

[31] For most studies in Figure 3-10 and Figure 3-11, 25th percentiles of exposure estimates are presented. The exception is Di et al., 2017b, for which Figure 3-10 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 concentrations). In addition, the authors of Di et al., 2017b provided population-weighted exposure values (Chan, 2019). The 10th and 25th percentiles of these population-weighted exposure estimates are 7.9 and 9.5 $\mu g/m^3$, respectively.

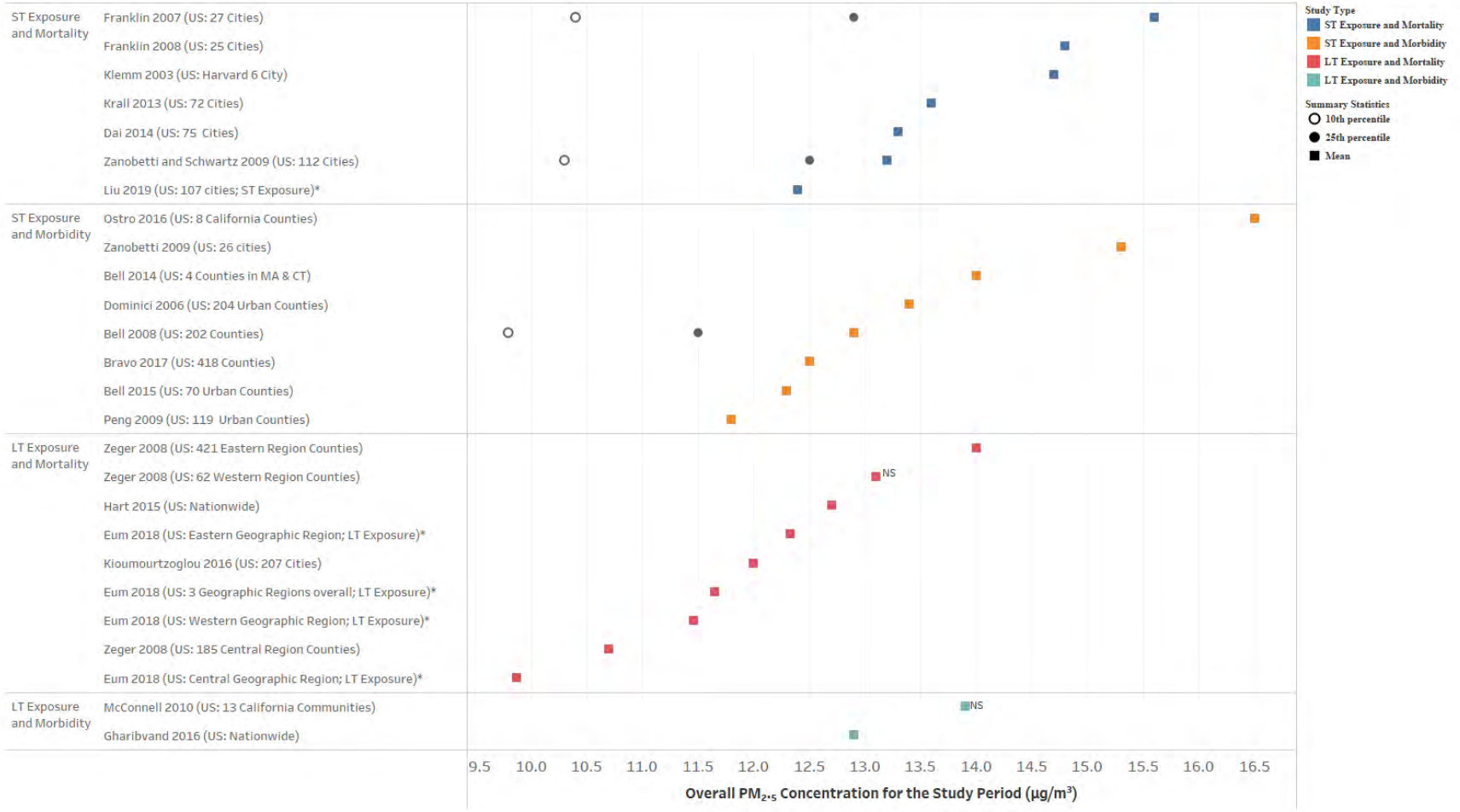


Figure 3-8. Monitor-based $PM_{2.5}$ concentrations in key U.S. epidemiologic studies. (Asterisks denote studies included in the draft ISA Supplement).

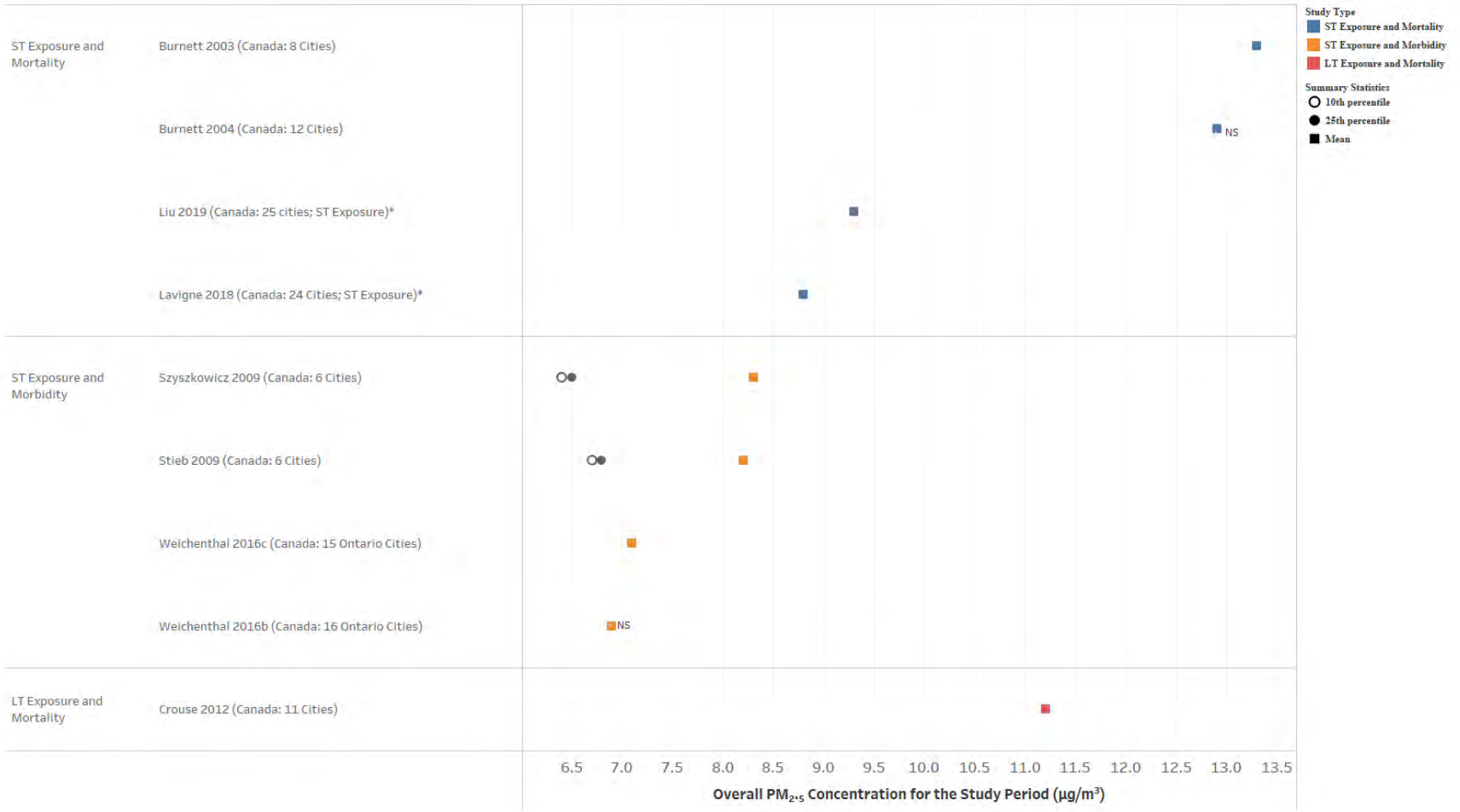


Figure 3-9. Monitor-based $PM_{2.5}$ concentrations in key Canadian epidemiologic studies. (Asterisks denote studies included in the draft ISA Supplement).

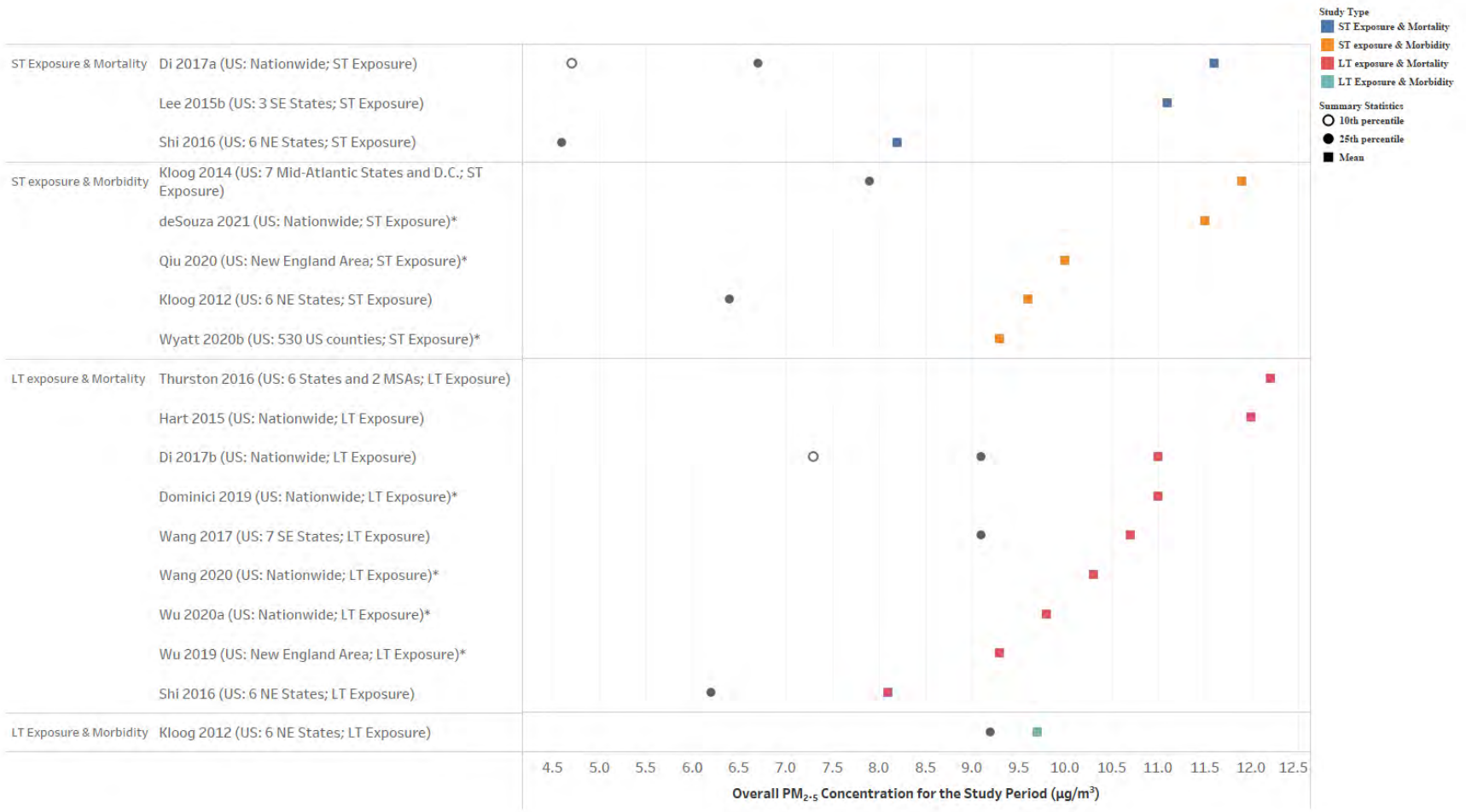


Figure 3-10. Hybrid model-predicted $PM_{2.5}$ concentrations in key U.S. epidemiologic studies. (Asterisks denote studies included in the draft ISA Supplement).

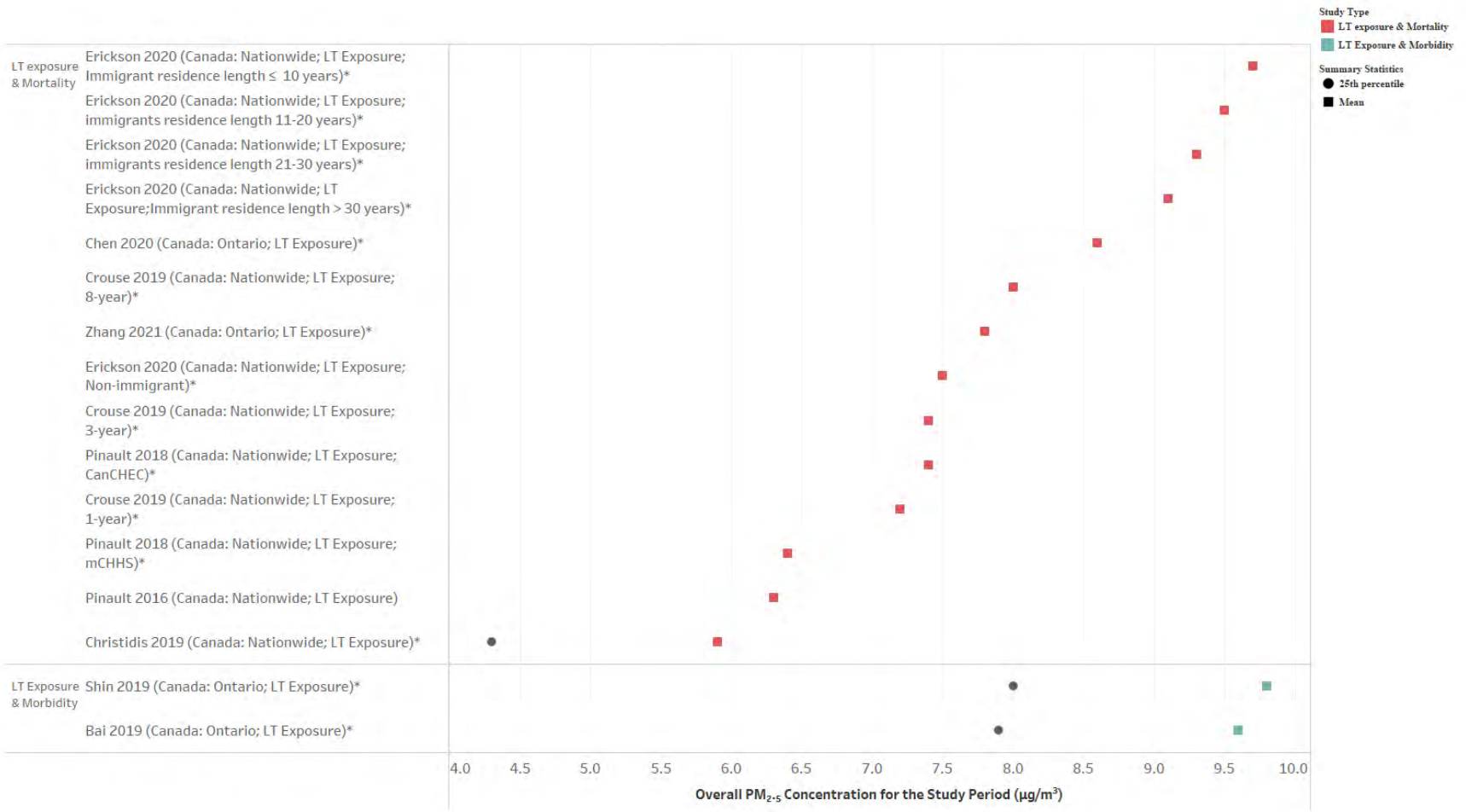


Figure 3-11. Hybrid model-predicted $PM_{2.5}$ concentrations in key Canadian epidemiologic studies. (Asterisks denote studies included in the draft ISA Supplement).

In further examining these data, we also ask:

- **For the key epidemiologic studies using hybrid modeling approaches, what are the study reported means for the general categories of methods of calculating the study mean and how do the study-reported means vary and compare to each other?**

Figure 3-12 and Figure 3-13 present the same key model-based epidemiologic studies from the figures above but focus on the U.S. studies and group them based on their approach to calculating the study-reported mean. For Figure 3-12, the studies are grouped by the geographical spatial scale at which the modeling was conducted (i.e., nationwide, regional, rural). Figure 3-13 presents the same key U.S. model-based epidemiologic studies, but subset by the method used to average grid cells in study-reported long-term mean $PM_{2.5}$ concentrations. For the key U.S. model-based epidemiologic studies, the various methods include the average of all grid cells; grid cells averaged up to ZIP code, postal code or census tract; or population-weighted grid cell averaged up to ZIP code or census tract. Lastly, Figure 3-14 subsets the key U.S. epidemiologic studies that used hybrid exposure models by both spatial scale and the method used to average grid cells in study-reported long-term mean $PM_{2.5}$ concentrations. Grouping the key epidemiologic studies in such ways allows for visual comparisons of the study-reported mean $PM_{2.5}$ concentrations across the different spatial scales and methods of averaging the grid cells.

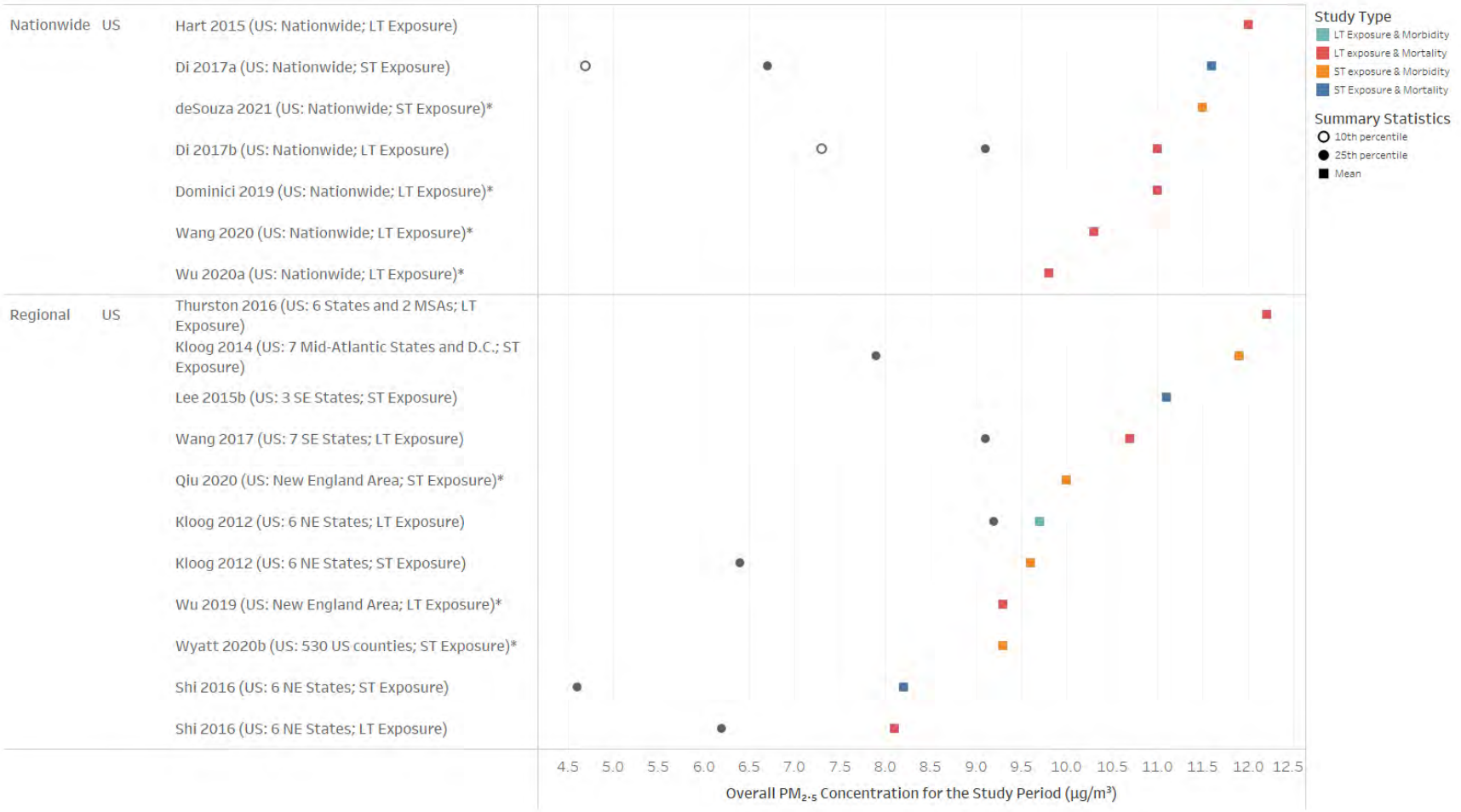


Figure 3-12. Hybrid model-predicted $PM_{2.5}$ concentrations in key U.S. epidemiologic studies, subset by spatial scale. (Asterisks denote studies included in the draft ISA Supplement).

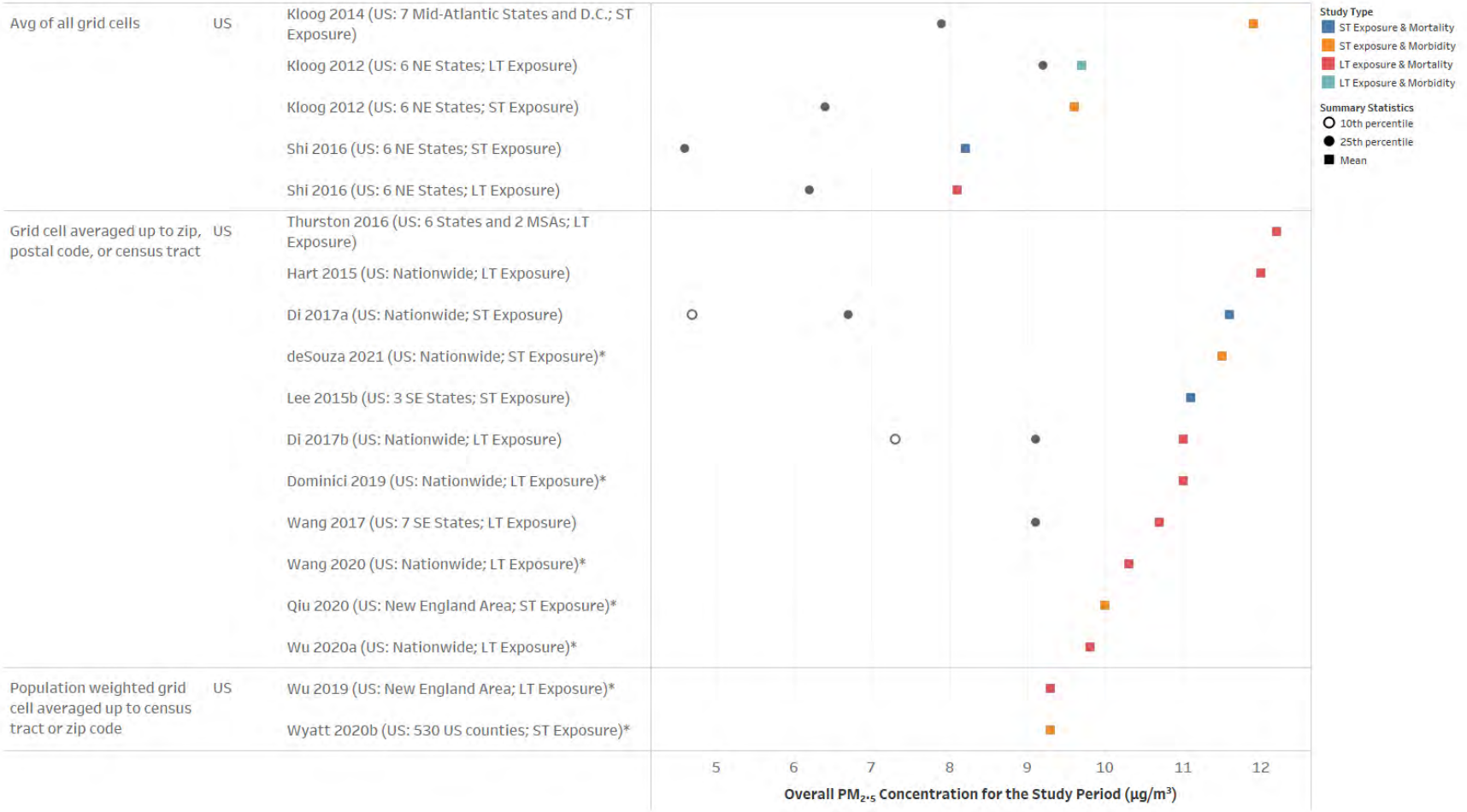


Figure 3-13. Hybrid model-predicted $PM_{2.5}$ concentrations in key U.S. epidemiologic studies, subset by method used to average grid cells in study-reported long-term mean $PM_{2.5}$ concentrations. (Asterisks denote studies included in the draft ISA Supplement).

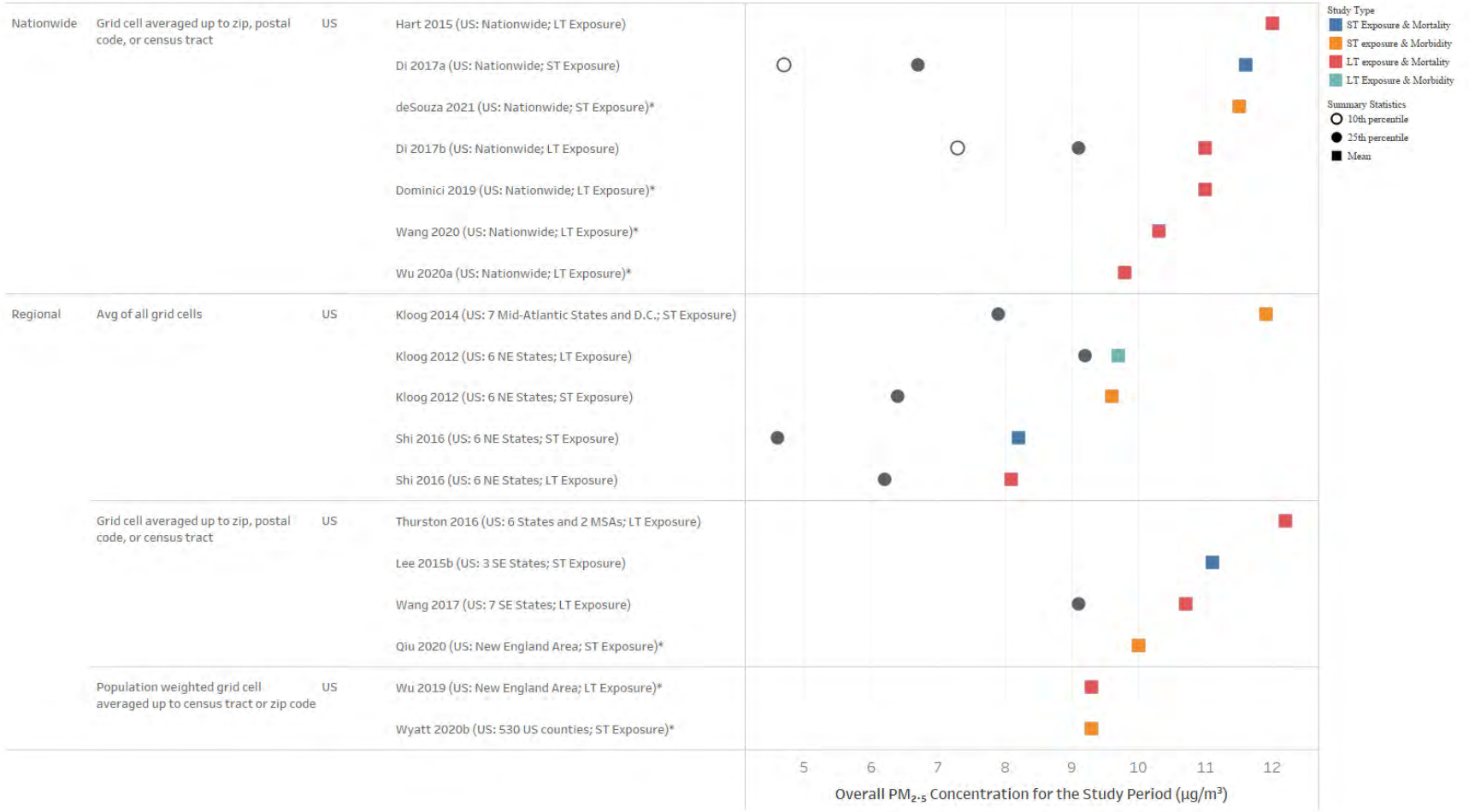


Figure 3-14. Hybrid model-predicted $PM_{2.5}$ concentrations in key U.S. epidemiologic studies, subset by spatial scale and method used to average grid cells in study-reported long-term mean $PM_{2.5}$ concentrations. (Asterisks denote studies included in the draft ISA Supplement).

Based on the information above with regard to the key U.S. and Canadian epidemiologic studies, we summarize some of our observations:

- For key U.S. epidemiologic studies that use monitors to estimate $PM_{2.5}$ exposures (Figure 3-8), overall mean $PM_{2.5}$ concentrations are generally at or above 9.9 $\mu g/m^3$.[32] Based on our air quality analyses, we would generally expect these values to be 10-20% lower than the corresponding area annual design value.
- For key U.S. epidemiologic studies that use hybrid model-predicted exposure (Figure 3-10), mean $PM_{2.5}$ concentrations range from just above 8.0 $\mu g/m^3$ to just above 12.0 $\mu g/m^3$.The majority of these studies estimate $PM_{2.5}$ exposure by averaging up from the grid cell spatial resolution used in the modeling approach to the spatial resolution of health study data (e.g., ZIP code or census tract). This incorporates an aspect of population weighting in the calculation of the mean. Based on our air quality analyses, we would expect these epidemiologic studies to report means similar to those from monitor-based studies and to generally be about 14-18% less than the area annual design value.
    - In studies that average up from the grid cell level to the ZIP code, postal code, or census tract level, mean $PM_{2.5}$ concentrations range from 9.8 $\mu g/m^3$ to 12.2 $\mu g/m^3$.
    - The one study that population weighted the grid cell prior to averaging up to the ZIP code or census tract level report mean $PM_{2.5}$ concentrations of 9.3 $\mu g/m^3$.
- The other set of key U.S. epidemiologic studies averaged up from the grid cell spatial resolution across the entire study area, whether that be the nation or a region of the country. Based on our air quality analyses (i.e., suggesting these means are 40-50% lower than the area annual design value), we would expect these epidemiologic studies to report some of the lowest mean values.
    - For these studies, the reported mean $PM_{2.5}$ concentrations range from 8.1 $\mu g/m^3$ to 11.9 $\mu g/m^3$.
- Of the key epidemiologic studies evaluated in the 2019 ISA and draft ISA Supplement, a subset of studies report $PM_{2.5}$ concentrations corresponding to the 25^{th} and 10^{th} percentiles of health data or exposure estimates to provide insight into the concentrations that comprise the lower quartiles of the air quality distributions.
    - In key U.S. epidemiologic studies that use monitors to estimate $PM_{2.5}$ exposures, 25^{th} 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 PM2.5 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).

[32] This is generally consistent with, but slightly below, the lowest study-reported mean $PM_{2.5}$ concentration from monitor-based studies available in the 2020 PA, which was 10.7 $\mu g/m^3$ (U.S. EPA, 2020, Figure 3-7).

- Of the key U.S. epidemiologic 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 key U.S. epidemiologic studies that use hybrid modeling approaches to estimate short-term $PM_{2.5}$ exposures, the ambient concentrations corresponding to 25^{th} 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 $\mu g/m^3$.[33] 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 $\mu g/m^3$.

- Generally, the study-reported mean concentrations in Canadian studies are lower than those reported in the U.S. studies for both monitor-based and hybrid model methods. However, based on our lack of information about how best to compare air quality gradients between the two countries, it is unclear how to view these Canadian study mean values in the context of a level of the annual standard in the U.S.
  - For the majority of key Canadian epidemiologic studies that use monitor-based exposure (Figure 3-9), mean $PM_{2.5}$ concentrations generally ranged from 7.0 $\mu g/m^3$ to 9.0 $\mu g/m^3$. For these studies, 25^{th} 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 $\mu g/m^3$.
  - For the key Canadian epidemiologic studies that use hybrid model-predicted exposure (Figure 3-11), the mean $PM_{2.5}$ concentrations are generally lower than in U.S. model-based studies (Figure 3-10), ranging from approximately 6.0 $\mu g/m^3$ to just below 10.0 $\mu g/m^3$.
  - The majority of the key Canadian epidemiologic studies that used hybrid modeling were completed at the nationwide scale, while four studies were completed at the regional geographic spatial scale. In addition, all the key Canadian epidemiologic studies, average up from the grid cell level to the spatial resolution of health study data (e.g., postal code).
  - The majority of studies estimating exposure nationwide range between just below 6.0 $\mu g/m^3$ to 8.0 $\mu g/m^3$. One study (Erickson et al. (2020)) presents an analysis related immigrant status and length of residence in Canada versus non-immigrant populations, which accounts for the four highest mean $PM_{2.5}$ concentrations in Figure 3-11, ranging between 9.0 $\mu g/m^3$ and 10.0 $\mu g/m^3$.
  - The four studies that estimate exposure at the regional scale report mean $PM_{2.5}$ concentrations that range from 7.8 $\mu g/m^3$ to 9.8 $\mu g/m^3$.

[33] As noted above, in this study (Shi et al., 2016), the authors report that most deaths occurred at or above the 75^{th} 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.

- In two Canadian studies with information available on the 25th percentile of health events, the ambient $PM_{2.5}$ concentration corresponding to that 25th percentile is approximately 8.0 $\mu g/m^3$ in two studies, and 4.3 $\mu g/m^3$ in a third study.

In addition to the key epidemiologic studies, the 2019 ISA and draft ISA Supplement also include a subset of studies that assess the relationship between $PM_{2.5}$ exposure and health effects that have emerged and so we ask:

- **To what extent has information emerged to further inform our understanding of $PM_{2.5}$ in ambient air and associations with health effects? Are there studies that explore alternative methods for assessing the relationship between $PM_{2.5}$ exposure and health effects or studies that observe changes in health effects with changes in $PM_{2.5}$ concentrations in ambient air over time?**

In addition to the expanded body of evidence from the key epidemiologic studies discussed above, there are also a subset of studies that have emerged that further inform our understanding of the relationship between $PM_{2.5}$ exposure and health effects (U.S. EPA, 2019, U.S. EPA,).

The first type are studies that examine health effect associations in analyses with the highest exposures excluded, restricting analyses to daily exposures less than the 24-hour primary $PM_{2.5}$ standard and annual exposures less than the annual $PM_{2.5}$ standard. The restricted analyses can be informative in assessing the nature of the association between long-term exposures (e.g., < 12.0 $\mu g/m^3$) or short-term exposures (e.g., < 35 $\mu g/m^3$) when looking only at exposures to lower concentrations, including whether the association persists in such restricted analyses compared to the same analyses for all exposures, as well as whether the association is stronger, in terms of magnitude and precision, than when completing the same analysis for all exposures. These studies, as assessed in the 2019 ISA and draft ISA Supplement, are summarized in Table 3-10 below.

Table 3-10. Epidemiologic studies examining the health impacts associated with ambient $PM_{2.5}$ concentrations when studies are conducted with restricted air quality exposures.

Citation	Study Area (health endpoint)	Years of $PM_{2.5}$ Air Quality (monitored)	AQ in restricted analysis (µg/m³)	Study-reported Mean in restricted analysis (µg/m³)	Study-reported Mean in main analysis (µg/m³)	Effect Estimate in restricted analysis (95% CI)	Effect Estimate in main analysis (95% CI)
U.S.-based Studies and Long-term Exposure (per 5 µg/m³)							
Di et al., 2017b	Nationwide (All-cause mortality 65+)	2000-2012	< 12.0	9.6	11.0	1.07 (1.06-1.07)	1.04 (1.04-1.04)
Dominici et al., 2019	Nationwide (All-cause mortality)	2000-2012	< 12	9.6	11.0	1.06 (1.06-1.07)	1.03 (1.03-1.04)
Shi et al., 2016	6 NE States	2003-2008	< 10.0	NR	8.1	1.04 (1.00, 1.09)	1.04 (1.01, 1.06)
Yazdi et al., 2019	7 SE States (CVD morbidity)	2000-2012	< 12	NR	NR	Stroke: 1.29 (1.27-1.31) MI: 1.18 (1.16-1.20) HF: 1.44 (1.43-1.46)	Stroke: 1.16 (1.16-1.17) MI: 1.14 (1.13-1.15) HF: 1.29 (1.29-1.30)
Canadian Studies and Long-term Exposure (per 5 µg/m³)							
Zhang et al., 2021	Ontario (Non-accidental and CVD mortality)	2000-2016	< 10.0 and < 8.8	NR	7.8	Non-accidental mortality: < 10.0: 1.22 (1.10-1.36); and < 8.8: 1.04 (0.91-1.17) CVD mortality: < 10.0: 1.38 (1.10-1.73); and < 8.8: 1.05 (0.80-1.38)	Non-accidental mortality: 1.20 (1.09-1.32) CVD mortality: 1.49 (1.22-1.83)
U.S. Studies and Short-term Exposure (per 10 µg/m³)							
deSouza et al., 2021	Nationwide (First CVD HA)	2000-2012	≤ 25	NR	11.5	1.3% (0.9-1.6 %)	0.9% (0.6-1.1 %)

Di et al., 2017a	Nationwide (All-cause mortality 65+)	2000-2012	<25.0	NR	11.6	1.61 (1.48-1.74)	1.18 (1.09-1.28)
Lee et al., 2015 [1]	3 SE States (Non-accidental)	2007-2011	In ZIP codes where annual average <12.0 and only on days < 35.0	NR	11.1	Non-accidental: 2.08% (1.99-2.17) %	Non-accidental: 1.56% (1.19-1.94%)
Lee et al., 2015 [2]	3 SE States (Non-accidental)	2007-2011	In ZIP codes where annual average < 12.0	NR	11.1	Non-accidental: 2.06% (1.97-2.15%)	Non-accidental: 1.56% (1.19-1.94%)
Shi et al., 2016	6 NE States	2003-2008	< 30.0	NR	8.2	2.14% (1.34-2.95%)	2.14% (1.38, 2.89%)
Wei et al., 2019	Nationwide (CVD HA)	2000-2012	≤ 25 (WHO air quality guideline value for daily $PM_{2.5}$)	NR	NR	Relative increase in risk for HA with 1 µg/m^3 increase in lag0-1 $PM^{2.5}$: MI: 0.16 (0.09, 0.24) CHF: 0.16 (0.11, 0.22)	Relative increase in risk for HA with 1 µg/m^3 increase in lag0-1 $PM^{2.5}$: MI: 0.11 (0.07, 0.16) CHF: 0.14 (0.10, 0.17)

[1] First, restricted ZIP code areas to where the annual average of predicted $PM_{2.5}$ is < 12 µg/m^3 to assess the acute effect of $PM_{2.5}$ on mortality only areas with annual average concentrations < 12 µg/m^3.
[2] In terms of daily standard, conducted analysis on the days < 35 µg/m^3 and only in ZIP codes with annual average concentrations < 12 µg/m^3.

1

There are a number of U.S. and Canadian studies that examine health effect associations in analyses with the highest exposures excluded. These restricted analyses provide support for positive and statistically significant effect estimates at lower mean $PM_{2.5}$ concentrations than their main effect analysis means as shown in Table 3-10 and in many cases, exhibit greater effect estimates in magnitude than their corresponding main analyses. With regard to these studies, we particularly note the following:

- In the four U.S. studies that estimate effects associated with long-term exposure to $PM_{2.5}$, the effect estimates are greater in the restricted analyses than in the main analyses.
    - Di et al. (2017a) and Dominici et al. (2019) report positive and statistically significant associations in analyses restricted to concentrations less than 12.0 $\mu g/m^3$ for all-cause mortality Di et al. (2017b) and stroke, MI, and HF Dominici et al. (2019), and effect estimates are greater in the restricted analyses than effect estimates reported in main analyses. In addition, both studies report mean $PM_{2.5}$ concentrations of 9.6 $\mu g/m^3$
    - Shi et al. (2016) and Yazdi et al. (2019) report positive and statistically significant associations in analyses restricted to concentrations less than 10.0 $\mu g/m^3$ and 12.0 $\mu g/m^3$, respectively. Shi et al. (2016) does not report overall mean $PM_{2.5}$ concentrations in restricted analyses, though such means are presumably somewhat below the main analysis reported mean of 8.1 $\mu g/m^3$. Yazdi et al. (2019) does not report the overall mean $PM_{2.5}$ concentration in either the restricted analysis or main analysis, but the effect estimates for stroke, MI, and HF are all higher in the restricted analyses compared to main analyses.
- While none of the U.S. studies of short-term exposure present mean $PM_{2.5}$ concentrations for the restricted analyses, these studies generally have mean 24-hour average $PM_{2.5}$ concentrations in the main analyses below 12.0 $\mu g/m^3$, and report increases in the effect estimates in the restricted analyses compared to the main analyses.
    - With the exception of Wei et al. (2019), short-term exposure studies report mean 24-hour average $PM_{2.5}$ concentration in main analyses all below 12.0 $\mu g/m^3$, and ranging from 8.2 $\mu g/m^3$ Shi et al. (2016) to 11.6 (Di et al. (2017a).
    - These studies, except for Shi et al. (2016), report increases in effect estimates in restricted analyses compared to main analyses. Shi et al. (2016) reports the same effect estimates for both the restricted and main analyses.
- In the one Canadian study of long-term $PM_{2.5}$ exposure, Zhang et al. (2021) conducted analyses where annual $PM_{2.5}$ concentrations were restricted to concentrations below 10.0 $\mu g/m^3$ and 8.8 $\mu g/m^3$, which presumably have lower mean concentrations than the mean of 7.8 $\mu g/m^3$ reported in the main analyses, though restricted analysis mean $PM_{2.5}$ concentrations are not reported.
    - Effect estimates for non-accidental mortality are greater in analyses restricted to $PM_{2.5}$ concentrations less than 10.0 $\mu g/m^3$, but less in analyses restricted to < 8.8 $\mu g/m^3$. Effect estimates for CVD mortality are lower in restricted analyses than the main analysis.

Overall, these studies provide additional information on the nature of the association between long- or short-term exposures when analyses are restricted to lower $PM_{2.5}$ concentrations. Further, these studies indicate that effect estimates are generally greater in magnitude in the restricted analyses for long- and short-term $PM_{2.5}$ exposure compared to the main analyses.

The second type of studies that have recently emerged and can further inform our understanding of the relationship between $PM_{2.5}$ exposure and health effects are those that employ causal modeling methods. Causal modeling methods seek to mimic randomized experiments through the use of study design and statistical methods, which reduces the potential bias of effects due to confounding. The studies that employ causal modeling methods assessed in the 2019 ISA and draft ISA Supplement are summarized in Table 3-11 below.

Table 3-11. Summary of information from studies that use causal modeling statistical methods.

Study Reference	Statistical Method [1]	Study Area	AQ Years	Health Endpoint (population)	Study-reported Mean (μg/m^3)	Results
Awad et al., 2019	IPW	U.S. Nationwide	2000-2012	LT mortality (65+)	Mean change in exposure the year before move and the second year after move: Whites: -0.73 Blacks: -0.90	Per a 10 μg/m^3 increase in annual $PM_{2.5}$ concentrations: White individuals: HR = 1.21 (95% CI: 1.20, 1.22) Black individuals: HR = 1.12 (95% CI: 1.08, 1.15) All-cause mortality: HR = 1.12 (95% CI: 1.08, 1.15)
Awad et al., 2019 (restricted	IPW	U.S. Nationwide	2000-2012	LT mortality (65+)	Restricted < 12.0: NR	Per a 10 μg/m^3 increase in annual $PM_{2.5}$ concentrations: White individuals: HR = 1.25 (95% CI: 1.24, 1.27) Black individuals: HR = 1.08 (95% CI: 1.01, 1.14)
Higbee et al., 2020	IPW	U.S. Nationwide	1986-2015	LT mortality (18+)	10.7	For a 10 μg/m^3 increase in annual $PM_{2.5}$ concentrations: All-cause mortality: HR = 1.12 (95% CI: 1.08, 1.15) Cardiopulmonary mortality: HR = 1.23 (95% CI: 1.17, 1.29)
Qiu et al., 2020	IPW	New England	2000-2012	ST CVD HA (65+)	AMI:10.3 CHF: 10.08 IS: 10.1	Percent increase HA rate for a 10 μg/m^3 increase in $PM_{2.5}$ concentrations AMI: 4.31% (95% CI: 2.21, 6.42) CHF: 3.95% (95% CI: 2.37,5.53) IS: 2.56% (95% CI: 0.44, 4.69)
Schwartz et al., 2018a	3 approaches: Instrumental approach Marginal structural models Time-series analysis	135 U.S. Cities	1999-2010	ST mortality (18+)	12.8	Percent change in daily mortality per 10 μg/m^3 increase in $PM_{2.5}$ concentrations Instrumental approach: 1.54% (95% CI: 1.12, 1.97) Marginal structural models: 0.75% (95% CI: 0.35, 1.15) Time-series: 0.60%: (95% CI: 0.34, 0.85%)
Schwartz et al., 2018a	3 approaches: Instrumental approach	135 U.S. Cities	1999-2010	ST mortality (18+)	Restricted < 25.0: NR	Percent change in daily mortality per 10 μg/m^3 increase in $PM_{2.5}$ concentrations Instrumental approach: 1.70% (95% CI: 1.11, 2.29) Marginal structural models: 0.83% (95% CI: 0.39,1.27)

(restricted analysis)	Marginal structural models Time-series analysis					Time-series: 0.62%: (95% CI: 0.32, 0.93)
Schwartz et al., 2018b	GPS IPW	Northeastern and Mid-Atlantic States	2000-2012	Life expectancy	NA	Estimated mean age at death for an annual average exposure of 12 μg/m^3 was 0.89 years (95% CI: 0.88,0.91) than estimated for a counterfactual $PM_{2.5}$ exposure of 7.5 μg/m^3
Schwartz et al., 2021	DID	U.S. Nationwide	2000-2016	LT probability of dying (65+)	10.3	Probability of dying in each year increased by 3.85x10^{-4} (95% CI 1.95x10^{-4}, 5.76x10^{-4}) for each 1 μg/m^3 increase in annual $PM_{2.5}$ concentrations
Schwartz et al., 2021 (restricted analysis)	DID	U.S. Nationwide	2000-2016	LT probability of dying (65+)	NR	Probability of dying in each year increased by 4.26x10^{-4} (95% CI 1.43x10^{-4}, 7.09x10^{-4}) for each 1 μg/m^3 increase in annual $PM_{2.5}$ concentrations
Wu et al., 2019	RC-GPS and 3 GPS approaches: Subclassification GPS IPTW GPS GPS matching	New England	2000-2012 (modeled)	LT mortality (65+)	NA	Exposure levels of low (≤ 8.0 μg/m^3) versus moderate $PM_{2.5}$ concentrations (8.0-10.0 g/m^3) to low exposure Subclassification: 1.025 (95% CI: 1.006,1.045) IPTW GPS: 1.022 (95% CI: 1.007, 1.038) Matching GPS: 1.028 (1.012, 1.045) Comparison of exposure levels of ≤ 8.0 μg/m^3 vs. ≥ 10.0 μg/m^3 Subclassification: 1.035 (95% CI: 0.999,1.072) IPTW GPS: 1.030 (95% CI: 1.005, 1.056) Matching GPS: 1.035 (95% CI: 1.015, 1.055)
Wu et al., 2020b	Three GPS approaches: GPS matching GPS weighting GPS adjustment	U.S. Nationwide	2000-2016 (modeled)	LT mortality (65+)	9.8	Reported hazard ratios for a decrease in mortality risk per 10 μg/m^3 decrease in annual $PM_{2.5}$ GPS matching: HR = 1.068 (95% CI: 1.054,1.083) GPS weighting: HR = 1.076 (95% CI: 1.065, 1.088) GPS adjustment: HR = 1.072 (95% CI: 1.061,1.082)
Wu et al., 2020a (restricted analysis)	Three GPS approaches: GPS matching GPS weighting	U.S. Nationwide	2000-2016 (modeled)	LT mortality (65+)	Restricted < 12.0: 8.4	Reported hazard ratios for a decrease in mortality risk per 10 μg/m^3 decrease in annual $PM_{2.5}$ GPS matching: HR = 1.261 (95% CI: 1.233,1.289) GPS weighting: HR = 1.268 (95% CI: 1.237, 1.300)

	GPS adjustment					GPS adjustment: HR = 1.231 (95% CI: 1.180,1.284)
Yazdi et al., 2021	Doubly Robust Additive Model (DRAM)	U.S. Nationwide	2000-2016 (modeled)	LT Cardiovascular hospitalization outcomes (65+)	10.21	% increase in the risk with 1 μg/m^3 increase in $PM_{2.5}$: MI: 0.002; Stroke: 0.009; AI: 0.006
Yitshak-Sade et al., 2019	DID	Northeastern and mid-Atlantic States (14 U.S. States)	2000-2013	LT mortality (65+)	Range: 6.5-14.5	4.04% (95% CI: 3.49,4.59) increase in mortality rates for an IQA (3 μg/m^3) increase in annual $PM_{2.5}$ concentrations

[1] GPS: generalized propensity score; IPW: inverse probability weighting; DID: Difference-in-difference; HR: hazard ratio; IRR: incidence rate ratio; IPTW: inverse probability treatment weighting; IV: instrument variable; OLS: Ordinary Least Squares; RC: regression calibration

1

The 2019 ISA and draft ISA Supplement assess epidemiologic studies that implemented causal modeling methods. As presented in Table 3-11 above, these studies employ a variety of statistical methods, such as GPS, IPW, and DID. We particularly note the following:

- These studies reported consistent results among large study populations across the U.S. The results from studies that use causal modeling methods further inform the relationship between long- and short-term $PM_{2.5}$ exposure and total mortality.
- Studies that employ causal methods to assess the association between long-term exposure to $PM_{2.5}$ and mortality provide additional support for the associations reported in the broader body of cohort studies that examined long-term $PM_{2.5}$ exposure and mortality.
    - For example, Wu et al., 2020a used three different causal modeling statistical approaches, in addition to two more traditional statistical method methods (Cox proportional hazards modeling and Poisson time-series regression model), finding consistent positive and statistically significant results between the five statistical methods and with HRs per a 10 $\mu g/m^3$ increase in $PM_{2.5}$ ranging from 1.062 (95% CI: 1.055,1.069) using the poisson statistical method to 1.076 (95% CI: 1.065, 1.088) with the GPS matching statistical method.

Lastly, there is also a smaller subset of epidemiologic studies, accountability analyses, that evaluated the potential for improvements in public health as ambient $PM_{2.5}$ concentrations have declined over time. Given the nature of these studies, the majority tend to focus on time periods in the past during which ambient $PM_{2.5}$ concentrations were substantially higher than those measured more recently (e.g., see Chapter 2, Figure 2-16). These studies, as assessed in the 2019 ISA and draft ISA Supplement, are summarized in Table 3-12 below.

Table 3-12. 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 Mean $PM_{2.5}$ Concentration ($\mu g/m^3$)	Ending Mean $PM_{2.5}$ Concentration ($\mu g/m^3$)	Study Results
Pope et al. (2009)	211 U.S. counties	1979-1983 compared to 1999-2000	20.6	14.1	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	11.6	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	14.4	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	11.9-17.8	Statistically significant improvements in 4-year growth of lung function
Wyatt et al., 2020b	2132 counties in the U.S. (population ≥20,000)	1990-2010	NR	NR	The annual change in cardiovascular mortality rate ranged from 6.5-7.6 fewer deaths/year (per 100,000 person-years) per 1 μg/m^3 decrease in $PM_{2.5}$ over time.
Bennett et al., 2019	U.S. Nationwide and 1339 U.S. counties	1999-2015	13.6 (Pop-weighted mean)	8.0 (Population-weighted mean; Mean range in counties: 2.8-13.2)	Reductions in $PM_{2.5}$ since 1999 have increased life expectancy in men and women in all but 14 counties where $PM_{2.5}$ increased slightly
Corrigan et al., 2018	619 U.S. counties	2000-2010	2000-2004: 12.0	2005-2010: 10.8	Fewer CV deaths per year for each 1 μg/m^3 decrease in $PM_{2.5}$.
Henneman et al., 2019	Multiple U.S. states	2005-2012	2005: 10.0	2012: 7.2	Reduced exposure to total $PM_{2.5}$ and coal emissions led to reduced rates total mortality and CVD HA.
Sanders et al., 2020	600-700 U.S. counties	2000-2013	Before 2006: Non-attainment: 15.3 and Attainment: 11.0	After 2006: Non-attainment: 12.0 Attainment: 9.3	By 2005 $PM_{2.5}$ designation status (attainment or non-attainment), $PM_{2.5}$ levels and corresponding mortality rates
Fan and Wang, 2020	Eastern US	1999-2013	NR	NR	Fewer CVD deaths per year for each 1

					μg/m^3 reduction in annual $PM_{2.5}$ concentrations
Peterson et al., 2020	2132 counties	1990-2010	NR	NR	Fewer CVD deaths for each 1 μg/m^3 reduction in annual $PM_{2.5}$ concentrations

The accountability studies assessed in the 2019 ISA and draft ISA Supplement provide support for the conclusion that public health benefits are associated with decreases in ambient $PM_{2.5}$ concentrations. In particular, we note the following key observations from these studies:

- Of the new studies evaluated in the 2019 ISA and draft ISA Supplement, Corrigan et al. (2018), Henneman et al. (2019) and Sanders et al. (2020) present analyses with starting concentrations below 12.0 μg/m^3.
    - Henneman et al. (2019) explored the changes in modeled $PM_{2.5}$ concentrations following the retirement of coal fired power plants in the U.S., and found that reductions from mean annual $PM_{2.5}$ concentrations of 10.0 μg/m^3 in 2005 to mean annual $PM_{2.5}$ concentrations of 7.2 μg/m^3 in 2012 from coal-fueled power plants resulted in corresponding reductions in the number of cardiovascular-related hospital admissions and total mortality in those aged 65 and older.
    - Corrigan et al. (2018) examined whether there was a change in the cardiovascular mortality rate before (2000-2004) and after (2005-2010) implementation of the first annual $PM_{2.5}$ NAAQS implementation based on mortality data from the National Center for Health Statistics. They reported 1.10 (95% CI: 0.37, 1.82) fewer cardiovascular deaths per year per 100,000 people for each 1 μg/m^3 reduction in annual $PM_{2.5}$ concentrations. When comparing whether counties met the annual $PM_{2.5}$ standard, there were 1.96 (95% CI: 0.77, 3.15) fewer cardiovascular deaths for each 1 μg/m^3 reduction in annual $PM_{2.5}$ concentrations between the two periods for attainment counties, whereas for non-attainment counties, there were 0.59 (95% CI: −0.54, 1.71) fewer cardiovascular deaths between the two periods.
    - Sanders et al. (2020) examined whether policy actions (i.e., the first annual $PM_{2.5}$ NAAQS implementation rule in 2005 for the 1997 annual $PM_{2.5}$ standard with a 3-year annual average of 15 μg/m^3) reduced $PM_{2.5}$ concentrations and mortality rates in Medicare beneficiaries between 2000-2013. They found evidence of changes in associations with mortality (a decreased mortality rate of ~ 0.5 per 1,000 in attainment and non-attainment areas) due to changes in annual $PM_{2.5}$ concentrations in both attainment and non-attainment areas, which had starting concentrations below 12.0 μg/m^3 following implementation of the annual $PM_{2.5}$ NAAQS in 2005. In addition, following implementation of the annual $PM_{2.5}$ NAAQS, annual $PM_{2.5}$

concentrations decreased by 1.59 μg/m^3 (95% CI: 1.39, 1.80) which corresponded to a reduction in mortality rates among individuals 65 years and older (0.93% [95% CI: 0.10%, 1.77%]) in non-attainment counties relative to attainment counties.

- Bennett et al. (2019) reports increases in life expectancy in all but 14 counties (1325 of 1339 counties) that have exhibited reductions in $PM_{2.5}$ concentrations from 1999 to 2015.
- While Fan and Wang (2020), Peterson et al. (2020), and Wyatt et al. (2020a) do not report starting and ending concentrations, these studies lend support to the conclusions that reductions in $PM_{2.5}$ concentrations lead to public health improvements, including reductions in cardiovascular mortality.

The information in Table 3-10, Table 3-11, and Table 3-12 provide additional support to inform the relationship between long- and short-term $PM_{2.5}$ exposure and total mortality. Analyses that are restricted only to concentrations at or below the levels of the current primary $PM_{2.5}$ standards find positive and significant associations with exposure to $PM_{2.5}$ and health outcomes. These restricted analyses often report greater effect estimates compared to effect estimates in the main analysis that uses the full distribution of $PM_{2.5}$ concentrations. Studies that use causal modeling methods to assess the relationship between $PM_{2.5}$ and health outcomes provide additional support for the associations reported in other epidemiologic studies. Finally, new studies assessed in the draft ISA Supplement evaluate the relationship between declines in ambient $PM_{2.5}$ concentrations over time and the potential for improvements in public health, and support the conclusion in the 2020 PA; improvements in air quality are associated with improvements in public health. Some of these new studies have lower starting concentrations than similar studies included in the 2019 ISA.

3.3.4 Uncertainties in the Health Effects Evidence

- **To what extent have important uncertainties identified in prior reviews been reduced and/or have additional uncertainties emerged?**

We have not identified any new uncertainties in the evidence since the 2020 review. However, we continue to recognize uncertainties that persist from the previous reviews. This array of important areas of uncertainty related to the current health effects evidence, including that assessed in the 2019 ISA and the draft ISA Supplement, is summarized below.

Although the epidemiologic studies clearly demonstrate associations between long- and short-term $PM_{2.5}$ exposures and health outcomes, as in previous reviews, we continue to recognize several uncertainties and limitations in the health effects evidence remain. Epidemiologic studies evaluating short-term $PM_{2.5}$exposure and health effects have reported heterogeneity in associations between cities and geographic regions within the U.S. Heterogeneity in the associations observed across epidemiologic studies may be due in part to exposure error related to measurement-related issues, the use of central fixed-site monitors to

represent population exposure to $PM_{2.5}$, and our limited understanding of factors that could be due to a number of factors including exposure error related to measurement-related issues, variability in $PM_{2.5}$ composition regionally, and factors that result in differential exposures (e.g., topography, the built environment, housing characteristics, personal activity patterns). Heterogeneity is expected when the methods or the underlying distribution of covariates vary across studies (U.S. EPA, 2019, p. 6-221). Studies assessed in the 2019 ISA and draft ISA Supplement have advanced the state of exposure science by presenting innovative methodologies to estimate PM exposure, detailing new and existing measurement and modeling methods, and further informing our understanding of the influence of exposure measurement error due to exposure estimation methods on the associations between $PM_{2.5}$ and health effects reported in epidemiologic studies (U.S. EPA, 2019, section 1.2.2; U.S. EPA, 2021a). Data from $PM_{2.5}$ monitors continue to be commonly used in health studies as a surrogate for $PM_{2.5}$ exposure, and often provide a reasonable representation of exposures throughout a study area (U.S. EPA, 2019, section 3.4.2.2; U.S. EPA, 2021a, section 3.2.2.2.2). However, an increasing number of studies employ hybrid modeling methods to estimate $PM_{2.5}$ exposure using data from several sources, often including satellites and models, in addition to ground-based monitors. These hybrid models typically have good cross-validation, especially for $PM_{2.5}$, and have the potential to reduce exposure measurement error and uncertainty in the health effect estimates from epidemiologic models of long-term exposure (U.S. EPA, 2019, section 3.5; U.S. EPA, 2021a, section 2.3.3).

While studies using hybrid modeling methods have demonstrated reduced exposure measurement error and uncertainty in the health effect estimates, these studies use a variety of approaches to estimate $PM_{2.5}$ concentrations and to assign exposure to assess the association between health outcomes and $PM_{2.5}$ exposure. This variability in methodology has inherent limitations and uncertainties, as described in more detail in section 2.3.3.1.5, and the performance of the modeling approaches depends on the availability of monitoring data which varies by location. Factors likely contributing to poorer model performance often coincide with relatively low ambient $PM_{2.5}$ concentrations, in areas where predicted exposures are at a greater distance to monitors, and under conditions where the reliability and availability of key datasets (e.g., air quality modeling) are limited. Thus, uncertainty in hybrid model predictions becomes an increasingly important consideration as lower predicted concentrations are considered.

Regardless of whether a study uses monitoring data or a hybrid modeling approach when estimating $PM_{2.5}$ exposures, one key limitation that persists is associated with the interpretation of the study-reported mean $PM_{2.5}$ concentrations and how they compare to design values, the

metric that describe the air quality status of a given area relative to the NAAQS.[34] 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 location (i.e., across multiple monitors or across modeled grid cells) and over time (i.e., over several years). For monitor-based studies, the comparison is somewhat more straightforward than for studies that use hybrid modeling methods, as the monitors used to estimate exposure in the epidemiologic studies are generally the same monitors that are used to calculate design values for a given area. It is expected that 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. 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 (U.S. EPA, 2020, 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. For studies that use hybrid modeling methods to estimate $PM_{2.5}$ concentrations, the comparison between study-reported mean $PM_{2.5}$ concentrations and design values is more complicated given the variability in the modeling methods, temporal scales (i.e., daily versus annual), and spatial scales (i.e., nationwide versus urban) across studies. A recent comparison between two hybrid modeling surfaces explored the impact of these factors on the resulting mean $PM_{2.5}$ concentrations and provided additional information about the relationship between mean concentrations from studies using hybrid modeling methods and design values (see section 2.3.3.1.4). However, the results of those analyses only reflect two surfaces and two types of approaches, so uncertainty remains in understanding the relationship between estimated modeled $PM_{2.5}$ concentrations and design values more broadly across hybrid modeling studies. Moreover, this analysis was completed using two hybrid modeling methods that estimate $PM_{2.5}$ concentrations in the U.S., thus an additional uncertainty includes understanding the relationship between modeled $PM_{2.5}$ concentrations and design values reported in Canada.

In addition, where $PM_{2.5}$ and other pollutants (e.g., ozone, nitrogen dioxide, and carbon monoxide) are correlated, it can be difficult to distinguish whether attenuation of effects in some studies results from copollutant confounding or collinearity with other pollutants in the ambient mixture (U.S. EPA, 2019, section 1.5.1; U.S. EPA, 2021a, section 2.2.1). Studies evaluated in

[34] 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 98th percentile of the annual distribution of 24-hour $PM_{2.5}$ concentrations, averaged over three years (Appendix N of 40 CFR Part 50).

the 2019 ISA and draft ISA Supplement further examined the potential confounding effects of both gaseous and particulate copollutants on the relationship between long- and short-term $PM_{2.5}$ exposure and health effects. The studies continue to provide evidence indicating that associations with $PM_{2.5}$ are relatively unchanged in copollutants models (U.S. EPA, 2019, section 1.5.1; U.S. EPA, 2021a, section 2.2.1). Another area of uncertainty is associated with other potential confounders, beyond copollutants. Some studies have expanded the examination of potential confounders to not only include copollutants, but also systematic evaluations of the potential impact of inadequate control from long-term temporal trends and weather (U.S. EPA, 2019, section 11.1.5.1). Analyses examining these covariates further confirm that the relationship between $PM_{2.5}$ exposure and mortality is unlikely to be biased by these factors. Other studies have explored the use of causal modeling statistical techniques to reduce uncertainties related to potential confounding that can further inform the causality determination for long-term and short-term $PM_{2.5}$ and mortality and cardiovascular effects (U.S. EPA, 2019, section 11.2.2.4, and U.S. EPA 2021, sections 3.1.1.3, 3.1.2.3, 3.2.1.2, and 3.2.2.3). These studies indicate that bias from unmeasured confounders can occur in either direction, although controlling for these confounders did not result in the elimination of the association, but instead provided additional support for associations between long-term $PM_{2.5}$ exposure and mortality when accounting for additional confounders (U.S. EPA, 2021a, section 3.2.2.2.6).

Another important limitation associated with the evidence is that, while epidemiologic studies indicate associations between $PM_{2.5}$ and health effects, they do not identify particular $PM_{2.5}$ exposures that cause effects. Rather, health effects can occur over the entire distribution 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 $PM_{2.5}$-related effects do not occur. Overall, evidence assessed in the 2019 ISA and draft ISA Supplement continues to indicate a linear, no-threshold concentration-response relationship for long- and short-term $PM_{2.5}$ exposure and all-cause or cause specific mortality. There is less certainty in the shape of the concentration-response curve at mean annual $PM_{2.5}$ concentrations generally below 8 $\mu g/m^3$, although some studies characterize the concentration-response function with certainty in the linear relationship below 8 $\mu g/m^3$ and down to as low as 5 $\mu g/m^3$ (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section 2.2.3.2).

3.4 RISK INFORMATION

To inform conclusions regarding the primary $PM_{2.5}$ standards that are "requisite" to protect 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 $\mu g/m^3$ and/or three-year 24-hour design values of 35 $\mu g/m^3$ (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 3.3.3.2), we assess $PM_{2.5}$-attributable risk associated with either:

- $PM_{2.5}$ 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 $\mu g/m^3$) or lower alternative annual and/or 24-hour standards.
- The change in risk associated with moving from $PM_{2.5}$ air quality "just meeting" the current standards to "just meeting" alternative annual and/or 24-hour standards.

These risk estimates, when considered alongside analyses of the evidence discussed in section 3.3.3, 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 3.4.1) and the results of the risk assessment (section 3.4.1.8). Additional detail on the risk assessment is provided in Appendix C.

3.4.1 Risk Assessment Overview

Risk assessments combine data from multiple sources and involve various assumptions and uncertainties. Below we summarize key aspects of the risk modeling approach. Input data for these analyses includes concentration-response functions from epidemiologic studies (section 3.4.1.1) for each health outcome (section 3.4.1.2) and ambient annual or 24-hour $PM_{2.5}$ concentrations (sections 3.4.1.3 and 3.4.1.4) for the study areas (section 3.4.1.5) utilized in the risk assessment. Quantitative and qualitative methods used to characterize variability and uncertainty in the risk estimates are discussed in section 3.4.1.7.

Information on other data inputs, such as baseline health incidence rate and population demographic information, can be found in the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits Technical Support Document (TSD)* (U.S. EPA, 2021b; associated with the 2021 Revised Cross-State Air Pollution Rule Update (86 FR 23054, April 30, 2021). Additional detail on the risk assessment approach is provided in Appendix C (section C.1).

3.4.1.1 Concentration-Response Functions

Concentration-response functions used in this risk assessment are from large, multicity U.S. epidemiologic studies that evaluate the relationship between $PM_{2.5}$ exposures and mortality. Specific epidemiologic studies and concentration-response functions used here to estimate risk were identified using criteria that take into account factors such as study design, geographic coverage, demographic populations, and health endpoints. Information about the studies used in this risk assessment is summarized in Table 3-13 and additional detail regarding the selection of epidemiologic studies and specification of concentration-response functions can be found in Appendix C (section C.1.1) and the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits TSD* (U.S. EPA, 2021b).

3.4.1.2 Health Outcomes

Consistent with the overall approach for this reconsideration, this risk assessment has a targeted scope that focuses on all-cause or nonaccidental mortality associated with long-term and short-term $PM_{2.5}$ exposures (Table 3-13 and Appendix C, section C.1.1).[35] Evidence for these outcomes supports the determination of a "causal relationship" in the 2019 ISA (U.S. EPA, 2019).[36]

Table 3-13. 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			
Di et al., 2017b	Medicare	65+	All-cause
Turner et al., 2016	ACS	30+	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), NPACT (National Particle Components Toxicity). See Appendix C Table C-1 for additional study details.

[35] Epidemiologic studies tend to attribute risk to either long- or short-term $PM_{2.5}$ exposures, but rarely to both, leading to uncertainties in the relationship between health effects from long- and short-term exposures. When biologically plausible pathways leading to health effects are similar, estimates of impacts from long-term exposures may include impacts due to short-term exposures and vice-versa. However, if pathways diverge, impacts due to long- and short-term exposures may be the sum, or even greater than the sum, of the two exposure durations.

[36] While the 2019 ISA also found that evidence supports the determination of a "causal relationship" between long- and short-term exposures and cardiovascular effects, cardiovascular mortality was not included as a health outcome as it will be captured in the estimates of all-cause mortality.

3.4.1.3 Air Quality Scenarios

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 then use air quality modeling to simulate air quality just meeting an alternative standard with a level of 10.0 $\mu g/m^3$ (annual) and 30 $\mu g/m^3$ (24-hour). In addition to the model-based approach, for the subset of 30 areas controlled by the annual standard we also employ linear interpolation and extrapolation to simulate just meeting alternative annual standards with levels of 11.0 (interpolated between 12.0 and 10.0 $\mu g/m^3$), 9.0 $\mu g/m^3$, and 8.0 $\mu g/m^3$ (both extrapolated from 12.0 and 10.0 $\mu g/m^3$) .[37] Figure 3-15 provides an example of the interpolation and extrapolation calculations performed for a single grid cell. In this example grid cell, modeled annual $PM_{2.5}$ concentrations are 11.23 when the corresponding design value monitor just meets the current annual standard and 9.87 when the corresponding design value monitor just meets the alternative annual standard of 10.0 $\mu g/m^3$. The interpolated and extrapolated values for the example grid cells are provided in green and blue text, respectively.[38]

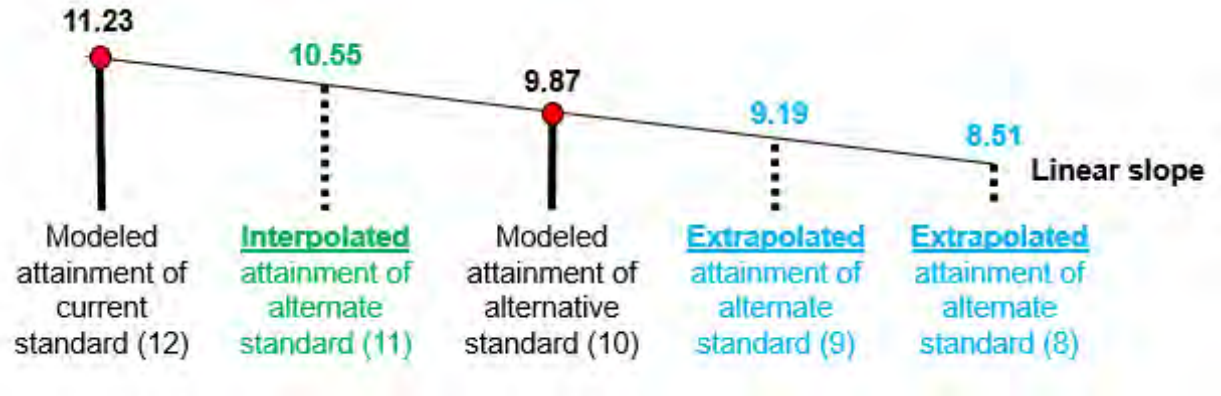


Figure 3-15. Illustration of approach to adjusting air quality to simulate just meeting annual standards with levels of 11.0, 9.0, and 8.0 $\mu g/m^3$.

[37] Modeled air quality surfaces are simulated to just meet standards at the design value monitors and not necessarily in all grid cells. As the extrapolated alternative annual standard decreases, the proportion of grid cells at or above the modeled standard increases. Appendix Figure C-31 provides the full distribution of grid cell concentrations at each modeled and extrapolated standard.

[38] Modeling to "just meet" annual standards involves adjusting the design value monitor to the standard, and not necessarily all grid cells modeled. Therefore, it is possible to have estimated $PM_{2.5}$ concentrations above the annual standard modeled in individual grid cells.

There is greater uncertainty regarding whether a revised 24-hour standard (i.e., with a lower level) is needed to further limit "peak" $PM_{2.5}$ concentration exposure[39] and whether a lower 24-hour standard level would most effectively reduce $PM_{2.5}$-associated health risks associated with "typical" daily exposures. However, we do estimate health risks associated with air quality adjusted to meet a revised 24-hour standard with a level of 30 $\mu g/m^3$, in conjunction with estimating the health risks associated with meeting a revised annual standard with a level of 10 $\mu g/m^3$.[40,41]

3.4.1.4 Model-Based Approaches to Adjusting Air Quality

Air quality modeling was used to develop 12 km gridded $PM_{2.5}$ concentration fields for the risk assessment in the 2020 PM PA, and the same air quality simulations used in that assessment are used here (U.S. EPA, 2020). A $PM_{2.5}$ concentration field for 2015 was developed using a Bayesian statistical model (Downscaler) that calibrates chemical transport model (CTM) predictions of $PM_{2.5}$ to surface measurements (section 2.3.3). The 2015 $PM_{2.5}$ concentration field was then adjusted 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 $PM_{2.5}$ concentration fields at 12 km resolution for study areas. The adjusted concentration fields correspond to:

(1) Just meeting the existing annual and 24-hour standards of 12.0 $\mu g/m^3$ and 35 $\mu g/m^3$, and

(2) Just meeting potential alternative annual and 24-hour standards of 10.0 $\mu g/m^3$ and 30 $\mu g/m^3$.

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. In this risk assessment, two different adjustment approaches were applied to provide two outcomes that could represent potential bounding scenarios of $PM_{2.5}$

[39] As noted in section 3.3.2.1, while controlled human exposure studies provided 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 the studies were well-above the ambient concentrations typically measured in locations meeting the existing standards.

[40] The simulated air quality surface, which just meets both an alternative annual standard of 10.0 $\mu g/m^3$ and alternative 24-hour standard of 30 $\mu g/m^3$, was subset into areas that are controlled by either the alternative annual standard of 10.0 $\mu g/m^3$ or 24-hour standard of 30 $\mu g/m^3$ to assess risk associated with just meeting each alternative standard.

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

concentrations changes across the study area. The two adjustment approaches used to guide the generation of these modeled surfaces were:

- *Reductions in primary $PM_{2.5}$ (Pri-PM)*: This modeling approach simulates air quality scenarios of interest by preferentially adjusting direct/primary PM emissions. As such, the changes in $PM_{2.5}$ tend to be more localized near the direct emissions sources of PM.[42]
- *Reductions in secondary $PM_{2.5}$ (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 secondary $PM_{2.5}$. In this case, the reductions in $PM_{2.5}$ tend to be more evenly spread across a study area.[43]

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 $PM_{2.5}$ concentrations.

3.4.1.5 Study Area Selection

The following factors were considered most important when selecting U.S. study areas for inclusion in the risk assessment:

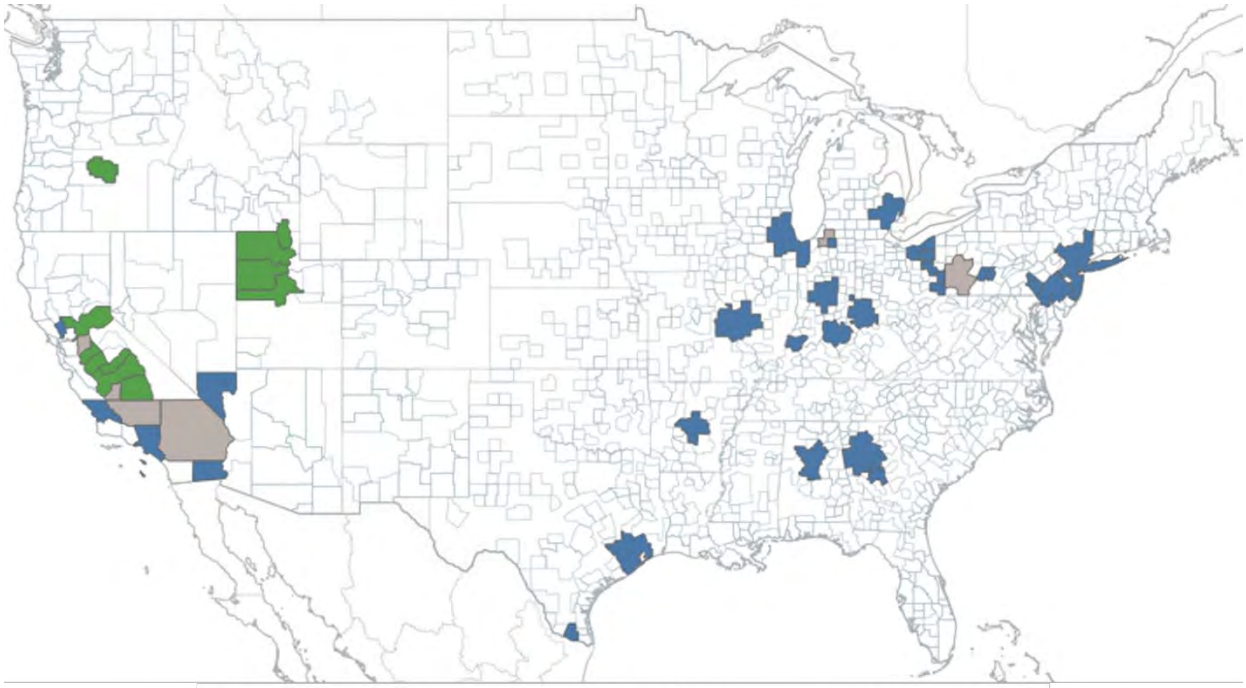
- *Available Ambient Monitors*: We have greater confidence in estimating and simulating air quality concentrations over areas with relatively dense ambient monitoring networks, as the modeled air quality surfaces can be compared with monitored concentrations (additional detail available in Appendix C, section C.1.4).
- *Geographical Diversity*: Risk assessments including areas that represent a variety of regions across the U.S. and a substantial portion of the U.S. population can be more representative.
- *Ambient $PM_{2.5}$ Air Quality Concentrations*: Based on 2014-2016 design values, only 16 CBSAs[44], also called urban study areas here, exceeded either or both the current annual and 24-hour $PM_{2.5}$ NAAQS. To include a larger portion of the U.S. in this risk assessment, we also identified CBSAs with ambient $PM_{2.5}$ concentrations below, but near, the current annual and/or 24-hour $PM_{2.5}$ NAAQS. 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 requiring 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). Areas that appeared to be strongly influenced by exceptional events were also excluded (section C.1.4). Using these criteria, 47 urban study areas were identified, which

[42] 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).

[43] 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. This behavior occurs in areas where emission changes in addition to NOx and SO2 would be needed to adjust design values to just meet the standard. (Appendix C, Figure C-19).

[44] 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.

include nearly 60 million people aged 30-99, or approximately 30% of the U.S population in this age range (Figure 3-16 and Appendix C, section C.1.3). Of the 47 study areas, there were 30 study areas where just meeting the current standards is controlled by the annual standard,[45] 11 study areas where just meeting the current standards is controlled by the daily standard,[46] and 6 study areas where the controlling standard differed depending on the air quality adjustment approach (Figure 3-16).[47]



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 3-16. Map of 47 urban study areas included in risk modeling.

[45] 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.

[46] 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.

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

3.4.1.6 At-Risk Analysis

To inform conclusions regarding the primary $PM_{2.5}$ standards that are "requisite" to protect public health (i.e., neither more nor less stringent than necessary; section 1.2) and provide an adequate margin of safety, it is important to consider the health risks of specific populations identified as at increased risk (at-risk) that would be allowed under current and alternative standards, recognizing associated uncertainties (section 3.4.1.8). Our consideration of estimated risks among potentially at-risk populations focuses on addressing the following policy-relevant questions:

- **How does $PM_{2.5}$ exposure and risk compare between demographic groups when air quality just meets the current and potential alternative primary $PM_{2.5}$ annual standards?**
- **To what extent are impacts estimated to change within each demographic group when air quality is adjusted to just meet potential alternative annual standards with lower levels?**

Assessing $PM_{2.5}$-attributable risk stratified by the value of another covariate (e.g., race or ethnicity) can provide insight into population-specific risk. As described in section 3.3.2, the 2019 ISA and draft ISA Supplement cite 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, 2019, p. 12-1; U.S. EPA, 2021a). Factors that may contribute to increased risk of $PM_{2.5}$-related health effects include lifestage (children and older adults), pre-existing diseases (cardiovascular disease and respiratory disease), race/ethnicity, and socioeconomic status. In considering the strength of the available scientific evidence and recognizing that this risk assessment is focused on the health endpoint of mortality, we assess long-term $PM_{2.5}$-attributable exposure and mortality risk, stratified by racial/ethnic demographics. Specifically, we evaluate exposure and risk, stratified by race-specific concentration-response functions when available, of White, Black, Asian, Native American, Non-Hispanic, and Hispanic individuals.

Concentration-response functions used in this at-risk analysis are from large, multicity U.S. epidemiologic studies that evaluate the relationship between $PM_{2.5}$ exposures and mortality. Eight epidemiologic long-term exposure studies of $PM_{2.5}$ exposure and all-cause, nonaccidental, or total mortality in nonwhite populations were identified in the 2019 ISA and draft ISA Supplement (U.S. EPA, 2019; U.S. EPA, 2021a). Associations from those eight studies relating long-term $PM_{2.5}$ exposure and mortality outcomes in nonwhite populations are available in Figure 3-17.

Specific epidemiologic studies and concentration-response functions used here to estimate risk were identified using criteria that take into account factors such as study design, geographic coverage, demographic populations, and health endpoints. Of the studies available

from the 2019 ISA, Di et al., 2017b was identified as best characterizing potentially at-risk non-White populations across the U.S.[48] Additional information on input parameters used in the at-risk analysis can be found in Appendix C, section C.3.

At-risk estimates presented in section 3.4.2.4, when considered alongside estimates of risk across all populations in the 47 study areas (sections 3.4.2.1, 3.4.2.2, and 3.4.2.3) are meant to inform conclusions on the primary annual $PM_{2.5}$ standards that would be requisite to protect the public health of nonwhite populations potentially at increased risk of long-term $PM_{2.5}$-related mortality effects.

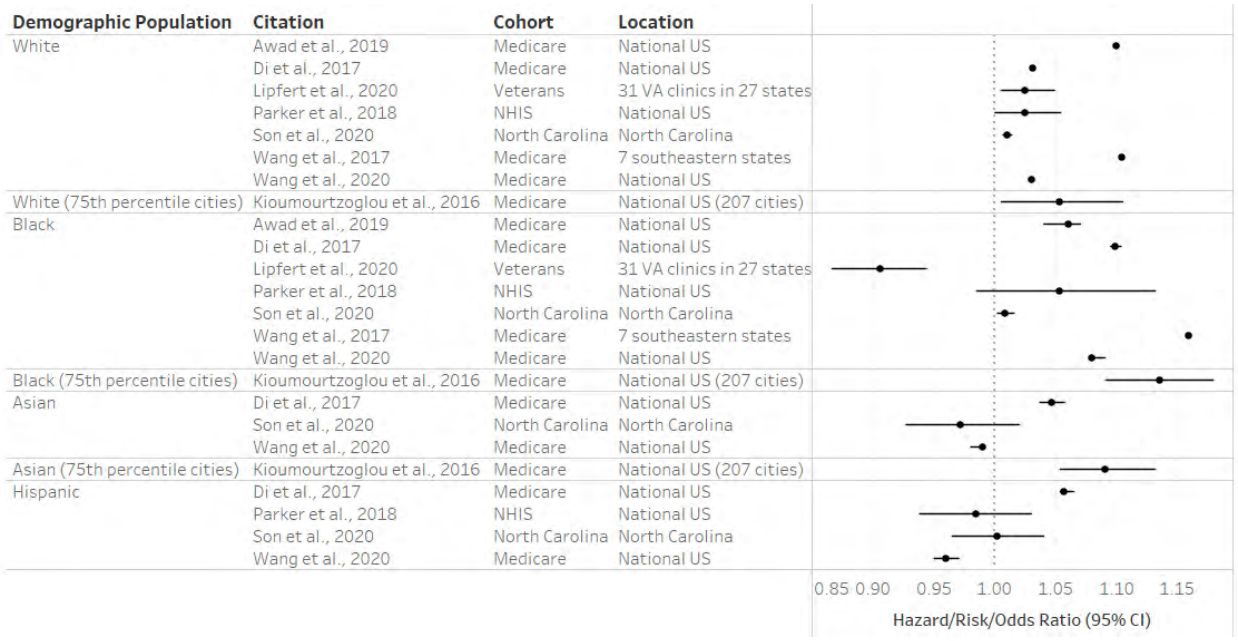


Figure 3-17. Available epidemiologic associations between long-term $PM_{2.5}$ exposure and mortality outcomes in demographic populations.[49]

3.4.1.7 Characterization of Variability and Uncertainty in the Risk Assessment

Both quantitative and qualitative methods have been used to characterize variability and uncertainty in the risk estimates (Appendix C, section C.3), including:

[48] Additional details on concentration-response function identification can be found in Appendix C, section C.3.2. Di et al., 2017b was identified as best characterizing potentially at-risk non-White populations across the U.S. using study and risk estimate criteria described in the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits TSD* (U.S. EPA, 2021b). Additional information on all available at-risk epidemiologic studies is available in Appendix C, section C.3.2.

[49] All studies estimated median or average long-term $PM_{2.5}$ exposures between 10-12 μg/m^3, other than Lipfert and Wyzga (2020), which reported an approximate average exposure concentration of 14 μg/m^3. Kioumourtzoglou et al., 2016 reported associations in cities ranking at or about the 75th percentile proportionally with regards to demographic population only. VA, Veterans Affairs; NHIS, National Health Insurance Service.

- *95th percentile confidence intervals*: We use an iterative Monte Carlo simulation that samples from the standard error associated with each epidemiologic concentration-response function. We present the resulting 2.5th and 97.5th percentile values from this distribution as a 95th percentile confidence interval around the risk estimate. Monte Carlo methods are a well-established means of characterizing random sampling error associated with concentration-response functions.
- *Health endpoint sensitivity analyses*: We include multiple concentration-response functions reflecting epidemiology studies differing in various ways, such as the population (e.g., geographic locations and demographics), exposure estimation methods (e.g., monitor-based or hybrid techniques), and potential confounders included in the epidemiologic model (e.g., ozone).[50]
- *Air quality adjustment sensitivity analyses*: We simulate just meeting the current and alternative standards using two approaches, which represent potential bounding scenarios of $PM_{2.5}$ concentration changes across the study areas. The Pri-PM adjustment method preferentially adjusts direct (i.e., primary, directly-emitted) $PM_{2.5}$ emissions, whereas the Sec-PM method preferentially adjusts SO_2 and NO_X precursor emissions to simulate changes in secondarily formed $PM_{2.5}$.
- *Qualitative uncertainty assessment:* We perform additional 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).

3.4.1.8 Characterization of Variability and Uncertainty in the At-Risk Analysis

While considering exposure and health risks of individual at-risk racial and ethnic populations can be policy-relevant, these estimates will be more uncertain than similar estimates from the overall risk assessment (sections 3.4.2.1 and 3.4.2.2). This is due to additional sources of uncertainty specific to the at-risk analysis, such as using concentration-response functions derived from smaller epidemiologic sample sizes, being combined with the sources of uncertainty that apply to the overall risk assessment. The augmentation of existing uncertainness is exemplified by the exposure estimates in the White populations in the simulated air quality scenarios. White populations make up a greater proportion of rural areas (~60% vs ~80%, USDA, 2018), and rural areas tend to have lower ambient $PM_{2.5}$ concentrations. Therefore, as these scenarios are restricted to the 47 urban study areas, we expect that the average exposure estimated in this assessment is an over-estimate of the overall national average exposure in the White population.

For characterizing risk in at-risk populations, we used air quality fields from the Pri-PM adjustment case alone, because the Pri-PM air quality adjustments are largely associated with emission reductions within the study areas, due to the local nature of air quality impacts from

[50] Additional information on long-term epidemiologic study identification can be found in the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits TSD* (U.S. EPA, 2021b). Specifically, additional information on the identified long-term epidemiologic studies can be found in the *Study Information Table* (U.S. EPA, 2021b).

primary PM sources[51]. In contrast, Sec-PM air quality adjustments may be strongly associated with sources located outside of the study areas. Since the at-risk analyses are performed for population groups within the 47 areas alone, the Pri-PM adjustment case (in which air quality adjustments are primarily associated with emission sources within the 47 areas) is most appropriate for this at-risk analysis. However, limiting the analysis to a single simulation decreases the potential representativeness of simulated $PM_{2.5}$ concentrations changes across the study area.

3.4.2 Results of the Risk Assessment

This section presents estimates of $PM_{2.5}$-associated mortality risks for populations in the identified urban study areas (additional results available in Appendix C, section C.2). Results are shown as point estimates with 95th percentile confidence intervals for air quality adjusted to simulate just meeting the current, and potential alternative, standards. We provide tables that include the total mortality risk associated with air quality just meeting the current or potential alternative standards, the change in mortality risk (also called delta risk) when moving from air quality just meeting the current standard to just meeting potential alternative standards, and the percent risk reduction when moving from air quality just meeting the current standard to just meeting potential alternative standards.[52] We also quantify the percent of baseline incidence, which estimates the percent of total incidence that is associated with ambient $PM_{2.5}$ exposure (e.g., percent of mortality attributable to $PM_{2.5}$ exposure out of all deaths in the specified population).[53] In addition to tables, we provide figures to illustrate how risks are distributed across annual average ambient PM_{25} concentrations. Figures present results for all-cause mortality associated with long-term $PM_{2.5}$ exposures, based on a key epidemiologic study by (Turner 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 urban study areas (section 3.4.2.1), the subset of 30 areas for which the annual $PM_{2.5}$ standard is controlling (section 3.4.2.2), and the subset of 11 areas for which the 24-hour $PM_{2.5}$ standard is controlling (section 3.4.2.3). Risk estimates from populations potentially at increased risk of PM-related effects are

[51] The Pri-PM and Sec-PM adjustment approaches are described in section 3.4.1.4.

[52] *Total risk* refers to risk associated with the full increment of exposure associated with each air quality scenario. 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. Percent risk reduction is calculated by dividing the delta risk by the total risk.

[53] In other words, the percent of the health effect attributable to $PM_{2.5}$ exposure. For example, risk results estimate that 6-8% of all-cause mortality in 2015 was associated with $PM_{2.5}$ exposure (Table 3-14).

available in section 3.4.2.4. Uncertainties in the risk assessment are summarized in section 3.4.2.5.

3.4.2.1 Summary of Risk Estimates for the Full Set of 47 Urban Study Areas

Risk estimates for the 47 urban study areas are presented in Table 3-14 and Table 3-15. Table 3-14 presents all-cause and non-accidental mortality risk estimates attributable to $PM_{2.5}$ when just meeting the current primary $PM_{2.5}$ standards and just meeting either an alternative modeled annual standard of 10.0 $\mu g/m^3$ or an alternative modeled 24-hour standard of 30 $\mu g/m^3$. Table 3-14 also provides the percent of total all-cause mortality attributable to $PM_{2.5}$ in 2015 estimated by each epidemiologic concentration-response function.

Table 3-15 presents the reduction in estimated risk when moving from air quality scenarios just meeting the current standard to air quality just meeting alternative standards. Areas are again subset into those just meeting either an alternative annual standard of 10.0 $\mu g/m^3$ or an alternative 24-hour standard of 30 $\mu g/m^3$, based on which standard is controlling in that study area. Smaller reductions estimated for the alternative 24-hour standard reflect the reduced number of study areas controlled by the 24-hour standard and the lesser population in those areas.

Key observations for the full set of 47 study areas from Table 3-14 and Table 3-15, which include approximately 30% of the U.S. population aged 30-99, are as follows:

- Substantially larger risk reductions are associated with lowering the annual standard than with lowering the 24-hour standard (Table 3-15). Impacts are estimated to decrease by 13-17% when air quality is adjusted to just meet an alternative annual standard with a level of 10.0 $\mu g/m^3$ or by 1-2% when adjusted to just meet an alternative 24-hour standard with a level of 30 $\mu g/m^3$. This corresponds to up to 7,440 (5,040-9,830) fewer deaths per year attributable to long-term $PM_{2.5}$ exposures.[54]
- Up to 45,100 deaths in 2015 are attributable to long-term $PM_{2.5}$ exposures associated with air quality just meeting the current annual and 24-hour $PM_{2.5}$ standards, with a 95th percentile confidence interval of 30,800-59,000. This constitutes up to 8% of total baseline mortality in adults age 30-99 (Table 3-14).

[54] 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 annual $PM_{2.5}$ design value of 12.0 $\mu g/m^3$ and air quality with a maximum three-year annual $PM_{2.5}$ 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 24-hour $PM_{2.5}$ design value of 35 $\mu g/m^3$ and air quality with a maximum three-year 24-hour $PM_{2.5}$ 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-hour 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 alternative standards evaluated.

- Short-term $PM_{2.5}$ exposures are estimated to be associated with up to 3,870 (2,570-5,160) deaths annually. This accounts for between 0.2-0.7% of mortality in adults age 30-99 in 2015.

Table 3-14. Estimates of $PM_{2.5}$-associated mortality for air quality adjusted to just meet the current or alternative standards (47 urban study areas).

Exposure	Study & Ages	Simulation Method	Total Mortality Under the Current Standard (12/35-0)	% of Baseline Mortality Attributable to the Current Standard	Total Mortality Under an Alternative Annual Standard (10-0)	Total Mortality Under an Alternative 24-Hr Standard (30-0)
Long-Term	Di (65-99)	Pri PM	40,600 (39,600 to 41,700)	7.4	35,400 (34,400 to 36,300)	40,100 (39,100 to 41,200)
		Sec PM	41,200 (40,200 to 42,300)	7.5	34,800 (33,900 to 35,700)	40,600 (39,500 to 41,600)
	Turner (30-99)	Pri PM	44,400 (30,300 to 58,200)	6.1	38,600 (26,300 to 50,700)	43,900 (30,000 to 57,500)
		Sec PM	45,100 (30,800 to 59,000)	6.2	38,000 (25,900 to 49,900)	44,400 (30,300 to 58,200)
Short-Term	Baxter (0-99)	Pri PM	2,490 (982 to 3,990)	0.4	2,160 (850 to 3,460)	2,460 (970 to 3,950)
		Sec PM	2,530 (997 to 4,050)	0.4	2,120 (837 to 3,400)	2,490 (982 to 3,990)
	Ito (0-99)	Pri PM	1,180 (-15.8 to 2,370)	0.2	1,020 (-13.7 to 2,050)	1,160 (-15.6 to 2,340)
		Sec PM	1,200 (-16.0 to 2,400)	0.2	1,000 (-13.5 to 2,020)	1,180 (-15.8 to 2,370)
	Zanobetti (65-99)	Pri PM	3,810 (2,530 to 5,080)	0.7	3,300 (2,190 to 4,400)	3,760 (2,500 to 5,020)
		Sec PM	3,870 (2,570 to 5,160)	0.7	3,250 (2,160 to 4,330)	3,810 (2,530 to 5,070)

Table 3-15. Estimated reduction in $PM_{2.5}$-associated mortality for alternative annual and 24-hour standards (47 urban study areas).

Exposure	Study & Ages	Simulation Method	Risk Change When Moving from the Current to an Alternative Annual Standard of 10	Risk Change When Moving from the Current to an Alternative 24-Hr Standard of 30	% Risk Reduction When Moving from the Current to an Alternative Annual Standard of 10	Risk Change When Moving from the Current to an Alternative 24-Hr Standard of 30
Long-Term	Di (65-99)	Pri PM	5,630 (5,490 to 5,780)	501 (488 to 514)	13.9	1.2
		Sec PM	6,820 (6,640 to 7,000)	675 (657 to 692)	16.6	1.6
	Turner (30-99)	Pri PM	6,120 (4,140 to 8,090)	555 (375 to 734)	13.8	1.2
		Sec PM	7,440 (5,040 to 9,830)	714 (483 to 943)	16.5	1.6
Short-Term	Baxter (0-99)	Pri PM	335 (132 to 537)	30.2 (11.9 to 48.4)	13.4	1.2
		Sec PM	408 (160 to 654)	38.7 (15.2 to 62.1)	16.1	1.5
	Ito (0-99)	Pri PM	158 (-2.12 to 317)	14.4 (-0.194 to 29.0)	13.4	1.2
		Sec PM	192 (-2.58 to 386)	18.4 (-0.246 to 36.9)	16.1	1.5
	Zanobetti (65-99)	Pri PM	513 (341 to 684)	45.5 (30.2 to 60.7)	13.5	1.2
		Sec PM	622 (413 to 830)	61.5 (40.8 to 82.0)	16.1	1.6

3.4.2.2 Summary of Risk Estimates for the 30 Areas Controlled by the Annual Standard

This section presents the results for the range of alternative annual standard levels for the 30 urban study areas for which the annual standard is controlling under all air quality scenarios evaluated.[55,56] Table 3-16 presents total all-cause and non-accidental mortality risk estimates attributable to $PM_{2.5}$ when just meeting the current standard of 12.0 $\mu g/m^3$ and just meeting potential alternative annual standards with levels of 11.0, 10.0, 9.0, and 8.0 $\mu g/m^3$. It also provides the percent of baseline risk attributable to $PM_{2.5}$ when just meeting the current annual standard. Table 3-17 presents the reduction in estimated mortality incidence and percent of risk reduction when moving from air quality scenarios just meeting the current annual standard to air quality just meeting the various alternative annual standards.

After presenting mortality impact results from the various epidemiologic studies in Table 3-16 and Table 3-17, we focus on a single epidemiologic concentration-response function from

[55] These 30 areas controlled by the annual standard under all scenarios evaluated include a population of approximately 48 million adults aged 30-99, which corresponds to about 75% of the population included in the full set of 47 areas or approximately 25% of the total U.S. population.

[56] Alternative annual air quality surfaces in addition to the modeled surface just meeting 10.0 $\mu g/m^3$ were developed using interpolation and extrapolation of modeled $PM_{2.5}$ concentrations (section 3.4.1.4 and Appendix C section C.1.4).

Turner et al. (2016) to provide additional insight into the distribution of health impacts across long-term ambient $PM_{2.5}$ concentrations.[57] Figure 3-18 presents distributions of total risk attributable to annual $PM_{2.5}$ concentration bins of 1 $\mu g/m^3$ when just meeting the current and alternative annual standards.[58] Figure 3-19 presents distributions as a heat map, again binned in 1 $\mu g/m^3$increments, associated with moving from just meeting the current standard to just meeting each alternative annual standard.[59]

Drawing from the information in Table 3-16, Table 3-17, Figure 3-18, and Figure 3-19 for the subset of 30 study areas (approximately 25% of the U.S. population) in which the annual standard is controlling, we note the following key observations:

- There is a potential for significant public health impacts in locations just meeting the current primary $PM_{2.5}$ standards. The majority of $PM_{2.5}$-associated deaths 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.
- Compared to the current annual standards, air quality adjusted to meet alternative annual standards with lower levels is associated with reductions in estimated all-cause mortality impacts (i.e., 7-9% reduction for an alternative annual level of 11.0 $\mu g/m^3$, 15-19% reduction for a level of 10.0 $\mu g/m^3$, 22-28% reduction for a level of 9.0 $\mu g/m^3$, and 30-37% reduction for a level of 8.0 $\mu g/m^3$) (Table 3-17 and Figure 3-18).
- 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 alternative annual standard, the majority of risk reduction occurs in grid cells with ambient $PM_{2.5}$ concentrations between the alternative standard and 2 $\mu g/m^3$ lower (e.g., for air quality adjusted to simulate just meeting an annual standard with a level of 8.0 $\mu g/m^3$, the

[57] The *Estimating $PM_{2.5}$ and Ozone- Attributable Health Benefits TSD* details the approach and criteria used to identify studies and concentration-response functions from the 2019 ISA used in this risk assessment (U.S. EPA, 2021b). Briefly, two studies were again identified as best characterizing mortality risk across the U.S., Di et al., 2017b and Turner et al., 2016. While both studies used sophisticated techniques to relate $PM_{2.5}$ exposure and all-cause mortality across large portions of the U.S population, Di et al., 2017b evaluated Medicare beneficiaries aged 65+, whereas Turner et al., 2016 included adults ages 30+ from the ACS cohort. The concentration-response function identified in the *Estimating $PM_{2.5}$ and Ozone- Attributable Health Benefits TSD* (U.S. EPA, 2021b) from Turner et al., 2016 was selected for use in this risk assessment due to the broader age range, although it should be noted that the concentration-response function from Di et al., 2017b typically generates mortality risk estimates within approximately 5% of the Turner et al., 2016 concentration-response function.

[58] Bins correspond to the lower whole number and include up to, but not including the next whole number. For example, the bin for 8 $\mu g/m^3$, includes all risk occurring at $PM_{2.5}$ concentrations from 8.00 $\mu g/m^3$ to 8.99 $\mu g/m^3$. Previously this data was presented as a line graph, which can be found in Appendix C, Figure C-30.

[59] As noted above, Figure 3-18 and Figure 3-19 present estimates of all-cause mortality associated with long-term $PM_{2.5}$ exposures, based on the study by Turner et al., 2016.

majority of risk reduction occurs in grid cells with ambient $PM_{2.5}$ concentrations between 6 and 8 $\mu g/m^3$) (Figure 3-18 and Figure 3-19).[60]

- For air quality just meeting the current annual standard, long-term $PM_{2.5}$ exposures are estimated to be associated with as many as 39,000 (26,000-51,000) total deaths from long-term exposure annually, accounting for approximately 6-8% of baseline mortality.

Table 3-16. 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.

Exposure	Study & Ages	Simulation Method	Total Risk Under the Current Standard (12/35-0)	% of Baseline Risk Attributable to the Current Standard	Total Risk Under an Alternative Annual Standard (11-0)	Total Risk Under an Alternative Annual Standard (10-0)	Total Risk Under an Alternative Annual Standard (9-0)	Total Risk Under an Alternative Annual Standard (8-0)
Long-Term	Di (65-99)	Pri PM	34,900 (34,000 to 35,800)	7.6	32,400 (31,600 to 33,300)	29,900 (29,200 to 30,700)	27,400 (26,700 to 28,100)	24,900 (24,200 to 25,500)
		Sec PM	35,600 (34,700 to 36,500)	7.7	32,500 (31,700 to 33,300)	29,400 (28,600 to 30,100)	26,300 (25,600 to 26,900)	23,100 (22,500 to 23,700)
	Turner (30-99)	Pri PM	38,200 (26,100 to 50,100)	6.3	35,500 (24,200 to 46,500)	32,700 (22,300 to 42,900)	29,900 (20,400 to 39,300)	27,200 (18,500 to 35,700)
		Sec PM	38,900 (26,600 to 51,000)	6.4	35,500 (24,200 to 46,600)	32,100 (21,900 to 42,100)	28,700 (19,500 to 37,600)	25,200 (17,100 to 33,100)
Short-Term	Baxter (0-99)	Pri PM	2,150 (846 to 3,440)	0.4	1,990 (784 to 3,190)	1,830 (721 to 2,930)	1,670 (658 to 2,680)	1,510 (595 to 2,420)
		Sec PM	2,190 (862 to 3,510)	0.4	1,990 (785 to 3,190)	1,790 (707 to 2,880)	1,600 (630 to 2,560)	1,400 (552 to 2,250)
	Ito (0-99)	Pri PM	1,010 (-13.6 to 2,040)	0.2	939 (-12.6 to 1,880)	864 (-11.6 to 1,730)	789 (-10.6 to 1,580)	713 (-9.57 to 1,430)
		Sec PM	1,030 (-13.9 to 2,070)	0.2	940 (-12.6 to 1,890)	847 (-11.4 to 1,700)	754 (-10.1 to 1,510)	661 (-8.87 to 1,330)
	Zanobetti (65-99)	Pri PM	3,280 (2,180 to 4,370)	0.7	3,040 (2,020 to 4,050)	2,790 (1,860 to 3,730)	2,550 (1,700 to 3,400)	2,310 (1,540 to 3,080)
		Sec PM	3,340 (2,220 to 4,450)	0.7	3,040 (2,020 to 4,050)	2,740 (1,820 to 3,650)	2,440 (1,620 to 3,260)	2,140 (1,420 to 2,860)

[60] Compared to adjusting primary $PM_{2.5}$ emissions, adjustment of PM precursor emissions resulted in substantially larger estimated risk reductions at 7 $\mu g/m^3$.

Table 3-17. 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.

Exposure	Study & Ages	Simulation Method	Risk Change When Moving from the Current to an Alternative Annual Standard of 11	Risk Change When Moving from the Current to an Alternative Annual Standard of 10	Risk Change When Moving from the Current to an Alternative Annual Standard of 9	Risk Change When Moving from the Current to an Alternative Annual Standard of 8	% Risk Reduction When Moving from the Current to an Alternative Annual Standard of 11	% Risk Reduction When Moving from the Current to an Alternative Annual Standard of 10	% Risk Reduction When Moving from the Current to an Alternative Annual Standard of 9	% Risk Reduction When Moving from the Current to an Alternative Annual Standard of 8
Long-Term	Di (65-99)	Pri PM	2,680 (2,610 to 2,750)	5,350 (5,210 to 5,490)	8,000 (7,790 to 8,210)	10,600 (10,400 to 10,900)	7.7	15.3	22.9	30.5
		Sec PM	3,320 (3,230 to 3,400)	6,610 (6,440 to 6,780)	9,880 (9,620 to 10,100)	13,100 (12,800 to 13,500)	9.3	18.6	27.8	36.9
	Turner (30-99)	Pri PM	2,920 (1,970 to 3,860)	5,830 (3,940 to 7,700)	8,720 (5,900 to 11,500)	11,600 (7,860 to 15,300)	7.6	15.2	22.8	30.3
		Sec PM	3,610 (2,440 to 4,770)	7,200 (4,870 to 9,510)	10,800 (7,290 to 14,200)	14,300 (9,710 to 18,900)	9.3	18.5	27.7	36.8
Short-Term	Baxter (0-99)	Pri PM	160 (62.8 to 256)	319 (126 to 512)	478 (188 to 767)	638 (251 to 1,020)	7.4	14.9	22.3	29.7
		Sec PM	197 (77.6 to 316)	394 (155 to 632)	592 (233 to 948)	789 (310 to 1,260)	9.0	18.0	27.0	36.0
	Ito (0-99)	Pri PM	75.2 (-1.01 to 151)	150 (-2.02 to 302)	226 (-3.03 to 453)	301 (-4.03 to 604)	7.4	14.8	22.3	29.7
		Sec PM	93.1 (-1.25 to 187)	186 (-2.49 to 374)	279 (-3.74 to 561)	372 (-4.99 to 748)	9.0	18.0	27.0	36.0
	Zanobetti (65-99)	Pri PM	244 (162 to 325)	487 (324 to 650)	731 (486 to 975)	974 (647 to 1,300)	7.4	14.9	22.3	29.7
		Sec PM	301 (200 to 402)	603 (400 to 804)	904 (600 to 1,210)	1,200 (800 to 1,610)	9.0	18.0	27.0	36.0

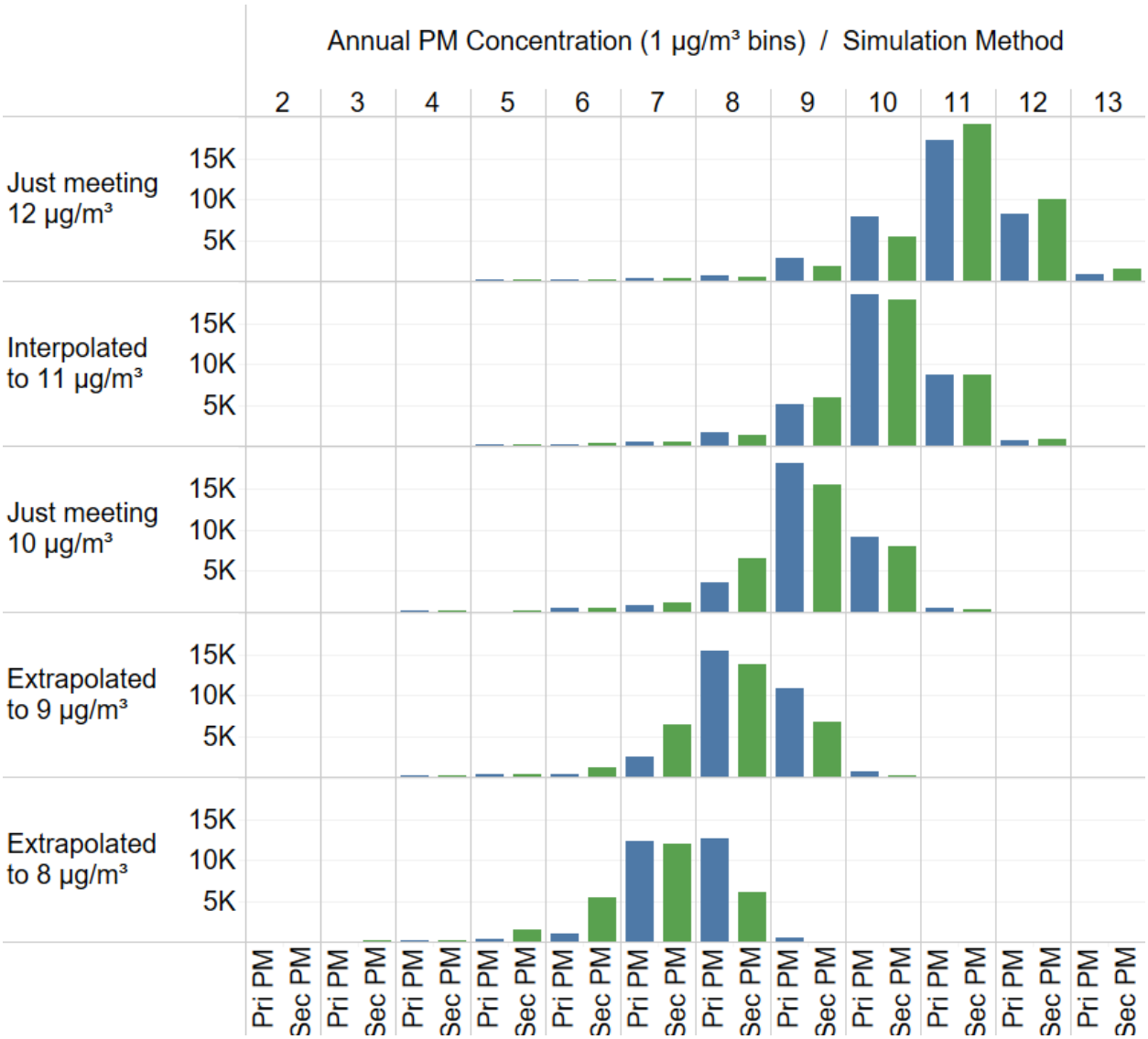


Figure 3-18. Distribution of total risk estimates ($PM_{2.5}$-attibutable mortality) for the current and alternative annual standards for the subset of 30 urban study areas where the annual standard is controlling (blue and green bars represent the Pri-$PM_{2.5}$ and Sec-$PM_{2.5}$ estimates, respectively).[61]

[61] Risk is estimated in this figure using Turner et al., 2016. Risk estimates are rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m^3 includes risk occurring at 10.0-10.9 µg/m^3). 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/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 µg/m^3, a single year can have grid cells with annual average concentrations above or below 12.0 µg/m^3.

Annual Standard Change	Simulation Method	Annual PM Concentration of Lower Standard (1 µg/m³ bins) 2	3	4	5	6	7	8	9	10	11	12	Sum
12-11 (interpolated) µg/m³	Pri PM		0	3	11	17	39	110	381	1,534	763	62	2,920
	Sec PM		0	4	9	26	40	122	628	1,836	858	89	3,611
12-10 µg/m³	Pri PM		1	18	12	81	116	569	3,205	1,720	103		5,826
	Sec PM		1	23	23	89	287	1,632	3,377	1,681	87		7,201
12-9 (extrapolated) µg/m³	Pri PM		3	27	82	106	596	4,467	3,252	185			8,718
	Sec PM	0	5	48	98	529	2,754	4,953	2,334	47			10,768
12-8 (extrapolated) µg/m³	Pri PM	0	11	85	161	368	5,324	5,408	238				11,595
	Sec PM	0	50	129	1,116	3,527	6,390	3,101					14,314

Figure 3-19. 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, 9.0, and 8.0 µg/m³ for the subset of 30 urban study areas where the annual standard is controlling.[62]

3.4.2.3 Summary of Risk Estimates for the 11 Areas Controlled by the 24-Hour Standard

Table 3-18 presents annual risk information for the subset of 11 urban study areas in which the 24-hour standard controls the simulated attainment of all modeled standard levels.[63] For air quality just meeting the current 24-hour standard, $PM_{2.5}$ exposures are estimated to be associated with as many as 2,570 (1,750-3,370) deaths annually, accounting for up to 7% of the baseline mortality in those 11 areas. Compared to the current standard, air quality just meeting an alternative 24-hr standard with a level of 30 µg/m³ is associated with reductions in estimated risk of 9-13%.

[62] 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 occurring at PM concentrations of 6.00 to 6.99 are shown in the bin at 6). Risk is estimated in this figure using Turner et al., 2016.

[63] These 11 areas controlled by the 24-hour standard under all scenarios evaluated include a population of approximately 10 million adults aged 30-99, or about 17% of the population included in the full set of 47 areas.

Table 3-18. 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.

Exposure	Study & Ages	Simulation Method	Total Risk Under the Current Standard (12/35-0)	% of Baseline	Total Risk Under an Alternative Annual Standard (30-0)	Risk Change When Moving from the Current to an Alternative 24-Hr Standard of 30	% Risk Reduction When Moving from the Current to an Alternative 24-Hr Standard of 30
Long-Term	Di (65-99)	Pri PM	2,320 (2,260 to 2,380)	6.7	2,040 (1,990 to 2,090)	304 (296 to 312)	13.1
		Sec PM	2,300 (2,250 to 2,360)	6.7	2,100 (2,050 to 2,150)	218 (212 to 224)	9.4
	Turner (30-99)	Pri PM	2,570 (1,750 to 3,370)	5.6	2,250 (1,530 to 2,960)	334 (226 to 442)	13.0
		Sec PM	2,550 (1,740 to 3,340)	5.6	2,320 (1,580 to 3,050)	241 (163 to 318)	9.4
Short-Term	Baxter (0-99)	Pri PM	142 (56.1 to 228)	0.3	124 (49.0 to 199)	18.1 (7.11 to 29.0)	12.7
		Sec PM	141 (55.6 to 226)	0.3	128 (50.5 to 206)	13.0 (5.12 to 20.9)	9.2
	Ito (0-99)	Pri PM	68.6 (-0.920 to 138)	0.1	59.9 (-0.803 to 120)	8.70 (-0.117 to 17.5)	12.7
		Sec PM	68.0 (-0.912 to 137)	0.1	61.8 (-0.828 to 124)	6.25 (-0.0838 to 12.6)	9.2
	Zanobetti (65-99)	Pri PM	217 (145 to 290)	0.6	190 (126 to 253)	27.7 (18.4 to 36.9)	12.7
		Sec PM	216 (143 to 287)	0.6	196 (130 to 261)	19.8 (13.1 to 26.4)	9.2

3.4.2.4 Summary of Risk Estimates for At-Risk Populations

Potential at-risk populations are summarized in section 3.3.2. Given that this risk and exposure assessment focuses on mortality endpoints, a quantitative assessment is supported by evidence in the 2019 ISA and draft ISA Supplement for racial and ethnic differences in $PM_{2.5}$ exposures and in $PM_{2.5}$-related health risk supports a quantitative assessment (U.S. EPA, 2019, section 12.5.4, U.S. EPA, 2021a, section 3.3.3.2).[64] Evidence strongly supports that non-White populations, such as Black and Hispanic populations, have higher $PM_{2.5}$ exposures than White and non-Hispanic populations, respectively, thus contributing to increased risk of PM-related effects. Additionally, Di et al., 2017b provides race- and ethnicity-stratified concentration-response functions for ages 65 and over. Therefore, we quantitatively assess risk for certain

[64] For characterizing risk in at-risk populations, we used air quality fields from the Pri-PM adjustment case alone. In the Pri-PM case, the air quality adjustments for a given area are largely associated with emission reductions within that area due to the local nature of air quality impacts from primary PM sources. For the Sec-PM case, the air quality adjustments may be strongly associated with sources located outside of the area. Since the at-risk analyses are performed for population groups within the 47 areas alone, the Pri-PM adjustment case (in which air quality adjustments are primarily associated with emission sources within the 47 areas) is most appropriate for the at-risk analysis.

racial and ethnic populations of older adults in the full set of 47 areas and the subset of 30 areas controlled by the annual $PM_{2.5}$ standard under all Pri-PM air quality simulations evaluated.[65] Additional information on this at-risk analysis is available throughout Appendix C, section C.2.

For this analysis, we first compare the estimated changes in air quality occurring within each demographic population when just meeting current and alternative annual $PM_{2.5}$ standards (Figure 3-20, left side).[66] Across all simulated air quality scenarios in the full set of 47 and subset of 30 study areas, Blacks experience the highest average $PM_{2.5}$ concentrations of the demographic groups analyzed. This increase was typically around 2-5% and was highest in modeling scenarios just meeting the current suite of standards. Native American populations typically experienced the lowest average $PM_{2.5}$ concentrations, especially in the full set of 47 study areas. White, Hispanic, and Asian populations were exposed to fairly similar average $PM_{2.5}$ concentrations, although White populations tended to be at the higher end of that range in the subset of 30 areas and the lower end of that range in the full set of 47 areas. Additionally, there is comparatively less disproportionate exposure between demographic populations as the alternative annual standard decreases.

While exposure is an important aspect to evaluate when considering potentially disproportionate impacts, risk estimates provide additional information. Notably, risk estimates also generate information regarding:

- The number of people affected by the air pollution reduction. In this instance, the population is further divided by demographic group.
- The relationship between exposure and health impact baseline incidence rates, or more specifically, the percentage change in the risk of an adverse health effect due to a one-unit change in ambient air pollution. These concentration-response functions are generally derived from epidemiologic studies.
- The average number of people who die in a given population over a given period of time. This is commonly referred to as the baseline mortality incidence rate.

For this quantitative analysis of demographic populations potentially at increased risk of $PM_{2.5}$ exposure, we utilize race-specific, or race-stratified, concentration-response functions and

[65] Each individual is categorized by both race and ethnicity in this analysis. In other words, the sum of White, Black, Asian, and Native American individuals equals the total population, as well as the sum of Hispanic and non-Hispanic individuals. Though Di et al., 2017b did not provide a non-Hispanic concentration-response relationship, results for non-Hispanics appears similar to Whites when the overall concentration-response relationship was applied to non-Hispanics (Appendix C Figures C-33 and C-34).

[66] Changes in air quality are estimated using the same approach used in the general risk assessment (sections 3.4.2.1, 3.4.2.2, and 3.4.2.3), summarized in section 3.4.1.4 and detailed in Appendix C.

baseline incidence rates, to more accurately estimate risk within each demographic group.[67] Population-normalized mortality risk occurring within each demographic population is available on the right side of Figure 3-20. Across all scenarios and demographic groups evaluated, Black populations are associated with the largest $PM_{2.5}$-attributable mortality risk rate per 100,000 people. An example of the 95th percentile confidence interval is available in Appendix Figure C-32.

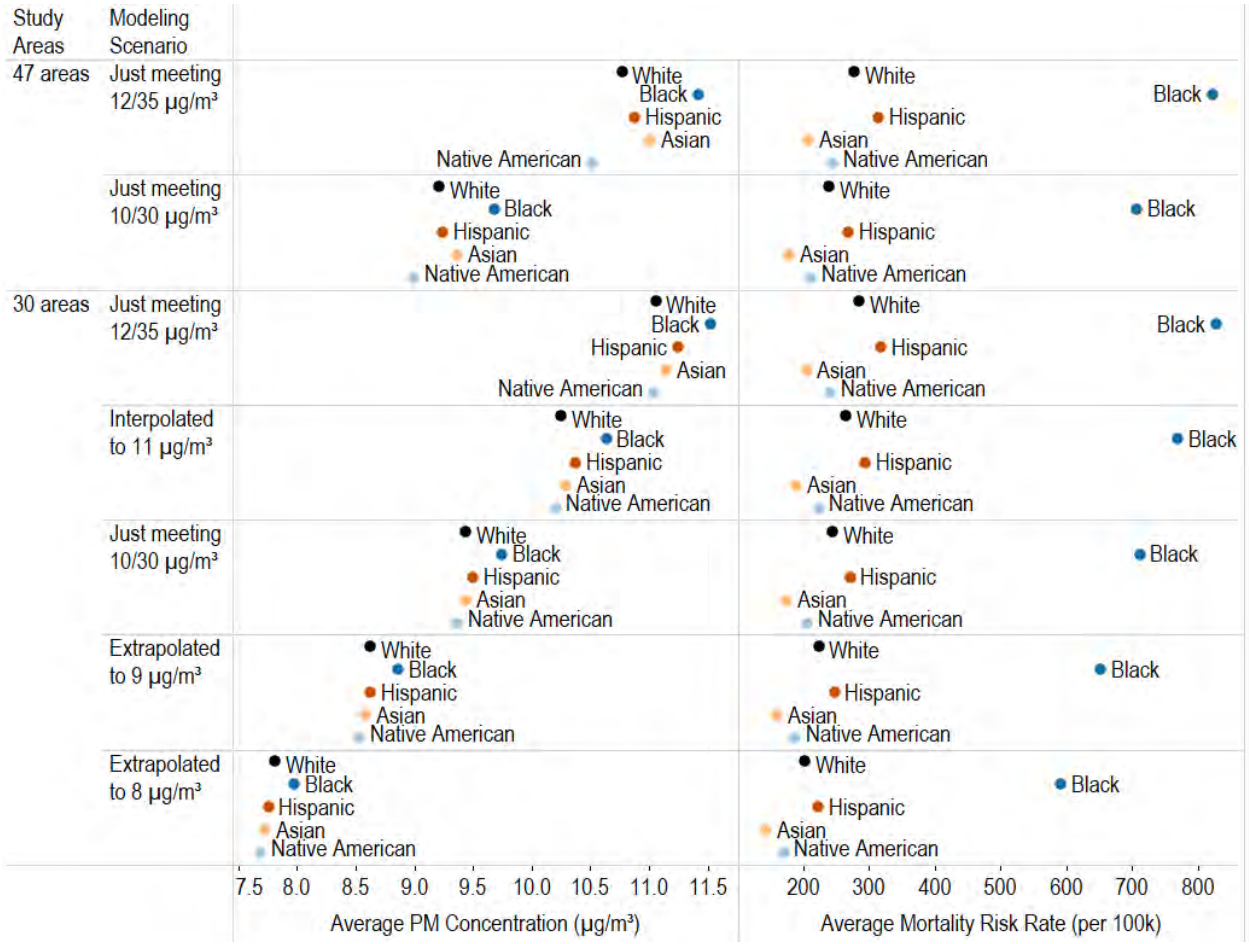


Figure 3-20. Average $PM_{2.5}$ exposure concentration and $PM_{2.5}$-attributable risk estimates by demographic population when just meeting current or alternative $PM_{2.5}$ standards.

We next estimate demographic-specific average exposure and risk changes when modeled air quality shifts from just meeting the current annual standard to just meeting potential

[67] Information on how the race-stratified concentration-response functions and baseline incidence rates impact the results can be found in Appendix C, section C.4. Briefly, race-stratified concentration-response functions increased risk estimated in nonwhite populations, with the greatest magnitude increase occurring in Black populations, and decreased risk estimated in White populations. Race-stratified baseline incidence rates decreased risk estimated in all demographic populations analyzed, with the greatest magnitude decreases occurring in White and Black populations.

alternative annual standard scenarios (Figure 3-21). Simulated $PM_{2.5}$ concentration reductions are shown on the left side of the figure and reductions in population-normalized mortality risk are shown on the right side. As the alternative annual PM standard decreases in the subset of 30 areas controlled by the annual standard, the average reduction in $PM_{2.5}$ concentration and mortality risk rates increase across all demographic populations assessed.

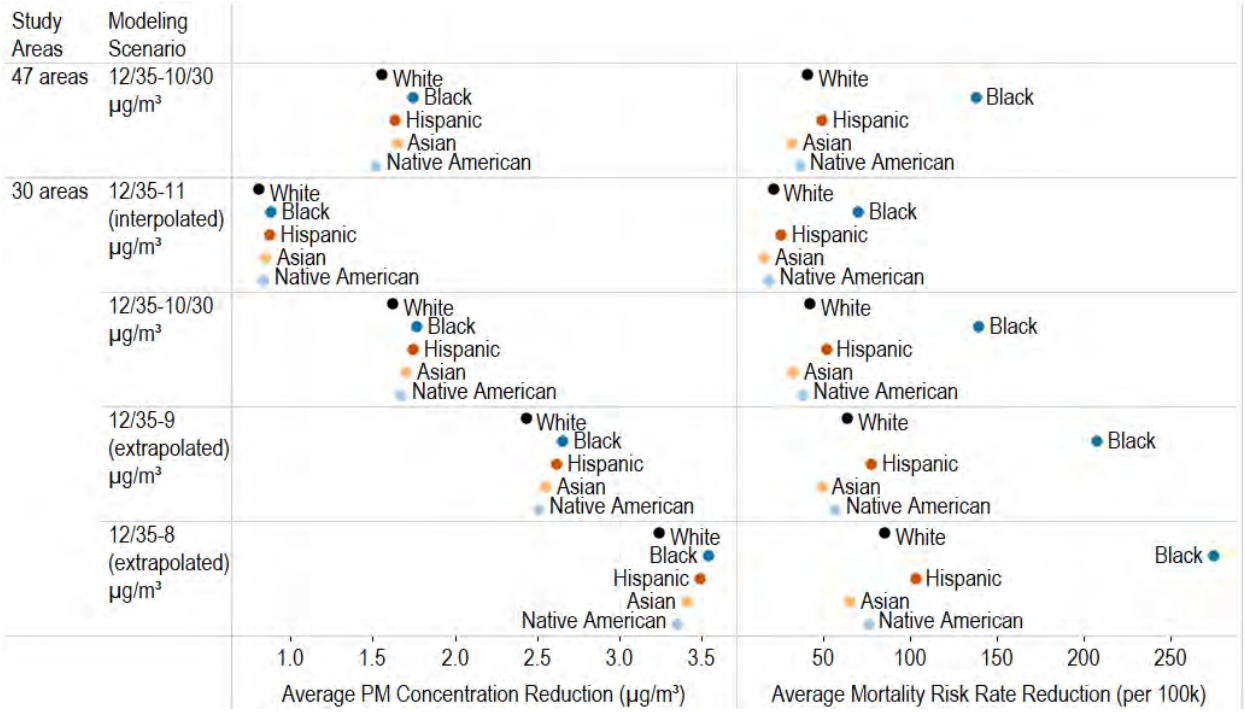


Figure 3-21. Average change in $PM_{2.5}$ exposure concentration and $PM_{2.5}$-attributable mortality risk estimates by demographic population when moving from the current to alternative $PM_{2.5}$ standards.

We also directly compare the reductions in average national $PM_{2.5}$ concentrations and risk rates within each demographic population. Table 3-19 and Table 3-20 provide the percent of national average $PM_{2.5}$-attributable exposures and risk reductions, when shifting from the current annual $PM_{2.5}$ standard (12.0 µg/m^3) to potential alternative annual $PM_{2.5}$ standards (11.0 µg/m^3, 10.0 µg/m^3, 9.0 µg/m^3, and 8.0 µg/m^3). The percent $PM_{2.5}$ and risk reductions are greater in the Black population than in the White population for each alternative standard evaluated for both the full set of study areas and the subset controlled by the annual standard. Additionally, the difference in percent risk reduction increases more in Blacks than in Whites as the potential alternative annual standard decreases. In other words, Blacks will experience proportionally greater benefit from successively lower annual standards, although even at an annual standard of 8 µg/m^3 Blacks will experience higher rates of premature mortality risk from $PM_{2.5}$ exposure than Whites.

Table 3-19. Average national percent $PM_{2.5}$ reduction in demographic populations aged 65 and over residing in the full set of 47 study areas and subset of 30 study areas controlled by the annual standard.

Ethnicity & Race	% PM Reduction from 12 μg/m³ to 11 (interpolated) μg/m³	% PM Reduction from 12 μg/m³ to 10 μg/m³		% PM Reduction from 12 μg/m³ to 9 (extrapolated) μg/m³	% PM Reduction from 12 μg/m³ to 8 (extrapolated) μg/m³
	30 areas	47 areas	30 areas	30 areas	30 areas
White	7	14	15	22	29
Black	8	15	15	23	31
Hispanic	8	15	16	23	31
Asian	8	15	15	23	31
Native American	8	14	15	23	30

Table 3-20. Average national percent $PM_{2.5}$ risk reduction in demographic populations aged 65 and over residing in the full set of 47 study areas and subset of 30 study areas controlled by the annual standard.

Ethnicity & Race	% Risk Reduction from 12 μg/m³ to 11 (interpolated) μg/m³	% Risk Reduction from 12 μg/m³ to 10 μg/m³		% Risk Reduction from 12 μg/m³ to 9 (extrapolated) μg/m³	% Risk Reduction from 12 μg/m³ to 8 (extrapolated) μg/m³
	30 areas	47 areas	30 areas	30 areas	30 areas
White	8	15	15	23	30
Black	9	17	17	25	33
Hispanic	8	16	16	25	33
Asian	8	16	16	24	32
Native American	8	15	16	24	32

While average exposure concentrations and risk estimates across demographic populations can convey some insight regarding whether certain populations may be disproportionately impacted, distributional information, while more complex, can provide a more comprehensive understanding of the analytical results. As such, we compare both estimated $PM_{2.5}$ exposures and mortality risk rates per 100k individuals to the running sum of each demographic population. To permit the direct comparison of demographic populations with different absolute numbers, populations are expressed as a percentage in Figure 3-22 and Figure 3-23.[68]

In both Figure 3-22 and Figure 3-23, $PM_{2.5}$ concentration information is on the left side and mortality risk estimates are on the right side. Recent conditions (2015) information for both exposure and risk can be found in Appendix C, section C.4, as well as sensitivity analyses

[68] Information on the absolute number of all-cause premature mortality cases within each racial and ethnic population demographic can be found in Appendix C Tables C-12 and C-13.

investigating the impact of race-stratified concentration-response functions and baseline incidence rates on the results. Cumulative distribution plots of $PM_{2.5}$ concentrations and population-normalized mortality risk reductions when shifting from the current to an alternative annual standard are available in Figure 3-23.

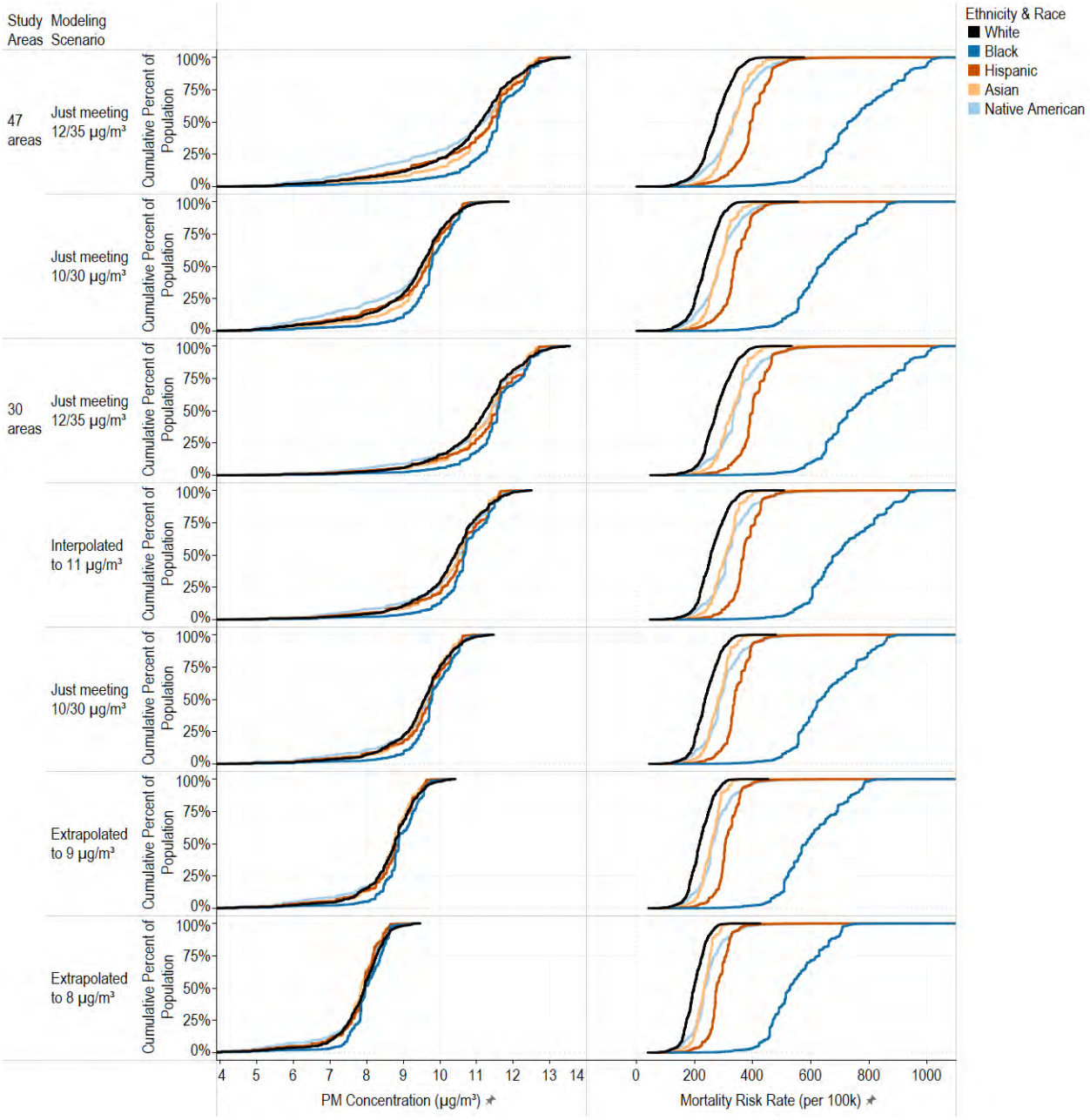


Figure 3-22. $PM_{2.5}$ exposure concentrations and $PM_{2.5}$-attributable mortality risk estimates by demographic population when just meeting current or alternative $PM_{2.5}$ standards.

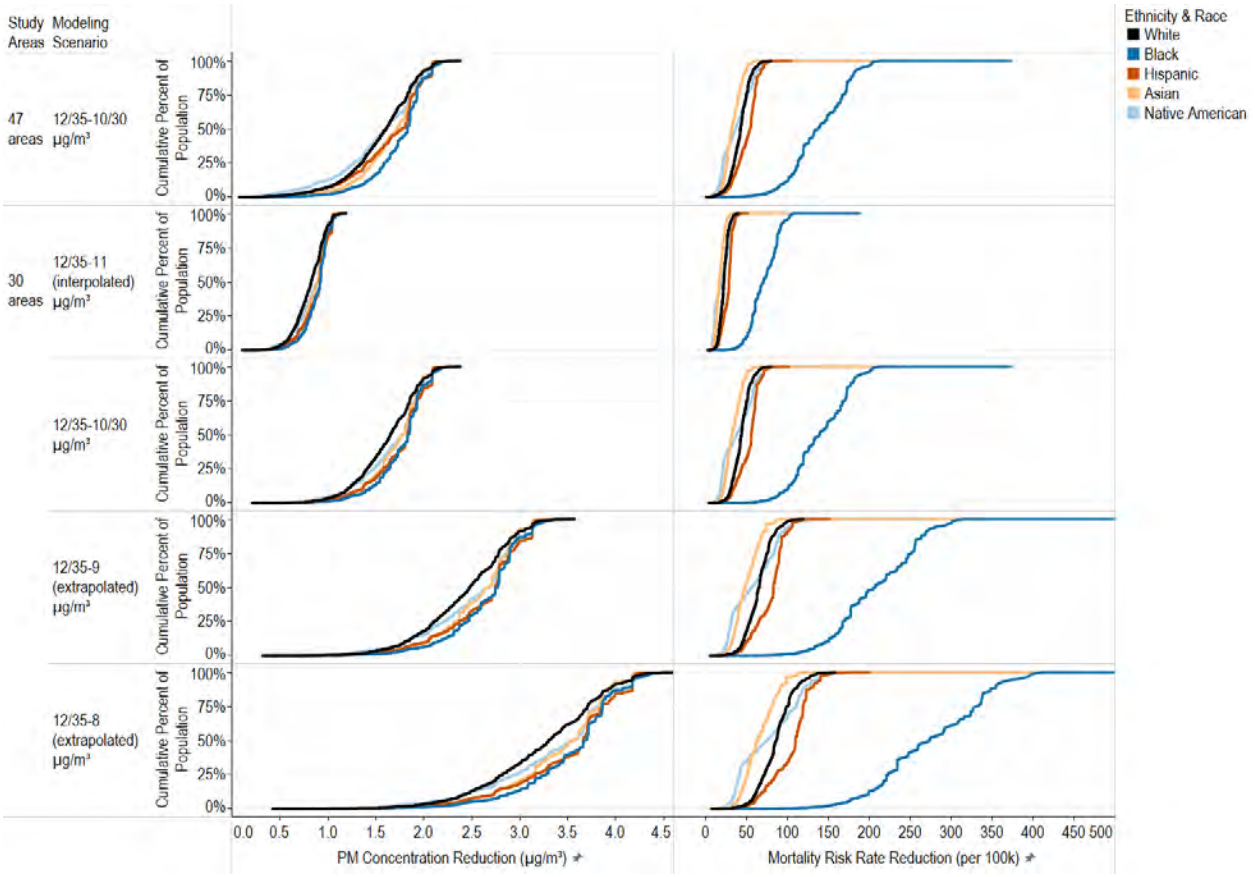


Figure 3-23. Change in $PM_{2.5}$ exposure concentrations and $PM_{2.5}$-attributable mortality risk estimates by demographic population when moving from the current to alternative $PM_{2.5}$ standards.

3.4.2.5 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 multiple concentration-response functions for the same health endpoint: 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. In most instances in this risk assessment, the concentration-response function used has only a small impact on risk estimates.
- Evaluating multiple methods for simulating air quality scenarios: The approach used to adjust air quality (i.e., Pri-PM and Sec-PM adjustments) has some impact on overall estimates of risk (e.g., Table 3-14). However, the adjustment approach has a larger impact on the distribution of risk reductions, particularly for alternative annual levels of 9.0 and 8.0 $\mu g/m^3$ (Figure 3-19).

- Characterizing the 95th percentile 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 (Table 3-14), with some concentration-response functions 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 all-cause mortality estimates based on Turner 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 evaluated in the original epidemiologic study.

- 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 also completed a qualitative characterization of sources of uncertainty including an assessment of both the magnitude and direction of impact of those uncertainties on risk estimates. The classification of the magnitude of impact for sources of uncertainty includes three levels: (a) low (unlikely to produce a sufficient impact on risk estimates to affect their interpretation), (b) medium (potential to have a sufficient impact to affect interpretation), and (c) high (likely to have an impact sufficient to affect interpretation). For several of the sources, we provide a classification between these levels (e.g., low-medium, medium-high).[69] The below uncertainties, as well as various additional sources of uncertainty, are detailed in the *Estimating $PM_{2.5}$ and Ozone- Attributable Health Benefits TSD* (U.S. EPA, 2021b).Sources of uncertainty with at least a low classification as to the magnitude of potential impacts include the following (from Appendix C, Table C-32):[70]

    - Use of air quality modeling to adjust $PM_{2.5}$ concentrations: The baseline and adjusted air quality concentration fields were developed using modeling to fill spatial and temporal gaps in monitoring and explore "what if" scenarios. State-of-the-science modeling methods were used, but modeling-related biases and errors introduce uncertainty into the $PM_{2.5}$ concentration estimates. In addition, due to the national scale of the assessment, scenarios are based on changing modeled emissions of primary $PM_{2.5}$ or NO_X and SO_2 from all anthropogenic sources throughout the U.S. by fixed percentages. Although this approach tends to target key emission sources in each study area, it does not tailor emission changes to specific sources. The two adjustment cases span a wide range of emission conditions, but these cases are necessarily a subset of the full set of possible emission scenarios that could be used to adjust $PM_{2.5}$ concentrations to simulate "just meeting" standards.

[69] Additional information is available in Appendix C, section C.3.

[70] We also identified several additional factors judged to have less than a medium classification of impact on the risk estimates generate, including: (a) the temporal mismatch between ambient air quality data characterizing exposure and mortality in long-term exposure-related epidemiology studies, (b) compositional and source differences in PM, (c) exposure measurement error in epidemiology studies assessing the relationship between mortality and exposure to ambient $PM_{2.5}$, (d) lag structure in short-term exposure-related mortality epidemiology studies, and (e) assumed causal association between PM and mortality that supports modeling changes in risk associated with future changes in ambient $PM_{2.5}$. See Table C-32 in Appendix C for additional discussion of these sources of uncertainty.

- Use of linear interpolation/extrapolation to adjust air quality: The use of interpolation and extrapolation to simulate just meeting annual standards with levels of 11.0, 9.0, and 8.0 $\mu g/m^3$ does not fully capture potential non-linearities associated with real-world changes in air quality.
- Potential confounding of the $PM_{2.5}$-mortality effect: Factors are considered potential confounders if demonstrated in the scientific literature to be related to the health effect and correlated with $PM_{2.5}$. Omitting potential confounders from analyses could either increase or decrease the magnitude of $PM_{2.5}$ effect estimates (e.g., Di et al., 2017b, supplemental Figure S2). Thus, not accounting for confounders can introduce uncertainty into effect estimates and, consequently, into the estimated impacts generated using those effect estimates. Confounders vary according to study design, exposure duration, and health effect. For studies of short-term exposures, confounders may include meteorology (e.g., temperature, humidity), day of week, season, medication use, allergen exposure, and long-term temporal trends. For studies of long-term exposures, confounders may include socioeconomic status, race, age, medication use, smoking status, stress, noise, and occupational exposures. While various approaches to control for potential confounders have been adopted across the studies used in the risk assessment, and across the broader body of $PM_{2.5}$ epidemiologic studies assessed in the 2019 ISA, no individual study adjusts for all potential confounders (U.S. EPA, 2019, Table A-1).
- Potential for exposure error: Epidemiologic studies have employed a variety of approaches to estimate population-level $PM_{2.5}$ exposures (e.g., stationary monitors and hybrid modeling approaches). These approaches are based on using measured and/or predicted ambient $PM_{2.5}$ concentrations as surrogates for population exposures. As such, exposure estimates in epidemiologic studies are subject to exposure error. The 2019 ISA notes that, while bias in either direction can occur, exposure error tends to result in underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, Hart et al. (2015) reports that correction for $PM_{2.5}$ exposure error using personal exposure information results in a moderately larger effect estimate for long-term $PM_{2.5}$ exposure and mortality, though with wider confidence intervals. Error in the underlying epidemiologic studies contributes to uncertainty in the risk estimates based on concentration-response relationships in those studies. Beyond the exposure error in concentration-response functions, the use of a different approach to represent exposures in the risk assessment (i.e., 12 x 12 km gridded surface based on modeling) could introduce additional error into risk estimates.
- Shape of the concentration-response relationship at low ambient PM concentrations: Interpreting the shapes of concentration-response relationships, particularly at $PM_{2.5}$ concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and

"linearize" population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015b, section 6.c).

Additional uncertainties are associated with the at-risk analysis. Importantly, the smaller population within each demographic group reduces statistical power. As this risk and exposure assessment focuses on urban areas, demographic groups that primarily reside in rural areas, such as Native Americans, are underrepresented.

3.4.3 Conclusions of the Risk Assessment

Although limitations in the underlying data and approaches lead to some uncertainty regarding estimates of $PM_{2.5}$-associated risk (summarized in section 3.4.1.7), 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. For example, when air quality in the 47 study areas is adjusted to simulate just meeting the current standards, the risk assessment estimates 40,600-45,100 long-term $PM_{2.5}$ exposure-related deaths in a single year, with confidence intervals ranging from 30,300-59,000 deaths (Table 3-14). Additionally, the at-risk assessment estimated that Black populations may experience disproportionally higher exposures and risk under simulated air quality conditions just meeting the current primary $PM_{2.5}$ annual standard as compared to White populations (section 3.4.2.4).[71]

Compared to the current annual standard, meeting a revised annual standard with a lower level is estimated to reduce $PM_{2.5}$-associated health risks in the 30 annually-controlled study areas by about 7-9% for a level of 11.0 $\mu g/m^3$, 15-19% for a level of 10.0 $\mu g/m^3$, 22-28% for a level of 9.0 $\mu g/m^3$, and 30-37% for a level of 8.0 $\mu g/m^3$. (Table 3-17)[72] Meeting a revised annual standard with a lower level may also reduce exposure and risk in Black populations slightly more so than in White populations in simulated scenarios just meeting alternative annual standards (section 3.4.2.4).

Revising the level of the 24-hour standard to 30 $\mu g/m^3$ is estimated to lower $PM_{2.5}$-associated risks across a more limited population and number of areas then revising the annual standard (section 3.4.2.3). Risk reduction predictions are largely confined to areas located in the

[71] Risk estimates in Black populations are largely due to race-specific concentration-response functions.

[72] Importantly, as the magnitude of estimated risk reductions increases with lower alternative annual standards, estimated risk reductions are associated with lower ambient $PM_{2.5}$ concentrations. Lower $PM_{2.5}$ concentrations may less closely align with those observed in the epidemiologic study from which the concentration-response function was obtained, contributing to uncertainty. Additional information on estimated ambient concentrations of the original Medicare and ACS cohorts evaluated by Di et al., 2017b and Turner et al., 2016, respectively, can be found in section 6.1.2.1 of the *Estimating $PM_{2.5}$ and Ozone- Attributable Health Benefits TSD* (*U.S. EPA, 2021b*).

western U.S., several of which are also likely to experience risk reductions upon meeting a revised annual standard.

3.5 KEY CONSIDERATIONS REGARDING THE ADEQUACY OF THE PRIMARY $PM_{2.5}$ STANDARDS

In considering the adequacy of the primary $PM_{2.5}$ standards, the overarching question we consider is:

- **Does the scientific evidence and risk-based information support or call into question the adequacy of the protection afforded by the current primary $PM_{2.5}$ standards?**

To assist us in interpreting the scientific evidence and the results of recent quantitative risk analyses to address this question, we have focused on a series of more specific questions, as detailed in sections 3.5.1 and 3.5.2 below. In considering the scientific and technical information, we consider both the information available at the time of the 2012 and 2020 reviews and information available in this reconsideration, which have been critically assessed in the 2019 ISA and the draft ISA Supplement. In so doing, a key consideration is whether the information in this reconsideration alters our overall conclusions from the 2020 review regarding health effects associated with $PM_{2.5}$ in ambient air.

3.5.1 Evidence-based Considerations

In considering the evidence with regard to the overarching question posed above regarding the adequacy of the current $PM_{2.5}$ standards, we address a series of more specific questions that focus on policy-relevant aspects of the evidence. These questions begin with consideration of the available evidence on health effects associated with exposure to $PM_{2.5}$. (section 3.5.1.1). The subsequent questions consider identification of populations at-risk of $PM_{2.5}$-related health effects (section 3.5.1.2), and the exposure durations and levels of $PM_{2.5}$ associated with health effects (section 3.5.1.3). Important uncertainties associated with the evidence are considered in section 3.5.1.4.

3.5.1.1 Health Effects Associated with Exposure to $PM_{2.5}$

In answering the overarching question above, we begin by considering the following question:

- **Is there newly available evidence that indicates the importance of certain particle characteristics (i.e., components or size fractions) other than $PM_{2.5}$ mass with regard to concentrations in ambient air, and potential for human exposures and health effects?**

No newly available evidence has been identified in this reconsideration regarding particle characteristics, such as components or size fractions, other than $PM_{2.5}$ mass with regard to

concentrations in ambient air, and potential for health effects. 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, 2019, section 1.5.4). The 2019 ISA specifically notes that "results of these studies confirm and further support the conclusion of the 2009 ISA that many $PM_{2.5}$ components and sources are associated with many health effects and that 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, 2019, section 1.5.4). In addition, the evidence for health effects following exposures specifically to the ultrafine fraction of fine particles continues to be far more limited than the evidence for $PM_{2.5}$ mass as a whole. As discussed in the 2019 ISA, the lack of a consistent UFP definition in health studies and across disciplines, together with a variety of approaches to administering and measuring UFP in those studies, contribute to such limitations (U.S. EPA, 2019, section 1.4.3). Thus, as was the case for previous reviews, the evidence base for health effects of fine particles does not support consideration of other PM characteristics, such as components, or size fractions. For these reasons, we continue to focus on the health effects associated with $PM_{2.5}$ mass.

- **Does the available scientific evidence alter our conclusions regarding the nature of health effects attributable to human exposure to $PM_{2.5}$ from ambient air?**

The scientific evidence, including that assessed in the 2019 ISA and draft ISA Supplement, is consistent with the conclusion reached in the previous reviews regarding health effects and PM exposures where a causal relationship was concluded. Specifically, as in prior reviews, it was concluded that there is a causal relationship between short- and long-term $PM_{2.5}$ exposures and mortality and cardiovascular effects (U.S. EPA, 2019, sections 11.1, 11.2, 6.1, 6.2; U.S. EPA, 2021a, sections 3.2.1, 3.2.2, 3.1.1, and 3.1.2). Further, a likely to be causal relationship was concluded for short- and long-term $PM_{2.5}$ exposures and respiratory effects (U.S. EPA, 2019, sections 5.1 and 5.2). Additionally, conclusions reached in the 2019 ISA differ with regard to cancer and nervous systems effects and long-term $PM_{2.5}$ exposure, based on evidence assessed in the 2019 ISA and it was concluded that there is a likely to be causal relationship (U.S. EPA, 2019, sections 10.2 and 8.2). The evidence base is concluded to be suggestive of, but not sufficient to infer, causal relationships between short- and long-term $PM_{2.5}$ exposures and metabolic effects (U.S. EPA, 2019, sections 7.1 and 7.2), reproduction and fertility (U.S. EPA, 2019, section 9.1.1), and pregnancy and birth outcomes (U.S. EPA, 2019, section 9.1.2). In addition, effects associated with short-term exposure to UFP and cardiovascular (U.S. EPA, 2019, section 6.5), respiratory (U.S. EPA, 2019, section 5.5), and nervous system effects (U.S. EPA, 2019, section 8.5), as well as long-term exposure to UFP and nervous system effects (U.S. EPA, 2019, section 8.6) are concluded to be suggest of, but not sufficient to infer,

causal relationship. As in the 2020 review, the strongest evidence, including with regard to quantitative characterizations of relationships between $PM_{2.5}$ exposure and effects, is for mortality and cardiovascular effects.

3.5.1.2 Populations At-Risk of $PM_{2.5}$-related Health Effects

Populations or lifestages can be at increased risk of an air pollutant-related health effect due to one or more factors. These factors can be intrinsic, such as physiological factors that may influence the internal dose or toxicity of a pollutant, or extrinsic, such as sociodemographic, or behavioral factors. The questions considered in this section address what the available evidence indicates regarding which populations are particularly at risk of health effects related to exposure to $PM_{2.5}$ in ambient air.

- **Does the current evidence alter our understanding of populations that are particularly at risk from $PM_{2.5}$ exposures? Is there evidence that suggests additional at-risk populations that should be given increased focus for this reconsideration?**

The current evidence does not alter our understanding of which populations are potentially at greater risk from health effects of $PM_{2.5}$ exposures. As in previous reviews, the 2019 ISA continues to provide support that factors that may contribute to increased risk of $PM_{2.5}$-related health effects include lifestage (children and older adults), pre-existing diseases (cardiovascular disease and respiratory disease), race/ethnicity, and socioeconomic status. Other factors that have the potential to contribute to increased risk, but for which the evidence is less clear, include obesity, diabetes, genetic factors, smoking status, sex, diet, and residential location (U.S. EPA, 2019, chapter 12).

In addition to these population groups, the 2019 ISA and draft ISA Supplement note that there is strong evidence for racial and ethnic differences in $PM_{2.5}$ exposures and $PM_{2.5}$-related health risk. There is strong evidence demonstrating that Black and Hispanic populations, in particular, have higher $PM_{2.5}$ exposures than non-Hispanic White populations (U.S. EPA, 2019, Figure 12-2; U.S. EPA, 2021a, Figure 3-38). Further, there is consistent evidence across multiple studies that demonstrate increased risk of $PM_{2.5}$-related health effects, with the strongest evidence for health risk disparities for mortality (U.S. EPA, 2019, section 12.5.4).

Studies assessed in the 2019 ISA and draft ISA Supplement also provide evidence of exposure and health risk disparities based on SES. The evidence indicates that lower SES communities are exposed to higher concentrations of $PM_{2.5}$ compared to higher SES communities (U.S. EPA, 2019, section 12.5.3; U.S. EPA, 2021a, section 3.3.3.1.1). Additionally, evidence supports the conclusions that lower SES is associated with cause-specific mortality and certain health endpoints (i.e., MI and CHF), but less so for all-cause or total (non-accidental) mortality (U.S. EPA, 2019, section 12.5.3; U.S. EPA, 2021a, section 3.3.3.1).

3.5.1.3 Exposure Concentrations Associated with Health Effects

In answering the overarching question with regard to the adequacy of the primary $PM_{2.5}$ standards, as described above, we next consider the scientific evidence and the support it provides for the occurrence of adverse public health effects and the associated exposure concentrations at which such effects occur. In so doing, we ask the following questions:

- **Does the current evidence alter our conclusions regarding the exposure duration and concentrations associated with health effects? To what extent does the scientific evidence indicate health effects attributable to exposures to $PM_{2.5}$ concentrations lower than previously reported and what are important uncertainties in that evidence?**

The evidence available in this reconsideration regarding $PM_{2.5}$ exposures associated with health effects affirms and strengthens the evidence available at the time of the 2020 review, taking into account studies that have become available since that time. Consistent with the evidence available in the 2020 review, and as assessed in the 2019 ISA and the draft ISA Supplement, the strong evidence base of epidemiologic studies report associations between long- and short-term $PM_{2.5}$ exposures and a variety of outcomes, including mortality and cardiovascular effects. Additionally, as detailed in section 3.3.1, animal toxicological studies and controlled human exposure studies continue to provide support understanding the effects of exposure to $PM_{2.5}$, and support for biologically plausible mechanisms through which adverse human health outcomes could occur. In addition, controlled human exposure studies have consistently reported that $PM_{2.5}$ exposures lasting from less than one hour up to five hours can impact cardiovascular function and provide some insight into how short-term exposure to $PM_{2.5}$ may impact cardiovascular function in ways that could lead to more serious outcomes.

The controlled human exposure studies, as discussed in detail in the 2019 ISA (U.S. EPA, 2019, section 6.1) and summarized above in section 3.3.3.1, have demonstrated effects on cardiovascular function following $PM_{2.5}$ exposures ranging from one to five hours, with the most consistent evidence for impaired vascular function (U.S. EPA, 2019, section 6.1.13.2). In addition, although less consistent, the 2019 ISA notes that studies examining $PM_{2.5}$ exposures also provide evidence for increased blood pressure (U.S. EPA, 2019, section 6.1.6.3), conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). The 2019 ISA concludes that, when taken as a whole, controlled human exposure studies demonstrate that exposure to $PM_{2.5}$ may impact cardiovascular function in ways that could lead to more serious outcomes (U.S. EPA, 2019, section 6.1.16). Thus, such studies can provide insight into the potential for specific $PM_{2.5}$ exposures to result in physiological

changes that could increase the risk of more serious effects, though the health relevance of the occurrence of these acute effects is less certain.

To provide some insight into what these studies may indicate regarding the primary $PM_{2.5}$ standards, air quality analyses examine monitored 2-hour $PM_{2.5}$ concentrations at sites meeting the current primary $PM_{2.5}$ standards (as described in section 2.3.2 and section A.3 of Appendix A).[73] The 2-hour $PM_{2.5}$ concentrations to which individuals were exposed in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards. For example, at air quality monitoring sites meeting the current primary $PM_{2.5}$ standards (i.e., the 24-hour standard and the annual standard), the 2-hour concentrations generally remain below 10 $\mu g/m^3$, and virtually never exceed 30 $\mu g/m^3$. Two-hour concentrations are higher at monitoring sites violating the current standards, but generally remain below 16 $\mu g/m^3$ and virtually never exceeding 80 $\mu g/m^3$. Thus, while controlled human exposure studies provide support for the biological mechanisms and plausibility of the serious cardiovascular effects associated with ambient $PM_{2.5}$ exposures in epidemiologic studies (U.S. EPA, 2019, chapter 6), the exposures evaluated in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards, and the results are variable across some of the controlled human exposure studies evaluated at near ambient $PM_{2.5}$ concentrations.

While controlled human exposure studies provide insight on the exposure concentrations that directly elicit health effects in humans, uncertainty exists in translating the observations in animal toxicology studies to potential adverse health effects in humans. The interpretation of the animal toxicology studies with regard to the potential implications for human health is complicated by the fact that the concentrations of $PM_{2.5}$ in animal toxicologic studies are much higher than those shown to elicit effects in human populations, and there are also significant anatomical and physiological differences between animal models and humans. Most of the animal toxicology 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, 2019, Table 1-2). Two exceptions are a study reporting impaired lung development following long-term exposures (i.e., 24 hours per day for several months prenatally and postnatally) to an average $PM_{2.5}$ 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 report serious effects following long-term exposures to $PM_{2.5}$ concentrations close to the

[73] In addition, 4-hour and 5-hour $PM_{2.5}$ concentrations at monitoring sites meeting or violating the current primary $PM_{2.5}$ standards were also evaluated (as described in section 2.3.2 and section A.3 of Appendix A).

ambient concentrations reported in some $PM_{2.5}$ epidemiologic studies (U.S. EPA, 2019, Table 1-2), though still above the ambient concentrations likely to occur in areas meeting the current primary standards. Thus, as is the case with controlled human exposure studies, animal toxicology studies support the plausibility of various adverse effects that have been linked to ambient $PM_{2.5}$ exposures (U.S. EPA, 2019)).

Epidemiologic studies in the U.S. and Canada, assessed in the 2019 ISA and draft ISA Supplement, continue to report positive and statistically significant associations between long- and short-term exposure to $PM_{2.5}$ and mortality and morbidity, including both new studies evaluated in the draft ISA Supplement related to total mortality and cardiovascular mortality and morbidity and studies that examined populations and lifestages that may be at comparatively higher risk of experiencing a $PM_{2.5}$-related health effects (e.g., older adults). Such studies employ various designs and examine a variety of health outcomes, geographic areas, and approaches to controlling for confounding variables. With regard to controlling for potential confounders in particular, key epidemiologic studies use a wide array of approaches. Time-series studies control for potential confounders that vary over short time intervals (e.g., including temperature, humidity, dew point temperature, and day of the week) while cohort studies control for community- and/or individual-level confounders that vary spatially (e.g., including income, race, age, socioeconomic status, smoking, body mass index, and annual weather variables such as temperature and humidity) (Appendix B, Table B-4). Sensitivity analyses indicate that adding covariates to control for potential confounders can either increase or decrease the magnitude of $PM_{2.5}$ effect estimates, depending on the covariate, and that none of the covariates examined can fully explain the association with mortality (e.g., Di et al., 2017b, Figure S2 in Supplementary Materials). Thus, while no individual study adjusts for all potential confounders, a broad range of approaches have been adopted across studies to examine confounding, supporting the robustness of reported associations.

Available studies additionally indicate that $PM_{2.5}$ health effect associations are robust across various approaches to estimating $PM_{2.5}$ exposures and across various exposure windows. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates the potential for exposure error in epidemiologic studies, such error does not call into question the fundamental findings of the broad body of $PM_{2.5}$ epidemiologic evidence. In fact, the 2019 ISA notes that while bias in either direction can occur, exposure error tends to lead to underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study reports that correction for $PM_{2.5}$ exposure error using personal exposure information results in a moderately larger effect estimate for long-term $PM_{2.5}$

exposure and mortality (Hart et al., 2015). While most $PM_{2.5}$ epidemiologic studies have not employed similar corrections for exposure error, several studies report that restricting analyses to populations in close proximity to a monitor (i.e., in order to reduce exposure error) result in larger $PM_{2.5}$ effect estimates (e.g., Willis et al., 2003; Kloog et al., 2013). The consistent reporting of $PM_{2.5}$ health effect associations across exposure estimation approaches, even in the face of exposure error, together with the larger effect estimates reported in some studies that have attempted to reduce exposure error, provides further support for the robustness of associations between $PM_{2.5}$ exposures and mortality and morbidity.

Consistent findings from the broad body of epidemiologic studies are also supported by an emerging body of studies employing causal modeling methods to further inform the causal nature of the relationship between long- or short-term term $PM_{2.5}$ exposure and mortality (U.S. EPA, 2019, sections 11.1.2.1, 11.2.2.4, U.S. EPA, 2021a, sections 3.1.1.3, 3.1.2.3, 3.2.1.3, and 3.2.2.3). These studies, summarized above in Table 3-11, used a variety of statistical methods to control for confounding bias and consistently report positive associations, which support the positive and significant effects seen in cohort studies associated with short- and long-term exposure to $PM_{2.5}$ and mortality.

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, 2019, section 1.5.3, U.S. EPA, 2021a, sections 2.2.3.1 and 2.2.3.2). While such analyses are complicated by the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), analyses that assess the concentration-response relationship support a linear, no-threshold effect down to 5.0 $\mu g/m^3$, though uncertainties increase at concentrations of less than 8.0 $\mu g/m^3$.

There are a number of U.S. and Canadian studies that examine health effect associations in analyses with the highest exposures excluded and report positive and statistically significant associations in analyses restricted to annual average $PM_{2.5}$ exposures at or below 12 $\mu g/m^3$ and or to daily exposures below 35 $\mu g/m^3$ (Table 3-10). While mean $PM_{2.5}$ concentrations for these restricted analyses may not be reported in most studies, we can presume that the mean $PM_{2.5}$ concentrations in the restricted analyses are less than the study-reported mean $PM_{2.5}$ concentrations in the main analyses, which range from 8.1 $\mu g/m^3$ to 11.6 $\mu g/m^3$ in the U.S., and was 7.8 $\mu g/m^3$ for the one study in Canada that included restricted analysis. It is important to note that even if we had information on $PM_{2.5}$ mean concentrations reported in restricted

analysis, we would not necessarily be able to use these means in a similar decision framework as was used in past reviews (section 3.3.3.2.1). given uncertainties associated with identifying the relationship between a calculated mean concentration that excludes specific daily or annual average concentrations above a certain threshold and the design value used to determine compliance with a standard (annual or 24-hour). However, restricted analyses do provide support for effects at lower concentrations, exhibiting associations for mean concentrations presumably below the mean concentrations for the main analyses.

Finally, accountability studies evaluate whether changes in air quality are associated with improvements in public health and a number of recent studies are evaluated in the draft ISA Supplement (summarized in Table 3-12 above). These studies exhibit positive and significant associations, including some studies that report starting $PM_{2.5}$ concentrations below 12.0 $\mu g/m^3$, indicating that public health improvements may occur following $PM_{2.5}$ reductions in areas that already meet the current annual $PM_{2.5}$ standard. For example, studies by Corrigan et al. (2018) and Sanders et al. (2020) both found improvements in mortality rates due to improvements in air quality in both attainment and nonattainment areas following implementation of the 1997 primary annual $PM_{2.5}$ NAAQS.[74] Other recent studies additionally report 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 reported in the epidemiologic evidence.

Consistent with previous reviews, we note that 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 can lead to the reported effects. Rather, health effects can occur over the entire distribution 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, 2019, section 1.5.3). However, the study-reported ambient $PM_{2.5}$ concentrations reflecting estimated exposure in the middle portion of the $PM_{2.5}$ air quality distribution, which corresponds to the bulk of the underlying data, which provide the strongest support for reported health effect associations and can inform our preliminary conclusions on the current and potential alternative standards. In using this information to inform our preliminary conclusions, we recognize that the mean $PM_{2.5}$ concentrations reported by key epidemiologic studies differ in how mean

[74] We note that the studies by Corrigan et al. (2018) and Sanders et al. (2020) report monitor-based average $PM_{2.5}$ concentrations, and that these studies do not report design values.

concentrations were calculated (Table 3-5, Table 3-6, Table 3-7, Table 3-8), as well as their interpretation in what means represent in the context of the current standards. To frame our evaluation of study-reported mean $PM_{2.5}$ concentrations, we specifically consider the following question:

- **How do the study-reported means from the key epidemiologic studies and the related air quality analyses that compare study means to area design values inform our consideration of the level of the current annual $PM_{2.5}$ standard?**

In the 2012 review, the Administrator recognized that evidence of an association between $PM_{2.5}$-related health effects and long- and short-term exposures in the epidemiologic studies were strongest at and around the long-term average where the data in the study are most concentrated. In so doing, she noted that the long-term mean $PM_{2.5}$ concentrations were available for the studies considered and represented the most robust data set to inform decisions on appropriate levels for the annual primary $PM_{2.5}$ standard, while also recognizing that this approach did not provide a bright line for reaching this decision (78 FR 3140, January 15, 2013). As detailed in section 3.3.3.2.1, the reported mean $PM_{2.5}$ concentrations derived from monitored observations are not the same as the mean $PM_{2.5}$ concentrations estimated using hybrid modeling methods, which are also not the same as design values used to determine whether an area meets or exceeds the $PM_{2.5}$ NAAQS. Additional analyses, new in this draft PA though similar to those in the 2012 review, examine how the calculation of the study mean varies across studies and how these metrics compare to the annual design value. The analysis indicates that study means from methods that use hybrid models to estimate exposures are generally lower in areas where urban and rural $PM_{2.5}$ concentrations are estimated, compared to hybrid modeled $PM_{2.5}$ concentrations in urban areas or concentrations that have been population-weighted. Moreover, the analysis indicates that hybrid modeling mean estimates are generally lower than the average of monitored $PM_{2.5}$ concentrations, which are both below the concentration measured at the highest monitor (i.e., the approach used to calculate the design value). In the national-scale analysis, where air quality analyses compared composite monitored $PM_{2.5}$ concentrations with annual $PM_{2.5}$ design values in the U.S., annual $PM_{2.5}$ design values were approximately 10% to 20% higher than concentrations averaged across multiple monitors in the same CBSA (section 2.3.3.1, Figure 2-28 and Table 2-2).

Further, with the expansion of studies that employ hybrid modeling methods to estimate $PM_{2.5}$ concentrations, Section 2.3.3.2.4 details a comparison of $PM_{2.5}$ fields in estimating

exposure relative to design values using the DI2019 and HA2020[75] surfaces, which are two air quality surfaces included in several of the key epidemiologic studies. This analysis illustrates that population-weighting the $PM_{2.5}$ concentrations in the hybrid modeling approaches has an effect on the resulting study-reported mean. Specifically, the analysis shows that area annual design values are 40% to 50% higher compared to the study-reported means when population-weighting is not employed. Additionally, when population-weighting is applied in studies using hybrid modeling approaches, average annual $PM_{2.5}$ design values are only 15% to 18% higher than the study-reported means. This suggests that whether a study using a hybrid modeling approach incorporated population-weighting is very important for understanding how to interpret the estimated $PM_{2.5}$ exposure concentrations, particularly for purposes of comparing those estimated concentrations to actual design values.

Thus, given the potentially large differences between study reported means and area annual design values, it is important to consider the manner in which $PM_{2.5}$ concentrations are estimated (e.g., monitored concentrations versus modeled concentrations) and the method by which means are calculated and reported as the overall mean $PM_{2.5}$ concentration (e.g., averaging across all grid cells in an urban area versus population-weighting). Additional analyses, new in this draft PA though similar to those in the 2012 review, suggest that area annual design values higher than the study-reported means by 10-20% (monitor-based studies), 14-18% (hybrid modeling with population-weighting) or 40-50% (hybrid modeling without population weighting). Grouping studies based on the approach used to estimate the mean, we note that the overall mean $PM_{2.5}$ concentrations in key U.S. epidemiologic studies are as follows:

- Range of monitor-based mean $PM_{2.5}$ concentrations is from 9.9 $\mu g/m^3$ to 16.5 $\mu g/m^3$ (range in 2020 PA: 10.7 $\mu g/m^3$ to 16.5 $\mu g/m^3$)
- Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling and apply population-weighting: 9.3 $\mu g/m^3$ to 12.3 $\mu g/m^3$
- Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling and do not apply population-weighting: 8.1 $\mu g/m^3$ to 11.9 $\mu g/m^3$

The mean $PM_{2.5}$ concentrations in Canadian studies are more difficult to compare to the annual design value used to determine compliance in the U.S. As we note above, the air quality analyses in section 3.3.3.2.1 are most relevant for interpreting U.S. epidemiologic studies. Given that we are lacking important pieces of information that allow us to do similar analyses for

[75] As discussed above in section 2.3.3.2.4, HA2020 refers to estimated $PM_{2.5}$ concentrations from a hybrid modeling approach developed by Hammer et al. (2020) and van Donkelaar et al. (2019), and which estimates Nationwide $PM_{2.5}$ concentrations from 2000-2016.

Canada, we are unable to provide specific quantitative insight into how the study reported means in the Canadian studies would compare to area design values in the U.S. However, we note that the overall mean $PM_{2.5}$ concentrations in key Canadian epidemiologic studies are similar to, though somewhat lower than, those from the U.S. studies:

- Range of monitor-based mean $PM_{2.5}$ concentrations: 6.9 $\mu g/m^3$ to 13.3 $\mu g/m^3$
- Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling (all of which average up to postal codes and thus include some aspects of population-weighting): 5.9 $\mu g/m^3$ to 9.8 $\mu g/m^3$

In the context of evaluating whether the newly available scientific information alters our conclusions from the 2020 review regarding the nature of health effects attributable to human exposure to $PM_{2.5}$ from ambient air, while the causality determinations have not changed, the number of studies that use hybrid modeling approaches has expanded. When using the information from the new air quality analyses to interpret key epidemiologic studies in the context of the primary standards, we note that they suggest that epidemiologic studies that use monitor-based estimates for $PM_{2.5}$ exposure or that calculate population-weighted averages from hybrid modeling approaches generally report mean concentrations that are more easily compared to an area annual design value (i.e., area annual design values are 10-20% greater than mean $PM_{2.5}$ concentrations). However, we also note that area annual design values tend to be substantially greater than mean concentrations in epidemiologic studies that use hybrid approaches and do not include population weighting (e.g. 40-50% greater). Thus, when 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 emphasize the importance of considering the broader relationships between mean $PM_{2.5}$ concentrations, averaged across space and over time using a variety of approaches, and $PM_{2.5}$ design values.

- **How do the study-reported $PM_{2.5}$ concentrations corresponding to the 25th and 10th percentiles of health data or exposure estimates provide insight to inform our consideration of the level of the current annual $PM_{2.5}$ standard?**

In the 2012 review, the 2011 PA noted the interrelatedness of the distributional statistics and a range of one standard deviation around the mean which contains approximately 68% of normally distributed data, in that one standard deviation below the mean falls between the 25th and 10th percentiles (U.S. EPA, 2011 p. 2-71). Given this, the 2011 PA provided information, as available for a subset of key epidemiologic studies, on the study-reported $PM_{2.5}$ concentrations corresponding to the 25th and 10th percentiles of health data or exposure estimates.

In that review, the Administrator placed some weight on studies that provided mean $PM_{2.5}$ concentrations around the 25th percentile of the distributions of deaths and cardiovascular-related hospitalizations and judged the region around the 25th percentile as a reasonable part of

the distribution to guide the decision on the appropriate standard level (78 FR 3161, January 15, 2013). Given the potential for consideration of this information in this reconsideration with regard to the adequacy of the standard level, we note that of the key epidemiologic studies evaluated in the 2019 ISA and draft ISA Supplement, a subset of studies report $PM_{2.5}$ concentrations corresponding to the 25th and 10th percentiles of health data or exposure estimates to provide insight into the concentrations that comprise the lower quartiles of the air quality distributions. In the key U.S. epidemiologic studies that report the 25th and 10th percentiles of health events corresponding to mean $PM_{2.5}$ concentrations (i.e., averaged over the study period for each study city), we note:

- Monitor-based 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 $\mu g/m^3$
- Monitor-based 10th percentiles of health events correspond to mean $PM_{2.5}$ concentrations: at or above 9.8 $\mu g/m^3$
- $PM_{2.5}$ concentrations corresponding to 25th percentiles of estimated exposures that use hybrid modeling approaches to estimate long-term $PM_{2.5}$ exposures range from 6.2 to 9.2 $\mu g/m^3$
- $PM_{2.5}$ concentrations corresponding to 25th percentiles of estimated exposures in studies that uses hybrid modeling to estimate short-term exposures: at or above 6.4 $\mu g/m^3$
- $PM_{2.5}$ concentration corresponding to the 25th percentile of estimated exposures in one study with lower concentrations is 4.6 $\mu g/m^3$
- $PM_{2.5}$ concentration corresponding to the 10th percentile in the two studies with available information on this percentile range from 4.7 $\mu g/m^3$ to 7.3 $\mu g/m^3$.

In thinking about these values relative to an area annual design value, we emphasize that the 25th and 10th percentiles provide information about the lower quartiles of the air quality distributions, while the study reported mean provides information about the average or typical exposures, and the corresponding area annual design value provides the highest average annual $PM_{2.5}$ concentration being measured. In this way, all of these metrics (i.e. lower percentiles, study mean, annual design value) have a relationship relative to the other.

3.5.1.4 Uncertainties in the Health Effects Evidence

A number of key uncertainties and limitations were identified in the previous review with respect to health effects evidence, as described in the 2020 PA. This section considers the currently available information, including that newly available in this reconsideration, with regard to such areas of uncertainty.

- **To what extent have previously identified uncertainties in the health effects evidence been reduced and/or have new uncertainties emerged?**

We continue to recognize uncertainties that persist from previous reviews. First, we note uncertainties related to the susceptibility of different population groups for which evidence is not as clear (e.g., based on differences in underlying factors such as obesity, smoking status and residential location). For human exposures studies, there are uncertainties related to mixed results seen at concentrations near ambient $PM_{2.5}$ levels. It is also unclear how the results alone and the importance of the effects observed in these studies, particularly in studies conducted at near-ambient $PM_{2.5}$ concentrations, should be interpreted with respect to adversity to public health. With respect to animal toxicology studies, while these studies also help establish biological plausibility, uncertainty exists in extrapolating the effects seen in animal toxicology studies, and the $PM_{2.5}$ concentrations that cause those effects to human populations.

Uncertainties associated with the epidemiologic evidence (e.g., the potential for copollutant confounding and exposure measurement error) remain, though new studies assessed in the draft ISA Supplement employ statistical methods like causal modeling methods, which have reduced some uncertainties related to potential confounding of effects. In so doing, however, we note the strength in the epidemiologic evidence in its support for determination of a causal relationship for mortality and cardiovascular effects as summarized in section 3.3.1 above.

With regard to controlling for potential confounders in particular, key epidemiologic studies use a wide array of approaches. Time-series studies control for potential confounders that vary over short time intervals (e.g., including temperature, humidity, dew point temperature, and day of the week), while cohort studies control for community- and/or individual-level confounders that vary spatially (e.g., including income, race, age, socioeconomic status, smoking, body mass index, and annual weather variables such as temperature and humidity) (Appendix B, Table B-4). Sensitivity analyses indicate that adding covariates to control for potential confounders can either increase or decrease the magnitude of $PM_{2.5}$ effect estimates, depending on the covariate, and that none of the covariates examined can fully explain the association with mortality (e.g., Di et al., 2017b, Figure S2 in Supplementary Materials). Thus, while no individual study adjusts for all potential confounders, a broad range of approaches have been adopted across studies to examine confounding, supporting the robustness of reported associations. Available studies additionally indicate that $PM_{2.5}$ health effect associations are robust across various approaches to estimating $PM_{2.5}$ exposures and across various exposure windows. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates

the potential for exposure error in epidemiologic studies, such error does not call into question the fundamental findings of the broad body of $PM_{2.5}$ epidemiologic evidence.

Additionally, 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, 2019, section 1.5.3, U.S. EPA, 2021a, sections 2.2.3.1 and 2.2.3.2). While such analyses are complicated by the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), analyses that assess the concentration-response relationship support a linear, no-threshold effect down to 5.0 $\mu g/m^3$, though uncertainties increase at concentrations of less than 8.0 $\mu g/m^3$.

While studies using hybrid modeling methods have demonstrated reduced exposure measurement error and uncertainty in the health effect estimates, these methodologies have inherent limitations and uncertainties, as described in more detail in section 2.3.3.1.5 and above in 3.3.4, and the performance of the modeling approaches depends on the availability of monitoring data which varies by location. Factors likely contributing to poorer model performance often coincide with relatively low ambient $PM_{2.5}$ concentrations, in areas where predicted exposures are at a greater distance to monitors, and under conditions where the reliability and availability of key datasets (e.g., air quality modeling) are limited. Thus, uncertainty in hybrid model predictions becomes an increasingly important consideration as lower predicted concentrations are considered.

In addition, limitations and or uncertainties exist in the analysis (section 2.3.3.2.4) evaluating the comparison of estimated $PM_{2.5}$ concentrations using hybrid modeling surfaces and their relationship to design values that should be considered. While design values in general are higher than estimated $PM_{2.5}$ concentrations using these two hybrid modeling approaches, it is important to recognize that these are just two hybrid modeling approaches and other models/approaches/spatial scales may result in somewhat different values. This analysis estimates $PM_{2.5}$ concentrations by CBSAs, but not every health study uses $PM_{2.5}$ estimates at this spatial scale, and spatial scales for exposure estimates can vary by study. As an example of this variation, in Di et al. (2016), an annual average $PM_{2.5}$ concentration was assigned to a person at-risk of death according to the ZIP code of the person's residence. The analysis completed was a nationwide analysis and ratios are based on national estimates. However, not all health studies are national studies and ratios in different parts of the country could be higher or lower, depending on factors like population, as well as rural versus urban areas. This analysis used specific air quality years (2000-2016) and other air quality year could result in higher or lower ratios.

Regardless of whether an epidemiologic study uses monitoring data or a hybrid modeling approach when estimating $PM_{2.5}$ exposures, one important challenge that persists is associated

with the interpretation of the study reported mean $PM_{2.5}$ concentrations and how they compare to design values. This is particularly true given the variability that exists across the various approaches to estimate exposure and to calculate the study reported mean. Further, with respect to interpreting the study reported mean concentrations from Canadian studies, using U.S. based analyses of hybrid modeling and their relationship to design values is complicated by differences between the U.S. and Canada as it relates to population densities, $PM_{2.5}$ concentration gradients, and source distributions in the two countries.

3.5.2 Risk-based Considerations

Our consideration of the scientific evidence available in this reconsideration, as at the time of the 2020 review, is informed by results from a quantitative analysis of risk. The overarching consideration in this section is whether the current risk information alters our overall conclusions regarding health risk associated with exposure to $PM_{2.5}$ in ambient air. As in our consideration of the evidence in section 3.5.1 above, we have focused the discussion regarding the risk information around key questions related to air quality conditions simulated to just meet existing and alternative primary $PM_{2.5}$ standards.

Prior to addressing the key risk questions, we provide a summary of important aspects of the assessment, including the study areas, air quality scenarios, and risk metrics (section 3.5.2.1). We then consider aspects of the questions beginning with the magnitude of risk estimated by both the overall assessment and for certain at-risk populations, followed by the key uncertainties associated with the quantitative analyses with regard to drawing conclusions as to the adequacy of protection afforded by the current primary $PM_{2.5}$ standards (section 3.5.2.2 and 3.5.2.3). We also consider uncertainties associated with the risk assessment (section 3.5.2.4). Lastly, we consider the risk estimates from the quantitative assessments with regard to the extent to which such estimates may be judged to be important from a public health perspective (section 3.5.2.5).

3.5.2.1 Risk Assessment Analyses

In the risk assessment conducted for this reconsideration, described in detail in section 3.4 above and Appendix C, we have estimated $PM_{2.5}$ health risks associated with air quality conditions that just meet the current primary $PM_{2.5}$ standards and potential alternative standard levels. These analyses inform our understanding of the health risks for all-cause or nonaccidental mortality associated with long- and short-term $PM_{2.5}$ exposures. These analyses estimate exposure and risk for populations in 47 urban study areas, as well as subsets of those study areas depending on which of the primary $PM_{2.5}$ standards is controlling in a given study area.

The 47 urban study areas were identified as they required relatively small adjustments (<20%) to just meet the current primary $PM_{2.5}$ standards and present a variety of circumstances with regard to risk associated with long- and short-term exposures to $PM_{2.5}$ in ambient air. This

set of study areas and the associated populations are intended to be informative to the EPA's consideration of potential risks that may be associated with the air quality conditions that meet the current and potential alternative primary $PM_{2.5}$ standards. The 47 study areas include nearly 60 million people ages 30 years or older and illustrate the differences likely to occur across various locations with such air quality as a result of area-specific differences in emissions, meteorological, and population characteristics. While the same conceptual air quality scenarios are simulated in all study areas (i.e., conditions that just meet the existing or alternate standards), source, meteorological and population characteristics in the study areas contribute to variability in the estimated magnitude of risk across study areas.

As an initial matter, we note that, consistent with the overall approach for this reconsideration, the risk assessment has a target scope that focuses on all-cause or nonaccidental mortality associated with long- and short-term $PM_{2.5}$ exposures (section 3.4.1.2). As noted in section 3.5.1 above, the evidence assessed in the 2019 ISA and draft ISA Supplement support a causal relationship between long- and short-term $PM_{2.5}$ exposures and mortality. Concentration-response functions used in the risk assessment are from large, multicity U.S. epidemiologic studies that evaluate the relationship between $PM_{2.5}$ exposures and mortality and were identified using criteria that take into account factors such as study design, geographic coverage, demographic populations, and health endpoints (U.S. EPA, 2021b, section 2.1).

In the risk assessment, air quality modeling was used to develop a $PM_{2.5}$ concentration field for 2015 (described in more detail in section 3.4.1.4 and Appendix C). The 2015 $PM_{2.5}$ concentration field was adjusted to simulate just meeting the existing annual and 24-hour standards of 12.0 $\mu g/m^3$ and 35 $\mu g/m^3$ and to just meeting potential alternative annual and 24-hour standards of 10.0 $\mu g/m^3$ and 30 $\mu g/m^3$. The adjustments made to the $PM_{2.5}$ concentration field are based on assumptions. Changes in $PM_{2.5}$, in reality, require specific information regarding emissions changes, with concentration gradients of $PM_{2.5}$ varying accordingly across an area. The risk assessment used two adjustment approaches to serve as bounding scenarios for the various ways an alternative standard may be met: (1) preferentially adjusting direct/primary PM emissions, for which changes in $PM_{2.5}$ tend to be more localized near the direct emissions sources of PM (Pri-PM), and (2) preferentially adjusting SO_2 and NO_X precursor emissions to simulate changes in secondarily formed $PM_{2.5}$, for which reductions in $PM_{2.5}$ tend to be more evenly spread across a study area (Sec-PM). In addition to the air quality modeling approach, linear interpolation and extrapolation were used to simulate just meeting alternative annual standards with levels of 11.0 (interpolated between 12.0 and 10.0 $\mu g/m^3$), 9.0 $\mu g/m^3$, and 8.0 $\mu g/m^3$ (both extrapolated from 12.0 and 10.0 $\mu g/m^3$) in the subset of study areas controlled by the annual standard.

Evidence strongly supports that different racial and ethnic groups, such as Black and Hispanic populations, have higher $PM_{2.5}$ exposures than White and non-Hispanic populations, respectively, thus contributing to increased risk of PM-related effects. In addition to the risk assessment described above, quantitative analyses for this reconsideration also assess long-term $PM_{2.5}$-attributable exposure and mortality risk, stratified by racial/ethnic demographics. Consistent with the overall risk assessment approach, the specific epidemiologic studies and concentration-response functions used in the at-risk analyses were selected to take into account factors such as study design, geographic coverage, demographic populations, and health endpoints. Of the available studies, Di et al., 2017b was identified as best characterizing populations potentially at increased risk of long-term exposure and all-cause mortality and provides race- and ethnicity-stratified concentration-response functions for ages 65 and over (section 3.4.1.6 and Appendix C). Risk is quantitatively assessed within racial and ethnic minority populations of older adults in the full set of 47 areas and the subset of 30 areas controlled by the annual $PM_{2.5}$ standard under Pri-PM air quality simulations. This analysis, when considered alongside estimates of risk across all populations in the 47 study areas, can help to inform preliminary conclusions on the annual primary $PM_{2.5}$ standards that would be requisite to protect the public health of nonwhite populations potentially at increased risk of long-term $PM_{2.5}$-related mortality effects.

3.5.2.2 Estimating Risk under the Current and Alternative Primary $PM_{2.5}$ Standards

In this section, we summarize the risk estimates associated with air quality scenarios just meeting the current primary $PM_{2.5}$ standards and potential alterative standard levels.

- **What are the estimated $PM_{2.5}$-associated health risks for air quality just meeting the current primary $PM_{2.5}$ standards?**

In considering the risk results, we focus first on estimates for the full set of 47 urban study areas. The risk assessment estimates that the current primary $PM_{2.5}$ standards could allow a substantial number of deaths in the U.S., with the large majority of those deaths associated with long-term $PM_{2.5}$ exposures. For example, when air quality in the 47 study areas is adjusted to just meet the current standards, the risk assessment estimates about 41,000 to 45,000 deaths from all-cause mortality in a single year (i.e., for long-term exposures; confidence intervals range from about 30,000 to 59,000) (section 3.4.2.1). For the 30 study areas[76] where just meeting the current

[76] These 30 areas controlled by the annual standard under all scenarios evaluated include a population of approximately 48 million adults aged 30-99, or about 75% of the population included in the full set of 47 areas.

standards is controlled by the annual standard,[77] long-term $PM_{2.5}$ exposures are estimated to be associated with as many as 39,000 (confidence intervals range from about 26,000 to 51,000) deaths from all-cause mortality in a single year (section 3.4.2.2). For the 11 study areas[78] where just meeting the current standards is controlled by the daily standard,[79] long-term $PM_{2.5}$ exposures are estimated to be associated with as many as 2,600 (confidence intervals ranging from 1,700 to 3,400) deaths in a single year (section 3.4.2.3). The risk assessment estimates far fewer deaths in a single year for short-term $PM_{2.5}$ exposures as compared to long-term $PM_{2.5}$ exposures, across all of the study area subsets.

While the absolute numbers of estimated deaths vary across exposure durations, populations, and concentration-response functions, the general magnitude of risk estimates supports the potential for significant public health impacts in locations meeting the current primary $PM_{2.5}$ 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 within the range of long-term average concentrations over which key epidemiologic studies provide strong support for reported positive and statistically significant health effect associations.

- **To what extent are risks estimated to decline when air quality is adjusted to just meet potential alternative standards with lower levels?**

In the 47 urban study areas, when air quality is simulated to just meet alternative standards, there are substantially larger risk reductions associated with lowering the annual standard then with lowering the 24-hour standard. Risks are estimated to decrease by 13-17% when air quality is adjusted to just meet an alternative annual standard with a level of 10.0 $\mu g/m^3$ or by 1-2% when adjusted to just meet an alternative 24-hour standard with a level of 30 $\mu g/m^3$ (section 3.4.2.1). The percentage decrease when just meet an alternative annual standard with a level of 10.0 $\mu g/m^3$ corresponds to approximately 7,400 fewer deaths per year (confidence intervals ranging from about 4,100 to 9,800) attributable to long-term $PM_{2.5}$ exposures.

[77] 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.

[78] These 11 areas controlled by the 24-hour standard under all scenarios evaluated include a population of approximately 10 million adults aged 30-99, or about 17% of the population included in the full set of 47 areas.

[79] 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.

In the 30 study areas where just meeting the current and alternative standards is controlled by the annual standard, air quality adjusted to meet alternative annual standards with lower levels is associated with reductions in estimated all-cause mortality risk. These reductions in risk for alternative annual levels are as follows: 7-9% reduction for an alternative annual level of 11.0 $\mu g/m^3$, 15-19% reduction for a level of 10.0 $\mu g/m^3$, 22-28% reduction for a level of 9.0 $\mu g/m^3$, and 30-37% reduction for a level of 8.0 $\mu g/m^3$ (section 3.4.2.2). For each of these standards, most of the risk remaining is estimated at annual average $PM_{2.5}$ concentrations that fall somewhat below the alternative standard levels.

3.5.2.3 At-Risk Analyses

As noted above, in addition to the risk assessment described in sections 3.4.1.1-3.4.1.5 and 3.4.2.1-3.4.2.3, risk was quantitatively assessed within racial and ethnic minority populations of older adults in the full set of 47 areas and the subset of 30 areas controlled by the annual $PM_{2.5}$ standard under all air quality simulations evaluated (sections 3.4.1.6 and 3.4.2.4).

- **What is the magnitude of population risk in at-risk populations in areas simulated to just meet the current primary $PM_{2.5}$ standards? To what extent are risks estimated to decline within each demographic group when air quality is adjusted to just meet potential alternative annual standards with lower levels?**

The at-risk analysis first compares the average estimated $PM_{2.5}$ exposure concentrations for each demographic population when just meeting the current and alternative annual $PM_{2.5}$ standards. Across all simulated air quality for both the full set of 47 and the subset of 30 study areas, Blacks experience the highest average $PM_{2.5}$ concentrations of the demographic groups analyzed. Native Americans experienced the lowest average $PM_{2.5}$ concentrations, particularly in the full set of 47 study areas. White, Hispanic, and Asian populations were exposed to similar average $PM_{2.5}$ concentrations. Additionally, as the levels of potential alternative annual $PM_{2.5}$ standards decrease, there is comparatively less disproportionate exposure between demographic populations (section 3.4.2.4).

Risk estimates can provide additional information beyond the exposure information to inform our understanding of potentially disproportionate impacts, in this instance by including demographic-specific information on baseline incidence and the relationship between exposure and health effect. Across all air quality scenarios and demographic groups evaluated, Black populations are associated with the largest $PM_{2.5}$-attributable mortality risk rate per 100,000 people, while White populations are associated with the smallest $PM_{2.5}$-attributative mortality risk rate (section 3.4.2.4, Figure 3-20). Generally, as the levels of potential alternative annual $PM_{2.5}$ standards decrease in the 30 areas controlled by the annual standard, the average reduction in $PM_{2.5}$ concentration and mortality risk rates increase across all demographic populations (section 3.4.2.4, Figure 3-21).

In comparing the reductions in average national $PM_{2.5}$ concentrations and risk rates within each demographic population, we note that the average percent $PM_{2.5}$ concentrations and risk reductions are slightly greater in the Black population than in the White population for each alternative standard evaluated (11.0 $\mu g/m^3$, 10.0 $\mu g/m^3$, 9.0 $\mu g/m^3$, and 8.0 $\mu g/m^3$), when shifting from the current annual $PM_{2.5}$ standard (12.0 $\mu g/m^3$) in the full set of 47 areas and the subset of 30 areas controlled by the annual standard. We further note that the difference in average percent risk reductions increases slightly more in Blacks than in Whites as the level of the potential alternative annual standard decreases (section 3.4.2.4, Table 3-19 and Table 3-20).

3.5.2.4 Uncertainties

In this section, we consider uncertainties associated with the quantitative estimates of risk in the overall risk assessment and from risk rates and exposure estimates in the at-risk analysis (sections 3.4.2.5, 3.4.1.7, and 3.4.1.8). Variability and uncertainty associated with the risk estimates are assessed using several quantitative and qualitative approaches, as described in more detail in section C.3 of Appendix C. Generally, the quantitative uncertainty characterization approaches include the following: (1) evaluating multiple concentration-response functions for the same health endpoint; (2) evaluating multiple methods for simulating air quality scenarios; and (3) characterizing the 95% confidence intervals associated with risk estimates. The qualitative uncertainty characterization approach is based on WHO (2008) guidance and on guidance documents developed by the EPA (U.S. EPA, 2001, U.S. EPA, 2004). This qualitative approach includes an assessment of both the magnitude and direction of impact of those uncertainties on risk estimates, including three levels of classification for the magnitude: low, medium, and high.[80]

- **What are the key uncertainties associated with the risk estimates and at-risk analysis, including those of particular significance with regard to drawing conclusions as to the adequacy of the protection afforded by the current primary $PM_{2.5}$ standards?**

Based on the uncertainty characterization and associated analyses in the risk assessment and consideration of associated policy implications, we recognize several areas of uncertainty as particularly important in our consideration of the risk estimates, as was also the case in previous reviews, and in the risk rates and exposure and risk reductions in the at-risk analysis.

[80] The classification of the magnitude of impact for sources of uncertainty includes three levels: (a) low (unlikely to produce a sufficient impact on risk estimates to affect their interpretation), (b) medium (potential to have a sufficient impact to affect interpretation), and (c) high (likely to have an impact sufficient to affect interpretation). For several of the sources, a classification was provided between these levels (e.g., low-medium, medium-high). More information is available in Appendix C, section C.3.

With regard to the concentration-response relationships, we recognize that the degree to which different concentration-response functions result in different risk estimates could reflect differences in study design and/or populations evaluated, as well as other factors. We also note uncertainty in the risk assessment associated with the interpretation of the shapes of concentration-response relationships, particularly at $PM_{2.5}$ concentrations near the lower end of the air quality distribution. This interpretation is complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and "linearize" population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015b, section 6.c). As described in section 3.3.1, the 2019 ISA concludes and the draft ISA Supplement provides further support that the majority of evidence of long-term $PM_{2.5}$ exposure and mortality supports a linear, no-threshold concentration-response relationship, though there is initial evidence indicating that the slope of the concentration-response curve may be steeper at lower concentrations for cardiovascular mortality (U.S. EPA, 2019, section 1.5.3.2; U.S. EPA, 2021a, section 3.2.2.2). The 2019 ISA and draft ISA Supplement note 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 (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section 3.2.2.2.7). As described in more detail in section 3.4.2.5 above and Appendix C, a portion of risk modeling in the risk assessment does include locations with annual ambient $PM_{2.5}$ concentrations adjusted to below 8 ug/m^3, so there is 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 $PM_{2.5}$ exposure and mortality, the 2019 ISA concludes and the draft ISA Supplement provides additional support that, while difficulties remain in assessing the shape of the $PM_{2.5}$-mortality concentration-response relationship 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 $\mu g/m^3$ (U.S. EPA, 2021a, section 3.2.1.2.6). However, we note that in most instances in the risk assessment for this reconsideration, the concentration-response function used had only a small impact on the risk estimates.

With regard to the method for simulating air quality scenarios, the approach used to adjust air quality (i.e., adjusting primary PM emissions or secondary PM emission precursors) had some impact on the overall risk estimates. We also note that there may be uncertainty associated with the methods used to simulate air quality scenarios just meeting the current and potential alternative primary $PM_{2.5}$ standards. The model-based methods for simulating air quality scenarios that just meet the current and alternative standards could contribute to

uncertainties associated with the $PM_{2.5}$ concentration estimates used in the risk assessment and at-risk analyses. While state-of-the-science modeling methods were used to fill in the spatial and temporal gaps in monitoring data, model-related biases and errors can introduce uncertainties. Additionally, the modeling scenarios are based on "across-the-board" changes in primary $PM_{2.5}$ or NO_X and SO_2 emissions from all anthropogenic sources throughout the U.S. by fixed percentages. While this approach tends to target the key sources in each area, emission changes are not tailored to specific periods or sources. Furthermore, while the two adjustment approaches that were applied span a wide range of emissions conditions, they represent a subset of the possible emissions cases that could be used to adjust $PM_{2.5}$ concentrations. In addition, when simulating air quality scenarios that just meet potential alternative annual $PM_{2.5}$ standards using linear extrapolation/interpolation, we recognize that this approach does not fully capture the potential non-linearities associated with real-world changes in air quality. However, it is important to note that the adjustment approach had a larger impact on the distribution of risk reductions, particularly for potential alternative annual standard levels of 9.0 and 8.0 $\mu g/m^3$.

It is important to note that the air quality adjustment approaches applied in the risk assessment differ from the development and modeling of emission control strategies that would occur in implementing a standard. In implementing a standard, an appropriately defined nonattainment area would reduce emissions of primary PM and/or PM precursors selected through analysis of site-specific conditions to meet a standard that is exceeded. In the risk assessment, gridded concentration fields over CBSAs were adjusted to higher or lower concentrations to correspond to just meet standards based on emission changes applied throughout the U.S. Two emission adjustment cases (primary PM and NO_X and SO_2) were used to provide concentration fields that span a wide range of realistic spatial patterns, but the air quality modeling for the risk assessment is not designed to reflect emission changes that might occur in implementing a standard. The Regulatory Impact Analysis (RIA) associated with NAAQS revisions provides illustrative estimates of emission changes needed to meet potential alternative standards and more closely reflects implementation considerations (U.S. EPA, 2013, U.S. EPA, 2015a).

We further note that there is considerable variation in the range of confidence intervals associated with the point estimates generated in the risk assessment, with some concentration-response functions displaying greater variability than others. A number of factors could potentially influence 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 evaluated in the original epidemiologic study.

There may also be uncertainty associated with the potential confounding of the $PM_{2.5}$-mortality effect and the omission of potential confounders from analyses could either increase or

decrease the magnitude of $PM_{2.5}$ effect estimates. Not accounting for confounders can introduce uncertainty into the effect estimates, and thereby introduce uncertainty into the risk estimates that are generated using those effect estimates. While various approaches to control for potential confounders have been adopted across the epidemiologic studies assessed in the 2019 ISA and draft ISA Supplement, and those used in the risk assessment, no individual study adjusts for all potential confounders.

In addition to the uncertainty associated with the risk assessment estimates, additional uncertainties are associated with the risk rates, exposure estimate, and risk reductions in the at-risk analysis. As an initial matter, we note that this analysis is based on race- and ethnicity-stratified concentration-response functions only for ages 65 and over (Di et al., 2017b). The use of one study in such an analysis introduces uncertainties and limitations in the broad applicability of such results in the context of the national U.S. population across demographic groups and age ranges. In addition, each non-White demographic group analyzed in the study comprised a smaller percentage of the full study population, which reduces analytical power. Finally, the risk and exposure assessment focuses on urban areas. This means that demographic groups that preferentially reside in rural areas, such as Native Americans, are underrepresented in this analysis. Additionally, average exposure concentrations estimated for demographic groups with substantial rural populations, such as Whites, may be overestimated in this urban analysis.

In summary, here we recognize several particularly important uncertainties that affect the quantitative estimates of risk rates and exposure in the at-risk analysis and their interpretation in the context of considering the current primary $PM_{2.5}$ standards. These include uncertainties related to the modeling and adjustment methods for simulating air quality scenarios; the potential influence of confounders on the relationship between $PM_{2.5}$ exposure and mortality; the interpretation of the shapes of concentration-response functions, particularly at lower concentrations; and limited availability of studies to inform the at-risk analysis.

3.5.2.5 Potential Public Health Implications

In considering the public health implications of the quantitative risk assessment and at-risk analysis that may inform the Administrator's judgments in this area, this section discusses the information pertaining to the following questions.

- **To what extent are the estimates of risk important from a public health perspective? What does the information available in this reconsideration indicate with regard to the size of the at-risk populations?**

Several factors are important to consideration of public health implications. These include the magnitude or severity of the effects associated with the estimated exposures, as well as their adversity at the individual and population scales. Other important considerations include

the size of the population estimated to experience such effects or to experience exposures associated with such effects. Thus, the discussion here reflects consideration of the risk-based evidence in the context of potential health implications in previous NAAQS decisions.

With regard to $PM_{2.5}$ concentrations in ambient air, the public health implications and potential public health impacts of interest in this reconsideration relate to those effects where a causal relationship with $PM_{2.5}$ exposure was concluded. These are mortality and cardiovascular effects related to both long- and short-term exposures, as summarized in section 3.3.1 above. Such effects, including more serious effects such as mortality, can be considered severe from a public health perspective.

In considering public health implications, it is important to consider impacts on population groups of differing susceptibility. The size of the at-risk populations (children, older adults, those with pre-existing cardiovascular or respiratory diseases) in the U.S. is substantial. As summarized in section 3.3.2, more than 22% of the population are children (<18 years old; approximately 73 million people) and about 16% are older adults (65+ years old; approximately 54 million people). For adults in the U.S. 18 years old and older, cardiovascular diseases are most prevalent in adult populations over the age of 65, with 29% of this age group reporting some type of heart disease (Table 3-3 above). Similarly, adults over the age of 65 also have a greater prevalence of respiratory diseases, particularly COPD reported as chronic bronchitis or emphysema, while the asthma prevalence is generally consistent across all adult age groups for those 18 years or older (Table 3-3). It is important to note that for older adults, the increased risk in this lifestage can likely be attributed to the gradual decline in physiological processes that occurs with aging, and some overlap exists between populations considered to be at-risk because of pre-existing disease and lifestage (U.S. EPA, 2019, p. 12-25).

Another factor that may contribute to differences $PM_{2.5}$ exposures and $PM_{2.5}$-related health risk is race/ethnicity. As described above in section 3.3.2 and in the 2019 ISA and draft ISA Supplement, there is strong evidence demonstrating that Black and Hispanic populations, in particular, have higher $PM_{2.5}$ exposures and health risk disparities compared to non-Hispanic White populations. In the U.S., more than 12% of the U.S. population (more than 40.5 million people) are Blacks and more than 18% are Hispanics (more than 60 million people), while 60% of the population (nearly 197 million people) are non-Hispanic Whites (Table 3-2). Black and Hispanic individuals of all ages make up a substantial portion of the population.

In considering the public health implications of the risk estimates across the study areas, we note the purpose for the study areas is to illustrate circumstances that may occur in areas that just meet the current or potential alternative standards, and not to estimate risk associated with conditions occuring in those specific locations currently. We note that some areas across the U.S. have air quality for $PM_{2.5}$ that is near or above the existing standards. Thus, the air quality and

exposure circumstances assessed in the study areas in the risk assessment are of particular importance in considering whether the currently available information calls into question the adequacy of the public health protection afforded by the current standards.

The risk estimates for the study areas assessed in this reconsideration reflect differences in exposure circumstances among those areas and illustrate the exposures and risks that might be expected to occur in other areas with such circumstances under air quality conditions that just meet the current standards or the alternative standards assessed. Thus, the exposure and risk estimates indicate the magnitude of exposure and risk that might be expected in many areas of the U.S. with $PM_{2.5}$ concentrations at or near the current or alternative standards. Although the methodologies and data used to estimate risks in this reconsideration differ in several ways from what was used in the 2020 review, the findings and considerations summarized here present a pattern of exposure and risk that is generally similar to that considered in the 2020 review, and indicate a level of protection generally consistent with that described in the 2020 PA.

In summary, the considerations raised here are important to conclusions regarding the public health significance of the risk assessment results. Specifically, we note that available evidence and information suggests that both long- and short-term $PM_{2.5}$ exposures are associated with adverse health effects, including more severe effects such as mortality. In addition, we note that such effects impact large segments of the U.S. population, including those populations that may have other factors that influence risk (i.e., lifestage, pre-existing cardiovascular and respiratory diseases, race/ethnicity), as well as disparities in PM2.5 exposures and health risks based on race and ethnicity. Therefore, we recognize that the air quality allowed by the current primary $PM_{2.5}$ standards could be judged to be associated with significant public health risk. We recognize that such conclusions also depend in part on public health policy judgments that will weigh in the Administrator's decision in this reconsideration with regard to the adequacy of protection afforded by the current standards. Such judgments that are common to NAAQS decisions include those related to public health implications of effects of differing severity. Such judgments also include those concerning the public health significance of effects at exposures for which evidence is limited or lacking, such as effects at lower concentrations than those demonstrated in the key epidemiologic studies and in those population groups for which population-specific information, such as concentration-response functions, are not available from the epidemiologic literature.

3.5.3 Preliminary Conclusions

This section describes our preliminary conclusions for the Administrator's consideration in this reconsideration of the primary $PM_{2.5}$ standards. These preliminary conclusions are based on considerations described in the sections above, and in the discussion below regarding the

scientific evidence (as summarized in the 2019 ISA (U.S. EPA, 2019) and the draft ISA Supplement (U.S. EPA, 2021a)), the quantitative assessments of $PM_{2.5}$-associated health risks, and analyses of $PM_{2.5}$ air quality.

3.5.3.1 Current Standards

In taking into consideration the discussions responding to specific questions above in this chapter, this section addresses the following overarching policy question.

- **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 considering this question, we recognize that, as is the case with NAAQS reviews in general, the extent to which the current primary $PM_{2.5}$ standards are judged to be adequate will depend on a variety of factors, including science policy judgments and public health policy judgments to be made by the Administrator. These factors include public health policy judgments concerning the appropriate $PM_{2.5}$ concentrations on which to place weight, as well as judgments on the public health significance of the effects that have been observed at the exposures evaluated in the health effects evidence. The factors relevant to judging the adequacy of the standards also include the interpretation of, and decisions as to the weight to place on, different aspects of the results of the risk assessment for the study areas included and the associated uncertainties. Thus, we recognize that the Administrator's conclusions regarding the adequacy of the current standards will depend in part on judgments regarding aspects of the evidence and risk estimates, and judgments about the degree of protection that is requisite to protect public health with an adequate margin of safety.

Our response to the overarching question above takes into consideration the discussions that address the specific policy-relevant questions in prior sections of this document (sections 3.3, 3.4, 3.5.1, and 3.5.2) and builds on the approach from previous reviews (summarized in section 3.1 above). We focus first on consideration of the evidence, including that assessed in the 2019 ISA and the draft ISA Supplement, and the extent to which it alters key conclusions supporting the current standards. We then turn to consideration of the quantitative estimates of risk developed in this reconsideration, including associated uncertainties and limitations, and the extent to which they indicate differing conclusions regarding the magnitude of risk, as well as level of protection from adverse effects, associated with the current standards. We additionally consider the key aspects of the evidence and risk estimates emphasized in establishing the current standards, and the associated public health policy judgments and judgments about the uncertainties inherent in the scientific evidence and quantitative analyses that are integral to decisions on the adequacy of the current primary $PM_{2.5}$ standards.

We first note that our approach recognizes that the current annual standard (based on arithmetic mean concentrations) and 24-hour standard (based on 98th percentile concentrations), together, are intended to provide public health protection against the full distribution of short- and long-term $PM_{2.5}$ exposures. In general, the annual standard is most effective at controlling exposures to "typical" daily $PM_{2.5}$ concentrations that are experienced over the year, while the 24-hour standard, with its 98th percentile form, is most effective at limiting peak daily or 24-hour $PM_{2.5}$ concentrations. In considering the combined effects of these 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, but also in fewer and lower short-term peak $PM_{2.5}$ concentrations. Additionally, changes designed to meet a lower 24-hour standard, with a 98th percentile form, would most effectively result in fewer and lower peak 24-hour $PM_{2.5}$ concentrations, but also have an effect on lowering the annual average $PM_{2.5}$ concentrations. Thus, our focus in evaluating the current primary standards is on the protection provided by the combination of the annual and 24-hour standards against the distribution of both short- and long-term $PM_{2.5}$ exposures.

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 reconsideration (i.e., assessed in U.S. EPA, 2019 and U.S. EPA, 2021a) and summarized above in section 3.3.1 and section 3.5.1) 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). As noted above, epidemiologic studies conducted in North America, Europe, or Asia 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, as well as evidence from panel studies, strengthens support for potential biological pathways through which $PM_{2.5}$ exposures could lead to the serious effects reported in many population-level epidemiologic studies, including support for pathways that could lead to cardiovascular, respiratory, nervous system, and cancer-related effects.

Epidemiologic studies in the U.S. report health effect associations with mortality and/or morbidity across multiple cities and in diverse populations, including in 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). Further, these studies use a variety of statistical designs, and employ a variety of methods to examine exposure measurement error as well as to control for confounding effects, including more recent causal modeling studies. Results of these

analyses support the robustness of the reported associations. Additional findings from an expanded body of studies that employ causal modeling and accountability methods further inform the causal nature of the relationship between long- or short-term term $PM_{2.5}$ exposure and mortality (U.S. EPA, 2019, sections 11.1.2.1, 11.2.2.4,U.S. EPA, 2021a, sections 3.1.1.3, 3.1.2.3, 3.2.1.3, and 3.2.2.3). These studies, summarized above in Table 3-11 and Table 3-12, examine both short- and long-term $PM_{2.5}$ exposure and cardiovascular effects and mortality, and using a variety of statistical methods to control for confounding bias, consistently report positive associations, which further supports the broader body of epidemiologic evidence for both cardiovascular effects and mortality. Moreover, 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 relationships 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, 2019, section 1.5.3, U.S. EPA, 2021a, sections 2.1.1.5.1 and 2.1.1.5.2). While such analyses are complicated by the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), several studies report positive and statistically significant associations in additional analyses restricted to annual average $PM_{2.5}$ exposures below 12 $\mu g/m^3$ or to daily exposures below 35 $\mu g/m^3$ as exhibited in Table 3-10.

These and other recent studies provide support for health effect associations at lower ambient $PM_{2.5}$ concentrations than in previous reviews. In this reconsideration, 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 3-8, Figure 3-9, Figure 3-10, Figure 3-11). Consistent with the 2012 review, it is important to consider the manner in which $PM_{2.5}$ mean concentrations are estimated (e.g., monitored concentrations versus modeled concentrations) and the method by which means are calculated and reported as the overall mean $PM_{2.5}$ concentration (e.g., averaging across all grid cells in an urban area versus population-weighting). Additional analyses, new in this draft PA though similar to those in the 2012 review, suggest that the area annual design value is generally greater than the study mean by 10-20% (monitor-based studies), 14-18% (hybrid modeling with population-weighting) or 40-50% (hybrid modeling without population weighting). We note this information relative to the overall mean $PM_{2.5}$ concentrations in key U.S. epidemiologic studies which are: 9.9 $\mu g/m^3$ to 16.5 $\mu g/m^3$ for monitor-based studies; 9.3 $\mu g/m^3$ to 12.3 $\mu g/m^3$ for studies that use hybrid modeling and apply population-weighting; and 8.1 $\mu g/m^3$ to 11.9 $\mu g/m^3$ for studies that use hybrid modeling and do not apply population-weighting. The study reported mean concentrations in Canadian studies are more difficult to compare to the area annual standard design value but are lower than those reported in the U.S. studies for both monitor-based and hybrid model methods, ranging from 7.0 $\mu g/m^3$ to 9.0 $\mu g/m^3$ in monitor-based studies, and 6.0 $\mu g/m^3$ to 10.0 $\mu g/m^3$ in model-based

studies. These mean values are consistent with the mean $PM_{2.5}$ concentrations reported in studies available at the time of the 2020 review (U.S. EPA, 2020, Figure 3-8).

In assessing the adequacy of the current standard, we examine a subset of studies, many of which are newly available in this reconsideration, that employ causal modeling methods to control for confounding bias (Table 3-11), which report positive and significant associations for a variety of health outcomes and support the positive and significant associations in analyses identified as key epidemiologic studies above. We also evaluate what the accountability studies may indicate with respect to improvements in public health with improvements in air quality. In so doing, we take note of two accountability studies (Sanders et al., 2020 and Corrigan et al., 2018) newly available in this reconsideration with starting concentrations at or below 12.0 $\mu g/m^3$ that indicate positive and significant associations with mortality and reductions in ambient $PM_{2.5}$ (Table 3-12). We further evaluate studies with analyses that restrict annual or daily $PM_{2.5}$ concentrations to values below the annual or daily $PM_{2.5}$ standard, respectively (Table 3-10). These restricted analyses indicate positive and significant associations, including mean $PM_{2.5}$ concentrations presumably below the mean reported $PM_{2.5}$ in the main cohort, where long-term mean $PM_{2.5}$ concentrations range from 8.2 $\mu g/m^3$ to 11.5 $\mu g/m^3$, as well as effect estimates that are generally greater in magnitude than effect estimates seen in main analyses.

In addition to the epidemiologic evidence, we examine experimental studies, including controlled human exposure studies and animal toxicological studies. As detailed in above in section 3.3.3.1 and section 3.5.1.3, these studies provide support for the effects of exposure to $PM_{2.5}$, and support for biologically plausible mechanisms through which adverse human health outcomes could occur. Exposures in controlled human exposure studies last from less than one hour and up to five hours, and indicate that the most consistent evidence is associated with cardiovascular effects, and more specifically, impaired vascular function. $PM_{2.5}$ exposures evaluated in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards. For example, at air quality monitoring sites meeting the current primary $PM_{2.5}$ standards (i.e., the 24-hour standard and the annual standard), the 2-hour concentrations generally remain below 10 $\mu g/m^3$, and virtually never exceed 30 $\mu g/m^3$. Two-hour concentrations are higher at monitoring sites violating the current standards, but generally remain below 16 $\mu g/m^3$ and virtually never exceed 80 $\mu g/m^3$. In addition, as noted earlier in section 3.3.3.1, chronic vascular dysfunction can be judged to be a biomarker of an adverse health effect from air pollution, but the health relevance of acute reductions in vascular function are less certain (Thurston et al., 2017). Thus, while these studies are important in establishing biological plausibility, it is unclear how the results alone and the importance of the effects observed in these studies, particularly in studies conducted at near-ambient $PM_{2.5}$ concentrations, should be interpreted with respect to adversity to public health..

In addition to the evidence above, 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., with the large majority of those deaths associated with long-term $PM_{2.5}$ exposures. For example, when air quality in the 47 study areas is adjusted to simulate just meeting the current standards, the risk assessment estimates 40,600-45,100 long-term $PM_{2.5}$ exposure-related deaths in a single year, with confidence intervals ranging from 30,300-59,000. While the absolute numbers of estimated deaths vary across exposure durations, populations, and concentration-response functions, the general magnitude of risk estimates supports the potential for significant public health impacts in locations meeting the current primary $PM_{2.5}$ 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 3.3, 3.4, and 3.5 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 assessed in the 2019 ISA and the draft ISA Supplement have reduced key 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 previous reviews.
    - Controlled human exposure studies and animal toxicological studies provide support for the effects of exposure to $PM_{2.5}$, and support for biologically plausible mechanisms through which adverse human health outcomes could occur.
    - Epidemiologic studies that use causal modeling methods have expanded since the 2020 PA and further inform the causal nature of the relationship between short- and long-term exposure to $PM_{2.5}$ and mortality and cardiovascular effects. These studies use a variety of statistical methods to reduce uncertainties with respect to confounding bias.
- Recent U.S. accountability studies provide support for improvements in public health, including reductions in mortality in studies with starting $PM_{2.5}$ concentrations at or below the current primary $PM_{2.5}$ annual standard. Some epidemiologic studies (Corrigan et al., 2018 and Sanders et al., 2020) that employ accountability methods using monitored data evaluate

the effect of the implementation of the 1997 annual $PM_{2.5}$ standard, finding evidence of reductions in mortality in areas with starting $PM_{2.5}$ concentrations at or below 12.0 $\mu g/m^3$.

- Studies that restrict analyses to air quality below the current daily or annual $PM_{2.5}$ standard exhibit positive and significant associations, which are often greater in magnitude than main analyses. Di et al. (2017b) and Dominici et al. (2019) report positive and statistically significant associations that are greater in analyses restricted below 12.0 $\mu g/m^3$ and report mean concentrations of 9.6 $\mu g/m^3$. In studies that restrict analyses < 35.0 $\mu g/m^3$ or lower, mean $PM_{2.5}$ concentrations are not reported, though such means are presumably somewhat below those based on the overall cohort, which range from 8.2 $\mu g/m^3$ to 11.5 $\mu g/m^3$, and effect estimates are generally great than those in the overall cohort. More specifically, one U.S. study by 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.
- Exposures in controlled human exposure studies last from less than one hour and up to five hours and indicate that the most consistent evidence is associated with cardiovascular effects, and more specifically, impaired vascular function. Further, air quality analyses suggest that the ambient concentrations in these studies typically do not occur in locations meeting the current primary standards, thus suggesting that the current primary $PM_{2.5}$ standards provide protection against these "peak" concentrations.
- We note the decision framework used in previous reviews that places significant weight on key epidemiologic studies and consider whether the mean concentrations in these studies would be allowed in areas meeting the current primary standard.
    - Such a decision framework placed significant weight on epidemiologic studies that assessed associations between $PM_{2.5}$ exposure and health outcomes that were most strongly supported by the body of scientific evidence and recognized there is significantly greater confidence in the magnitude and significance of observed associations for the part of the air quality distribution corresponding to where the bulk of the health events in each study have been observed, generally at or around the mean concentration.
    - Additional analyses, new in this draft PA though similar to analyses in the 2012 review, suggest that the area annual design value is greater than the study reported mean values by 10-20% (monitor-based studies), 14-18% (hybrid modeling with population-weighting) or 40-50% (hybrid modeling without population weighting).
    - Focusing on the key epidemiologic studies available in this reconsideration, the overall mean $PM_{2.5}$ concentrations in key U.S. epidemiologic studies are as follows:
        - Range of monitor-based mean $PM_{2.5}$ concentrations is from 9.9 $\mu g/m^3$ to 16.5 $\mu g/m^3$ (range in 2020 PA: 10.7 $\mu g/m^3$ to 16.5 $\mu g/m^3$)
        - Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling and apply population-weighting: 9.3 $\mu g/m^3$ to 12.3 $\mu g/m^3$
        - Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling and do not apply population-weighting: 8.1 $\mu g/m^3$ to 11.9 $\mu g/m^3$

- Though the Canadian studies are more difficult to utilize for comparison to the annual design value used to determine compliance in the U.S., the overall mean $PM_{2.5}$ concentrations in key Canadian epidemiologic studies are within the range, though somewhat lower than those from the U.S. studies, and are as follows:
    - Range of monitor-based mean $PM_{2.5}$ concentrations is from 6.9 $\mu g/m^3$ to 13.3 $\mu g/m^3$
    - Range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling (all of which average up to postal codes and thus include some aspects of population-weighting) is 5.9 $\mu g/m^3$ to 9.8 $\mu g/m^3$
- Past decision frameworks also placed some weight on considering the annual standard level relative to the 25th and 10th percentile of health events while also noting that epidemiologic studies provide more limited support for health effect associations based on air quality distributions at these lower $PM_{2.5}$ concentration percentiles.
    - In key U.S. epidemiologic studies that use monitors 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 $\mu g/m^3$
    - Of the key U.S. epidemiologic studies that use hybrid modeling approaches to estimate long-term $PM_{2.5}$ exposures and do not apply population-weighting, the ambient $PM_{2.5}$ concentrations corresponding to 25th percentiles of estimated exposures range from 4.6 $\mu g/m^3$ to 9.2 $\mu g/m^3$, while in studies that do apply population-weighting, 25th percentiles range from 6.7 $\mu g/m^3$ to 9.1 $\mu g/m^3$. In the two studies (each apply population-weighting) with information available on the 10th percentile of health events, the ambient $PM_{2.5}$ concentrations corresponding to the 10th percentile are 4.7 $\mu g/m^3$ and 7.3 $\mu g/m^3$.

- 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. Further, the risk assessment estimated that Black populations may experience disproportionally higher exposures and risk under simulated air quality conditions just meeting the current primary $PM_{2.5}$ annual standard as compared to White populations.

When taken together, we reach the conclusion that the available scientific evidence, air quality analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the combination of the current annual and 24-hour primary $PM_{2.5}$ standards. In particular, we note the information and analyses new to this reconsideration (and discussed in detail above) in reaching this conclusion.

3.5.3.2 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 reconsideration. 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 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.

3.5.3.2.1 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 2012 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 2012 review, studies assessed the 2019 ISA continue to provide strong support for health effects following long- and short-term $PM_{2.5}$ exposures (U.S. EPA, 2019). While some studies evaluate the health effects of particular sources of fine particles, or of particular fine particle components, evidence from these studies does not identify any one source or component that is a better predictor of health effects than $PM_{2.5}$ mass (U.S. EPA, 2019, section 1.5.4). As summarized in section 3.5.1 above, the 2019 ISA the evidence confirms and further supports that many $PM_{2.5}$ components and sources are associated with health effects, and 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, 2019, section 1.5.4). Further, 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, and the varying definitions of UFP, as well as differences in approaches to administering and measuring UFP, contribute to such limitations (U.S. EPA, 2019, section 1.4.3). Thus, for reasons similar to those discussed in the 2020 review (85 FR 82715, December 18, 2020), 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.

3.5.3.2.2 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 this reconsideration, 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, 2019, section 1.5.2.1). In addition, studies of sub-daily exposures typically examine subclinical effects, rather than the more serious population-level effects that have been reported to be associated with 24-hour exposures (e.g., mortality, hospitalizations). Taken together, the 2019 ISA concludes that epidemiologic studies do not indicate sub-daily averaging periods are more closely associated with health effects than the 24-hour average exposure metric (U.S. EPA, 2019, section 1.5.2.1).

Additionally, while recent controlled human exposure studies provide consistent evidence for cardiovascular effects following $PM_{2.5}$ 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 3.3.3.1). 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 2019 ISA, and the observations noted above, we reach the conclusion that the available evidence continues to provide strong support for consideration of retaining the current annual and 24-hour averaging times. The available evidence suggests that $PM_{2.5}$ 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 2020 review (78 FR 82715, December 18, 2020), we reach the preliminary conclusion that 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.

3.5.3.2.3 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 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 61117, October 17, 2006).[81]

In the 2012 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 2011 PA concluded that spatial averaging could result in disproportionate impacts in minority populations and populations with lower SES. The Administrator concluded that public health would not be protected with an adequate margin of safety in all locations, as required by law, if disproportionately higher $PM_{2.5}$ 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 2012 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 2012 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).

In the 2020 review, the Administrator noted that the scientific evidence continued to provide strong support for health effect associations for both long-term (e.g., annual or multi-year) and short-term (e.g., mostly 24-hour) exposures to $PM_{2.5}$ and judged that the evidence did not support considering alternative averaging times (85 FR 82715, December 18, 2020). For

[81] 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).

reasons consistent with those in the 2012 review, the Administrator judged that the current annual and 24-hour averaging times remained appropriate.

The information available in this reconsideration continues to support the current forms of the annual and 24-hour $PM_{2.5}$ standards. As discussed above (section 3.3.1), 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 3.3.3.2.1). The form of the current annual standard (i.e., arithmetic mean, averaged over three years) remains appropriate for targeting "typical" daily and annual 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 3.3.3.1). Thus, the evidence also supports retaining a standard focused on providing supplemental protection against short-term peak exposures. The information available in this reconsideration continues to support 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 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.

3.5.3.2.4 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.

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, 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 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, 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 key observations in section 3.5.3.1 (rather than repeating them here) and note more specifically, related to those observations that:

Mean $PM_{2.5}$ Concentrations in Key Epidemiologic Studies and Relationships between Mean $PM_{2.5}$ Concentrations and Annual Design Values

- 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 (which is measured at the peak monitor). 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 in an area (section 2.3.3.1, Figure 2-28; Table 2-2). Additional analyses also support differences between annual $PM_{2.5}$ design values and long-term mean $PM_{2.5}$ concentrations in hybrid modeling studies, with the extent of the difference depending on the methods used to estimate mean $PM_{2.5}$ concentrations. These analyses suggest that the area annual design values are generally higher than the study mean by 14-18% (hybrid modeling with population-weighting) or 40-50% higher (hybrid modeling without population-weighting) (section 2.3.3.2.4, Table 2-4).

- Most key U.S. epidemiologic studies indicate consistently positive and statistically significant health effect associations based on air quality distributions with overall mean $PM_{2.5}$ concentrations at or above 9.3 $\mu g/m^3$ (9.9 $\mu g/m^3$ based on U.S. studies that use monitors to estimate $PM_{2.5}$ exposures). Other key epidemiologic studies (which do not incorporate population-weighting into their calculation of the study mean) report mean $PM_{2.5}$ concentrations to be as low as 8.1 $\mu g/m^3$ with the air quality analyses suggesting that areas included in these studies would have corresponding area annual design values generally 40-50% higher than the study reported mean concentrations.

- Though the mean $PM_{2.5}$ concentrations from Canadian studies are more difficult to directly compare to the annual design value used to determine compliance in the U.S., the overall mean $PM_{2.5}$ concentrations in key Canadian epidemiologic studies are close to, though somewhat lower than, those from the U.S. studies. The range of monitor-based mean $PM_{2.5}$ concentrations is from 6.9 $\mu g/m^3$ to 13.3 $\mu g/m^3$ while the range of mean $PM_{2.5}$ concentrations in studies that use hybrid modeling (all of which average up to postal codes and thus include some aspects of population-weighting) is 5.9 $\mu g/m^3$ to 9.8 $\mu g/m^3$.

- Epidemiologic studies provide more limited support for health effect associations based on air quality distributions at lower $PM_{2.5}$ percentile concentrations. In assessing the 25^{th} percentile of data, $PM_{2.5}$ concentrations in key U.S. epidemiologic studies that use hybrid modeling methods and do not apply some aspects of population-weighting range from 4.6 $\mu g/m^3$ to 9.2 $\mu g/m^3$, while those that apply some aspects of population weighting range from 6.7 $\mu g/m^3$ to 9.1 $\mu g/m^3$. In U.S. studies that use monitored values have 25^{th} percentiles ranging from 11.5 $\mu g/m^3$ to just below 13.0 $\mu g/m^3$. In Canada two monitored studies report 25^{th} percentile concentrations around 6.5 $\mu g/m^3$, while hybrid modeled studies in Canada, all of which average up to postal codes and thus include some aspects of population-weighting, report 25^{th} percentile concentrations around 8.0 $\mu g/m^3$ in two studies, and 4.3 $\mu g/m^3$ in one study.

Scientific Evidence and Associated Uncertainties Supporting Associations at Lower Concentrations

- Recent evidence further demonstrates that associations with mortality remain robust in copollutants analyses (U.S. EPA, 2019, section 11.2.3), and that associations persist in

analyses restricted to long-term exposures below 12 μg/m^3 (Di et al., 2017b) or 10 μg/m^3 (Shi et al., 2016) (i.e., indicating that risks are not disproportionately driven by the upper portions of the air quality distribution).

- 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, 2019, section 1.5.3, U.S. EPA, 2021a, section 2.2.3.1 and 2.2.3.2). While such analyses are complicated by the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), analyses that assess the concentration-response relationship support a linear, no-threshold effect down to 5.0 μg/m^3, though uncertainties increase at concentrations of less than 8.0 μg/m^3.

- While there is no specific point in the air quality distribution of any epidemiologic study that represents a "bright line" at and above which effects have been observed and below which effects have not been observed, there is significantly greater confidence in the magnitude and significance of observed associations for the part of the air quality distribution corresponding to where the bulk of the health events in each study have been observed, generally at or around the mean concentration, with more limited support for health effect associations based on air quality distributions at lower $PM_{2.5}$ percentile concentrations.

- Controlled human exposure studies demonstrate consistent evidence of effects at higher concentrations (e.g., > 120 μg/m^3) and provide support for biological plausibility for more serious effects (e.g., hospital admissions) (U.S. EPA, 2019, Figure 6-1).

Scientific Evidence on Short-term Exposures and $PM_{2.5}$ Exposures Shown to Cause Effects

- 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, 2019, 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) (Figure 2-19, Figure A-2, Figure A-3).

$PM_{2.5}$-Associated Risk Estimates

- The risk assessment estimates that, compared to the current standards, potential alternative annual standards with levels from 11.0 down to 8.0 μg/m^3 could reduce $PM_{2.5}$-associated mortality broadly across the United States. Meeting a revised annual standard with a lower level is estimated to reduce $PM_{2.5}$-associated health risks in the 30 annually-controlled study areas by about 7-9% for a level of 11.0 μg/m^3, 15-19% for a level of 10.0 μg/m^3, 22-28% for a level of 9.0 μg/m^3, and 30-37% for a level of 8.0 μg/m^3, compared to the current annual standard.

- Revising the level of the 24-hour standard to 30 μg/m^3 is estimated to lower $PM_{2.5}$-associated risks across a more limited population and number of areas than revising the annual standard (section 3.4.2.3). Risk reduction predictions are largely confined to areas located in the western U.S., several of which are also likely to experience risk reductions upon meeting a revised annual standard.

- The at-risk assessment estimated that Black populations may experience disproportionally higher exposures and risk under simulated air quality conditions just meeting the current primary $PM_{2.5}$ annual standard as compared to White populations. Meeting a revised annual standard with a lower level may also proportionally reduce exposure and risk in Black populations slightly more so than in White populations in simulated scenarios just meeting alternative annual standards.
- Uncertainties in risk estimates (e.g., in the size of risk estimates) can result from a number of factors, including assumptions about the shape of the C-R relationship with mortality at low ambient PM concentrations, the potential for confounding and/or exposure measurement error in the underlying epidemiologic studies, and the methods used to adjust $PM_{2.5}$ air quality. In considering such uncertainties, we recognize that the risk estimates can help to place the evidence for specific effects into a broader public health context, but should be considered along with the inherent uncertainties and limitations of such analyses when informing judgments about the potential for additional public health protection associated with $PM_{2.5}$ exposure and related health effects.

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. 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.

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. 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, a level below 12 $\mu g/m^3$ and 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 distributions below those present in most key epidemiologic studies, recognizing the general relationships demonstrated in the air quality analyses between study mean calculation and the annual standard and noting the values of the study reported means as listed below:

    - The monitor-based key epidemiologic studies report mean $PM_{2.5}$ concentrations from 9.9 $\mu g/m^3$ to 16.5 $\mu g/m^3$;
    - The key epidemiologic studies that incorporate hybrid modeling and population-weight study mean $PM_{2.5}$ concentrations report means from 9.3 $\mu g/m^3$ to 12.2 $\mu g/m^3$.
- Noting that given the differences between population densities, $PM_{2.5}$ concentration gradients, and source distributions between the U.S. and Canada, it may be inappropriate to draw a direct comparison between the Canadian study means and the annual design value metric used for compliance in the U.S., but also noting that the study reported means from the Canadian studies are similar, though somewhat lower, than those in the U.S.
- Setting a standard level within the starting range of the mean $PM_{2.5}$ concentrations evaluated in accountability studies, recognizing that some of the studies that report public health improvements with improvements to air quality have starting concentrations that range between 10.0 $\mu g/m^3$ to 12.0 $\mu g/m^3$ (Table 3-12).
- Setting a standard estimated to reduce $PM_{2.5}$-associated health risks, such that a substantial portion of the risk reduction that would be accomplished is estimated at annual average $PM_{2.5}$ concentrations within the range of overall means for which key epidemiologic studies indicate consistently positive and statistically significant health effect associations ($\geq$ about 8 $\mu g/m^3$) while also noting important uncertainties inherent in the risk assessment as described in detail in sections 3.4.1.7 and 3.4.1.8. Further, the at-risk analyses indicate that the average percent reduction in $PM_{2.5}$ concentrations and risk are slightly greater in the Black population than in the White population for each alternative standard evaluated (11.0 $\mu g/m^3$ and 10.0 $\mu g/m^3$), when shifting from the current annual $PM_{2.5}$ standard (12.0 $\mu g/m^3$) in the full set of 47 areas and the subset of 30 areas controlled by the annual standard (section 3.4).
- Noting a number of uncertainties associated with the scientific evidence and risk information including: (1) there are few key epidemiologic studies (and only one key U.S. study) that 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 3.3.3.2.1); (2) 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 3.3.3.2.1); and (3) there is increasing uncertainty in quantitative estimates of $PM_{2.5}$-associated mortality risk for standard levels below 10.0 $\mu g/m^3$, given that a substantial proportion of the risk reductions estimated for lower standard levels occur at annual average $PM_{2.5}$ concentrations below 8 $\mu g/m^3$, 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 3.4.1.7).

In contrast, an annual standard with a level below 10.0 $\mu g/m^3$ and as low as 8.0 $\mu g/m^3$, 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 $\mu g/m^3$) and less weight is placed on the

limitations in the evidence that contribute to greater uncertainty at lower concentrations. For example, a level below 10.0 $\mu g/m^3$ could be supported to the extent greater weight is placed on the following:

- Setting the annual standard at or below most or all of the study reported means, including means of hybrid modeling studies that did not use population weighted approaches, such that the standard would be expected to maintain the $PM_{2.5}$ air quality distributions further below those present in most key epidemiologic studies and noting that the relationships between study mean calculation and the annual standard in the draft PA analyses are approximations and less weight should be placed on them and the mathematical approach used to calculate the mean.
- Results of the key Canadian epidemiologic studies, which report mean $PM_{2.5}$ concentrations that are lower than those reported in U.S. studies and for which the $PM_{2.5}$ concentrations generally range from 7.0 $\mu g/m^3$ to 9.0 $\mu g/m^3$ (monitor-based) and 6.0 $\mu g/m^3$ to 10.0 $\mu g/m^3$ (hybrid model-based and all of which apply some aspects of population-weighting) (section 3.3.3.2.1);
- Consideration of the air quality distribution below the mean for which key epidemiologic studies have reported associations with health effects. The ambient $PM_{2.5}$ concentrations around the 25th percentile of underlying data, which range from 11.5 $\mu g/m^3$ to 12.9 $\mu g/m^3$ in U.S. monitor-based studies, from 6.5 $\mu g/m^3$ to 6.8 $\mu g/m^3$ in Canadian monitor-based studies, from 4.6 $\mu g/m^3$ to 9.2 $\mu g/m^3$. In key U.S. epidemiologic studies that use hybrid modeling methods and do not apply some aspects of population-weighting range from 4.6 $\mu g/m^3$ to 9.2 $\mu g/m^3$, while those that apply some aspects of population weighting range from 6.7 $\mu g/m^3$ to 9.1 $\mu g/m^3$ while hybrid modeled studies in Canada, all of which average up to postal codes and thus include some aspects of population-weighting, report 25th percentile concentrations around 8.0 $\mu g/m^3$ in two studies, and 4.3 $\mu g/m^3$ in one study (section 3.3.3.2.1);
- Noting studies that examined the shapes of concentration-response functions over the full distribution of ambient $PM_{2.5}$ concentrations and concluded that while the concentration-response relationship support a linear, no-threshold effect down to 5.0 $\mu g/m^3$, uncertainties increase at concentrations of less than 8.0 $\mu g/m^3$; and also noting that the $PM_{2.5}$ exposure concentrations in an area with a design value of less than 8.0 $\mu g/m^3$ would reflect a distribution of air quality that would be mostly associated with average daily concentrations below 8.0 $\mu g/m^3$.
- The potential for continued public health improvements with improvements in air quality below the lowest starting concentration evaluated in accountability studies, which was approximately 10.0 $\mu g/m^3$ (Table 3-12);
- Studies that restrict analyses to air quality associated with levels below the current annual standard and report positive and significant associations, often with effect estimates that are greater in magnitude than those reported in the main analysis. Although the mean of the restricted analyses are generally not reported, in one key U.S. epidemiologic study, the mean concentration when restricting annual average $PM_{2.5}$ concentrations to below 12.0 $\mu g/m^3$ was presumably lower than the overall mean concentration of 8.1 $\mu g/m^3$ reported in the main analysis (Shi et al., 2016) (Table 3-10);

- The potential public health importance of the additional reductions in $PM_{2.5}$-associated health risks estimated for a level below 10.0 $\mu g/m^3$ $\mu g/m^3$ and the potential for continued improvements below the lowest level examined in the risk assessment (8.0 $\mu g/m^3$). Further, the at-risk analyses indicate that the average percent reduction in $PM_{2.5}$ concentrations and risk are slightly greater in the Black population than in the White population for each alternative standard evaluated (9.0 $\mu g/m^3$ and 8.0 $\mu g/m^3$), when shifting from the current annual $PM_{2.5}$ standard (12.0 $\mu g/m^3$) in the full set of 47 areas and the subset of 30 areas controlled by the annual standard (section 3.4).

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. With respect to current and recent air quality relationships, we note that 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 $PM_{2.5}$ design values below 35 $\mu g/m^3$ (and below 30 $\mu g/m^3$ in most areas) (chapter 2, Figure 2-18). The exceptions are a few CBSAs in the western U.S.

As in previous reviews, we recognize that the annual standard would generally be the controlling standard across much of the U.S., except for certain areas where there are high seasonal emissions (e.g., wood smoke) and conducive meteorology (e.g., temperature inversions) or where there are more unique source-oriented influences (e.g., near manufacturing sources). In such areas, the 24-hour standard is the generally controlling standard, though the number of these areas in the U.S. is small. Thus, as was the approach in multiple recent reviews, we 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). 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 previous reviews (78 FR 3161-3162, January 15, 2013; 85 FR 82715, December 18, 2020), we focus on 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.

Taking into account this approach, 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 that 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:

- Controlled human exposure studies 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). In the studies evaluated at near ambient $PM_{2.5}$ concentrations, results are mixed but they do 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).
- Animal toxicologic studies provide evidence of effects related to short-term exposures to $PM_{2.5}$ at concentrations ranging from 100 to > 1,000 $\mu g/m^3$ and providing further evidence to support the biological mechanisms and plausibility of various adverse effects associated with short-term exposures.
- The body of epidemiologic evidence provides limited support for judging adequacy of the level of the 24-hour standard. As discussed in detail above (section 3.3.3.2.1), 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. Additionally, the magnitudes of the associations in restricted analyses are similar to or larger than the magnitudes of the associations based on the full cohorts (Table 3-10), suggesting that, at a minimum, short-term exposures to peak $PM_{2.5}$ concentrations are not disproportionately responsible for reported health effect associations.

Based on the evidence above, we assessed the protection provided by the current standards against the concentrations seen in the human exposure studies. The air quality analyses included in this draft PA show that 2-hour ambient concentrations of $PM_{2.5}$ at monitoring sites meeting the current standards almost never exceed 30 $\mu g/m^3$ (Figure 2-19). In fact, even the extreme upper end of the distribution of 2-hour $PM_{2.5}$ concentrations at sites meeting the current standards remain well-below the $PM_{2.5}$ exposure concentrations consistently shown to elicit effects (i.e., 99.9^{th} percentile of 2-hour concentrations at these sites is 62 $\mu g/m^3$ during the warm season). We also note some caution in placing too much weigh on the need to provide protection against any of the exposures observed in the clinical studies given that it is unclear how the results alone and the importance of the effects observed in these studies, particularly in the studies conducted at near-ambient $PM_{2.5}$ concentrations, should be interpreted with respect to adversity to public health.

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 average short- and long-term $PM_{2.5}$ exposures 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 the epidemiologic studies do support the need to consider increasing protection against the typical daily and annual $PM_{2.5}$ exposures that provide strong support for reported health effect associations, these studies do not provide the same support for a need for increasing protection against short-term, peak exposures. Further, the epidemiologic studies do not indicate that the reported health effect associations in these studies are strongly influenced by exposures to the peak concentrations in the air quality distribution. Also, while animal toxicologic studies provide evidence to support the biological mechanisms and plausibility of various adverse effects associated with short-term exposures, they provide limited support for judging adequacy of the level of the 24-hour standard. Human clinical 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, though these concentrations are well above those typically measured in areas meeting the current standards, suggesting that the current standards are providing protection against these exposures. As such, the available evidence supports the need for the current 24-hour standard to protect against peak concentrations but does not clearly support the need for a lower level of that standard. 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 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 this reconsideration, consideration of 24-hour standard levels 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 3.4.2).

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 $PM_{2.5}$ 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.

3.6 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

In this section, we identify key areas for additional research and data collection for fine particles, based on the uncertainties and limitations that remain in the evidence and technical information. Additional research in these areas could reduce uncertainties and limitations in future reviews of the primary $PM_{2.5}$ 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 exposures near ambient $PM_{2.5}$ concentrations (e.g., Wyatt et al. (2020a) 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 near ambient $PM_{2.5}$ concentrations.
- Additional research into "causal inference" methods in epidemiologic studies to evaluate the causal nature of relationships between $PM_{2.5}$ exposure and mortality or morbidity.
- Additional research into "accountability" or "quasi-experimental" epidemiologic studies with 'starting $PM_{2.5}$ concentrations' below 12.0 $\mu g/m^3$.
- 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 and the composition of ultrafine particles, 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.
- Future research to examine $PM_{2.5}$ exposure and associated effects in pregnant women, and birth outcomes, as well as future research and data collection to examine developmental outcomes and different life stages

In addition to research and data collection, additional information that could be reported in epidemiologic studies may help to reduce uncertainties and limitations in future reviews of the primary $PM_{2.5}$ standards. This information includes:

- Descriptive statistics of $PM_{2.5}$ concentrations that are used in epidemiologic studies to evaluate associations between $PM_{2.5}$ and health effects (e.g., minimum, maximum, 10^{th} percentile, 25^{th} percentile, mean, median, 75^{th} percentile).

- More detailed information on the methods used to calculate the mean $PM_{2.5}$ concentrations that are reported in the study (e.g., whether population-weighting was applied, how the $PM_{2.5}$ concentrations estimated from hybrid modeling are averaged prior to being assigned to health events).
    - Noting whether the mean $PM_{2.5}$ concentration reported is the concentration across the area evaluated or if the mean $PM_{2.5}$ concentration reported is based only $PM_{2.5}$ concentrations used in analyses to assess the association between health outcomes and $PM_{2.5}$.
- In analyses restrict $PM_{2.5}$ concentrations below specific concentrations (e.g., below annual averages of 12.0 $\mu g/m^3$ or below daily averages of 35 $\mu g/m^3$) reporting of the Mean $PM_{2.5}$ concentrations in the restricted analysis could be helpful.

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4 RECONSIDERATION OF THE PRIMARY STANDARD FOR PM_{10}

This chapter presents and evaluates the policy implications of the scientific and technical information pertaining to reconsideration of the 2020 final decision on the primary PM_{10} standard. In so doing, the chapter presents key aspects of the health effects evidence of $PM_{10-2.5}$, as documented in the 2019 ISA, with support from the prior ISA and AQCDs, and associated public health implications. This information provides the basis for our evaluation of the scientific information regarding health effects of PM_{10} in ambient air and the potential for effects to occur under air quality conditions associated with the existing standard, as well as the associated implications for public health. Our evaluation is framed around key policy-relevant questions derived from the IRP (U.S. EPA, 2016, section 2.1) for the review completed in 2020, and the scientific conclusions regarding the relationship between short- and long-term $PM_{10-2.5}$ exposure and health effects detailed in the 2019 ISA, while also taking into account conclusions reached in previous reviews. In this way, we identify key policy-relevant issues and summary conclusions regarding the public health protection provided by the current standard as the Administrator reconsiders the final 2020 decision on the primary PM_{10} standard.

As described in Chapter 1, the scope of the updated scientific evaluation of the health effects evidence for PM_{10} is based on those health effects categories where the 2019 ISA concluded a causal relationship exists. Therefore, the draft ISA Supplement does not include an evaluation of additional studies for $PM_{10-2.5}$ and the 2019 ISA continues to serve as the scientific foundation for assessing the adequacy of the primary PM_{10} standard in this reconsideration of the 2020 final decision (U.S. EPA, 2019, section 1.7; U.S. EPA, 2021). As such, this chapter draws heavily from the 2020 PA in identifying and summarizing key issues related to this reconsideration of the primary PM_{10} standard.

Within this chapter, background information on the current standard is summarized in section 4.1. The general approach for evaluating the available information in this reconsideration, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 4.2. Key aspects of the available health effects evidence presented in the 2019 ISA and considered in the 2020 PA are addressed in section 4.3. Section 4.4 summarizes the key evidence-based considerations identified in our evaluation and presents associated preliminary conclusions on the adequacy of the current standard. Key remaining uncertainties and areas for future research are identified in section 4.5.

4.1 BACKGROUND ON THE CURRENT STANDARD

With the 2020 final decision on the PM NAAQS, 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 short-term exposures to $PM_{10-2.5}$ (85 FR 82725, December 18, 2020). This decision was based on the scientific information available at that time, as well as the Administrator's judgments regarding the health effects evidence and the appropriate degree of public health protection for the existing standard.

The health effects evidence assessed in the 2019 ISA included an expanded body of scientific evidence linking short-term $PM_{10-2.5}$ to health outcomes such as premature death and hospital visits (U.S. EPA, 2009, U.S. EPA, 2019). This evidence base assessed the causal nature of relationships between short-term exposure to $PM_{10-2.5}$ and a broad range of health effects (U.S. EPA, 2020, section 1.4.2). These effects associated with short-term exposure ranged from hospital admissions and emergency department visits for cardiovascular effects (documented in epidemiologic studies that reported $PM_{10-2.5}$ associations with cardiovascular hospital admissions and emergency department visits in study locations with mean 24-hour average $PM_{10-2.5}$ concentrations ranging from 7.4 to 13 $\mu g/m^3$) and respiratory effects (documented in epidemiologic studies that reported $PM_{10-2.5}$ associations with respiratory hospital admissions and emergency department visits in study locations with mean 24-hour average concentrations ranging from 5.6 to 16.2 $\mu g/m^3$) to mortality (documented in epidemiologic studies that reported $PM_{10-2.5}$ associations with mortality in study areas with mean 24-hour average concentrations ranging from 6.1 $\mu g/m^3$ to 16.4 $\mu g/m^3$). In addition to the epidemiologic studies, the evidence base included few controlled human exposure studies and animal toxicologic studies that provided insight into the biological plausibility of these effects. Collectively, the epidemiologic studies, controlled human exposure, and animal toxicological studies, with their inherent uncertainties, contributed to the causality determinations of "suggestive of, but not sufficient to infer, a causal relationship" between short-term exposures to $PM_{10-2.5}$ and cardiovascular effects, respiratory effects, and mortality (U.S. EPA, 2009, U.S. EPA, 2019, section 1.4.2).

Building on the evidence considered in the 2012 review, the primary focus in the 2020 review was on multi-city and single-city epidemiologic studies that evaluated associations between short-term $PM_{10-2.5}$ and mortality, cardiovascular effects (hospital admissions and emergency department visits), and respiratory effects. Despite differences in the approaches used to estimate ambient $PM_{10-2.5}$ concentrations, the majority of the studies reported positive, though often not statistically significant, associations with short-term $PM_{10-2.5}$ exposures. Most $PM_{10-2.5}$ effect estimates remained positive in copollutant models that included either gaseous pollutants or other particulate matter size fractions (e.g., $PM_{2.5}$). In U.S. study locations likely to have met

the PM_{10} standard during the study period, a few studies reported positive associations between $PM_{10-2.5}$ and mortality that were statistically significant and remained so in copollutant models (U.S. EPA, 2009, U.S. EPA, 2019).

In addition to the epidemiologic studies, there were a small number of controlled human exposure studies assessed in the 2019 ISA that reported alterations in heart rate variability or increased pulmonary inflammation following short-term exposure to $PM_{10-2.5}$, providing some support for the associations in the epidemiologic studies. Toxicological studies that examined the effects of $PM_{10-2.5}$ used intratracheal instillation as opposed to inhalation. Therefore, these studies provided limited evidence for the biological plausibility of $PM_{10-2.5}$-induced effects (U.S. EPA, 2009, U.S. EPA, 2019).

Although the scientific evidence available in the 2019 ISA expanded the understanding of health effects associated with $PM_{10-2.5}$ exposures, a number of important uncertainties remained. These uncertainties, and their implications for interpreting the scientific evidence, include the following:

- The potential for confounding by copollutants, notably $PM_{2.5}$, was addressed with copollutant models in a relatively small number of $PM_{10-2.5}$ epidemiologic studies (U.S. EPA, 2009, U.S. EPA, 2019). This was particularly important given the relatively small body of experimental evidence (i.e., controlled human exposure and animal toxicological studies) available to support the independent effect of $PM_{10-2.5}$ on human health. This increases the uncertainty regarding the extent to which $PM_{10-2.5}$ itself, rather than one or more cooccurring pollutants, is responsible for the mortality and morbidity effects reported in epidemiologic studies.
- There was greater spatial variability in $PM_{10-2.5}$ concentrations than $PM_{2.5}$ concentrations, resulting in increased exposure error for $PM_{10-2.5}$ (U.S. EPA, 2009, U.S. EPA, 2019). Available measurements did not provide sufficient information to adequately characterize the spatial distribution of $PM_{10-2.5}$ concentrations (U.S. EPA, 2009, U.S. EPA, 2019). The limitations in estimates of ambient $PM_{10-2.5}$ concentrations "would tend to increase uncertainty and make it more difficult to detect effects of $PM_{10-2.5}$ in epidemiologic studies" (U.S. EPA, 2009, U.S. EPA, 2019).
- The distributions of $PM_{10-2.5}$ concentrations over which reported health outcomes occur remain highly uncertain. Only a relatively small number of $PM_{10-2.5}$ monitoring sites were operating at the time of the 2012 review and such sites had only been in operation for a relatively short period of time, limiting the spatial and temporal coverage for routine measurement of $PM_{10-2.5}$ concentrations. Given these limitations in routine monitoring, epidemiologic studies employed a number of different approaches for estimating $PM_{10-2.5}$ concentrations. Given the relatively small number of $PM_{10-2.5}$ monitoring sites, the relatively large spatial variability in ambient $PM_{10-2.5}$ concentrations, the use of different approaches to estimating ambient $PM_{10-2.5}$ concentrations across epidemiologic studies, and the limitations inherent in such estimates, the distributions of $PM_{10-2.5}$ concentrations over which reported health outcomes occur remain highly uncertain (U.S. EPA, 2009, U.S. EPA, 2019).

- There was relatively little information on the chemical and biological composition of $PM_{10-2.5}$ and the effects associated with the various components (U.S. EPA, 2019). Without more information on the chemical speciation of $PM_{10-2.5}$, the apparent variability in associations with health effects across locations was difficult to characterize (U.S. EPA, 2009, U.S. EPA, 2019).

Consistent with the general approach routinely employed in NAAQS reviews, the initial consideration in the 2020 review of the primary PM_{10} standard was with regard to the adequacy of protection provided by the then-existing standard. Key aspects of that consideration are summarized in section 4.1.1 below.

4.1.1 Considerations Regarding the Adequacy of the Existing Standards in the 2020 Review

In the 2020 final decision, 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 $PM_{10-2.5}$ (85 FR 82727, December 18, 2020). In reaching his decision, the Administrator specifically noted that, while the health effects evidence was somewhat expanded since the prior reviews, the overall conclusions in the 2019 ISA, including uncertainties and limitations, were generally consistent with what was considered in the 2012 review (85 FR 82725, December 18, 2020). In addition, the Administrator recognized that there were still a number of uncertainties and limitations associated with the available evidence.

With regard to the evidence on $PM_{10-2.5}$-related health effects, the Administrator noted that epidemiologic studies continued to report positive associations with mortality and morbidity in cities across North America, Europe, and Asia, where $PM_{10-2.5}$ sources and composition were expected to vary widely. While significant uncertainties remained in the 2020 review, the Administrator recognized that this expanded body of evidence had broadened the range of effects that have been linked with $PM_{10-2.5}$ exposures. The studies evaluated in the 2019 ISA expanded the scientific foundation presented in the 2009 ISA and led to revised causality determinations (and new determinations) for long-term $PM_{10-2.5}$ exposures and mortality, cardiovascular effects, metabolic effects, nervous system effects, and cancer (85 FR 82726, December 18, 2020). Drawing from his consideration of this evidence, the Administrator concluded that the scientific information available since the time of the last review supported a decision to maintain a primary PM_{10} standard to provide public health protection against $PM_{10-2.5}$ exposures, regardless of location, source of origin, or particle composition (85 FR 82726, December 18, 2020).

With regard to uncertainties in the available evidence, the Administrator first noted that a number of limitations were identified in the 2012 review related to: (1) estimates of ambient $PM_{10-2.5}$ concentrations used in epidemiologic studies; (2) limited evaluation of copollutant

models to address the potential for confounding; and (3) limited experimental studies supporting biological plausibility for $PM_{10-2.5}$-related effects. Despite the expanded body of evidence for $PM_{10-2.5}$ exposures and health effects, the Administrator recognized that uncertainties in the 2020 review continued to include those associated with the exposure estimates used in epidemiologic studies, the independence of the $PM_{10-2.5}$ health effect associations, and the biologically plausible pathways for $PM_{10-2.5}$ health effects (85 FR 82726, December 18, 2020). These uncertainties contributed to the 2019 ISA determinations that the evidence is "suggestive of, but not sufficient to infer" causal relationships (85 FR 82726, December 18, 2020).

Further, consistent with the approach in reaching the 2012 decision, the approach for the 2020 PM NAAQS review did not include quantitative assessments of estimated exposures or risks allowed by the existing standard or potential alternative standards. Further, the available evidence in the 2019 ISA did not provide support for evaluating air quality distributions in locations of individual epidemiologic studies as was done in the 2012 review (78 FR 3176, January 15, 2013). The substantial uncertainty in such analyses, if conducted based on the available $PM_{10-2.5}$ health studies, would have been of limited utility for informing conclusions on the primary PM_{10} standard.

In the 2020 decision, for all of the reasons discussed above and recognizing the CASAC conclusion that the evidence provided support for retaining the current standard, the Administrator concluded that it was appropriate to retain the existing primary PM_{10} standard, without revision. His decision was consistent with the CASAC advice related to the primary PM_{10} standard. Specifically, the CASAC agreed with the 2020 PA conclusions that, while these effects are important, the "evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard" and "supports consideration of retaining the current standard in this review" (Cox, 2019a, p. 3 of letter). Thus, the Administrator concluded that the primary PM_{10} standard (in all of its elements) 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.

4.2 GENERAL APPROACH AND KEY ISSUES IN THIS RECONSIDERATION OF THE 2020 FINAL DECISION

As is the case for all such reviews, this reconsideration of the 2020 final decision on the primary PM_{10} standard is most fundamentally based on using the Agency's assessment of the scientific evidence and quantitative information, if available, to inform the Administrator's judgments regarding a primary standard that is requisite to protect public health with an adequate margin of safety. The approach for this reconsideration builds on the substantial assessments and evaluations performed over previous reviews (U.S. EPA, 2011, U.S. EPA, 2020). As noted

above, the draft ISA Supplement does not include an evaluation of studies for $PM_{10-2.5}$ and the 2019 ISA continues to serve as the scientific foundation for this reconsideration. Given that there is no new evidence for $PM_{10-2.5}$-related health effects assessed in the draft ISA Supplement that would inform quantitative assessments or preliminary conclusions on the current primary PM_{10} standard since the completion of the 2020 review, this draft PA draws from the evaluation of the health effects evidence for $PM_{10-2.5}$-related effects in the 2019 ISA and considerations of such effects in the 2020 PA (U.S. EPA, 2020).

The evaluations in this draft PA of the health effects evidence assessed in the 2019 ISA are intended to inform the Administrator's public health policy judgments and conclusions as a part of this reconsideration of the 2020 final decision, including his decision as to whether to retain or revise the primary PM_{10} standard. The draft PA evaluations consider the potential implications of various aspects of the scientific evidence and the associated uncertainties and limitations. In so doing, the approach for this draft PA involves evaluating the available scientific and technical information to address a series of key policy-relevant questions using evidence-based considerations. Consideration of the full set of evidence in this reconsideration will inform the answer to the following initial overarching question:

- **Does the 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 $PM_{10-2.5}$?**

In reflecting on this question, we consider the body of scientific evidence, assessed in the 2019 ISA, including whether it supports or calls into question the scientific conclusions reached in previous reviews regarding health effects related to exposure to $PM_{10-2.5}$ in ambient air. Information available in the 2019 ISA that may be informative to public health judgments regarding significance or adversity of key effects will also be considered. Further, in considering this question with regard to the primary PM_{10} standard, as in all NAAQS reviews, we give particular attention to exposures and health risks to at-risk populations (including at-risk lifestages). Evaluation of the scientific information with regard to this consideration of the current standard will focus on key policy-relevant issues by addressing a series of questions including the extent to which the available scientific evidence supports retaining or altering the conclusions in the prior reviews regarding health effects attributed to $PM_{10-2.5}$ exposures. Furthermore, this draft PA will examine whether the previously identified uncertainties have been reduced and if new uncertainties have been identified.

The general approach to reaching preliminary conclusions on the current primary PM_{10} standard is summarized in Figure 4-1:

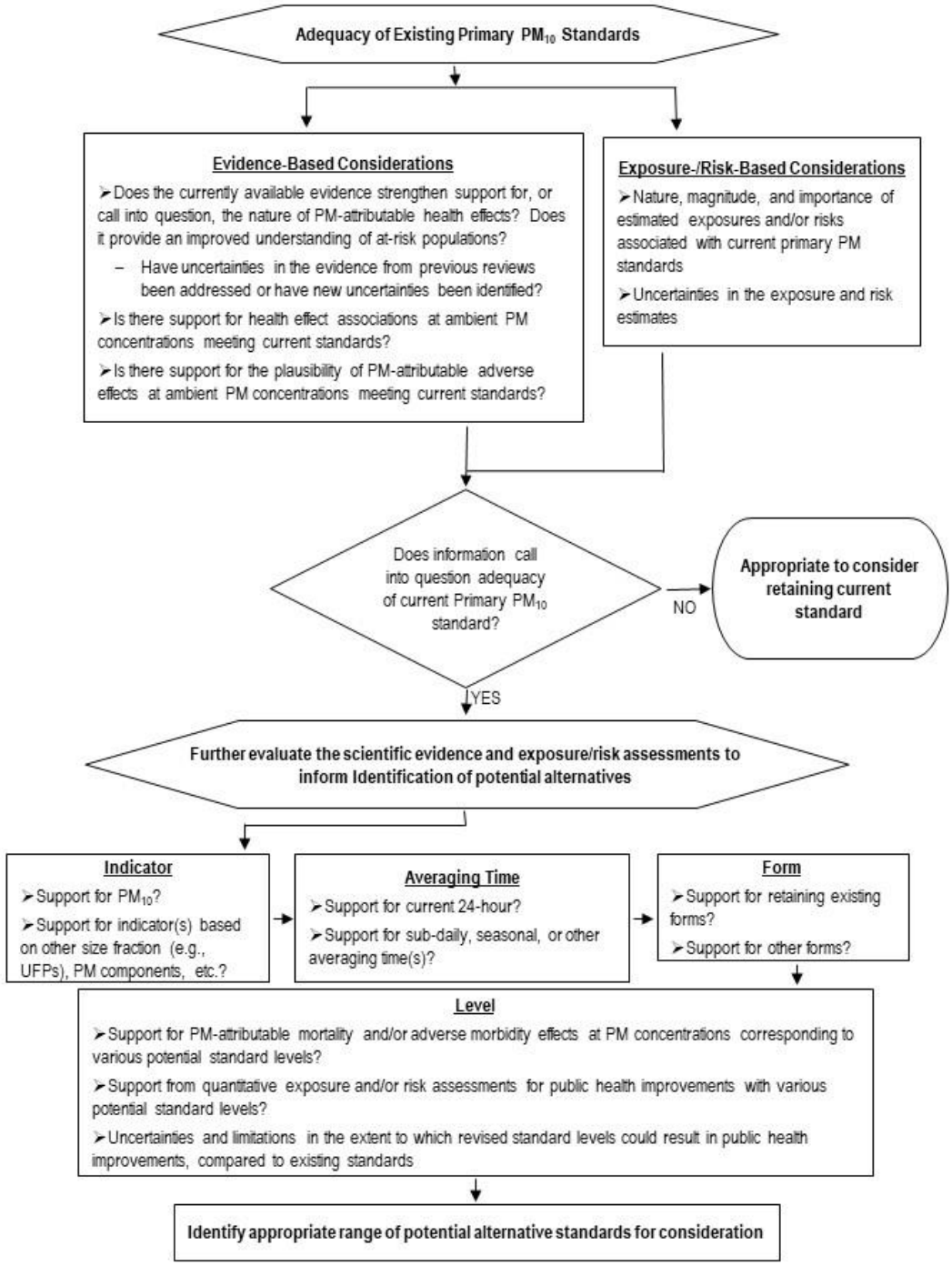

Figure 4-1. Overview of general approach for the reconsideration of the 2020 final decision on the primary PM_{10} standard.

The Agency's approach to reviewing the primary standards is consistent with the requirements of the provisions of the CAA related to the review of the NAAQS and with how the EPA and the courts have historically interpreted the CAA. As discussed in section 1.1 above, these provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite (i.e., neither more nor less stringent than necessary) to protect public health with an adequate margin of safety. Consistent with the Agency's approach across all NAAQS reviews, the approach of this draft PA to informing these judgments is based on a recognition that the available health effects evidence generally reflects continuums that include ambient air exposures for which scientists generally agree health effects are likely to occur through lower levels at which the likelihood and magnitude of response become increasingly uncertain. The CAA does not require the Administrator to establish a primary standard at a zero-risk level or at background concentration levels, but rather at a level that reduces risk sufficiently so as to protect public health, including the health of sensitive groups,[1] with an adequate margin of safety.

The decisions on the adequacy of the current primary PM_{10} standard and on any alternative standards considered in a review are largely public health policy judgments made by the Administrator. The four basic elements of the NAAQS (i.e., indicator, averaging time, form, and level) are generally considered collectively in evaluating the health protection afforded by the current standard, and by any alternatives considered. The Administrator's final decisions in a review draw upon the scientific evidence for health effects, quantitative analyses of population exposures and/or health risks, as available, and judgments about how to consider the uncertainties and limitations that are inherent in the scientific evidence and quantitative analyses.

4.3 HEALTH EFFECTS EVIDENCE

This section draws from the EPA's synthesis and assessment of the scientific evidence presented in the 2019 ISA (U.S. EPA, 2019) to consider the following policy-relevant questions:

- **To what extent does the available scientific evidence strengthen, or otherwise alter, our conclusions from previous reviews 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 response to the overarching question on the adequacy of the current primary PM_{10} standard, posed at the beginning of this chapter. In section 4.3.1

[1] More than one population group may be identified as sensitive or at-risk in a NAAQS review. Decisions on NAAQS reflect consideration of the degree to which protection is provided for these sensitive population groups. To the extent that any particular population group is not among the identified sensitive groups, a decision that provides protection for the sensitive groups would be expected to also provide protection for other population groups.

below, we consider the nature of the effects attributable to long-term and short-term $PM_{10-2.5}$ exposures.

4.3.1 Nature of Effects

As noted above, for the health effect categories and exposure duration combinations evaluated, the 2019 ISA concludes that the evidence supports causality determinations for $PM_{10-2.5}$ that are "suggestive of, but not sufficient to infer, a causal relationship." These health effect categories, along with their corresponding causality determinations from the 2009 ISA, are highlighted below in Table 4-1 (adapted from U.S. EPA, 2019, Table 1-4).

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

Health Outcome	Exposure Duration	2009 PM ISA	2019 PM ISA
Mortality	Long-term	Inadequate	Suggestive of, but not sufficient to infer
	Short-term	Suggestive of, but not sufficient to infer	
Cardiovascular effects	Long-term	Inadequate	
	Short-term	Suggestive of, but not sufficient to infer	
Respiratory effects	Short-term	Suggestive of, but not sufficient to infer	
Cancer	Long-term	Inadequate	
Nervous System effects	Long-term	---	
Metabolic effects	Long-term	---	

While the evidence supporting the causal nature of relationships between exposure to $PM_{10-2.5}$ has been strengthened for some of the health effect categories listed in Table 4-1 since the 2009 ISA, the 2019 ISA concludes that overall "the uncertainties in the evidence identified in the 2009 PM ISA have, to date, still not been addressed" (U.S. EPA, 2019, section 1.4.2, p. 1-41). Specifically, epidemiologic studies available in the 2012 review relied on various methods to estimate $PM_{10-2.5}$ concentrations, and these methods had not been systematically compared to evaluate spatial and temporal correlations in $PM_{10-2.5}$ concentrations. 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, 2019, section 1.4.2). As described in the 2019 ISA, there continues to be variability across epidemiologic studies in the approaches used to estimate PM_{10-}

$_{2.5}$ concentrations. Additionally, some 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. The various methods used to estimate $PM_{10-2.5}$ concentrations have not been systematically evaluated (U.S. EPA, 2019, section 3.3.1.1), contributing to uncertainty regarding the spatial and temporal correlations in $PM_{10-2.5}$ concentrations across methods and in the $PM_{10-2.5}$ exposure estimates used in epidemiologic studies (U.S. EPA, 2019, section 2.5.1.2.3). Given the greater spatial and temporal variability of $PM_{10-2.5}$ and the lower number of $PM_{10-2.5}$ monitoring sites, compared to $PM_{2.5}$, this uncertainty is particularly important for the coarse size fraction.

Beyond the 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 health effects following $PM_{10-2.5}$ exposures also continue to contribute 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. On the other hand, uncertainty related to the biological plausibility of effects attributed to $PM_{10-2.5}$ exposures results from the small number of controlled human exposure and animal toxicology[2] studies that have evaluated the health effects of experimental $PM_{10-2.5}$ inhalation exposures. The evidence supporting the 2019 ISA's "suggestive of, but not sufficient to infer, a causal relationship" causality determinations for $PM_{10-2.5}$, including uncertainties in this evidence, is summarized in sections 4.3.1.1 to 4.3.1.6 below.

4.3.1.1 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 ISA concluded that the evidence was "inadequate to determine if a causal relationship exists" (U.S. EPA, 2009, U.S. EPA, 2019). As reported in the 2019 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, 2019, Table 11-11). The examination of copollutant models in these studies remains limited and, when included, $PM_{10-2.5}$ effect estimates were often attenuated after adjusting for $PM_{2.5}$ (U.S. EPA, 2019, Table 11-11). Across studies, $PM_{10-2.5}$ exposure concentrations were estimated using a variety of approaches, including direct

[2] Compared to humans, rats and mice have small nasal passages, allowing smaller fractions of inhaled $PM_{10-2.5}$ to penetrate into the thoracic regions of the lungs of rats and mice (U.S. EPA, 2019, section 4.1.6), contributing to the relatively limited evaluation of $PM_{10-2.5}$ exposures in animal studies.

measurements from dichotomous samplers, calculating the difference between PM_{10} and $PM_{2.5}$ concentrations measured at collocated monitors, and calculating the difference between 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, 2019, section 3.3.1.1 and Table 11-11). The 2019 ISA concludes that this uncertainty "reduces the confidence in the associations observed across studies" (U.S. EPA, 2019, p. 11-125). The 2019 ISA additionally concludes that the evidence for long-term $PM_{10-2.5}$ exposures and cardiovascular effects, respiratory morbidity, and metabolic disease evidence provides limited biological plausibility for $PM_{10-2.5}$-related mortality (U.S. EPA, 2019, sections 11.4.1 and 11.4). Taken together, the 2019 ISA concludes that, "this body of evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{10-2.5}$ exposure and total mortality" (U.S. EPA, 2019, p. 11-125).

Short-term exposures

The 2009 ISA concluded that the evidence is "suggestive of a causal relationship between short-term exposure to $PM_{10-2.5}$ and mortality" (U.S. EPA, 2009). The 2019 ISA included multicity epidemiologic studies conducted primarily in Europe and Asia which continue to provide consistent evidence of positive associations between short-term $PM_{10-2.5}$ exposure and total (nonaccidental) mortality (U.S. EPA, 2019, 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. Studies considered in the 2019 ISA continue to 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, 2019, section 11.3.4.1, Figure 11-28, Table 11-10). The 2019 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, 2019, section 11.3.7). The 2019 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, 2019, section 11.3.7). Based on the overall evidence, the 2019 ISA concludes that, "this body of evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term $PM_{10-2.5}$ exposure and total mortality" (U.S. EPA, 2019, p. 11-120).

4.3.1.2 Cardiovascular Effects

Long-term exposures

In the 2009 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, 2019, section 6.4).

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, 2019, Table 6-70). The evidence for associations with cardiovascular morbidity has grown since the 2009 ISA and, while results across studies are not entirely consistent, some epidemiologic studies report positive associations with ischemic heart disease (IHD) and myocardial infarction (MI) (U.S. EPA, 2019, Figure 6-34); stroke (U.S. EPA, 2019, Figure 6-35); atherosclerosis; venous thromboembolism (VTE); and blood pressure and hypertension (U.S. EPA, 2019, Section 6.4.6). $PM_{10-2.5}$ cardiovascular mortality effect estimates are often attenuated, but remain positive, in models that adjust for $PM_{2.5}$. For morbidity outcomes, associations are inconsistent in models that adjust for $PM_{2.5}$, NO_2, and chronic noise pollution (U.S. EPA, 2019, p. 6-276). The lack of toxicological evidence for long-term $PM_{10-2.5}$ exposures represents a substantial data gap (U.S. EPA, 2019, section 6.4.10), resulting in the 2019 ISA conclusion that "evidence from experimental animal studies is of insufficient quantity to establish biological plausibility" (U.S. EPA, 2019, p. 6-277). Based largely on the observation of positive associations in some epidemiologic studies, the 2019 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, 2019, p. 6-277).

Short-term exposures

The 2009 ISA concluded 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 prior reviews 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, U.S. EPA, 2019). In the 2019 ISA, key uncertainties include the potential for exposure measurement error, copollutant confounding, and limited evidence of biological plausibility for cardiovascular effects following inhalation exposure (U.S. EPA, 2019, section 6.3.13).

The evidence for short-term $PM_{10-2.5}$ exposure and cardiovascular outcomes has expanded since the 2009 ISA, though important uncertainties remain. The 2019 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, the evidence is limited to suggest that these associations were biologically plausible, or independent of copollutant confounding. The 2019 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 2019 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, 2019, p.6-254).

4.3.1.3 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 concluded that the relationship between short-term exposure to $PM_{10-2.5}$ and respiratory effects is "suggestive of a causal relationship" (U.S. EPA, 2009). 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, 2019, Table 5-36). As noted above for other endpoints, one source of uncertainty in these epidemiologic studies is the lack of a systematic evaluation of the various methods used to estimate $PM_{10-2.5}$ concentrations as well as the resulting uncertainty in the spatial and temporal variability in $PM_{10-2.5}$ concentrations compared to $PM_{2.5}$ (U.S. EPA, 2019,

sections 2.5.1.2.3 and 3.3.1.1). Taken together, the 2019 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, 2019, p. 5-270).

4.3.1.4 Cancer

Long-term exposures

In the 2012 review, few studies examined 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). The scientific information assessed in the 2019 ISA 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 (U.S. EPA, 2019). Additionally, 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, 2019, 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, 2019, section 10.3.4). Taken together, the small number of epidemiologic and experimental studies, along with uncertainty with respect to exposure measurement error, contribute to the determination in the 2019 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, 2019, p. 10-87).

4.3.1.5 Metabolic Effects

Long-term exposures

The 2009 ISA did not make a causality determination for $PM_{10-2.5}$-related metabolic effects. One epidemiologic study is assessed in the 2019 ISA that reports an association between long-term $PM_{10-2.5}$ exposure and diabetes incidence, while additional cross-sectional studies report associations with effects on glucose or insulin homeostasis (U.S. EPA, 2019, section 7.4). As discussed above for other outcomes, uncertainties with the epidemiologic evidence include the potential for copollutant confounding and exposure measurement error (U.S. EPA, 2019, Tables 7-14 and 7-15). The evidence base to support the biological plausibility of metabolic effects following $PM_{10-2.5}$ 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, 2019, sections 7.4.1 and 7.4.3). Based on the expanded, though still limited evidence base, the 2019 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, 2019, p. 7-56).

4.3.1.6 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 2019 ISA, available epidemiologic studies report associations between $PM_{10-2.5}$ and impaired cognition and anxiety in adults in longitudinal analyses (U.S. EPA, 2019, Table 8-25, section 8.4.5). Associations of long-term exposure with neurodevelopmental effects are not consistently reported in children (U.S. EPA, 2019, sections 8.4.4 and 8.4.5). Uncertainties in these studies include the potential for copollutant confounding, as no studies examined copollutants models (U.S. EPA, 2019, section 8.4.5), and for exposure measurement error, given the use of various model-based subtraction methods to estimate $PM_{10-2.5}$ concentrations (U.S. EPA, 2019, Table 8-25). In addition, there is only limited animal toxicological evidence supporting the biological plausibility of nervous system effects (U.S. EPA, 2019, sections 8.4.1 and 8.4.5). Overall, the 2019 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, 2019, p. 8-75).

4.3.1.7 Preliminary Conclusions Drawn from the Evidence

With the evidence available in this reconsideration, as assessed in the 2019 ISA (U.S. EPA, 2019) and summarized in subsections 4.3.1.1 to 4.3.1.6 above, we revisit the policy-relevant questions posed at the beginning of this section:

- **To what extent does the available scientific evidence strengthen, or otherwise alter, our conclusions from previous reviews 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?**

For each of these categories of effects listed above, the 2019 ISA concludes that the evidence is "suggestive of, but not sufficient to infer, a causal relationship" (U.S. EPA, 2019). As summarized in the sections above, key uncertainties in the evidence result 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. The evidence base for several $PM_{10-2.5}$-related health effects has expanded over time, broadening our understanding of the range of health effects linked to $PM_{10-2.5}$ exposures. This includes additional evidence for the relationships between long-term exposures and cardiovascular effects, metabolic

effects, nervous system effects, cancer, and mortality. However, the 2019 ISA identifies a number of key limitations in the evidence, including 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 contribute to uncertainty in the biological plausibility of the $PM_{10-2.5}$-related effects reported in epidemiologic studies.

These uncertainties contribute to the conclusions in the 2019 ISA that the evidence for the $PM_{10-2.5}$-related health effects discussed in this section for both short- and long-term exposures is "suggestive of, but not sufficient to infer, a causal relationship."

4.4 PRELIMINARY CONCLUSIONS ON THE ADEQUACY OF THE CURRENT PRIMARY PM_{10} STANDARD

This section describes our preliminary conclusions regarding the adequacy of the current primary PM_{10} standard. Our approach to reaching preliminary conclusions considers the EPA's assessment of the scientific evidence for $PM_{10-2.5}$-related health effects in the 2019 ISA. We revisit the overarching question for this chapter:

- **Does the 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 $PM_{10-2.5}$?**

As an initial matter, we note that the scope of the updated scientific evaluation of the health effects evidence for PM_{10} is based on those health effects categories where the 2019 ISA concludes a causal relationship exists. Therefore, the draft ISA Supplement does not include an evaluation of additional studies for $PM_{10-2.5}$ and the 2019 ISA continues to serve as the scientific foundation for assessing the adequacy of the primary PM_{10} standard in this reconsideration of the 2020 final decision (U.S. EPA, 2019, section 1.7; U.S. EPA, 2021). As such, this section describing our preliminary conclusions regarding the adequacy of the current primary PM_{10} standard draws heavily from the conclusions in the 2020 PA related to the primary PM_{10} standard (U.S. EPA, 2020, section 4.4). Lastly, we recognize that a final decision on the primary PM_{10} standard in this reconsideration will be largely a public health policy judgement in which the Administrator weighs the evidence, including its associated uncertainties.

With respect to the indicator, we note that the evidence continues to support retaining 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 reflect differences in the strength of $PM_{10-2.5}$ health effects evidence.

Regarding evidence for $PM_{10-2.5}$-related health effects, we note that 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. The epidemiologic studies considered in the 2019 ISA continue to report positive associations with mortality or morbidity in cities across North America, Europe, and Asia, where $PM_{10-2.5}$ sources and composition are expected to vary widely. Such studies provide an important part of the body of evidence supporting the strengthened causality determinations (and new determinations) for long-term $PM_{10-2.5}$ exposures and mortality, cardiovascular effects, metabolic effects, nervous system effects and cancer (U.S. EPA, 2019). Although most of these studies examined $PM_{10-2.5}$ health effect associations in urban areas, some studies have also linked mortality and morbidity with relatively high ambient concentrations of particles of non-urban crustal origin from dust storm events (U.S. EPA, 2019). Drawing from this evidence, we note continued support for maintaining 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). Thus, the scientific evidence evaluated for this reconsideration does not call into question the decision in the 2020 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.

With regard to uncertainties, the 2019 ISA notes that important uncertainties remain in the evidence base for $PM_{10-2.5}$-related health effects. As summarized in section 4.3.1 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 2019 ISA for consideration in reaching preliminary conclusions in this reconsideration is subject to the same broad uncertainties present in the 2012 review (U.S. EPA, 2019). Consistent with the assessment of the evidence in the 2009 ISA, these uncertainties contribute to the determinations in the 2019 ISA that the evidence for key $PM_{10-2.5}$-related health effects is "suggestive of, but not sufficient to infer" causal relationships (U.S. EPA, 2019). Drawing from this information, we reach the preliminary conclusion that, as in previous reviews, 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 2020 review to retain the current primary PM_{10} standard in order to protect against $PM_{10-2.5}$ exposures. Specifically, while the 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 previous reviews, we reach the preliminary conclusion that the evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard. Furthermore, the available evidence in this reconsideration of the 2020 final decision supports retaining the current standard. As such, we have not evaluated alternative standards in this updated PA.

4.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

As discussed above, a number of key uncertainties and limitations in the health evidence have been considered, consistent with those identified in the 2009 ISA and 2019 ISA. In this section, we highlight areas for future health-related research and data collection activities from the 2020 PA to address these uncertainties and limitations in the evidence (U.S. EPA, 2020, section 4.5). 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 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, 2019, U.S. EPA, 2009 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 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, 2019, U.S. EPA, 2009, section 2.5.1.1.3). Starting in 2011, $PM_{10-2.5}$ has been monitored at NCore stations, IMPROVE stations, and several sites run by State and local agencies. Furthermore, there has been an increase in the deployment of $PM_{2.5}$ FEM monitors that also measure $PM_{10-2.5}$. 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.

- Evaluate and expand the $PM_{10-2.5}$ network, along with speciation of $PM_{10-2.5}$ including multi-elements, major ions, carbon (including carbonate carbon), and bioaerosols.

- Characterize $PM_{10-2.5}$ in different health-relevant exposure environments (e.g., city center, suburban, roadside, agricultural, and rural areas) for mass, elements (including potential toxic species), carbonaceous materials (including selected organic compounds and carbonate), water-soluble ions, and bioaerosols (including endotoxins, 1,3 beta glucans, and total protein).

- 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 RECONSIDERATION OF THE SECONDARY STANDARDS FOR PM

This chapter presents and evaluates the policy implications of the scientific and technical information pertaining to this reconsideration of the 2020 final decision on the secondary PM standards. In so doing, the chapter presents key aspects of the evidence for the welfare effects of PM documented in the 2019 ISA and draft ISA Supplement, with support from the prior ISA and AQCDs, and associated public welfare implications, as well as key aspects of quantitative analyses of recent air quality that is presented in the appendix associated with this chapter. As described in detail in section 1.4.2, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. The selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for welfare effects, the focus within the draft ISA Supplement is on visibility effects. The draft ISA Supplement does not include an evaluation of studies on climate or materials effects. Together, the scientific evidence and quantitative information provides the foundation for our evaluation of the scientific information regarding welfare effects of PM in ambient air and the potential for welfare effects to occur under air quality conditions associated with the current standards, as well as the associated public welfare implications. Our evaluation is framed around key policy-relevant questions derived from the questions included in the IRP (U.S. EPA, 2016) for the review completed in 2020 and also takes into account the conclusions reached in the review. In this way we identify key policy-relevant considerations and summary conclusions regarding the public welfare protection provided by the currents standards for the Administrator's consideration in this reconsideration of the 2020 final decision on the secondary PM standards.

Within this chapter, background information on the current standards, including key considerations in reaching the final decision in the 2020 review, is summarized in section 5.1. The general approach for considering the information in this reconsideration of the 2020 final decision, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 5.2. Key aspects of the welfare effects evidence, quantitative information, and associated public welfare implications and uncertainties are addressed in section 5.3. Section 5.3.1 presents our consideration of the available scientific evidence and quantitative information for visibility effects, while section 5.3.2 considers the scientific evidence for each of the non-

visibility welfare effects (climate effects and materials effects) separately.[1] Section 5.4 summarizes the key evidence- and quantitative-based considerations identified in our evaluation and presents associated summary conclusions of this analysis. Key remaining uncertainties and areas for future research are identified in section 5.5.

5.1 BACKGROUND ON THE CURRENT STANDARDS

The current secondary PM standards were affirmed in 2020 based on the scientific and technical information available at that time, as well as the Administrator's judgments regarding the available welfare effects evidence, the appropriate degree of public welfare protection for the existing standards, and available air quality information on visibility impairment that may be allowed by such a standard (85 FR 82684, December 18, 2020). The welfare effects evidence base available in the 2020 review included several decades of extensive research on the visibility and non-visibility effects (climate effects, materials effects, and ecological effects) of PM, conducted both in and outside of the U.S., that documents the impacts of PM (U.S. EPA, 2019; U.S. EPA, 2009; U.S. EPA, 2004b; U.S. EPA, 2004a). With the 2020 decision, the EPA retained the secondary 24-hour $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$, the annual $PM_{2.5}$ standard, with its level of 15.0 $\mu g/m^3$, and the 24-hour PM_{10} standard, with its level of 150 $\mu g/m^3$. The sections below focus on the key considerations, and the Administrator's conclusions, for climate and materials effects (section 5.1.1) and visibility effects (section 5.1.2) in the 2020 review.

5.1.1 Non-Visibility Effects

In light of the robust evidence base, the 2019 ISA concluded there to be causal relationships between PM and climate effects and material effects (U.S. EPA, 2019, sections 13.3.9 and 13.4.2). For climate effects, the 2019 ISA concluded that aerosols[2] alter climate processes directly through radiative forcing and by indirect effects on cloud brightness, changes

[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, oxides of sulfur and PM. 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 reconsideration of the 2020 final decision 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.

[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 reconsideration of the 2020 final decision on the secondary PM NAAQS the discussion on climate effects of PM uses the term PM throughout for consistency with the 2019 ISA (U.S. EPA, 2019) as well as to emphasize that the climate processes altered by aerosols are generally altered by the PM portion of the aerosol. Exceptions to this practice include the discussion of climate effects in the 2012 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).

in precipitation, and possible changes in cloud lifetimes (U.S. EPA, 2019, section 13.3.9). 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, 2019, section 13.3.5). For materials effects, the 2019 ISA 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, 2019, section 13.4.2). Additionally, the deposition of PM from ambient air can reduce the aesthetic appeal of buildings and objects through soiling.

The 2020 decision on the adequacy of the secondary standards for climate and materials effects was a public welfare policy judgment made by the Administrator, which drew upon the available scientific evidence for PM-attributable climate and materials effects and recognized that the evidence did not support a quantitative assessment of exposures and public welfare risks based on impacts to climate and materials. 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. He noted that a number of uncertainties in the scientific information affected our ability to quantitatively evaluate the standards in this regard. For example, the 2019 ISA and 2020 PA noted the spatial and temporal heterogeneity of PM components that contribute to climate forcing, uncertainties in the measurement of aerosol components, inadequate consideration of aerosol impacts in climate modeling, insufficient data on local and regional microclimate variations and heterogeneity of cloud formations (U.S. EPA, 2019, section 13.3.9). In light of these uncertainties and the lack of sufficient data, the 2020 PA concluded that "the data remain insufficient to conduct quantitative analyses for PM effects on climate in the current review" (U.S. EPA, 2020, pp. 5-34 to 5-35) 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, 2020, section 5.4).

With regard to materials effects, the Administrator noted that the 2020 PA noted that quantitative relationships were lacking between characteristics of PM 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, 2020, p. 5-35). The 2020 PA concluded that none of the evidence available called into question the adequacy of the existing secondary PM standards to protect against material effects (U.S. EPA, 2020, section 5.4).

The 2020 final decision was based on a thorough review in the 2019 ISA of the scientific information on PM-induced climate and materials effects. The decision also took into account: (1) assessments in the 2020 PA of the most policy-relevant information in the 2019 ISA regarding evidence of adverse effects of PM to climate and materials, (2) uncertainties in the available evidence to inform a quantitative assessment of PM-related climate and materials effects, (3) CASAC advice and recommendations, and (4) public comments received during the development of these documents and on the proposal notice.

Consistent with the general approach routinely employed in NAAQS reviews, the initial consideration in the 2020 review of the secondary standards was with regard to the adequacy of protection provided by the then-existing standards. Key aspects of that consideration are summarized in section 5.1.1.1 below.

5.1.1.1 Considerations Regarding Adequacy of the Existing Standards for Non-Visibility Effects in the 2020 Review

In considering non-visibility welfare effects in the 2020 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, "it is generally appropriate to retain the existing standards and that there is insufficient information to establish any distinct secondary PM standards to address climate and materials effects of PM" (85 FR 82744, December 18, 2020).

With regard to climate, the Administrator recognized that there were a number of improvements and refinements to climate models since the 2012 review. However, while the evidence continued to support a causal relationship between PM and climate effects, the Administrator noted that significant limitations continued to exist related to quantifying the contributions of direct and indirect effects of PM and PM components on climate forcing (U.S. EPA, 2020, sections 5.2.2.1.1 and 5.4). He also recognized that the models continued to exhibit considerable variability in estimates of PM-related climate impacts as regional scales (e.g., ~100 km) as compared to simulations at global scales. Therefore, the resulting uncertainty led the Administrator to conclude that the available scientific information in the 2020 review remained insufficient to quantify climate impacts associated with particular concentrations of PM in ambient air (U.S. EPA, 2020, section 5.2.2.2.1) or to evaluate or consider a level of PM air quality in the U.S. to protect against climate effects and that there was insufficient information available to base a national ambient standard on climate impacts (85 FR 82744, December 18, 2020).

With regard to materials effects, the Administrator noted that the evidence available in the 2019 ISA continued to support a causal relationship between materials effects and PM deposition (U.S. EPA, 2019, section 13.4). He recognized that the deposition of fine and coarse

particles to materials can lead to physical damage and/or impaired aesthetic qualities. Particles can contribute to materials damage by adding to the natural weathering processes and by promoting the corrosion of metals, the degradation of building materials, and the weakening of material components. While some new information was available in the 2019 ISA, the information was from studies primarily conducted outside of the U.S. in areas where PM concentrations in ambient air are typically higher than those observed in the U.S. (U.S. EPA, 2020, section 13.4). Additionally, the information assessed in the 2019 ISA did not support quantitative analyses of PM-related materials effects in the 2020 review (U.S. EPA, section 5.2.2.2.2). Given the limited amount of information available and its inherent uncertainties and limitations, the Administrator concluded that he was unable to relate soiling or damage to specific levels of PM in ambient air or to evaluate or consider a level of air quality to protect against such materials effects, and that there was insufficient information available to support a distinct national ambient standard based on materials effects (85 FR 82744, December 18, 2020).

In the 2020 decision, for all of the reasons discussed above and recognizing the CASAC conclusion that the evidence provided support for retaining the current secondary PM standards, the Administrator concluded that it was appropriate to retain the existing secondary PM standards, without revision. His decision was consistent with the CASAC advice related to non-visibility effects. Specifically, the CASAC agreed with the 2020 PA conclusions that, while these effects are important, "the available evidence does not call into question the protection afforded by the current secondary PM standards" and recommended that the secondary standards "should be retained" (Cox, 2019a, p. 3 of letter). For climate and materials effects, this conclusion reflected his judgment that, although it remains important to maintain secondary $PM_{2.5}$ and PM_{10} standards to provide some degree of control over long- and short-term concentrations of both fine and coarse particles, there was insufficient information to establish distinct secondary PM standards to address non-visibility PM-related welfare effects (85 FR 82744, December 18, 2020). Thus, the Administrator concluded that it was appropriate to retain all aspects of the existing 24-hour $PM_{2.5}$, annual $PM_{2.5}$, and 24-hour 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 $\mu g/m^3$ while revising only the form of the standard to remove the option for spatial averaging (85 FR 82744, December 18, 2020).

5.1.2 Visibility Effects

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. Given the strength of the evidence base, the 2019 ISA concluded that, "the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment" (U.S. EPA, 2019, section 13.2.6). 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 2012 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; 2019 ISA, section 13.2.4.1).

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). Consistent with the evidence available in the 2012, the 2019 ISA evaluated available visibility preference studies that were part of the overall body of evidence, and these preference studies were considered in the 2020 PA (U.S. EPA, 2020, pp. 5-15 to 5-17). 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]

The 2020 decision on the adequacy of the secondary standards with regard to visibility effects was a public welfare policy judgment made by the Administrator, which drew upon the available scientific evidence for PM-related visibility effects and on analyses of visibility impairment, as well as judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. Consistent with the approach in the 2012 review, the analyses utilized a $PM_{2.5}$ visibility index based on an algorithm, known as the

[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; 2019 ISA, section 13.2.1). 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.

[4] Preference studies were available in four urban areas. Three western preference studies were available, including one in Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009). More details about these studies are available in Appendix D.

IMPROVE algorithm,[5] that 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. The quantitative analyses focused on $PM_{2.5}$ based on conclusions in the 2019 ISA that 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). The 2019 ISA also concluded that hygroscopic particles like ammonium sulfate, ammonium nitrate, and sea salt increase in size as relative humidity increases, leading to increased light scattering (U.S. EPA, 2019, section 13.2.3). Included in this decision were judgments on the weight to place on the visibility preference studies; on the weight to give associated uncertainties, including those related to variability in visibility preferences across the studies in different areas of the U.S.; variability in in occurrence of visibility impairment in areas of the U.S., especially in urban areas; and on the extent to which such effects in such areas may be considered adverse to public welfare.

The 2020 final decision was based on a thorough review in the 2019 ISA of the scientific information on PM-related visibility effects. The decision also took into account: (1) assessments in the 2020 PA of the most policy-relevant information in the 2019 ISA regarding evidence of adverse effects of PM on visibility; (2) air quality analyses of the $PM_{2.5}$ visibility index and design values based on the form and averaging time of the existing standard; (3) CASAC advice and recommendations; and (4) public comments received during the development of these documents and on the 2020 proposal notice.

Consistent with the general approach routinely employed in NAAQS reviews, the initial consideration in the 2020 review of the secondary PM standards was with regard to the adequacy of the protection provided by the then-existing standards. Key aspects of that consideration are summarized in section 5.1.2.1 below.

5.1.2.1 Consideration Regarding the Adequacy of the Existing Standards for Visibility Effects in the 2020 Review

In considering the visibility effects in the 2020 review, the Administrator noted the long-standing body of evidence for PM-related visibility impairment. This evidence, which is based on the fundamental relationship between light extinction and PM mass, demonstrated that ambient PM can impair visibility in both urban and remote areas, and had changed very little since the 2012 review (U.S. EPA, 2019, section 13.1; U.S. EPA, 2009a, section 9.2.5). The evidence related to public perception of visibility impairment was from studies from four areas in North America. These studies provided information to inform our understanding of levels of

[5] 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).

visibility impairment that the public judged to be "acceptable" (U.S. EPA, 2010b; 85 FR 24131, April 30, 2020). In considering these public preference studies, the Administrator noted that, as described in the 2019 ISA, no new visibility studies had been conducted in the U.S. and there was little newly available information with regard to acceptable levels of visibility impairment in the U.S. The Administrator recognized that visibility impairment can have implications for people's enjoyment of daily activities and their overall well-being, and therefore, considered the degree to which the current secondary standards protect against PM-related visibility impairment.

Consistent with the 2012 review, in the 2020 review, the Administrator first concluded that a target level of protection for a secondary PM standard is most appropriately defined in terms of a visibility index 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. In defining a target level of protection, the Administrator considered the specific aspects of such an index, including the appropriate indicator, averaging time, form and level (78 FR 82742-82744, December 18, 2020).

First, with regard to indicator, the Administrator noted that in the 2012 review, the EPA used an index based on estimates of light extinction by $PM_{2.5}$ components calculated using an adjusted version of the IMPROVE algorithm, which allows the estimation of light extinction using routinely monitored components of $PM_{2.5}$ and $PM_{10-2.5}$, along with estimates of relative humidity. The Administrator recognized that, while there have been some revisions to the IMPROVE algorithm since the time of the 2012 review, our fundamental understanding of the relationship between PM in ambient air and light extinction had changed little and the various IMPROVE algorithms appropriately reflected this relationship across the U.S. In the absence of a monitoring network for direct measurement of light extinction, he concluded that calculated light extinction indicator that utilizes the IMPROVE algorithms continued to provide a reasonable basis for defining a target level of protection against PM-related visibility impairment (78 FR 82742-82744, December 18, 2020).

In further defining the characteristics of a visibility index, the Administrator next considered the appropriate averaging time, form, and level of the index. Given the available scientific information in the review, and in considering the CASAC's advice and public comments, the Administrator concluded that, consistent with the decision in the 2012 review, a visibility index with a 24-hour averaging time and a form based on the 3-year average of annual 90^{th} percentile values remained reasonable. With regard to the averaging time and form of such an index, the Administrator noted analyses conducted in the last review that demonstrated relatively strong correlations between 24-hour and subdaily (i.e., 4-hour average) $PM_{2.5}$ light extinction (78 FR 3226, January 15, 2013), indicating that a 24-hour averaging time is an

appropriate surrogate for the sub-daily time periods of the perception of PM-related visibility impairment and the relevant exposure periods for segments of the viewing public. This decision in the 2020 review also recognized that a 24-hour averaging time may be less influenced by atypical conditions and/or atypical instrument performance (78 FR 3226, January 15, 2013). The Administrator recognized that there was no new information to support updated analyses of this nature, and therefore, he believed these analyses continued to provide support for consideration of a 24-hour averaging time for a visibility index in this review. With regard to the statistical form of the index, the Administrator noted that, consistent with the 2012 review: (1) A multi-year percentile form offers greater stability from the occasional effect of interannual meteorological variability (78 FR 3198, January 15, 2013; U.S. EPA, 2011, p. 4–58); (2) a 90th percentile represents the median of the distribution of the 20 percent worst visibility days, which are targeted in Federal Class I areas by the Regional Haze Program; and (3) public preference studies did not provide information to identify a different target than that identified for Federal Class I areas (U.S. EPA, 2011, p. 4–59). Therefore, the Administrator judged that a visibility index based on estimates of light extinction, with a 24-hour averaging time and a 90th percentile form, averaged over three years, remained appropriate (78 FR 82742-82744, December 18, 2020).

With regard to the level of a visibility index, consistent with the 2012 review, the Administrator judged that it was appropriate to establish a target level of protection of 30 deciviews (dv),[6] [7] reflecting the upper end of the range of visibility impairment judged to be acceptable by at least 50% of study participants in the available public preference studies (78 FR 3226, January 15, 2013). As described above, the 2011 PA identified a range of levels from 20 to 30 dv based on the responses in the public preference studies available at that time. At the time of the 2012 review, the Administrator noted a number of uncertainties and limitations in public preference studies, including the small number of stated preference studies available, the relatively small number of study participants, 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. In considering the available preference studies, with their inherent uncertainties and limitations, the prior Administrator concluded that the substantial degree of variability and uncertainty in the public preference studies should be reflected in a target level of protection based on the upper end of the range of candidate protection levels (CPLs).

[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 megameters (Mm^{-1}), respectively.

Given that there were no new preference studies available in 2020 review, the Administrator's judgments were based on the same studies, with the same range of levels, available in the 2012 review. As identified in the 2020 PA (U.S. EPA, 2020, section 5.5), there were a number of limitations and uncertainties associated with these studies, including the following:

- Available studies may not represent the full range of preferences for visibility in the U.S. population, particularly given the potential variability in preferences based on the conditions commonly encountered and the scenes being viewed.
- Available preference studies were conducted 15 to 30 years ago and may not accurately represent the current day preferences of people in the U.S.
- The variety of methods used in the preference studies may potentially influence the responses as to what level of impairment is deemed acceptable.
- Factors that are not captured in the methods of the preference studies, such as the time of day when light extinction is the greatest or the frequency of impairment episodes, may influence people's judgment on acceptable visibility (U.S. EPA, 2020, section 5.2.1.1).

Therefore, in considering the scientific information, with its uncertainties and limitations, as well as public comments on the level of the target level of protection against visibility impairment, the Administrator concluded that it is appropriate to again use a level of 30 dv for the visibility index (78 FR 82742-82744, December 18, 2020).

Having concluded that the protection provided by a standard defined in terms of a $PM_{2.5}$ visibility index, with a 24-hour averaging time, and a 90^{th} percentile form, averaged over 3 years, set at a level of 30 dv, was requisite to protect public welfare with regard to visual air quality, the Administrator next considered the degree of protection from visibility impairment afforded by the existing suite of secondary PM standards.

In this context, the Administrator considered the updated analyses of visibility impairment presented in the 2020 PA (U.S. EPA, 2020, section 5.2.1.2), which reflected a number of improvements since the 2012 review. Specifically, the updated analyses examined multiple versions of the IMPROVE equation, including the version incorporating revisions since the time of the 2012 review. These updated analyses provided a further understanding of how variation in the inputs to the algorithms affect the estimates of light extinction (U.S. EPA, 2020, Appendix D). Additionally, for a subset of monitoring sites with available $PM_{10-2.5}$ data, the updated analyses better characterized the influence of coarse PM on light extinction than in the 2012 review (U.S. EPA, 2020, section 5.2.1.2).

The results of the updated analyses in the 2020 PA were consistent with those from the 2012 review. Regardless of which version of the IMPROVE equation was used, the analyses demonstrated that, based on 2015–2017 data, the 3-year visibility metric was at or below about 30 dv in all areas meeting the current 24-hour $PM_{2.5}$ standard, and below 25 dv in most of those

areas. In locations with available $PM_{10-2.5}$ monitoring, which met both the current 24-hour secondary $PM_{2.5}$ and PM_{10} standards, 3-year visibility index metrics were at or below 30 dv regardless of whether the coarse fraction was included as an input to the algorithm for estimating light extinction (U.S. EPA, 2020, section 5.2.1.2). While the inclusion of the coarse fraction had a relatively modest impact on the estimates of light extinction, the Administrator recognized the continued importance of the PM_{10} standard given the potential for larger impacts on light extinction in areas with higher coarse particle concentrations, which were not included in the analyses in the 2020 PA due to a lack of available data (U.S. EPA, 2019, section 13.2.4.1; U.S. EPA, 2020, section 5.2.1.2). He 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. 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 provided 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 he judged appropriate (78 FR 82742-82744, December 18, 2020).

5.2 GENERAL APPROACH AND KEY ISSUES IN THIS RECONSIDERATION OF THE 2020 FINAL DECISION

This reconsideration of the 2020 final decision on the secondary PM standards is most fundamentally based on using the Agency's assessment of the scientific evidence and associated quantitative analyses to inform the Administrator's judgments regarding secondary standards that are requisite to protect public welfare from known or anticipated adverse effects. This draft PA is intended to help bridge the gap between the scientific evidence and information assessed in the 2019 ISA and draft ISA Supplement and the judgments required of the Administrator in determining whether it is appropriate to retain or revised the secondary PM NAAQS. The approach planned for this reconsideration of the 2020 final decision on the secondary PM standards will build on previous reviews, including the substantial assessments and evaluations performed in those reviews, and taking into account scientific information and air quality data to inform our understanding of the key policy-relevant issues in this reconsideration.

The evaluations in this draft PA, of the scientific assessments in the 2019 ISA and draft ISA Supplement[8] augmented by quantitative air quality analyses, are intended to inform the Administrator's public welfare policy judgments and conclusions, including his decisions as to whether to retain or revise these standards. The draft PA considers the potential implications of various aspects of the scientific evidence, the air quality information, and the associated uncertainties and limitations. In so doing, the approach for this draft PA involves evaluating the scientific and technical information to address a series of key policy-relevant questions using both evidence- and quantitative-based considerations. Together, consideration of the full set of evidence and information in this reconsideration will inform the answer to the following initial overarching question for the reconsideration:

- **Do the scientific evidence and quantitative information support or call into question the adequacy of the protection afforded by the current secondary PM standards?**

In reflecting on this question in the remaining sections of this chapter, we consider the body of scientific evidence assessed in the 2019 ISA and draft ISA Supplement and considered as basis for developing or interpreting air quality analyses, including whether it supports or calls into question the scientific conclusions reached in the 2020 review regarding welfare effects related to exposure to PM in ambient air. Information in this reconsideration of the 2020 final decision that may be informative to public policy judgments on the significance or adversity of key effects on the public welfare is also considered. Additionally, the quantitative information, whether newly developed in this reconsideration or predominantly developed in the past and interpreted in light of current information, is considered, including with regard to the extent to which it may continue to support judgments made in previous reviews.

The approach to reaching conclusions on the current secondary PM standards and, as appropriate, on potential alternative standards, including consideration of policy-relevant questions that frame the current reconsideration, is illustrated in Figure 5-1.

[8] As noted above and described in detail in section 1.4.2, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. The selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for welfare effects, the focus within the draft ISA Supplement is on visibility effects. The draft ISA Supplement does not include an evaluation of studies on climate or materials effects.

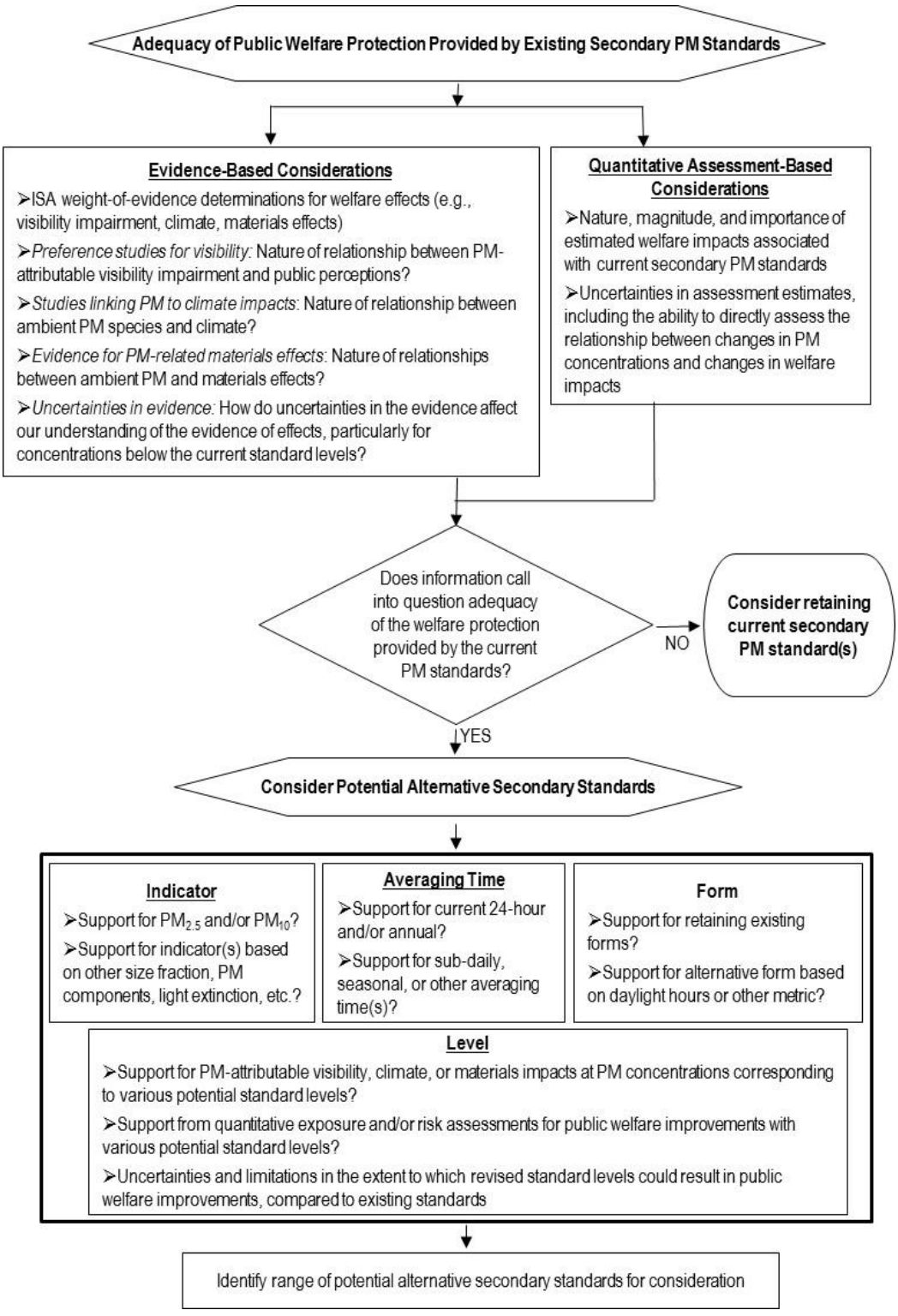

Figure 5-1. Overview of general approach for the reconsideration of the 2020 final decision on the secondary PM standards.

The Agency's approach in its reconsideration of the 2020 final decision on the secondary standards is consistent with the requirements of the provisions of the CAA related to the review of NAAQS and with how the EPA and the courts have historically interpreted the CAA. As discussed in section 2.1 above, these provisions require the Administrator to establish secondary standards that, in the Administrator's judgment, are requisite (i.e., neither more nor less stringent than necessary) to protect the public welfare from known or anticipated adverse effects associated with the presence of the pollutant in ambient air. In so doing, the Administrator considers advice from the CASAC and public comment.

Consistent with the Agency's approach across all NAAQS reviews, the approach of this draft PA to informing the Administrator's judgments in this reconsideration of the 2020 final decision on the secondary PM standards is based on a recognition that the evidence generally reflects continuums that include ambient air exposures for which scientists generally agree that effects are likely to occur through lower levels at which the likelihood and magnitude of response become increasingly uncertain. The CAA does not require that standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect the public welfare from known or anticipated adverse effects. The Agency's decisions on the adequacy of the current secondary standards and, as appropriate, on any potential alternative standards considered in a review, are largely public welfare policy judgments made by the Administrator. The four basic elements of the NAAQS (i.e., indicator, averaging time, form, and level) are considered collectively in evaluating the protection afforded by the current standard, or any alternative standards considered. Thus, the Administrator's final decisions in such reviews draw upon the scientific information and analyses about welfare effects, environmental exposures and risks, and associated welfare significance, as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and analyses.

5.3 WELFARE EFFECTS AND QUANTITATIVE INFORMATION

In considering the evidence for welfare effects attributable to PM presented in the 2019 ISA and the draft ISA Supplement, this section poses the following policy-relevant questions:

- **Does the 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 scientific evidence and quantitative information, as discussed below. In considering the scientific and technical information, we reflect upon both the information in previous reviews and information that is assessed and presented in the 2019 ISA (U.S. EPA, 2019) and in the draft ISA Supplement (U.S. EPA, 2021), 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 2019 ISA for welfare effects. As in previous reviews, the evidence is sufficient to support a causal relationship between PM and visibility effects (section 5.3.1), climate effects (section 5.3.2) and materials effects (section 5.3.2).

While the 2019 ISA provides the broad scientific foundation for this reconsideration, we recognized that additional literature has become available since the cutoff date of the 2019 ISA that expands the body of evidence related to visibility effects that can inform the Administrator's judgments on the adequacy of the current secondary PM standards. As such, the draft ISA Supplement builds on the information in the 2019 ISA with a target identification and evaluation of new scientific information regarding visibility effects (U.S. EPA, 2021, section 1.2). As described in chapter 1, the selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. The draft ISA Supplement focuses on U.S. and Canadian studies that provide new information on public preference for visibility impairment and/or developed new methodologies or conducted quantitative analyses of light extinction (U.S. EPA, 2021, section 1.2). Such studies of visibility effects and quantitative relationships between visibility impairment and PM in ambient air were considered to be of greatest utility in informing the Administrator's conclusions on the adequacy of the current secondary PM standards. The visibility effects evidence presented within the 2019 ISA, along with the targeted identification and evaluation of new scientific information in the draft ISA Supplement, provides the scientific basis for the reconsideration of the 2020 final decision on the primary $PM_{2.5}$ standards. For climate and materials effects, the 2020 PA concluded that there were substantial uncertainties associated with the quantitative relationships with PM concentrations and the concentration patterns that limited the ability quantitatively assess the public welfare protection provided by the standards from these effects. Therefore, for climate and materials effects, we draw heavily from the 2020 PA in our evaluation of the information related to these effects and in reaching preliminary conclusions in this draft PA.

Table 5-1. Key causality determinations for PM-related welfare effects.

Effect	2009 PM ISA	2019 PM ISA
Visibility effects	Causal	Causal
Climate effects	Causal	Causal
Materials effects	Causal	Causal

5.3.1 Visibility Effects

In the sections below, we consider the nature of visibility-related effects attributable to PM (section 5.3.1.1) and the quantitative information (section 5.3.1.2).

5.3.1.1 Nature of Effects

In considering the evidence of visibility welfare effects attributable to PM as presented in the 2019 ISA and the draft ISA Supplement, this section addresses the following policy-relevant question:

- **Does the available scientific evidence alter our conclusions from the 2020 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 2019 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 2012 review (U.S. EPA, 2019, section 13.2.6). These conclusions are based on strong and consistent evidence that ambient PM can impair visibility in both urban and remote areas (U.S. EPA, 2009, section 9.2.5).

These subsequent questions consider the characterization and quantification of light extinction and preferences associated with varying degrees of visibility impairment.

- **To what extent is 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, 2019, section 13.2.1). The impact of PM on light scattering depends on particle size and composition, as well as relative humidity. All particles scatter light, as described by the Mie theory, which relates light scattering to particle size, shape and index of refraction (U.S. EPA, 2019, section 13.2.3; Van de Hulst,

1981; Mie, 1908). Fine particles scatter more light than coarse particles on a per unit mass basis and include sulfates, nitrates, organics, light-absorbing carbon, and soil (Malm et al., 1994). Hygroscopic particles like ammonium sulfate, ammonium nitrate, and sea salt increase in size as relative humidity increases, leading to increased light scattering (U.S. EPA, 2019, section 13.2.3).

Direct measurements of PM light extinction, scattering, and absorption are considered more accurate for quantifying visibility impairment than PM mass-based estimates because they do not depend on assumptions about particle characteristics (e.g., size, shape, density, component mixture, etc.). Measurements of light extinction can be made with high time resolution, allowing for characterization of subdaily temporal patterns of visibility impairment. Measurement methods include transmissometers for measurement of light extinction and the determination of visual range and integrating nephelometers for measurement of light scattering, as well as teleradiometers and telephotometers, and photography and photographic modeling (U.S. EPA, 2009; U.S. EPA, 2004b). While some recent research confirms and adds to the body of knowledge regarding direct measurements as is described in the 2019 ISA and draft ISA Supplement, no major new developments have been made with these measurement methods since prior reviews (U.S. EPA, 2019, section 13.2.2.2; U.S. EPA, 2021, section 4.2).

A theoretical relationship between light extinction and PM characteristics has been derived from Mie theory (U.S. EPA, 2019, Equation 13-5) and can be used to estimate light extinction by combining mass scattering efficiencies of particles with particle concentrations (U.S. EPA, 2019, section 13.2.3; U.S. EPA, 2009, sections 9.2.2.2 and 9.2.3.1). However, routine ambient air monitoring rarely includes measurements of particle size and composition information with sufficient detail for these calculations. Accordingly, a much simpler algorithm has been developed to make estimating light extinction more practical.

The algorithm, known as the IMPROVE algorithm,[9] estimates light extinction (b_{ext}, measured 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

[9] 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).

calculation using particle size distributions associated with each of the major aerosol components (U.S. EPA, 2019, section 13.2.3.1, section 13.2.3.3).

The *original IMPROVE algorithm* (Equation D-1 in Appendix D), so referenced here to distinguish it from subsequent variations developed later, was found to underestimate the highest light scattering values and overestimate the lowest values at IMPROVE monitors throughout the U.S. (Malm and Hand, 2007; Ryan et al., 2005; Lowenthal and Kumar, 2004) and at sites in China (U.S. EPA, 2019, section 13.2.3.3). To resolve these biases, a *revised IMPROVE equation*, shown in Equation D-2 in Appendix D, was developed (Pitchford et al., 2007) that divides PM components into smaller and larger sizes of particles in $PM_{2.5}$, with separate mass scattering efficiencies and hygroscopic growth functions for each size category. The revised IMPROVE equation was described in detail in the 2009 ISA (U.S. EPA, 2009) and at that time, it both reduced bias at the lowest and highest scattering values and improved the accuracy of the calculated light b_{ext}. However, poorer precision was observed with the revised IMPROVE equation compared to the original IMPROVE equation (U.S. EPA, 2009).[10] Recent research suggests that changes in PM composition in ambient air can impact the accuracy of estimating light extinction using the IMPROVE algorithms (U.S. EPA, 2021, section 4.2.2). As an example, a study by Prenni et al. (2019) found that the relationship between directly measured light scattering and estimated light scattering using the revised IMPROVE equation has changed over time in recent years. In particular, Prenni et al. (2019) compared estimated light extinction using the revised IMPROVE equation with measured light extinction using nephelometers from 2001-2016 and found that the revised IMPROVE equation underestimated light extinction at many sites, especially for locations that experienced large decreases in sulfate and organic mass concentrations. They further found that the underestimation results from splitting the components into smaller and larger sizes of particles, with too much of the mass being allocated to the smaller size fraction which has a lower dry mass scattering efficiency (U.S. EPA, 2021, section 4.2.2; Prenni et al., 2019).

Since the 2012 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

[10] 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.

closer to 2.1 than the value of 1.8 used in the revised IMPROVE equation.[11] They also observed that water soluble organic matter absorbs water as a function of relative humidity, which is not accounted for in either the original or revised IMPROVE equations and was therefore underestimated in these equations. They further suggested that light scattering by sulfate was overestimated because the assumption that all sulfate is fully neutralized ammonium sulfate is not always true (U.S. EPA, 2019, section 13.2.3.3). Modifications based on these points are reflected in Equation D-3 in Appendix D.

In summary, rather than altering our understanding from previous reviews, 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 refinements to the inputs of the IMPROVE equation. Accordingly, as in previous reviews, this reconsideration focuses on calculated light extinction when quantifying visibility impairment resulting from recent concentrations of PM in ambient air.

- **What does the 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 2019 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 monitoring sites, which are mostly rural and urban, respectively.[12] The data from these sites for the periods of 2005-2008 and 2011-2014 were used in the 2019 ISA to identify differences in species contributing to light extinction in urban and rural areas by region and season. This is an expansion over the analysis in the 2009 ISA, in that the measurements at that time were primarily based measurements from monitors located in rural areas and at remote sites (U.S. EPA, 2019, section 13.2.4.1, Figures 13-1 through 13-14).

Focusing on the more recent time period of 2011-2014, some major differences in estimated light extinction are apparent among regions of the U.S. Annual average calculated b_{ext} was considerably greater in the East and Midwest than in the Southwest. Based on IMPROVE

[11] 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.

[12] Monitors were grouped into 28 IMPROVE regions and 31 CSN regions based on site location and PM concentrations for major species. For comparison purposes, and where possible, CSN regions were defined similarly to those for the IMPROVE network (Hand et al., 2011; U.S. EPA, 2019, section 13.2.4.1).

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,[13] 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, 2019, section 13.2.4.1, Figure 13-1, Figure 13-3, Figure 13-5).

Consistent with the analysis in the 2019 ISA, a recent study analyzed national and regional trends in light extinction based on reconstructed total light extinction estimated from IMPROVE data using 5-year aggregates of annual mean b_{ext} (Mm^{-1}) for 2000-2004 and 2014-2018 (U.S. EPA, 2021, section 4.2.2). Hand et al. (2020) found that, for 2000-2004, the highest levels of b_{ext} occurred in the Appalachian Mountains and Ohio River valley (~100 Mm^{-1} or greater), with decreasing values in the central U.S (~70 Mm^{-1}). Values of b_{ext} in the East significantly decreased over time, reduced to ~50 Mm^{-1} in the 2014-2018 time period, likely corresponding to decreases in sulfate concentrations over time. However, for 2014-2018, the highest values of b_{ext} were in the central U.S. (50-60 Mm^{-1}), which is an area with high agricultural activity and nitrate and ammonium concentrations. During both time periods, lower b_{ext} occurred in the western U.S. (20-30 Mm^{-1}), with improvements in b_{ext} closer to the West Coast in 2014-2018 compared to 2004-2008.

Moreover, Hand et al. (2020) also explored changes in b_{ext} over time as relative trends (% yr^{-1}) and found spatial variability in long-term and short-term trends. Generally, similar magnitudes and spatial variability were found for both long-term and short-term trends, with the strongest reductions in b_{ext} across the eastern U.S. (-4% yr^{-1} or greater) and along the West Coast, particularly in Southern California. There was less improvement in the Intermountain West[14] (-2% yr^{-1}), although air quality in these areas have been increasingly impacted by wildfire activity and biomass smoke in recent years (Hand et al., 2020). Decreased trends also occurred across the Southwest, but at a lower rate than in the Eastern U.S. Over the entire continental U.S., on average, b_{ext} decreased at a rate of -2.8% yr^{-1} from 2002 to 2018 and -18% yr^{-1} from 1992 to 2018, with much of the improvement occurring in the eastern U.S. (U.S. EPA, 2021, section 4.2.2; Hand et al., 2020).

Components of $PM_{2.5}$ contributing to light extinction vary regionally. For example, in the analysis completed in the 2019 ISA, in the Eastern regions, ammonium sulfate accounted for approximately 35 to 60% of the annual average b_{ext}, with the greatest contributions typically

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

[14] The Intermountain West area includes Idaho, Montana, northern Wyoming, and portions of northern California.

occurring in the summer (U.S. EPA, 2019, section 13.2.4.1). 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 on average than ammonium sulfate. Ammonium nitrate also contributed approximately 10% to 35% of annual average b_{ext}, with much higher concentrations in the winter than in the summer (U.S. EPA, 2019, section 13.2.4.1). In the Northwest, POM was the largest contributor to annual average b_{ext}, up to 70%, in most urban and rural regions with the greatest contributions in the fall. This seasonal contribution of POM may be related to wildfires. A few exceptions included Boise and sites in North Dakota, where ammonium nitrate was the greatest contributor, and sites in the Alaska IMPROVE region, where ammonium sulfate was the greatest contributor (U.S. EPA, 2019, section 13.2.4.1). In the Southwest, based on IMPROVE data, ammonium sulfate or POM were generally the greatest contributors to annual average b_{ext}, with nearly equivalent contributions in several regions. Based on CSN data, ammonium nitrate was often the greatest contributor, with especially high b_{ext} contributions in the winter. While $PM_{10-2.5}$ mass scattering was relatively small in the eastern and northwestern U.S., in the Southwest, $PM_{10-2.5}$ mass scattering contributed to more than 20% of light extinction (U.S. EPA, 2019, section 13.2.4.1).

Differences also exist between the urban CSN and the mainly rural IMPROVE data. Light extinction is generally higher in CSN regions than the geographically corresponding IMPROVE regions. Annual average b_{ext} was greater than 50 Mm^{-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, 2019, Figure 13-11). Ammonium nitrate also accounted for more light extinction in the CSN regions, while it was only a top contributor to b_{ext} in one IMPROVE region (U.S. EPA, 2019, section 13.2.4.1).

From the 2005-2008 time period to the 2011-2014 time period, the annual average b_{ext} in most CSN regions in the Eastern U.S. decreased by more than 20 Mm^{-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, 2019, section 13.2.4.1).

Additionally, Hand et al. (2020) observed that changes in PM composition in ambient air also affect trends for annual, regional mean speciated b_{ext} at IMPROVE monitoring locations across the U.S. In the East, annual mean total b_{ext} decreased by -4.3% yr^{-1} during from 2002 to

2018, much of which is attributable to reductions of light extinction from ammonium sulfate. Light extinction was also decreased for ammonium nitrate, although at a lower rate and a lower magnitude than ammonium sulfate. Light extinction by POM, EC, and fine dust also decreased over time, while light extinction by coarse PM increased slightly. In the Intermountain West and Southwest, annual mean total b_{ext} decreased by -0.9% yr^{-1} from 2002 to 2018. The composition of PM in these regions are different than in the East, and while light extinction from ammonium sulfate and ammonium nitrate generally decreased over these time periods, their contribution to light extinction in the Intermountain West and Southwest is less than in the East. Light extinction by POM, EC, and fine dust decreased over time, while the trend for coarse PM remained relatively the same, although the composition of the particles responsible for light extinction in these areas shifted towards a more carbon-dominated composition over time. It is also important to note that the trends observed in the Intermountain West and Southwest regions are likely influenced by biomass smoke, as wildfire smoke emissions are the largest contributor to light extinction by POM and the impacts of wildfires on air quality in these regions has increased in recent years (Hand et al., 2020). Light extinction levels in the West Coast region were higher than in the Intermountain West and Southwest regions, but generally decreased over time (-1.5% yr^{-1}). Light extinction by ammonium nitrate decreased at the highest rate in the West Coast region, and was the only area were the rate decreased at a greater rate than ammonium sulfate. Light extinction by EC and fine dust also decreased, while the trend for POM generally remained flat and light extinction by coarse mass increased slightly. The mix of positive and negative trends in the West Coast region are likely due to the influence of biomass smoke in northern California and Oregon, in particular during 2017 and 2018, as well as reductions in NO_X emissions in Southern California and reductions in light extinction by ammonium sulfate across the region (U.S. EPA, 2021, section 4.2.2; Hand et al., 2020).

Since the completion of the 2019 ISA, additional research has emerged that explores the impact of wildfire smoke and biomass smoke on PM composition in the U.S. The increases in PM emissions from these sources coincides with decreases in SO_2 and NO_X emissions, which influences the contribution of different PM species to light extinction. The evidence suggests that PM emissions from wildfire and biomass smoke can impact visibility impairment due to general changes in the dominant PM species in the ambient air during these events, as well as the influence of particle size and aging of the PM over time (U.S. EPA, 2021, section 4.2.2; Laing et al., 2016; Kleinman et al., 2020).

In summary, the spatial and temporal analysis of PM monitoring network data in the 2019 ISA and recent evidence presented in the draft ISA Supplement emphasize 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 spatially and temporally.

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 recent 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 2012 review, visibility preference studies were available from four areas in North America,[15] as described in section 5.1.2 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 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).[16] 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).

[15] As noted above, preference studies were available in four urban areas in the last review: Denver, Colorado (Ely et al., 1991, Pryor, 1996), Vancouver, British Columbia, Canada (Pryor, 1996), Phoenix, Arizona (BBC Research & Consulting, 2003), and Washington, DC (Abt Associates, 2001; Smith and Howell, 2009). More details about these studies are available in Appendix D.

[16] 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).

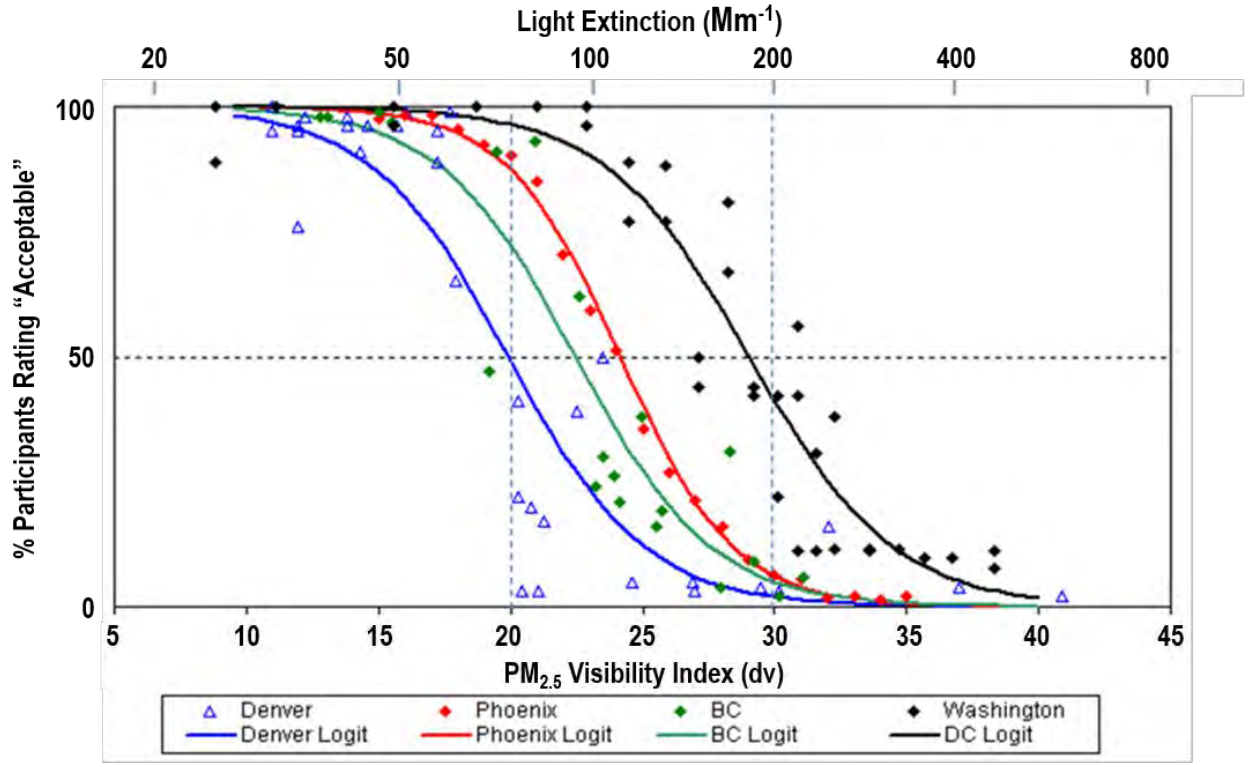

Figure 5-2. Relationship of viewer acceptability ratings to light extinction. (Source: U.S. EPA, 2011, Figure 4-2; U.S. EPA, 2010, Figure 2-16)

Since the completion of the 2012 review, there has been very little research on visibility preferences, with one visibility preference study conducted in the Grand Canyon, AZ (Malm et al., 2019) and one in Beijing, China (Fajardo et al., 2013). The Grand Canyon study reported a lower range of acceptable visibility impairment among participants than was found in preference studies previously conducted in the U.S. (Malm et al., 2019). The Malm et al. (2019) study design is similar to that used in the public preference studies discussed above, but differs from those studies in that this study was conducted in a Federal Class I area, as opposed to in an urban area, with a scene depicted in the photographs that did not include urban features.[17] The Malm et al. (2019) study also used a much lower range of superimposed "haze" than the preference studies discussed above, which may bias the participant responses given the generally lower visibility range presented compared to the other studies.[18]

[17] The Grand Canyon study used a single scene looking west down the canyon with a small landscape feature of a 100-km-distant mountain (Mount Trumbull), along with other closer landscape features. The scenes presented in the previously available visibility preference studies are presented in more detail in Table D-9 in Appendix D.

[18] The Grand Canyon study superimposed light extinction ranging from 3 dv to 20 dv on the image slides shown to participants compared to the previously available preference studies. In those studies, the visibility ranges

The study conducted in Beijing 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 is typical in the U.S. (U.S. EPA, 2019, section 13.2.5).

Similarly, there is little recent information regarding acceptable levels of visibility impairment in the U.S. One study explored alternate methods for evaluating "acceptable" levels of visual air quality from the preference studies, including the use of scene-specific visibility indices as potential indicators of visibility levels as perceived by the observer (Malm et al., 2019). In addition to measures of atmospheric haze, such as atmospheric extinction, used in previously available preference studies, other indices for visual air quality include color and achromatic contrast of single landscape figures, average and equivalent contrast of an entire scene, edge detection algorithms such as the Sobel index, and just-noticeable difference or change indexes. The results reported by Malm et al. (2019) suggest that scene-dependent metrics, such as contrast, may be useful alternate predictors of preference levels compared to universal metrics like light extinction (U.S. EPA, 2021, section 4.2.1). This is because extinction alone is not a measure of "haze," but of light attenuation per unit distance, and visible "haze" is dependent on both light extinction and distance to a landscape feature (U.S. EPA, 2021, section 4.2.1).

- **To what extent have important uncertainties in the evidence from the last review been addressed, and have new uncertainties emerged?**

Since the 2012 review, some refinements have been made to the IMPROVE equation to better estimate light extinction, but there has been no expansion of monitoring efforts for direct measurement of light extinction. At the time of the 2012 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 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 past reviews persist in this reconsideration.

- 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 preference studies may not capture the range of preferences of people in the U.S.
- Most of t7he 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

presented were as low as 9 dv and as high as 45 dv. The visibility ranges presented in the previously available visibility preference studies are described in more detail in Table D-9 in Appendix D.

over the last several decades, the older studies may not reflect current preferences of people in the U.S. Newer studies may not capture the extent to which preferences may be changing over time.

- The 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 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 studies).
- Methods for quantitatively evaluating people's judgments on acceptability are evolving but are still inconsistent in their application across studies. Variability in quantitative methods for comparing visual air quality in public preference studies may influence the consistency and comparability of results and the interpretation of these results in the context of regional or national preferences for visibility impairment in urban, non-urban, and Federal Class I areas.

Overall, the body of evidence regarding visibility effects remains largely unchanged since the time of the 2012 review. While one new study provides refinements to the methods for estimating light extinction, uncertainties and limitations in the scientific evidence during the previous reviews remain.

5.3.1.2 Quantitative and Air Quality Information

Beyond our consideration of the scientific evidence, discussed in section 5.3.1.1 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 2012 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 2012 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 available since the 2012 review includes an updated equation for estimating light extinction, summarized in section 5.3.1.1 above and described in the 2019 ISA, 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 reconsideration based on the 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 2012 and 2020 reviews, we have conducted analyses examining the relationship between PM mass concentrations and calculated light extinction using the 3-year design values[19] for the current secondary standards and a 3-year average visibility metric based on light extinction estimated using IMPROVE equations using air quality data for 2017 to 2019.[20] 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.

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 60 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 2017-2019 period and met strict criteria for PM species for this analysis.[21] We present results for these 60 sites using the original IMPROVE equation, with modifications to the equation consistent with those made in evaluating light extinction in the 2012 review (described in detail in section D.1 of Appendix D). We then present results for these 60 sites with light extinction calculated using the Lowenthal and Kumar (2016) IMPROVE equation described in section 5.3.1.1 above.

[19] A design value is a statistic that summarizes the air quality data for a given area in terms of the indicator, averaging time, and form of the standard. Design values can be compared to the level of the standard and are typically used to designate areas as meeting or not meeting the standard and assess progress towards meeting the NAAQS.

[20] This is the 3-year visibility metric that was used to evaluate visibility impairment in the 2012 and 2020 reviews. Given that there has been little new research since the time of the 2012 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 2012 review.

[21] For this analysis, completeness criteria for speciated PM data at these sites included having all 12 quarters in the 2017-2019 period with at least 11 days in each quarter with a valid $PM_{2.5}$ and $PM_{10-2.5}$ mass, sulfate, nitrate, organic carbon, elemental carbon, sea salt (chlorine or chloride), and fine soil (aluminum, silica, calcium, iron, and titanium) measurement.

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 2012 review (Kelly et al., 2012; 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 26 dv (Figure 5-3; 78 FR 3218, January 15, 2013). For the four locations that exceed the current 24-hour $PM_{2.5}$ standard, light extinction estimates range from 22 dv to 29 dv (Figure 5-3). These findings are consistent with the findings of the analysis using the same IMPROVE equation in the 2012 review with data from 102 sites with data from 2008-2010 and in the 2020 review with data from 67 sites with data from 2015-2017. This indicates similar findings from this analysis as was the case with the similar analysis in the 2012 and 2020 reviews, i.e., the updated quantitative analysis shows that the 3-year visibility metric was no higher than 30 dv[22] at sites meeting the current secondary PM standards, and at most such sites the 3-year visibility index values are much lower (e.g., an average of 20 dv across the 60 sites).

[22] 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 2012 review (78 FR 3191, January 15, 2013).

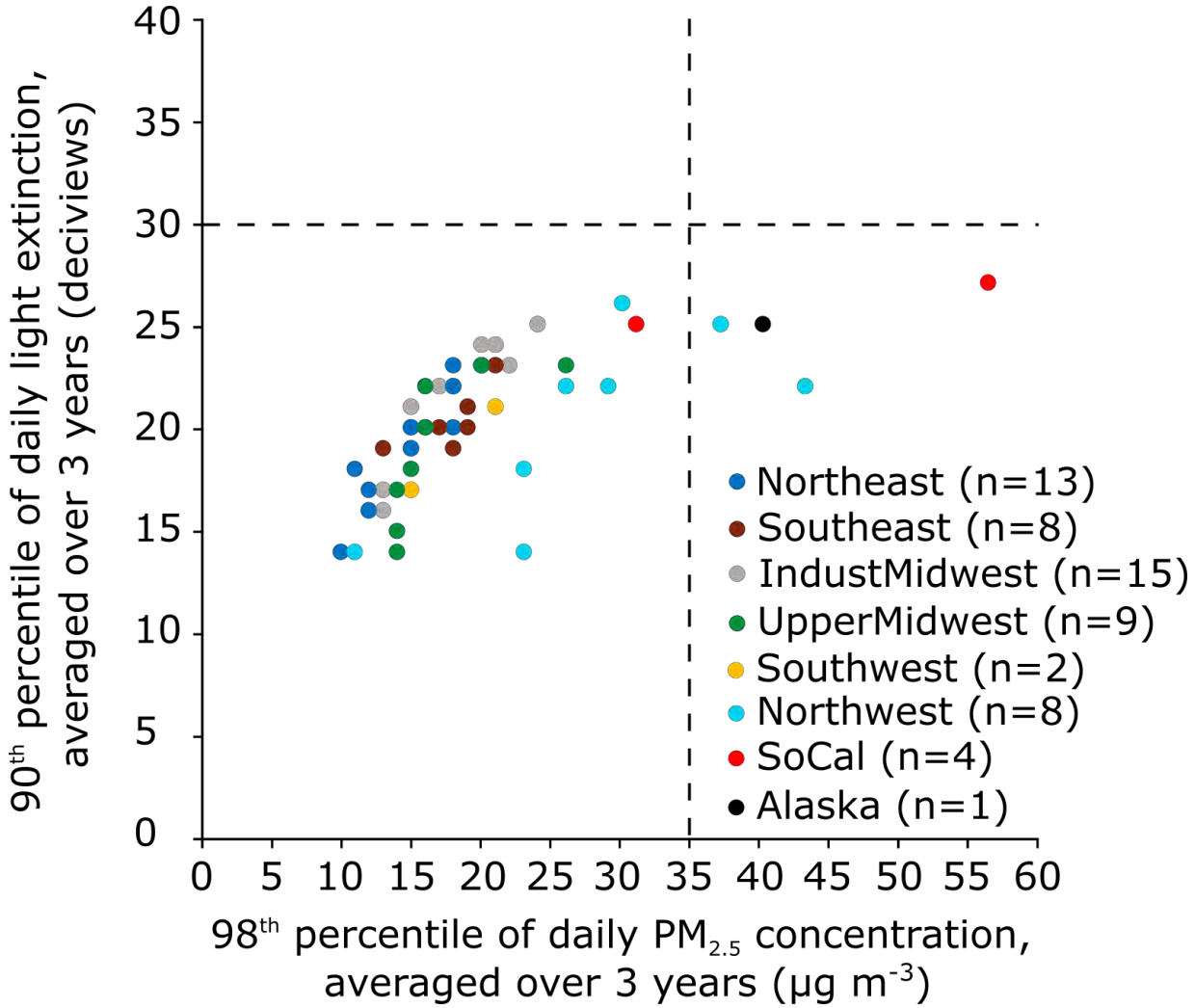

Figure 5-3. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2017-2019 using the original IMPROVE equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ standard (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 5.3.1.1, 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 5.3.1.1, 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.

Using the Lowenthal and Kumar (2016) equation, for those sites that meet the current 24-hour $PM_{2.5}$ standard, the 3-year visibility metric is at or below 28 dv when light extinction is calculated. For those sites that exceed the current 24-hour $PM_{2.5}$ standard, three of these sites have a 3-year visibility metric ranging between 26 dv and 30 dv, while one site in Fresno, California that exceeds the current 24-hour $PM_{2.5}$ standard and has a 3-year visibility index value of 32 dv (compared to 29 dv when light extinction is calculated with the original IMPROVE equation) (see Table D-3 in Appendix D). At this site, it is likely that the 3-year visibility metric using the Lowenthal and Kumar (2016) equation would be below 30 dv if $PM_{2.5}$ concentrations were reduced such that the 24-hour $PM_{2.5}$ level of 35 $\mu g/m^3$ was attained.

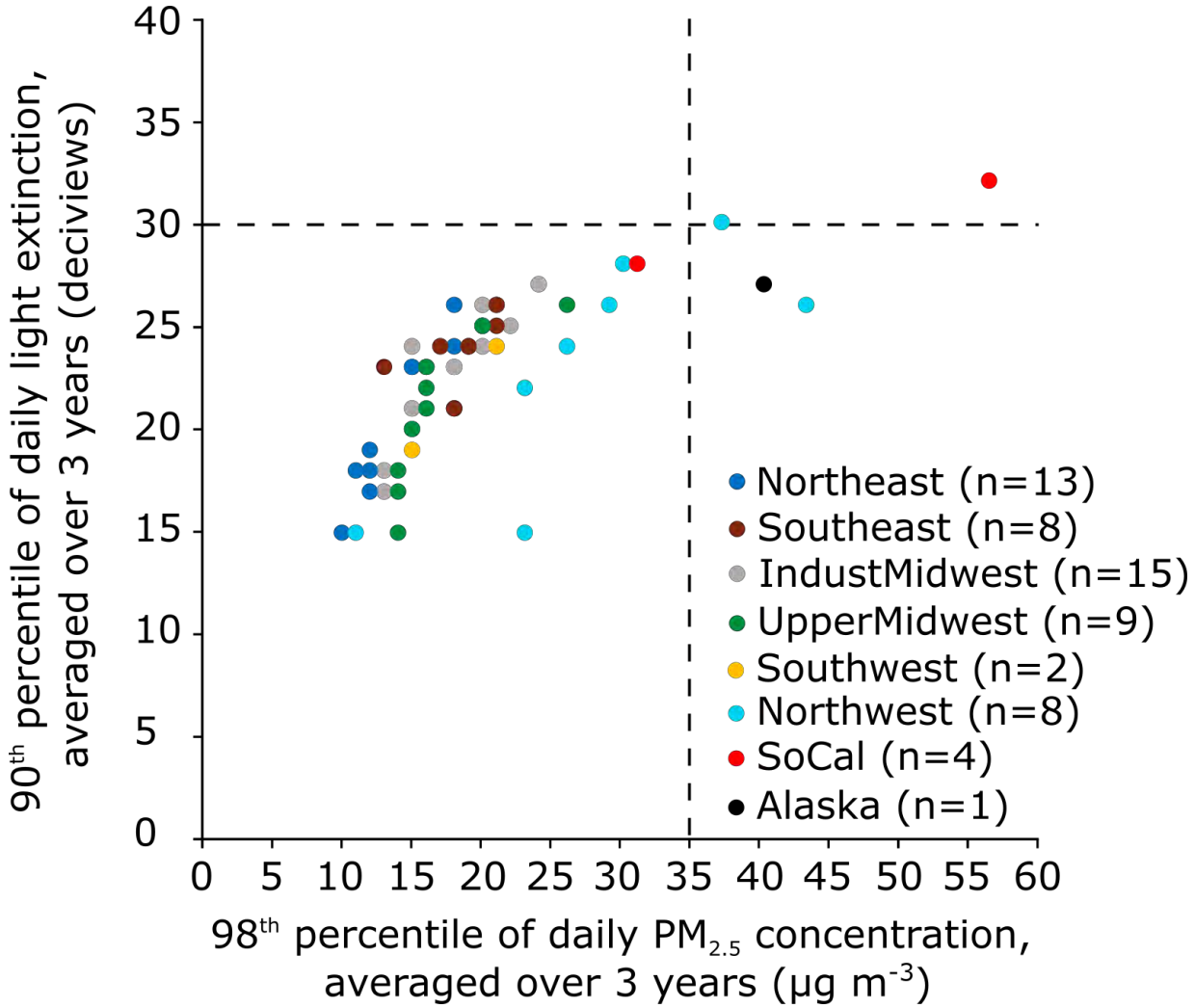

Figure 5-4. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2015-2017 using the Lowenthal and Kumar equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ 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 2012 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, 2012a, p. IV-5). The information and analyses 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 2012 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 2012 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 2012 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. The analysis in the 2020 review addressed light extinction at 20 of the 67 $PM_{2.5}$ sites where collocated $PM_{10\text{-}2.5}$ monitoring data were available. Since the 2020 review, $PM_{10\text{-}2.5}$ monitoring data are available at more locations and the analyses presented in this draft PA include those for light extinction estimated with coarse and fine PM at all 60 sites. 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 (U.S. EPA, 2020, section 5.2.1.2). However, we note that in our analysis, only a few sites were in locations that would be expected to have high concentrations of coarse PM, such as the Southwest. These results are consistent with those in the analyses in the 2019 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, 2019, section 13.2.4.1, p. 13-36).

In summary, the findings of these updated quantitative analyses are generally consistent with those in the 2012 and 2020 reviews. The 3-year visibility metric was generally below 26 dv in most areas that meet the current 24-hour $PM_{2.5}$ standard. 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.

5.3.2 Non-Visibility Effects

5.3.2.1 Nature of Effects

In considering the evidence for non-visibility welfare effects attributable to PM as presented in the 2019 ISA, this section poses the following policy-relevant questions:

- **To what extent has the 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?**

As an initial matter, we note that the draft ISA Supplement does not include an evaluation of additional studies for climate and materials effects and the causality determinations from PM-related climate and materials effects presented in the 2019 ISA continue to serve as the scientific foundation for these effects. As such, the sections below that address these questions for PM and climate effects (section 5.3.2.1.1) and materials effects (section 5.3.2.1.2) draw from the evaluation of the welfare effects evidence for PM-related climate and materials effects in the 2019 ISA and considerations of such effects in the 2020 PA (U.S. EPA, 2020).

5.3.2.1.1 Climate Effects

In considering the evidence of climate effects attributable to PM, this section poses the following policy-relevant question:

- **To what extent is 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 2012 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).[23] Since the 2012 review, climate impacts have been

[23] 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

extensively studied and the 2019 ISA concludes that "overall the evidence is sufficient to conclude that a causal relationship exists between PM and climate effects" (U.S. EPA, 2019, section 13.3.9). Recent research reinforces and strengthens the evidence evaluated in the 2009 ISA. Recent 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 2012 review, the 2009 ISA relied heavily on the Fourth IPCC Assessment Report (AR4); since the 2012 review, the IPCC issued an updated report, as described in the 2019 ISA. The Fifth IPCC Assessment Report (AR5; IPCC, 2013) reports on the key scientific advances in understanding the climate effects of PM since AR4. The 2019 ISA draws substantially upon AR5 in summarizing these effects.

Atmospheric PM has the potential to affect climate in multiple ways, including absorbing and scattering of incoming solar radiation, alterations in terrestrial radiation, effects on the hydrological cycle, and changes in cloud properties (U.S. EPA, 2019, section 13.3.1). Atmospheric PM interacts with incoming solar radiation. Many species of PM (e.g., sulfate and nitrate) efficiently scatter solar energy. By enhancing reflection of solar energy back to space, scattering PM exerts a cooling effect on the surface below. Certain species of PM such as black carbon (BC), brown carbon (BrC), or dust can also absorb incoming sunlight. A recent study found that whether absorbing PM warms or cools the underlying surface depends on several factors, including the altitude of the PM layer relative to cloud cover and the albedo of the surface (Ban-Weiss et al., 2014). PM also perturbs incoming solar energy by influencing cloud cover and cloud lifetime. For example, PM provides nuclei upon which water vapor condenses, forming cloud droplets. Finally, absorbing PM deposited on snow and ice can diminish surface albedo and lead to regional warming (U.S. EPA, 2019, section 13.3.2).

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 ±

forcing indicates net energy trapped in the Earth system and suggests warming of the Earth's surface, whereas a negative forcing indicates net loss of energy and suggests cooling (U.S. EPA, 2019, section 13.3.2.2).

0.5 watts per square meter (Wm^{-2}) (Boucher, 2013), which is slightly reduced compared to AR4. Estimates of effective radiative forcing[24] from aerosol-radiation interactions (ERFari), which include the rapid feedback effects of temperature and cloud cover, rely mainly on model simulations, as this forcing is complex and difficult to observe (U.S. EPA, 2019, section 13.3.4.1). The IPCC AR5 best estimate for ERFari is -0.45 ± 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, 2019, section 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, 2019, section 13.3.4.2).[25] Studies have also calculated the combined effective radiative forcing from aerosol-radiation and aerosol-cloud interactions (ERFari+aci) (U.S. EPA, 2019, section 13.3.4.3). IPCC AR5 reports a best estimate of ERFari+aci of -0.90 (-1.9 to -0.1) 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 heating. The absorbed energy can then melt the snow and ice cover and further depress the albedo, resulting in a positive feedback loop (U.S. EPA, 2019, section 13.3.3.3; Bond et al., 2013; U.S. EPA, 2012b). Deposition of absorbing PM, such as BC, may also affect surface temperatures over glacial regions (U.S. EPA, 2019, section 13.3.3.3). The IPCC AR5 best estimate of RF from the albedo effect is +0.04 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 2012 review, there are still significant uncertainties associated with the accurate measurement of PM contributions to the direct and indirect effects of PM on climate.

[24] Effective radiative forcing (ERF), new in the IPCC AR5, takes into account not just the instantaneous forcing but also a set of climate feedbacks, involving atmospheric temperature, cloud cover, and water vapor, that occur naturally in response to the initial radiative perturbation (U.S. EPA, 2019, section 13.3.2.2).

[25] While the 2019 ISA includes estimates of RFaci and ERFaci from a number of studies (U.S. EPA, 2019, sections 13.3.4.2, 13.3.4.3, 13.3.3.3), this draft PA focuses on the single best estimate with a range of uncertainty, as reported in IPCC AR5 (Boucher, 2013).

- **To what extent does the information provide evidence of a quantitative relationship between specific PM constituents (i.e., BC, OC, sulfate) and climate-related effects?**

Since the 2012 review, a number of 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, 2019, 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, 2019, section 13.3.5.1). Sulfate particles are highly reflective. Consistent with other recent estimates, on a global scale, the IPCC AR5 estimates that sulfate contributes more than other PM types to RF, with 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} (Adams et al., 2001, Zelinka et al., 2014).

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, 2019, section 13.3.5.2; Hauglustaine et al., 2014; Lee et al., 2013; Shindell et al., 2013). Warmer global temperatures, however, may decrease nitrate abundance given that it is highly volatile at higher temperatures (Tai et al., 2010). The IPCC AR5 estimates RFari of nitrate of -0.11 (-0.3 to -0.03) Wm^{-2} (Boucher, 2013), which is one-fourth of the RFari of sulfate.

Primary organic carbonaceous PM, including BrC, are emitted from wildfires, agricultural fires, and fossil fuel and biofuel combustion. Secondary organic aerosols (SOA) form when anthropogenic or biogenic nonmethane hydrocarbons are oxidized in the atmosphere, leading to less volatile products that may partition into PM (U.S. EPA, 2019, section 13.3.5.3). Organic particles are generally reflective, but in the case of BrC, a portion is significantly absorbing at shorter wavelengths (<400 nm). The IPCC AR5 estimates an RFari for primary

organic PM from fossil fuel combustion and biofuel use of -0.09 (-0.16 to -0.03) 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, 2019, section 13.3.5.4, section 13.3.4.4).

Dust, or mineral dust, is mobilized from dry or disturbed soils as a result of both meteorological and anthropogenic activities. Dust has traditionally been classified as scattering, but a recent study found that dust may be substantially coarser than currently represented in climate models, and thus more light-absorbing (Kok et al., 2017). The IPCC AR5 estimates RFari as -0.1 ± 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, 2019, section 13.3.5.5).

Recent research expands upon the evidence from the 2012 review. Consistent with the evidence in the 2012 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 the 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 2012 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, 2019, section 13.3.6). The complex climate system interactions lead to variation among climate models, with some studies showing relatively close correlation between forcing and surface response temperatures (e.g., 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, 2019, section 13.3.7). While evidence described in the 2019 ISA 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 prior reviews been reduced and/or have new uncertainties emerged?**

Since 2009, significant progress has been made in evaluating PM-related climate effects and uncertainties. The IPCC AR5 states that "climate-relevant aerosol processes are better understood, and climate-relevant aerosol properties are better observed, than at the time of the AR4" (Boucher, 2013). However, significant uncertainties remain that make it difficult to quantify the climate effects of PM. Such uncertainties include those related to our understanding of:

- The magnitude of PM radiative forcing and the portion of that associated with anthropogenic emissions;
- The contribution of regional differences in PM concentrations, and of individual components, to radiative forcing;
- The mechanisms of climate responses and feedbacks resulting from PM-related radiative forcing; and,
- The process by which PM interacts with clouds and how to represent such interactions in climate models.

While research has progressed significantly since the 2012 review, substantial uncertainties still remain with respect to key processes linking PM and climate, because of the small scale of PM-relevant atmospheric processes compared to the resolution of state-of-the-art models, and because of the complex cascade of indirect impacts and feedbacks in the climate

system that result from an initial PM-related radiative perturbation (U.S. EPA, 2019, section 13.3.9).

5.3.2.1.2 Materials Effects

In considering the evidence on materials effects attributable to PM, this section poses the following policy-relevant question:

- **To what extent is 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 2012 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 2012 review, the evidence in the 2019 ISA continues to support prior conclusions regarding materials effects associated with PM deposition. Effects of deposited PM, particularly sulfates and nitrates,[26] 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 evidence on materials effects of PM are from outside the U.S. on buildings and other items of cultural heritage; however, they provide limited new data for consideration. (U.S. EPA, 2019, section 13.4).

Materials damage from PM generally involves one or both of two processes: soiling and corrosion (U.S. EPA, 2019, section 13.4.2). Soiling and corrosion are complex, interdependent processes, typically beginning with deposition of atmospheric PM or SO_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, 2019, section 13.4.2).

Soiling is the result of PM accumulation on an object that alters its optical characteristics or appearance. These soiling effects can affect 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

[26] In the case of materials effects, it is difficult to isolate the effects of gaseous and particulate nitrogen and sulfur wet deposition so both will be considered along with other PM-related deposition effects on materials.

of black crusts on surfaces, and the buildup of microbial biofilms[27] can discolor surfaces by trapping PM more efficiently (U.S. EPA, 2009, p. 9-195; U.S. EPA, 2019, section 13.4.2). The presence of PM may alter light transmission or change the reflectivity of a surface. Additionally, the organic or nutrient content of deposited PM may enhance microbial growth on surfaces.

Since the 2012 review, very little 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, 2019, 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) (Grossi et al., 2007, McAlister et al., 2008). 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 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 the relationship between the concentrations of elements in the black crusts and local environmental conditions. Recent research has also used isotope tracers to distinguish between contributions from local sources versus atmospheric pollution to black crusts on historical monuments in France (Kloppmann et al., 2011). A study in Portugal found that biological activity played a major role in soiling, specifically in the development of colored layers and in the detachment process (de Oliveira et al., 2011). Another study found damage to cement renders, often used for restoration, consolidation, and decorative purposes on buildings, following exposure to sulfuric acid, resulting in the formation of gypsum (Lanzon and Garcia-Ruiz, 2010).

Corrosion of stone and the decay of stone building materials by acid deposition and sulfate salts were described in the 2009 ISA (U.S. EPA, 2009, section 9.5.3). Since that time, advances have been made on the quantification of degradation rates and further characterization of the factors that influence damage of stone materials (U.S. EPA, 2019, section 13.4.2). Decay rates of marble grave stones were found to be greater in heavily polluted areas compared to a relatively pristine area (Mooers et al., 2016). The time of wetness and the number of

[27] 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.

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, 2019, section 13.4.2). Other studies have found that soiling of building materials can be attributed to enhanced biological processes and colonization, including the development and thickening of biofilms, resulting from the deposition of PM components and atmospheric gases (U.S. EPA, 2019, section 13.4.2).

Since the 2012 review, other materials have been studied for damage attributable to PM, including glass and photovoltaic panels. Soiling of glass can affect its optical and thermal properties, and can lead to increased cleaning costs and frequency. The development of haze[28] 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.

[28] In this discussion of non-visibility welfare effects (section 5.3.2), haze is used as it has been defined in the scientific literature on soiling of glass, i.e., the ratio of diffuse transmitted light to direct transmitted light (Lombardo et al., 2010). This differs from the definition of haze as used in the discussion of visibility welfare effects in section 5.3.1, where it is used as a qualitative description of the blockage of sunlight by dust, smoke, and pollution.

In five sites in the U.S. representing different meteorological and climatological conditions,[29] 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).

- **To what extent has 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 nitrogen and sulfur species?**

Some progress has been made since the 2012 review in the development of dose-response relationships for soiling of building materials, although some key relationships remain poorly characterized. The first general dose-response relationships for soiling of materials were generated by measuring contrast reflectance of a soiled surface to the reflectance of the unsoiled substrate for different materials, including acrylic house paint, cedar siding, concrete, brick, limestone, asphalt shingles, and window glass with varying total suspended particulate (TSP) concentrations (Beloin and Haynie, 1975; U.S. EPA, 2019, section 13.4.3). Continued efforts to develop dose-response curves for soiling have led to some advancements for modern materials, but these relationships remain poorly characterized for limestone. One 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-

[29] Of the five sites studied, three were in rural, suburban, and urban areas representing a semi-arid environment (Front Range of Colorado), one site represented a hot and humid environment (Cocoa, Florida), and one represented a hot and arid environment (Albuquerque, New Mexico) (U.S. EPA, 2019, section 13.4.2; Boyle et al., 2017).

soda-lime window glass soiling by PM_{10}, NO_2, and SO_2 was quantified based on 31 different locations (Lombardo et al., 2010; U.S. EPA, 2019, section 13.4.3, Figure 13-32, Equation 13-8). The development of this dose-response relationship required several years of observation time and had inconsistent data reporting across the locations.

Since the 2012 review, there has also been progress in developing methods to more rapidly evaluate soiling of different materials by PM mixtures. Modern buildings typically have simpler lines, less detailed surfaces, and a greater use of glass, tile, and metal, which are easier to clean than stone. There have also been major changes in the types of materials used for buildings, including a variety of polymers available for use as coatings and sealants. New economic and environmental considerations beyond aesthetic appeal and structural damage are emerging (U.S. EPA, 2019, section 13.4.3). Changes in building materials and design, coupled with new approaches in quantifying the dose-response relationship between PM and materials effects, may reduce the amount of time needed for observations to support the development of material-specific dose-response relationships.

In addition to dose-response functions, damage functions have also been used to quantify material decay as a function of pollutant type and load. Damage can be determined from sample surveys or inspection of actual damage and a damage function can be developed to link the rate of material damage to time of replacement or maintenance. A cost function can then link the time for replacement and maintenance to a monetary cost, and an economic function links cost to the dose of pollution based on the dose-response relationship (U.S. EPA, 2019, section 13.4.3). Damage functions are difficult to assess because it depends on human perception of the level of soiling deemed to be acceptable and evidence in this area remains limited. As described in the 2019 ISA, 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 prior reviews been reduced and/or have new uncertainties emerged?**

While there are a number of studies in the 2019 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 2012 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 assessed in the 2019 ISA 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.

5.3.2.2 Quantitative Information

Beyond our consideration of the scientific evidence, discussed above in section 5.3.2.1 **Error! Reference source not found.** 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.

5.3.2.2.1 Climate Effects

While expanded since the 2012 review, our current understanding of PM-related climate effects is still limited by significant uncertainties. Large spatial and temporal heterogeneities in direct and indirect PM climate forcing can occur for a number of reasons, including the frequency and distribution of emissions of key PM components contributing to climate forcing, the chemical and microphysical processing that occurs in the atmosphere, and the atmospheric lifetime of PM relative to other pollutants contributing to climate forcing (U.S. EPA, 2019, section 13.3). These issues particularly introduce uncertainty at the local and regional scales in the U.S. that would likely be most relevant to a quantitative assessment of the potential effects of a national PM standard on climate in this review. Limitations and uncertainties in the evidence make it difficult to quantify the impact of PM on climate and in particular how changes in the level of PM mass in ambient air would result in changes to climate in the U.S. Thus, as in the 2012 review, the data remain insufficient to conduct quantitative analyses for PM effects on climate.

5.3.2.2.2 Materials Effects

As at the time of the 2012 review, sufficient evidence is not available to conduct a quantitative assessment of PM-related soiling and corrosion effects. While soiling associated with PM can lead to increased cleaning frequency and repainting of surfaces, no quantitative relationships have been established between characteristics of PM or the frequency of cleaning or repainting that would help inform our understanding of the public welfare implications of

soiling (U.S. EPA, 2019, section 13.4). Similarly, while some information is available with regard to microbial deterioration of surfaces and the contribution of carbonaceous PM to the formation of black crusts that contribute to soiling, the available evidence does not support quantitative analyses (U.S. EPA, 2019, section 13.4). While some evidence is available with respect to PM-attributable materials effects, the data are insufficient to conduct quantitative analyses for PM effects on materials.

5.4 PRELIMINARY CONCLUSIONS REGARDING THE ADEQUACY OF 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 2019 ISA and draft ISA Supplement, as well as 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 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 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 5.3.1 and 5.3.2) and the approach described in section 5.2 that builds on the approach from previous reviews. With respect to the evidence-based considerations, we note that the evidence, while somewhat expanded since previous reviews, does not include evidence of effects at lower concentrations or other welfare effects of PM than those identified at the time of prior reviews. 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 evidence continues to support a causal relationship. With respect to evidence for visibility effects of PM, we note that the evidence, while somewhat expanded since the 2012 review, does not include evidence of effects at lower concentrations than those identified at the time of the 2012 review. Consistent with the evidence available at the time of the 2012 review, significant limitations remain in directly measuring light extinction. However, a number of small refinements have been made to the algorithm commonly used to estimate light extinction (U.S. EPA, 2019, section 13.2.3.3; section 5.3.1.1 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 limited new research is available on methods of characterizing visibility or on how visibility is valued by the public, such as visibility preference studies. Thus, while limited new research has further informed our understanding of the influence of atmospheric components of $PM_{2.5}$ 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 2012 and 2020 reviews. We recognize that conclusions reached regarding visibility in previous reviews 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 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, consistent with the approach in the 2020 review, we have utilized both the most recently published equations as well as alternatives considered in the 2012 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 analyses on which the decisions were based in the 2012 and 2020 reviews, 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 and 2020 reviews with regard to the adequacy of visibility protection provided by the secondary PM standards, both Administrators identified 30 dv as an appropriate target level of protection. We have not identified new information available since the completion of the 2020 review in this reconsideration of the 2020 final decision 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 that all sites meet this target level of protection while also meeting the current daily standards. 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 reconsideration of the 2020 final decision 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 available evidence continues to support causal relationships between climate effects and PM and materials effects and PM. The evidence related to climate effects and PM, while expanded since previous reviews, 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 2012 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 2012 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 recent 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 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 information to provide support for different conclusions than were reached in the 2012 and 2020 reviews with regard to climate-related effects of PM in ambient air.

In considering the evidence related to materials effects and PM, we note that there is some 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 recent 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 previous reviews, documents a causal role for PM in ambient air. This evidence, however, as in the 2012 and 2020 reviews, 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 available information 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 conclusion for the Administrator's consideration is that it is appropriate to consider retaining the current secondary PM standards, without revision. In so concluding, we recognize, as noted above, that the final decision on this reconsideration 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.

5.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

In this section, we highlight key uncertainties in the available information related to the effects of PM on public welfare. Such key uncertainties and areas for future research, model development, and data gathering are outlined below. We note, however, that a full set of research recommendations is beyond the scope of this discussion. Rather, listed below are key uncertainties, research questions and data gaps that have been thus far highlighted in this review of the secondary PM standards.

- A critical aspect of our consideration of the evidence and quantitative information for visibility impairment is our understanding of human perception of visibility impairment in the preference studies. This is essential to the Administrator's consideration of the public welfare implications of visibility effects and to decisions on the adequacy of protection provided by the secondary PM standards from them. Additional information related to several areas would reduce uncertainty in 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 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.

- The development and implementation of 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;
  - 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 PM concentrations or to establish a quantitative relationship between PM concentrations in ambient air and materials degradation. Additional information would reduce uncertainty in in our interpretation of the available information, including in the following areas:
  - Identifying quantitative relationships between particle size, PM concentration, chemical concentrations, and frequency of repainting and repair;
  - Understanding human perceptions of reduced aesthetic appeal of buildings, and other objects to PM-related materials effects; and
  - Characterizing deposition rates of airborne PM to surfaces and the interaction of co-pollutants.

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APPENDIX A. SUPPLEMENTAL INFORMATION ON PM AIR QUALITY ANALYSES

This appendix provides supplemental information on the data sources and methods used to generate the figures and table presented in Chapter 2 of this 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. Section A.7 described the methods used for the comparison on $PM_{2.5}$ fields in estimating exposure and relative to design values.

A.1 DATA SOURCES AND METHODS FOR GENERATING NATIONAL $PM_{2.5}$, PM_{10}, $PM_{10-2.5}$, 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 2000-2019 from the EPA's Air Quality System (AQS, https://www.epa.gov/aqs)
- PM_{10} 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 2000-2019 from the EPA's Air Quality System (AQS, https://www.epa.gov/aqs)
- $PM_{10-2.5}$ 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 2000-2019 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 2017-2019.

- The 2000-2019 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 2017-2019 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 show 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 2000-2019 from the EPA's Air Quality System (AQS, https://www.epa.gov/aqs)

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 and 5-hour averages were calculated for periods with each hourly average available. Only sites with a valid annual or 24-hour design value for 2017-2019 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. Frequency distributions of 5-hour averages are presented in Figure A-2.

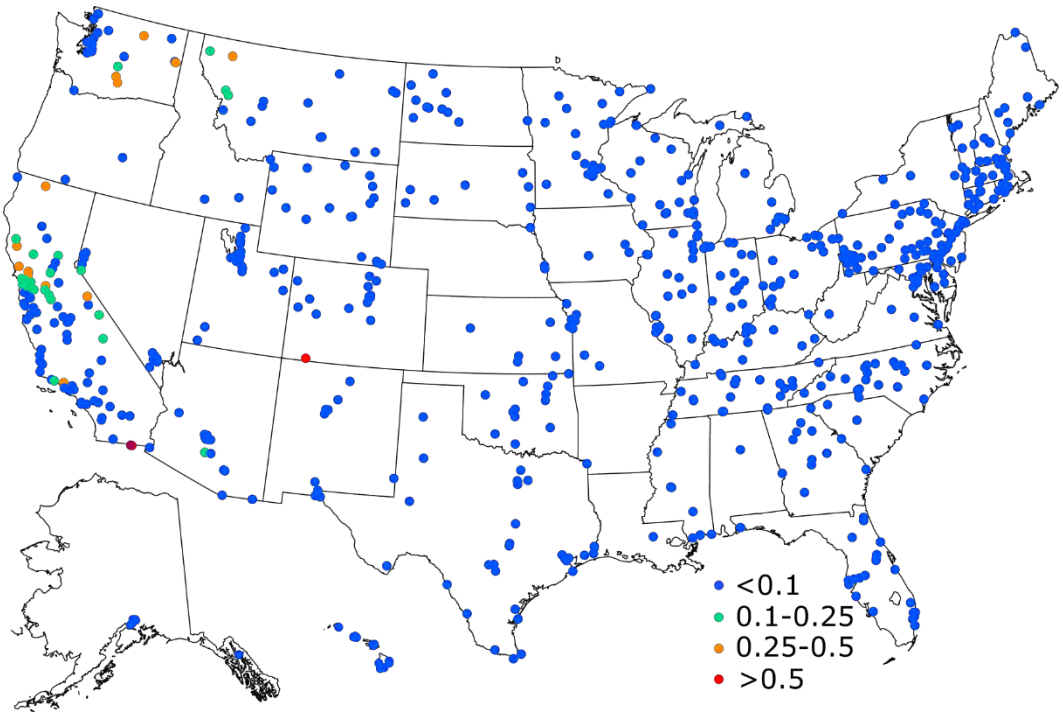

Figure A-1. Percentages of 2017-2019 2-hour average $PM_{2.5}$ mass concentrations above 140 µg/m³.

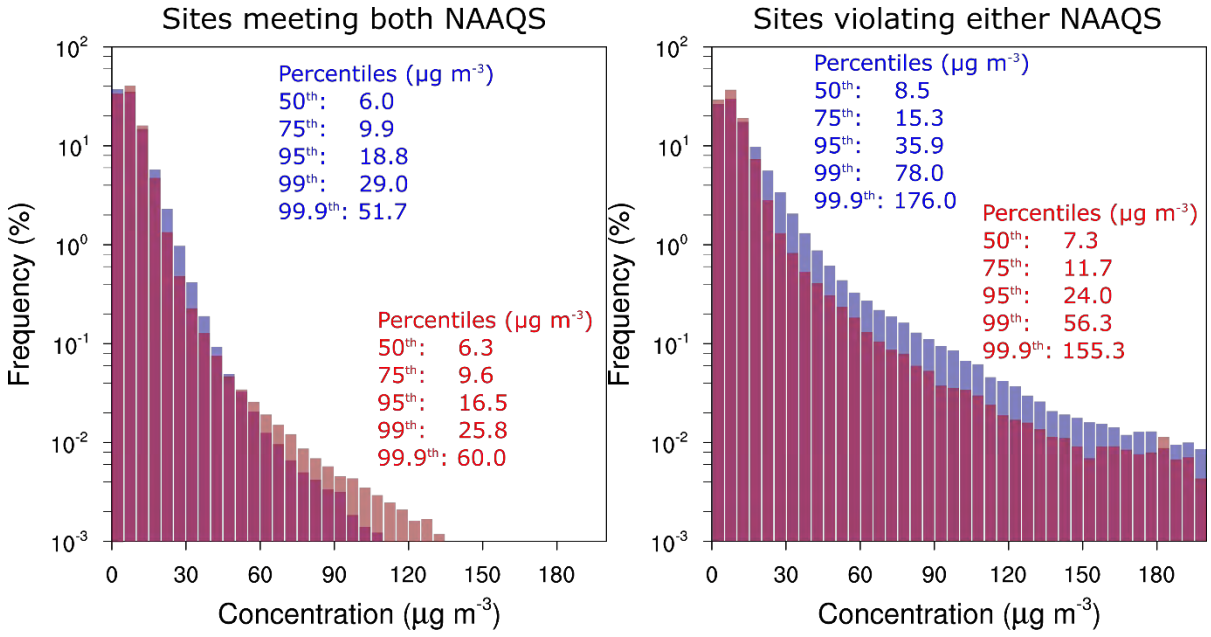

Figure A-2. Frequency distribution of 2017-2019 4-hour averages for sites meeting both or violating either $PM_{2.5}$ NAAQS for October to March (blue) and April to September (red).

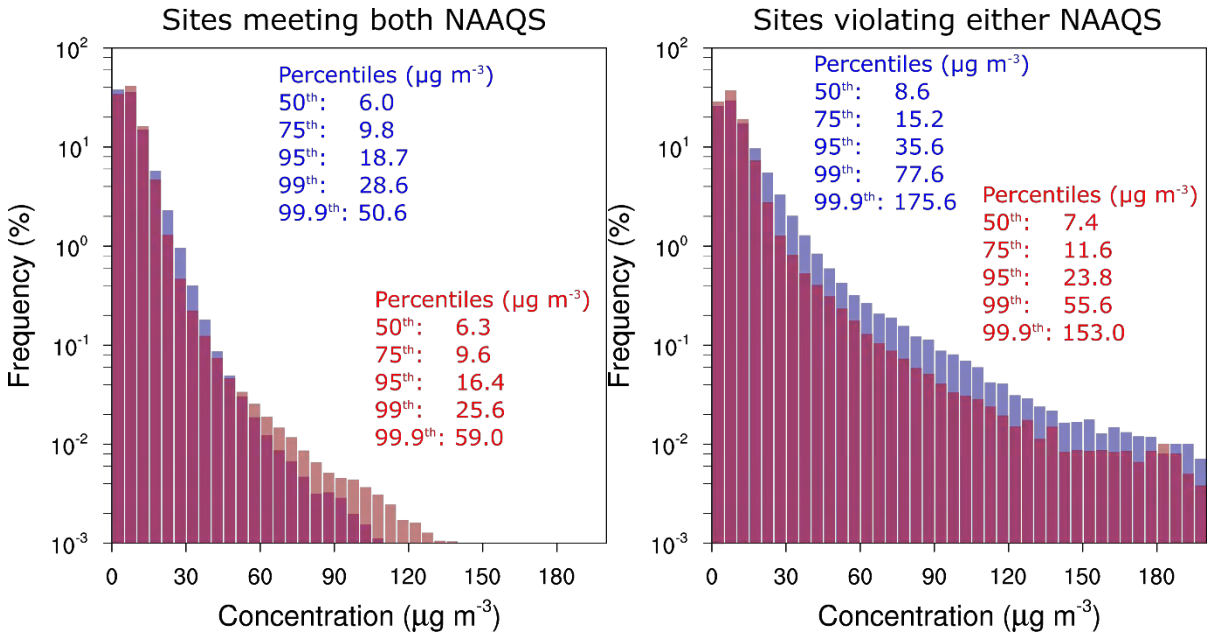

Figure A-3. Frequency distribution of 2017-2019 5-hour averages for sites meeting both or violating either $PM_{2.5}$ NAAQS for October to March (blue) and April to September (red).

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 2000-2019. 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 2-28, Figure 2-29, and Table 2-2.
- 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 2-38: 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 2-39: 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 2-40: 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 2-41.

A.7 COMPARISON OF $PM_{2.5}$ FIELDS IN ESTIMATING EXPOSURE AND RELATIVE TO DESIGN VALUES: METHODS

Section 2.3.3.2.4 outlines analyses comparing the $PM_{2.5}$ concentrations in estimating exposure relative to design values. Below details the data sources and methods used.

To calculate annual average concentrations over the U.S. for 2000-2016, gridded concentration fields were obtained based on the DI2019 (Di et al., 2019) and the HA2020 (Hammer et al., 2020) and (Van Donkelaar et al., 2019) methods. The DI2019 concentrations were acquired from a Google Drive and the HA2020 concentrations (version V4.NA.03) were acquired from a web link. To identify grid cells that fall within the contiguous U.S. and Core Based Statistical Areas (CBSAs) boundaries, cartographic boundary shapefiles ("cb_2017_us_state_5m" and "cb_2017_us_cbsa_5m") were downloaded from the census.gov website. The concentration data and shapefiles were read into R version 3.62 (R Core Team, 2019), and grid cells within the contiguous U.S. and CBSAs were identified using the Simple Features package version 0.8-0 (Pebesma, 2018) in R. Average concentrations were then calculated for each year and for each region (i.e., contiguous U.S. and CBSAs within the contiguous U.S.) using the dplyr package version 0.8.3 (Wickham et al., 2019) in R.

To generate the population-weighting for the DI2019 and HA2020 $PM_{2.5}$ concentrations, 2015 gridded population counts at 0.05×0.05° from the fourth version of the Gridded Population of the World (GPWv4; https://sedac.ciesin.columbia.edu/data/collection/gpw-v4) were spatially-collocated with the $PM_{2.5}$ concentrations surfaces after conversion to latitude-longitude coordinates. A similar CBSA filtering was performed for the gridded population and spatially-collocated $PM_{2.5}$ surfaces from DI2019 and HA2020 and the fractional population for each grid was multiplied by the $PM_{2.5}$ concentrations within each CBSA.

Regulatory 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 $PM_{2.5}$, by CBSA, for each 3-year period of available hybrid modeling surface data from the EPA's Air Quality System (AQS, https://www.epa.gov/aqs). Within each CBSA, by each 3-year period, the ratio of design values to estimated $PM_{2.5}$ concentrations was calculated.

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APPENDIX B. SUPPLEMENTAL STUDY INFORMATION: SELECTION CRITERIA, STUDY METHODS AND DETAILS

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This appendix presents supplemental information on the key epidemiologic studies evaluated in section 3.3.3 of this draft PA. Section B.1 provides supplemental information on the forest plots presented in Figures 3-3 to 3-6. Section B.2 provide supplemental information on the study-reported $PM_{2.5}$ concentrations presented in Figure 3-8, Figure 3-9, while section B.3 provides supplemental information on studies presented Figure 3-10 and Figure 3-11. Section B.4 provides details on key elements of epidemiologic studies, including the study design and details on the statistical analyses employed, including control for confounding effects.

B.1 FOREST PLOTS

Figure 3-3 through 3-6 in Chapter 3 present forest plots that include the effect estimates and 95% confidence intervals from 92 epidemiologic studies that were assessed in the 2019 ISA and draft ISA Supplement that have the potential to be most informative in reaching conclusions on the adequacy of the current primary $PM_{2.5}$ standards. Epidemiologic studies included in these figures support "causal" or "likely to be causal" relationships with PM exposures in the 2019 ISA and include mortality (all-cause mortality, cardiovascular (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 (U.S. EPA, 2019). For some of the major cohort studies included in the 2009 ISA, like the American Cancer Society (ACS) cohort, we included more recent 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., 2015a, Turner et al. (2016), Jerrett et al. (2016), and Thurston et al. (2016b)), as well as a reanalysis (Krewski et al. (2009) of the original ACS dataset, including an extended follow-up period, that was evaluated in the 2009 ISA (U.S. EPA, 2009)).

B.2 MONITORED $PM_{2.5}$ CONCENTRATIONS IN KEY EPIDEMIOLOGIC STUDIES

Based on the 92 key studies identified in Figure 3-3 to Figure 3-6, a subset of studies are depicted in Figure 3-8 and Figure 3-9 and 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 plots include studies that report significant effect estimates (29 studies) and studies that report non-significant effect estimates (4 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[1] are not likely to reflect the full ranges of ambient $PM_{2.5}$ concentrations that contributed to reported associations.[2]

Some of the key epidemiologic studies assessed in the 2019 ISA also provide city-specific study mean concentrations and city-specific health events, but this information was not available in studies evaluated in the draft ISA Supplement. $PM_{2.5}$ exposure estimates corresponding to the 10^{th} and 25^{th} percentiles of those events were calculated 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 are presented in Figure 3-8 and Figure 3-9 and the cumulative percent values closest to the associated 10^{th} and 25^{th} percentile values are shown in Table B-1.[3] 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] The following studies do not have an overlap between the years of $PM_{2.5}$ air quality data and the years during which health effects are reported: Miller et al., 2007 ; Hart et al., 2011 ; Thurston et al., 2013; Weichenthal et al., 2014; Pope et al., 2015b ; Villeneuve et al., 2015; Turner et al., 2016; Weichenthal et al., 2016a; Pinault et al., 2017; Parker et al., 2018; and Pope et al., 2019.

[2] 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.

[3] 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 3-8 and Figure 3-9 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 3-10 and Figure 3-11 focus on multicity/multistate studies in the U.S. and Canada, that are part of the evidence supporting "causal" or "likely to be causal" determinations in the 2019 ISA and that use hybrid modeling methods to estimate $PM_{2.5}$ exposures, as well as studies assessed in the draft ISA Supplement. In addition, as detailed in section 3.2.3.2.1, for studies included in Figure 3-10 and Figure 3-11 we also consider the approach used to estimate $PM_{2.5}$ concentrations and the approach used to validate hybrid model predictions when determining those studies that we identify as key epidemiologic studies. Such studies are identified as those that use hybrid modeling approaches for which recent methods and models were used (e.g., recent versions and configurations of the air quality models); studies that are fused with $PM_{2.5}$ data from national monitoring networks (i.e., FRM/FEM data); and studies that reported a thorough model performance evaluation for core years of the study.[4]

Figure 3-10 and Figure 3-11 present 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, along with other percentiles, are illustrated in Figure B-1 and Figure B-2 (Jenkins, 2019a, Jenkins, 2019b). The study authors for Di et al. (2017b) additionally provided information on population weighted percentile values corresponding to long-term $PM_{2.5}$ exposure (Chan, 2019). These are presented in Table B-2. For other studies included in Figure 3-10 and 3-11 [Bai et al., 2019, Erickson et al., 2019, Kloog et al. (2012), Kloog et al. (2014), Shi et al. (2016), U.S. EPA, 2021, and Wang et al. (2017)], 25^{th} percentiles of exposure estimates were derived from study manuscripts of air quality descriptive statistics and can be found in Table B-3.

[4] The following studies do not meet these criteria: Bravo et al., 2017, Crouse et al., 2015; Puett et al., 2009, Puett et al., 2011, Hystad et al., 2012; Hystad et al., 2013, Hayes et al., 2020; Elliott et al., 2020; Lefler et al., 2019;; Pappin et al., 2019; Cakmak et al., 2018; Fisher et al., 2019; Sun et al., 2019; McClure et al., 2017; Loop et al., 2018 ; and Honda et al., 2017.

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.

Table B-2. Population weighted percentiles of annual $PM_{2.5}$ concentrations by zip code corresponding to long-term exposure estimates in Di et al., 2017b.

Percentile	Population Weighted $PM_{2.5}$ ($\mu g/m^3$)
0.0	0.0
5.0	7.1
10.0	7.9
15.0	8.6
20.0	9.1
25.0	9.5
30.0	9.9
35.0	10.3
40.0	10.6
45.0	11.0
50.0	11.4
55.0	11.7
60.0	12.1
65.0	12.5
70.0	12.9
75.0	13.4
80.0	13.9
85.0	14.4
90.0	15.1
95.0	16.1
100.0	32.6

Percentiles of $PM_{2.5}$ 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 various percentiles of deaths at the zip-county level in Di et al., 2017a.

Table B-3. $PM_{2.5}$ 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
Bai et al. (2019)		7.9
Christidis et al. (2019)		4.3
Shin et al. (2019)		8

B.4 DETAILS OF KEY EPIDEMIOLOGIC STUDIES, INCLUDING STUDY DESIGN, EXPOSURE METRIC, AND STATISTICAL ANALYSIS

Table B-4 below summarizes additional details related to the designs of the U.S. and Canadian epidemiologic studies included in Figure 3-3 to 3-6, and Figure 3-8 to Figure 3-11, as well as studies included in the risk assessment (Table 3-13).

Table B-4. Study characteristics from key studies.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Baxter et al., 2017	ST	All-cause mortality	77 US Cities	Time Series study (NCHS)	EPA's National and State Local Ambient Monitoring Stations providing integrated daily measurements and operated more than 6 months or had more than 30 observations (2001-2005) considered. Monitors representing the general population exposure in the cities were selected. For this correlation was assessed between each pair of monitors within the county and the ones uncorrelated (coefficient<0.8 with majority of other monitors) were excluded. Once appropriate valid monitors were identified the summary measure of $PM_{2.5}$ concentration over the county was calculated. 2-day moving average (lag 0-1 days) of $PM_{2.5}$ conc included in the model.	Poisson regression model and meta-regression In stage 1, ran single city Poisson time-series models; adjusted for temperature and dew point temperature, including variables for previous day temperature, temporal trends, and trends by age. In stage 2, meta-regression with cluster analysis (5 clusters) based on characteristics of residential infiltration.	Average daily $PM_{2.5}$ values were calculated for each city. First, a global mean and variance were created within each city for the entire time period. Using the valid monitor measurements. Next, all values were standardized and average $PM_{2.5}$ within a given day in each city was calculated. Finally, the standardized daily value was reversed to calculate average daily $PM_{2.5}$ for each city.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Bell et al., 2008	ST	CVD HA Age 65+	202 US Counties with populations≥ 200,000	Time Series study (MEDICARE enrollees)	$PM_{2.5}$ concentrations obtained from EPA monitors providing data daily or every 3 days for the period 1999-2005. Used 10% trimmed mean to calculate daily average across monitors after correction for yearly monitor averages (to protect against outliers as applied in Dominici et al. 2006). Used lag0 $PM_{2.5}$ in the model.	2-stage Bayesian hierarchical model In stage 1, adjusted for temperature and dew point temperature, including variables for previous day's conditions, day-of-the-week, temporal trends, and differential temporal trends by age. In stage 2, county-specific estimates were combined, accounting for their statistical uncertainty.	Average daily $PM_{2.5}$ concentrations for each county used to calculate overall mean for the study area and duration.

Bell et al., 2014	ST	CVD, Asthma, and COPD HA Age 65+	4 Counties in MA and CT	Time-series study (MEDICARE enrollees)	$PM_{2.5}$ Teflon filter samples (measuring $PM_{2.5}$ total mass) obtained from CT and MA DEP for the period of 2000-2004. Used data from five monitoring locations (providing daily or every third day data) within four county regions. Assigned daily $PM_{2.5}$ concentration from a single monitor to three counties. For Fairfield County with two monitors: daily $PM_{2.5}$ concentration was calculated by using population-weighted averaging of census tract $PM_{2.5}$ concentrations. First, each census tract in the Fairfield county (209 tracts in total) was assigned the $PM_{2.5}$ exposure of the nearest monitor. Then, $PM_{2.5}$ exposures for all tracts were averaged and weighted by each tract's 2000 U.S. census population to calculate a county-level exposure for the Fairfield county.	Log-linear Poisson regression analysis Adjusted for temperature and dew point temperature, including previous day's temperature and dew point temperature, day-of-the-week temporal trends, and region.	Daily $PM_{2.5}$ concentrations for all four counties (three with single monitor and one with two monitors that used population weighted approach) over the period of 2000-2004 were used to calculate the overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					Explored various lags and presented lag0 $PM_{2.5}$ model.		
Bell et al., 2015	ST	HF HA 65+	213 U.S. Counties	Time-series study (MEDICARE enrollees)	Daily monitored $PM_{2.5}$ data from the US EPA AQS monitors for the period of 1999-2010. On average, county-level $PM_{2.5}$ data was available for 56.5% of study days (range: 7.8%-99.9%; no imputation done for missing data). For each county, daily $PM_{2.5}$ measurement was calculated by averaging the $PM_{2.5}$ values from all monitors within a county in a given day. Explored various lags and presented lag0 $PM_{2.5}$ model.	2-stage Bayesian hierarchical model The stage 1 model included county-specific model adjusted for weather (temperature, dew point, previous days' temperature, and dew point), day-of-the-week, and temporal trends. In stage 2 county-specific effect estimates were pulled together to present overall association.	Daily $PM_{2.5}$ concentrations for 213 counties over the period of 1999-2010 were used to calculate region-specific mean $PM_{2.5}$, and overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Bravo et al., 2017	ST	CVD HA Age 65+	418 U.S. Counties	Time-series study (MEDICARE enrollees)	Daily (24-hr) monitored $PM_{2.5}$ data from the US EPA AQS monitors (NAMS/SLAMS) obtained for the period of 2002-2006. Approximately 80% of $PM_{2.5}$ monitors recorded observation once every 3 days. For each county (>=50K population), daily (24-hr) $PM_{2.5}$ concentration was calculated by averaging multiple monitor measurements for the same day. Explored various lags and distributed lags of $PM_{2.5}$ exposure.	2-stage Bayesian hierarchical model The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of $PM_{2.5}$ conc simultaneously in the county-specific model. The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.	Daily $PM_{2.5}$ concentrations for 418 counties over the period of 2002-2006 were used to calculate overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Bravo et al., 2017	ST	CVD HA Age 65+	708 U.S. Counties	Time-series study (MEDICARE enrollees)	Daily $PM_{2.5}$ concentrations were estimated at census tract centroids using the downscaler method (input from the US EPA AQS NAMS/SLAMS monitoring data, and gridded 12x12 km CMAQ) for the period of 2002-2006. County-level daily $PM_{2.5}$ exposures were calculated from a pop-weighted averages of $PM_{2.5}$ concentrations predicted at census tract within each county using 2000 U.S. Census data. CMAQds was generated for all days in the study period 2002-2006. CMAQds-subset was calculated by taking population-weighted county level exposures only for counties and days with monitoring data (n=418 counties. Explored various lags and distributed lags of $PM_{2.5}$ exposure.	2-stage Bayesian hierarchical model The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of $PM_{2.5}$ conc simultaneously in the county-specific model. The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.	24-hr average $PM_{2.5}$ concentrations for 708 counties over the period of 2002-2006 were used to calculate overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Burnett and Goldberg, 2003	ST	All-cause mortality	8 Canadian Cities	Time-series study	$PM_{2.5}$ data obtained from dichotomous sampler with Teflon filters operating on 6-day schedule for the period of 1986-1996. Each city had one sampler and two cities have two samplers. If two samplers then data was averaged between the samplers and assigned to the city. Lag 1 explored.	Generalized additive model (GAM) analysis to generate pooled estimate of air pollution effect among the eight cities. The model adjusted for day-of-the-week, temporal trends, and weather variables (daily average temperature, daily average relative humidity, and barometric pressure lagged 0 and 1 days).	Daily $PM_{2.5}$ concentrations (day before the death) for 8 Canadian cities over the period of 1986-1996 were averaged to get overall mean for the study area and period
Burnett et al., 2004	ST	All-cause mortality	12 Canadian Cities	Time-series study (data from Statistics Canada)	Monitoring data available for 12 cities from the Statistics Canada for the period of 1981-1999. $PM_{2.5}$ data available every 6^{th}-day sampling schedule. Daily $PM_{2.5}$ concentrations were calculated for each city by averaging data over all monitors with each city. Explore various lags and moving average and presented data for lag 1 for $PM_{2.5}$.	Random-effects regression model. Adjusted for temporal trends in mortality and effects of weather using humidex index at lag 0 and lag 1 (a measure of combined effect of temperature and humidity)	Daily $PM_{2.5}$ concentrations for all 12 cities over the period of 1981-1999 were used along with population information to calculate an overall population weighted $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Cakmak et al., 2018	LT	Non-accidental, CVD, respiratory and lung cancer mortality	Canada Nationwide	Cohort study (CanCHEC)	$PM_{2.5}$ estimates obtained from median satellite-derived concentrations for the period of 1998 – 2011. The concentration was determined at 10 km2 resolution as detailed in (van Donkelaar, 2010). Changes in $PM_{2.5}$ between 1998 and 2006 was inferred using satellite instruments, MISR and SeaWiFS (Boys, 2014). Annual estimates of $PM_{2.5}$ concentration was assigned to participants based on postal code of residence and was used to calculate 7-year moving average (at least 4 out of 7 years of data is available) $PM_{2.5}$ concentration for each year of follow-up in the study.	Cox proportional hazards models to estimate the relationship between long-term exposure and date of death accounting for residential mobility. Model adjusted for individual-level covariates (aboriginal ancestry, minority status, marital status, education, immigrant status and income)	The 7-year moving averages for study participants were then used to calculate overall mean $PM_{2.5}$ concentration (all and by geographic zones)

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Chen et al., 2020	LT	CVD mortality	Ontario, Canada	Cohort study (ONPHEC)	$PM_{2.5}$ concentration estimated from multiple satellite retrievals of AOD combined with geophysical relationship between AOD and $PM_{2.5}$ simulated by GEOS-Chem, which were then calibrated with surface measurements by GWR as detailed in (van Donkelaar, 2019). Annual estimates of exposure to $PM_{2.5}$ and the composition for each participant was estimated by interpolating the annual mean concentrations of $PM_{2.5}$ and the corresponding proportion of $PM_{2.5}$ attributed to the seven major components to the centroid of their residential postal code for that year, thereby accounting for residential mobility.	Component-adjusted approach that jointly estimated the health impacts of $PM_{2.5}$ and its major components while allowing for a potential nonlinear $PM_{2.5}$−outcome relationship. Compared this approach with three traditional approaches using Cox Hazard models. Adjusted for individual-level covariates, four time-varying variables for neighborhood-level SES, area-level indicators.	Annual $PM_{2.5}$ concentrations in the Ontario region were then used to calculate overall mean $PM_{2.5}$ concentration for the study location and period.

Christidis et al., 2019	LT	Non-accidental mortality	Canada Nationwide	Cohort study (mCHHS)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Spatial variation from modeled surface used with simulate $PM_{2.5}$ and constrained with local ground-based monitors to estimate $PM_{2.5}$ concentrations through 2015 (Meng, 2019). Linked postal codes to $PM_{2.5}$ concentrations using points of latitude and longitude. When multiple points of latitude and longitude was available for a single urban postal code, equal weighting of the multiple air pollutant values was used to provide a singular value. In rural communities, population-weighted average of the values associated with duplicate postal codes was used. Used population-weighing to average multiple values to create inputs for partial postal codes (2 to 5 digit). For each individual and year of follow-up, $PM_{2.5}$	Cox proportional hazard models to assess the relationship between $PM_{2.5}$ exposure and non-accidental death in low-exposure environment. C-R relationship observed using Shape constrained health impact function (SCHIF Model) Adjusted for socio-economic, behavioral, and time-varying contextual covariates	3-year moving average $PM_{2.5}$ concentrations were for the participants used to calculate overall mean $PM_{2.5}$ concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					estimates was calculated as 3-year moving average with one-year lag.		

Crouse et al., 2012	LT	All-cause mortality	11 Canadian Cities	National Cohort study (Subset of Canadian census mortality follow-up study; 43%; non-immigrant population)	Monitor data from ground-based stations available for 11 cities for the 15-yr period including the 5-yr prior to baseline and 10-yr of follow-up (1987-2001) from Statistics Canada. $PM_{2.5}$ data available every 6th-day sampling schedule. To address missing monthly $PM_{2.5}$ data for some stations, data from all stations within 6-km of each other were pooled to calculate monthly, seasonal, annual and five-yr (1987-1991, 1992-1996, 1997-2001) means at each monitored location. Mean annual concentration (averaged over 1987-2001) from ground-based monitors was then assigned to the cohort member based on the 11 census divisions of their residence. A second set of exposure (10x10 km) was created using estimates of $PM_{2.5}$ from remote sensing during period 2001-2006 to calculate 6-yr average. The mean concentration of $PM_{2.5}$ within boundaries of each enumeration area	2 different modelling approach. Approach 1: Cox proportional hazards model, and Approach 2: nested, spatial random-effects Cox model with spatial clusters. Models adjusted for individual-level covariates, urban/rural indicator, and ecological covariates (% unemployed, % without high school diploma, lowest income quintile, and rural/urban indicator).	Annual$PM_{2.5}$ concentrations for the study participants were used to calculate overall mean $PM_{2.5}$ for the study population and duration.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					was calculated by overlaying PM surface over the surface of enumeration area across country. Satellite derived $PM_{2.5}$ estimate was then assigned to participants based on their enumeration area of residence in 1991.		

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Crouse et al., 2019	LT	Non-accidental, CVD, respiratory mortality, and lung cancer	Canada Nationwide	Cohort study (CanCHEC)	$PM_{2.5}$ concentrations derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Spatial variation from modeled surface used with simulate $PM_{2.5}$ and constrained with local ground-based monitors to estimate $PM_{2.5}$ concentrations through 2015 as detailed in (Meng, 2019). Linked postal codes to $PM_{2.5}$ concentrations from grid cells. Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate moving average at various temporal and spatial scales based on the location and year of follow-up.	Cox Hazard model to assess the relationship between $PM_{2.5}$ exposure at different temporal and spatial scales. Adjusted for individual-level variables (aboriginal identity, visible minority status, marital status, highest level of education, employment status, and household income adequacy quintiles)	The average annual $PM_{2.5}$ concentrations were used to calculate overall mean $PM_{2.5}$ concentration for the study period at various temporal and spatial scales.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Dai et al., 2014	ST	All-cause, CVD, and Respiratory mortality	75 U.S. Cities (with available daily mortality data and $PM_{2.5}$ data for at least 400 days between 2000 and 2006)	Time-series study (NCHS)	Monitored data obtained from US EPA AQS for the period of 2000-2006. Daily $PM_{2.5}$ concentrations from each monitor assigned to corresponding city. For cities with more than one sampling site, concentration data were averaged across all monitors within the city. Used average of 2-day lag (lag 01) $PM_{2.5}$.	Two stage: Stage 1. City-specific season-stratified time-series analysis using Poisson regression in GAM Model adjusted for 24-hr average temperature from closest weather station to the city center at lag0 and lag1, temporal trends, and day-of-the-week. Stage 2. Multivariate random effects meta-analysis to combined 300 (i.e. 75 cities * 4 seasons) effect estimates to obtain overall association.	Daily $PM_{2.5}$ concentrations for all 75 cities over the period of 2000-2006 were used to calculate an overall mean $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
deSouza et al., 2021	ST	First CVD HA	US Nationwide	Time-stratified case–crossover design (MEDICAID)	$PM_{2.5}$ concentration were derived for 1 km2 grid cells in the continental United States by integrating remote sensing, outputs from a chemical transport model, and other variables such as meteorological and land-use variables (Di et al. 2019); from an ensemble-based model that integrated multiple machine learning algorithms for the period of 2000-2012. Daily $PM_{2.5}$ estimates of all grid cells averaged at-Zip code were assigned to study participants based on the zip code of residence. Used lag01 average exposure in the model.	Conditional logistic regression models to estimate the associations between short-term exposure to $PM_{2.5}$ and CVD hospitalization rates. Adjusted for individual-level covariates, air and dew-point temperature.	Daily $PM_{2.5}$ concentration from case days were then used to calculate overall case day mean $PM_{2.5}$ concentration for the study location and period.

Di et al., 2017b	LT	All-cause mortality 65+	US Nationwide	Cohort (MEDICARE enrollees)	Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of $PM_{2.5}$. The neural network was fit with monitored $PM_{2.5}$ data and daily $PM_{2.5}$ concentrations were predicted for nationwide grids that were 1x1 km. While not explicitly detailed in the study, it was assumed that the 1 km x 1 km grid cells were averaged up to the zip code spatial resolution. For each calendar year during which a person was at risk of death the annual average $PM_{2.5}$ concentration was assigned according to the ZIP Code of the person's residence. As part of a sensitivity analysis, monitored $PM_{2.5}$ data was matched with each person in the study within a distance of 50 km of the nearest monitoring site.	Two-pollutant Cox proportional hazards model with generalized estimating equation to account for correlation between ZIP codes. Accounted for individual variables, (sex, race, Medicaid eligibility, and average age at study entry), zip code-level variables (% Hispanic, % Black, median household income, median value of housing, % > 65 living below poverty level, % > 65 with less than high school education, % of owner-occupied housing units, and population density), county-level variables (county-level BMI and % ever smokers), hospital service area-level variables (% low-density lipoprotein level measured, % glycated hemoglobin level measured, and % >1 ambulatory visits), 32 km2 gridded weather and 1 km2 gridded pollution variables (annual average $PM_{2.5}$ concentration, annual average temperature, and annual average humidity), monitor level air pollution variables ($PM_{2.5}$ monitored data), and a regional dummy variable.	Average $PM_{2.5}$ concentrations for all Zip Codes (entire US or ZIP codes with study participants only) from 2000 to 2012 were used to calculate overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Di et al., 2017b (< 12 ug/m3)					Analysis restricted to persons-years with $PM_{2.5}$ exposures lower than 12 ug/m^3		

Di et al., 2017a	ST	All-cause mortality 65+	US Nationwide	Case-crossover study (MEDICARE enrollees)	Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of $PM_{2.5}$. The neural network was fit with monitored $PM_{2.5}$ data and daily $PM_{2.5}$ concentrations were predicted for nationwide grids that were 1x1 km. For each case day (date of death) and its control days, the 24-hour $PM_{2.5}$ concentrations were assigned based on zip code of residence of the individual. As part of a sensitivity analysis, monitored $PM_{2.5}$ data was matched with each person in the study within a distance of 50 km of the nearest monitoring site, and cross-validation was performed between predicted and monitored concentrations. Used average of 2-day lag (lag 01) $PM_{2.5}$.	Conditional logistic regression. "Case Day" defined as death. For the same person, compared daily air pollution exposure on the case day vs. daily air pollution exposure on "control days." Control days were chosen (1) on the same day of the week as the case day to control for potential confounding effect by day of week; (2) before and after the case day to control for time trend; and (3) only in the same month as the case day to control for seasonal and sub-seasonal patterns. Individual-level covariates and zip code-level covariates that did not vary day to day (e.g., age, sex, race/ethnicity, SES, smoking, and other behavioral risk factors) were not considered to be confounders as they remain constant when comparing case days vs control days. The regression model adjusted for air and dew point temperature.	The case and control days $PM_{2.5}$ concentrations for study participants were averaged to get mean $PM_{2.5}$ concentration for the study area and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Dominici et al., 2006	ST	HF and COPD HA 65+	204 Urban U.S. counties	Time-series study (MEDICARE enrollees)	Monitored $PM_{2.5}$ concentrations available from US EPA AQS for the period of 1999-2002. Of the 204 counties (>200,000 population), 90 counties had daily $PM_{2.5}$ data across the study period and the remaining counties had $PM_{2.5}$ data collected once every 3 days for at least 1 full year. To protect against consequences of outliers, used 10% trimmed mean to calculate daily average across monitors after correction for yearly averages for each monitor. Various lags (lag 0, 1, 2 days) and distributed lags assessed and presented.	2-stage Bayesian hierarchical models to estimate county-specific, region-specific, and national-average associations. Stage 1 model included single lag and distributed lag over-dispersed Poisson regression models to estimate county-specific risk. Models adjusted for temperature and dew point on the same day and the 3 previous days, calendar time to control for seasonality and other time-varying influences, daily numbers of individuals at risk, and day-of-the-week. In Stage 2, to produce a national average estimate, Bayesian hierarchical models were used to combine RRs across counties and accounting for within-county statistical error and for between-county variability or heterogeneity. To produce regional estimates. The Stage 2 hierarchical models described above was used for 7 regions separately.	Daily $PM_{2.5}$ concentrations for all 204 US counties over the period of 1999-2002 were used to calculate an overall mean $PM_{2.5}$ concentration for the study regions and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Dominici et al., 2019	LT	Non-accidental mortality	Nationwide	Cohort study (MEDICARE)	Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data was used to predict daily concentrations of $PM_{2.5}$ (Di et al. 2017). Daily $PM_{2.5}$ concentrations were predicted for nationwide grids at1 km2 resolution for the period 2000–2012.	Survival analyses using the Andersen-Gill method, a variant of the traditional Cox proportional hazards model C-R relationship assessed fitting a log-linear model with thin-plate splines. Adjusted for individual-level covariates, county-and ZIP code-level variables, meteorological variables, and other area-level variables.	Daily $PM_{2.5}$ concentrations over all ZIP codes were used to calculate overall mean $PM_{2.5}$ for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Erickson et al., 2020	LT	Non-accidental, CVD, respiratory mortality, and lung cancer	Canada Nationwide	Cohort study (CanCHEC)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Linked postal codes to $PM_{2.5}$ concentrations from grid cells. Annual $PM_{2.5}$ estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year moving average based on the location and year of follow-up.	Cox proportional hazards models to examine the associations between ambient $PM_{2.5}$ exposure and non-accidental and cause-specific mortality. Adjusted for individual-level and contextual-level covariates.	The average $PM_{2.5}$ concentrations were used to calculate overall mean $PM_{2.5}$ concentration for the study period by immigrant status and duration in Canada.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Eum et al., 2018	LT	All-cause mortality	Geographic regions: "East" of the Mississippi River, "Center" between the Mississippi River and the Sierra Nevada mountain range, and "West" of the Sierra Nevada mountain range	Cohort study (MEDICARE)	$PM_{2.5}$ concentration obtained from US EPA's AQS for the period of 2000-2012. Monitoring sites with daily measurements for at least 8 calendar years with each year having 9+ months and with 4+ daily measurements included. 798 sites then were used to calculate long-term concentration (yearly moving average with 350+ days of valid data) using Greven et al. Annual average assigned to individuals that lived in ZIP codes with centroids within 6 miles of a valid monitor.	Age-stratified log-linear model including offset terms for the size of the population as a base model. Also included the temporal and spatiotemporal components. Ran base model using data for entire 13-year study period (2000-2012) and for shorter periods ranging between 3 and 12 years and compared MRRs to assess temporal confounding. In addition to base model, also assessed temporal confounding using three approaches (decomposition-based, residual-based, and spline models) Adjusted for individual-covariates, as well as county-level behavioral covariates, % of non-whites, smoking status, comorbidities, access to health care, income, and BMI.	Annual average $PM_{2.5}$ concentrations were used to calculate overall mean concentration for the study location (all and by study region) and study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Fisher et al., 2019	ST	Stroke (Self-reported stroke adjudicated by physician medical record)	Nationwide	Time-stratified case-crossover study (HPFS)	Validated national-scale, log-normal ordinary kriging model for $PM_{2.5}$ were used to estimate daily $PM_{2.5}$ concentration. US EPA's AQS data used to calculate monitor specific daily averages (monitors >=18 hours measures). These inputs were then used to produced kriged surfaces of daily mean $PM_{2.5}$ concentrations at the geocoded residential addresses of all HPFS participants for the period 1999-2010. Lag periods up to 3 days prior to the stroke event and a 4-day average used in model.	Conditional logistic regression models Adjusted for mean daily temperature, and stratified models to examine effect modification by individual-level characteristics.	Daily $PM_{2.5}$ concentration on the case day were used to calculate overall case day $PM_{2.5}$ mean for the study period.

Franklin et al., 2007	ST	All-cause, CVD, and Respiratory mortality	27 U.S. communities (with $PM_{2.5}$ monitoring and daily mortality data for at least 2 years of 6-year study period 1997-2000)	Case-crossover study (NCHS)	Monitored daily $PM_{2.5}$ concentrations available from US EPA AQS (NAMS/SLAMS) for the period of 1997-2000. Data for Boston area available from Harvard University. To determine which monitors in the county are representative of exposure for a general population in the county, correlation was assessed between monitor pairs and excluded the monitors with $r<0.8$ for 2 or more monitor pairs. Once appropriate monitors were identified then a summary measure of $PM_{2.5}$ conc for the county was calculated using alternate averaging method described in Schwartz 2000 to account for data availability variation (daily vs 3-6 days for each monitors in the county) and calculate daily average $PM_{2.5}$ conc for each of the 27 counties and corresponding communities.	2-stage time-stratified analysis: 1) Conditional logistic regression analysis to generate community specific estimates; 2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate. Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as "deaths" and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1, while effect modification of community-specific characteristics including geographic location, annual $PM_{2.5}$ concentration > 15 ug/m^3 and central AC prevalence was used in stage 2.	Daily $PM_{2.5}$ concentrations for all 27 US communities over the period of 1997-2000 were used to calculate an overall mean $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					Calculated and presented various lags and averages for $PM_{2.5}$.		

Franklin et al., 2008	ST	All-cause, CVD, and Respiratory mortality	25 U.S communities (with $PM_{2.5}$ monitoring and daily mortality data for at least 4 years of 6-year period between 2000-2005)	Case-crossover study (NCHS)	Monitored daily $PM_{2.5}$ concentrations available from US EPA AQS (NAMS/SLAMS) for the period of 2000-2005. Data for Boston area available from Harvard University. To determine which monitors in the county are representative of exposure for a general population in the county, correlation was assessed between monitor pairs and excluded the monitors with $r<0.8$ for 2 or more monitor pairs. Once appropriate monitors were identified then a summary measure of $PM_{2.5}$ conc for the county was calculated using alternate averaging method described in Schwartz 2000 to account for data availability variation (daily vs 3-6 days for each monitors in the county) and calculate daily average $PM_{2.5}$ conc for each of the 27 counties and corresponding communities.	2-stage time-stratified analysis: 1) Conditional logistic regression analysis to generate community specific estimates; 2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate. Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as "deaths" and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1.	Daily $PM_{2.5}$ concentrations for all 25 US communities over the period of 2000-2005 were used to calculate an overall mean $PM_{2.5}$ concentration for the study location and period (overall and by seasons).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					Calculated and presented various lags and averages for $PM_{2.5}$.		
Gharibvand et al., 2016	LT	Lung cancer incidence	US Nationwide	Cohort study (AHSMOG-2 study)	Monitor data obtained from US EPA AQS for the period of 2000-2001 (2-year prior to start of the study). Using monitored $PM_{2.5}$ data, inverse distance weighted interpolations methods, monthly pollution surfaces for $PM_{2.5}$ were created for the US. Monthly exposure averages were based on daily $PM_{2.5}$ measurements. Only months with at least 75% valid data were included in the exposure estimation. Participants were assigned monthly exposure based on their baseline residential address.	Cox proportional hazards model Covariates included sex, race, smoking status, years since participant quit smoking, average number of cigarettes per day during all smoking years, and education level. Additional covariates included calendar time, alcohol consumption, family income, BMI, physical activity, and marital status. 3 variables identified a priori as either as confounders or effect modifiers: hours/day spent outdoors, years of pre-study residence length at enrollment address, and moving distance from enrollment address during follow-up.	Monthly $PM_{2.5}$ concentrations for study participants were used to calculate overall 2-yr mean $PM_{2.5}$ for the study period 2000-2001.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Hart et al., 2015 (monitored)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	Monitored data obtained from US EPA AQS for the period 1999-2006. Monthly average $PM_{2.5}$ concentration calculated from the nearest monitoring location for all addresses. The monthly data was again averaged to get the previous 12-month moving average at each residential address prior to mortality. Nearest monitor exposures were validated against personal exposures to $PM_{2.5}$ of ambient origin.	Cox proportional hazards model. Information on potential confounders was available every two years (4 years for diet information) and each woman was assigned updated covariate values for each questionnaire cycle. Confounders examined include age, race, region, season, physical activity, BMI, hypercholesterolemia, family history of MI, smoking history, Current smoking status, diet, SES (education level, occupation of	Monthly $PM_{2.5}$ concentrations at residence locations during the follow-up period of 2000 to 2012 were averaged to calculate overall mean $PM_{2.5}$ exposure for the participants included in the study.

Hart et al., 2015 (modeled)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	Spatio-temporal models (developed using monitored data from US EPA AQS, the IMPROVE network, and also included meteorological and GIS-derived covariates, such as urban land use within 1 km, elevation, tract- and county-level population density, distance to the nearest road for road classes A1-A3 and point-source emission density within 7.5 km) was used to estimate monthly $PM_{2.5}$ exposures at each geocoded address. The monthly data was again averaged to get the previous 12-month moving average prior to mortality for each residential address. Modeled exposures were validated against personal exposures to $PM_{2.5}$ of ambient origin. Previous 12-month moving average of exposure either from nearest monitor or spatio-temporal models were	both of the nurses' parents when she was 16, marital status, and husband's education if applicable). Also adjusted for area-level SES (census tract level median income and house value), and long-term temporal trends. Risk set regression calibration for time-varying exposures was used to correct for bias due to exposure measurement error in the hazard ratios of all-cause mortality using the personal exposure validation data.	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					assigned to study participants.		
Hayes et al., 2020	LT	CVD mortality	6 US states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and 2 urban areas (Atlanta, GA, and Detroit, MI,)	Cohort study (NIH-AARP Diet and Health study)	Modelled (Hybrid land use regression geostatistical model developed by Kim et al. 2017) for the period of 1980-2010. Mean annual estimates of $PM_{2.5}$ for each census tract in the US from spatio-temporal model were used till 1998. For period 1999-2010, monitored US EPA monitor and IMPROVE network was used to derive annual average estimates. Annual average $PM_{2.5}$ concentrations assigned at census tract level lagged by 1 year in time-dependent manner. Annual $PM_{2.5}$ exposure analyzed as continuous and categorical <8, 8-<12, 12-<20, and 20+ ug/m3 variables.	Cox regression modelling with time-dependent covariates. Adjusted for individual-level variables (age, race/ethnicity, education, marital status, BMI, alcohol, and smoking status), as well as census tract variables.	Annual $PM_{2.5}$ concentrations of the study participants for the year 2000 was used to calculate overall mean $PM_{2.5}$ concentration for the period 2000.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Ito et al., 2013[5]	ST	All-cause mortality	150 U.S. cities	Time-series study	24-hr average $PM_{2.5}$ mass data in a given city, and when data from multiple monitors were available in a given city, computed the average of the daily values after standardizing each site's data using the mean and standard deviation of the sites data. Pollutant concentration is expressed in the model as a deviation from the monthly mean to reduce the influence of the seasonal cycles of the pollutants on the overall associations and help focus on the short-term associations.	Poisson regression analysis First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach Adjusted for temporal trends (annual cycles and influenza epidemics), immediate and delayed temperature, and day-of-week pattern, for entire years (2001-2006) and for warm (April-September) and cold (October-March) seasons. In second stage, assessed effect modification using land-use variables and average air pollution levels.	

[5] This study is not referenced individually in the ISA, but is study 3 of the National Particle Component Toxicity (NPACT) Initiative published in HEI (Lippmann et al., 2013).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Jerrett et al., 2016	LT	IHD mortality 30+	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Multiple exposure estimation approaches evaluated within the study – risk assessment uses results based on an ensemble approach that incorporates chemical transport modeling, land use data, satellite data, and data from ground-based monitors	Cox proportional hazards regression Covariates included current and former smoking status as well as smoking duration, amount, age started, second hand cigarette smoke (hours/day exposed), exposure to $PM_{2.5}$ in the workplace for each of the subject's major lifetime occupation, self-reported exposure to dust/fumes at work, marital status, level of education, BMI, alcohol consumption, dietary vegetable/fruit/fiber index, dietary fat index, missing nutrition information. Ecologic characteristics included median household income, percentage of people with < 125% of poverty-level income, percentage of persons > 16 who are unemployed, percentage of adults with < 12^{th} grade education, and percentage of population who were Black or Hispanic.	

Kioumourtzoglou et al., 2016	LT	All-cause mortality 65+	207 U.S. cities	Open Cohort study (MEDICARE enrollees)	Monitored data available from US EPA AQS for the period of 2000-2010. City-specific annual and 2-year $PM_{2.5}$ averages was calculated using data from all available monitors in each city.	2-stage approach for modelling. In Stage 1, Cox proportional hazards model was fit for each city stratified by age, gender, race, and follow-up time in study. Control for slowly varying potential confounders (e.g., SES) and confounders that vary across subjects, city, and time. City-characteristics for: proportion of city population > 65, median household income, proportion in poverty, proportion of city families in poverty, proportion of white, black, and Asian residents, proportion of residents with/without high-school degrees and a college degree, and city-specific smoking and obesity rates. Population-weighted city averages were developed based on census data at the county level. Also included average annual temperature in the model. In stage 2, combined the city-specific estimates using a random effects meta-analysis to generate region-specific effects. Assessed effect modification by annual temperature levels, and population and city characteristics (greenness,	Annual $PM_{2.5}$ concentrations for 207 cities during the period of 2000 to 2010 were averaged to calculate overall mean $PM_{2.5}$ exposure for the study location (all and region specific) and study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
						poverty, racial composition, etc.).	
Klemm and Mason, 2003	ST	All-cause mortality	Harvard Six-City study reanalysis	Time-series study	24-hour $PM_{2.5}$ concentration obtained from Dichotomous samplers placed at the central residential monitoring sites in each of the six cities. Integrated 24-hour samples were collected daily for part of the study periods but were collected at least every other day until the late 1980s.	Generalized additive and Generalized linear models Model adjusted for temporal trends, day-of-the-week, weather (average daily temperature and average daily dew point temperature).	Daily $PM_{2.5}$ concentration of six cities over the period of 1979-1988 were used to calculate overall mean, median and percentiles of $PM_{2.5}$ exposure for the study location (all and by study center) and period.

Kloog et al., 2012	ST, LT	CVD HA Age 65+	New England Area with 6 U.S. States	Mixed study design (with time series and cohort components)	Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground $PM_{2.5}$ measurements. Incorporated land use regressions and meteorological variables (temperature, wind speed, visibility, elevation, distance to major road, percent of open space, point emissions and area emissions) for the period of 2000-2006. Model predicted daily $PM_{2.5}$ concentrations at a 10 x 10 km spatial resolution. The $PM_{2.5}$ concentration then was matched to ZIP codes based on spatial location and date. Short-term exposure: used the mean of $PM_{2.5}$ on the day of admission and day before admission. Long-term exposure: calculated as the mean exposure in each zip-code across the 7-year study period. Short term exposure was defined as the difference between the two-day average and the long-term average.	Equivalence between Poisson regression and the piecewise constant proportional hazard model to model the time to a hospital admission as a function of both long-term and short-term exposure simultaneously and enabling simultaneously examination of short term and long-term associations with hospital admissions (Hierarchical mixed Poisson regression model). The model adjusts for temperature, age, percent minorities, median income, and percent of people with no high school education.	Daily $PM_{2.5}$ concentration of all grids within the NE area for the acute (0 day lag) and chronic (365 day moving average) were used to calculate overall mean short- and long-term $PM_{2.5}$ exposure respectively, for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Kloog et al., 2014	ST	CVD and COPD HA Age 65+	7 U.S. Mid-Atlantic States and D.C.	Case-crossover design (MEDICARE enrollees)	Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground $PM_{2.5}$ measurements. Incorporated land use regression (elevation, distance to major roads, percent of open space, point emissions and area emissions) and meteorological variables (temperature, wind speed, relative humidity, and visibility) for the period of 2000-2006. Model used to predict daily $PM_{2.5}$ concentrations at a 10 x 10 km spatial resolution. Daily predicted $PM_{2.5}$ exposure estimates at grids were matched to zip codes. Average of 2-day lag (lag 0 and 1) $PM_{2.5}$ used.	Conditional logistic regression analysis Temperature with the same moving average as $PM_{2.5}$ was included in the model as a potential confounder. Study design samples only cases and compares each subject's exposure experience in a time period just before a case-defining event with the subject's exposure at other times, eliminating confounding (unmeasured or measured) that do not vary over time. Cases were matched on day of the week and defined the relevant exposure time window as the mean exposure of the day of and day before the patient's hospital admission. Effect modification: 1) assessed whether subject residence within 30 km of a monitor or farther modified the $PM_{2.5}$ association; 2) examined interaction between exposure and income level and gender.	2-day moving average of $PM_{2.5}$ concentration of all grids within the mid-Atlantic states were used to calculate overall mean (all area and rural/urban areas) $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Krall et al., 2013	ST	All-cause mortality	72 Urban U.S. Communities	Time-series study (NCHS)	Monitored data available from US EPA AQS for the period of 2000-2005. Excluded data from source-oriented monitors that may not be representative of typical population exposures. Daily community-level pollutant exposure as the arithmetic mean of daily monitor observations within the community. For communities with single monitor pollutant concentration represented concentrations recorded by that monitor. Used lag 1 $PM_{2.5}$ in model.	Log-linear Poisson Regression Model Model adjusted for temperature and previous day's temperature, long-term and seasonal trends, age, and day-of-the-week. Also included interaction term for pollutant concentration and seasons.	Daily $PM_{2.5}$ concentration of 72 US urban communities were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Krall et al., 2018	ST	ED Visits for CVD (CHF, Cardiac dysrhythmia, IHD, Stroke) or RD (asthma/wheeze, COPD, pneumonia, URI)	Multi-city (5 Metropolitan areas)	Time-series study (Electronic billing of ED visits)	$PM_{2.5}$ concentrations obtained from ambient monitoring stations located within each of the metropolitan areas were fused with Community Multi-Scale Air Quality model estimates (Friberg et al, 2016, 2017) to obtain population-weighted average estimates of the 24-hour average $PM_{2.5}$ concentrations.	Poisson time- series regression model accounting for over-dispersion (Peng et al. 2009; Krall et al. 2013) to calculate city specific associations. To calculate overall and posterior city-specific associations, applied Bayesian hierarchical models (Everson and Morris 2000). Adjusted for weekday, season, holidays, metrology, temporal trends.	Daily (24-hr) PM concentrations for specific cities were used to calculate overall $PM_{2.5}$ concentration by city for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Lavigne et al., 2018	ST	Non-accidental, CVD, and respiratory mortality	24 Canadian cities	Case-crossover study	Daily (24-hour) average $PM_{2.5}$ concentrations obtained from monitors in Canada's NAPS network and were used to estimate $PM_{2.5}$ concentrations for the period of 1998-2011. Exposure estimates were assigned to each study participant based on the monitoring station(s) located in participants' city of residence. If $PM_{2.5}$ measurements were available from multiple monitors in a single city, daily concentrations were averaged across monitors.	Conditional logistic regression analysis. Performed stratified analyses examining the relationship between $PM_{2.5}$ and mortality across tertiles of Oxidant capacity.	Daily $PM_{2.5}$ concentrations in 24 Canadian cities were used to calculate overall mean $PM_{2.5}$ concentration over the study location and period.

Lee et al., 2015	ST	All-cause, Cardiovascular, respiratory mortality	3 U.S. Southeast States	Case-crossover design (Dept. of Pub Health data)	Spatio-temporal model that used satellite AOD data to predict daily $PM_{2.5}$ at 1X1 km resolution for the period of 2007-2011. Daily $PM_{2.5}$ concentration at 1km grids were aggregated into the zip code level. For this, 1 km grid cells were matched to zip code area by assigning the centroid of each 1 km grid cell to the centroid of the closest zip code. Zip code areas that contained one or more 1 km grid cells were given the averaged $PM_{2.5}$ and zip codes that were smaller than 1 km2 were given the predictions from the closest grid cell. Finally, $PM_{2.5}$ concentrations from zip codes were assigned to the study participants based on their residence zip code and for specific days. For sensitivity: Daily monitored $PM_{2.5}$ concentrations from the nearest EPA and IMPROVE monitors from	Conditional logistic regression Model adjusted for temperature and day of the week Also ran stratified analysis by age, sex, race, education, and primary cause of death. Analysis also restricted for zip codes where annual average of $PM_{2.5}$ <12 or daily average <35 separately. Sensitivity analysis: potential non-linear relationship between temp and mortality modelled using natural spline to the temperature term.	Daily $PM_{2.5}$ concentrations for ZIP code from 2007-2011 were averaged to get overall mean $PM_{2.5}$ (all states and by state)

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					resident zip code (no distance limit) were identified and assigned to individuals. Used lag0 and lag1 in model.		

Lefler et al., 2019	LT	All-cause mortality, Cardiopulmonary mortality	Nationwide	Cohort study (NHIS)	Annual average $PM_{2.5}$ was modeled using regulatory monitors and land use data as described in (Kim, 2018). $PM_{2.5}$ exposure prior to 1999 were estimated using PM10 data. Estimates for each pollutant-year through 2015 were generated at the census-block level using year-2010 Census block centroids. Tract-level estimates for year 2000 Census tracts and year-2010 Census tracts were estimated by mapping year-2010 Census blocks to census tracts and then calculating a population-weighted average of the census blocks within a census tract. $PM_{2.5}$ exposure estimates were assigned to home census tracts as either 2-year (i.e., cohort year and previous year) or 5-year (i.e., cohort year and previous 4 years) average $PM_{2.5}$ concentrations, 17-year average $PM_{2.5}$ concentrations (1999 – 2015), or 28-year average	Cox hazard model 2 versions: Basic PH model, and complex PH model using SURVEYPHREG. Basic model adjusted for age, sex, and race/ethnicity. Complex model adjusted for complex survey design. Both models controlled for marital status, household income, education, smoking status, BMI, urban/rural, census regions and survey year.	Annual $PM_{2.5}$ concentration were for participants were used to calculate overall mean concentration for the17-year study period 1999-2015.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
					$PM_{2.5}$ concentrations (1988 – 2015).		
Lepeule et al., 2012	LT	All-cause, Cardiovascular, lung cancer mortality	HARVARD 6 cities	Prospective Cohort/Longitudinal follow-up study (HARVARD 6 cities data)	$PM_{2.5}$ data from monitors in the participant's city. $PM_{2.5}$ data 1979-1986/1988 from monitors, end of monitoring to 1998 estimated from PM10 using US EPA monitors, 1999-2009 direct $PM_{2.5}$ measurement from US EPA monitors. 1-yr or 1-3yr or 1-5 yr. moving $PM_{2.5}$ averages were assigned to participants based on city of residence.	Cox proportional hazard models, Poisson survival analysis Stratified analysis by sex, age, and time in the study (1-yr interval). Confounders included: Baseline information on smoking status, smoking pack-years, education, linear and quadratic term for BMI. Also explored effect modification of $PM_{2.5}$ on mortality by smoking status at enrollment, as well as time period in study.	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Liu et al., 2019	ST	All-cause and cause-specific mortality	107 U.S. Cities	Time-series study (MCC Collaborative Research Network)	Monitored $PM_{2.5}$ concentration obtained from MCC database for the period of 1987-2006. Hourly data was used to calculate 24-hr daily average. Daily $PM_{2.5}$ concentrations were averaged across stations within each city. Finally, 2-day moving average for the city was calculated. 2-day moving average (lag01) was used in model.	Used two-stage analytic protocol, which had been developed and widely applied in previous multicity time-series studies. First stage estimated city-specific association using quasi-Poisson generalized additive models. Second stage used random-effects models to pool the estimates of the city-specific associations. Two-stage regional analysis was also performed by WHO regions. Also explored the shape of the relationship using C-R curves with PM term appearing with a B-spline function with two knots at 25th and 75th percentiles.	Daily $PM_{2.5}$ concentration were used to calculate overall mean concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Malig et al., 2013	ST	Respiratory morbidity (Asthma and COPD ED and HA)	35 CA counties (9 counties included for $PM_{2.5}$ analysis)	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	$PM_{2.5}$ data obtained from California Air Resources Board. Same day lag and various days lags average were calculated for $PM_{2.5}$. Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid. Only participants living in zip codes within 20 km of $PM_{2.5}$ monitors were included to increase validity of pollution exposure metrics.	County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis Time-invariant confounders and seasonal trends were controlled for given the study design. Other confounders included in the models were: other gaseous pollutants including ozone, linear and squared term for daily average temperature. Stratified analysis also by distance to monitor within 10 km vs. 10-20 km	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
McConnell et al., 2010	LT	Asthma Incidence	13 CA communities	Cohort Study (CHS)	$PM_{2.5}$ concentration data measured in central site monitors in each community (for 9 of 13 communities since 1994 and others different time period). This study considered 2003-2004 $PM_{2.5}$ measurements at each community monitor. Average annual $PM_{2.5}$ concentration from each community was assigned to study participants based on their community of residence.	Multi-level Cox proportional hazard model accounting for residual variation in time to asthma onset and clustering of children around schools and communities Models adjusted for: secondhand smoke, pets in home, race/ethnicity, age at study entry, sex, and random effects for community and school.	Average annual $PM_{2.5}$ concentrations assigned to study participants were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Ostro et al., 2016	ST	Asthma and COPD ED	8 metropolitan areas/counties in CA	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	$PM_{2.5}$ (24-hour average) data obtained from U.S. EPA provided by California Air Resources Board for the period of 2005-2009. Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid. Only participants living in zip codes within 20 km of $PM_{2.5}$ monitors were included to increase validity of pollution exposure metrics. Used lag0, lag1 and lag2 in model.	County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis Time-invariant confounders and seasonal trends were controlled for given the study design. Other confounders included in the models were: linear and squared term for lag0 temperature, day of the week.	Daily $PM_{2.5}$ concentrations for all 8 metropolitan counties over the period of 2005-2009 were used to calculate an overall mean $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Pappin et al., 2019	LT	Non-accidental mortality	Canada Nationwide	Cohort study (CanCHEC)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). For $PM_{2.5}$ concentrations prior to 1998, back casting method employed that applied observed trends in ground monitoring data for $PM_{2.5}$ to adjust pre-gridded $PM_{2.5}$ estimates (Meng, 2019). Annual M2.5 estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year moving average based on the location and year of follow-up.	Cox Hazard model and DAG approach. Also performed C-R analysis using a 3 step approaches: (1) fit the data using restricted cubic splines (RCS) with a large number of knots; (2) smooth potential erratic predictions from the large number of knots using monotonically increasing smoothing splines (MISS); and (3) fit the shape constrained health impact function (SCHIF) to the MISS predictions. Cox model stratified by age, sex, and immigration status separately by CanCHEC cohorts. Two covariate adjustment models. First based on DAG and controlled for airshed, urban form, CMA/CA size. Second model "full" model adjusted for individual-level variables (income, education, occupation, marital status).	The annual average $PM_{2.5}$ concentrations were used to calculate overall mean $PM_{2.5}$ concentration for the study cohorts and periods.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Peng et al., 2009	ST	CVD HA Age 65+	119 U.S. Urban counties>150,000 populations	Time-series analysis (MEDICARE enrollees)	$PM_{2.5}$ data (daily or every 3 days) obtained from US EPA's AQS and STN for the period of 2000-2006. Countywide $PM_{2.5}$ total mass concentration was calculated by averaging the daily $PM_{2.5}$ values from all the monitors in a county. Used lag0, lag1 and lag2 in model.	Log-linear Poisson Regression analysis Adjusted for potential confounders including weather, day of the week, unobserved seasonal factors. In county-specific regression model, following indicators were included: indicator for the day of the weeks, a smooth function of time per calendar year to control for seasonality and long-term trends, a smooth function of current-day temperature, a smooth function of the 3-day running mean temperature, a smooth function of current-day dew-point temperature, and a smooth function of the 3-day running mean dew-point temperature. To model smooth functions, we used a natural spline basis.	Daily $PM_{2.5}$ concentrations for all 119 counties over the period of 2000-2006 were used to calculate an overall median $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Pinault et al., 2016	LT	All-cause, CVD and lung cancer mortality	Multicity Canada	Prospective Cohort Study (subset of participants of the Canadian Community Health Survey)	$PM_{2.5}$ concentration derived from MODIS. Geographically weighted regression including monitoring and land use data was applied to the estimates from MODIS to produce average $PM_{2.5}$ concentration at 1 km2 resolution. These model estimates extended to 1998-2003 using inter-annual variation of Boys et al. Participants were assigned exposure based on their postal code of residence. For each year in the cohort, respondents were assigned a $PM_{2.5}$ concentration corresponding to the mean of the three previous years to the follow-up year.	Cox proportional hazards models Models were stratified by age (5-yr interval) and sex. Models adjusted for individual socioeconomic covariates and behavioral (BMI, smoking and alcohol consumption, fruit, and vegetable consumption) covariates, ecological variables including neighborhood socioeconomic status (both social and material deprivation).	Annual 3-year $PM_{2.5}$ average concentration for the study participants were used to calculate overall $PM_{2.5}$ concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Pinault et al., 2017	LT	Non-accidental, CVD, respiratory and lung cancer mortality	Canada Nationwide	Cohort study (CanCHEC)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). $PM_{2.5}$ concentrations extended back to 1998 by applying interannual variation of a publishing $PM_{2.5}$ dataset (Boys, 2014). Annual $PM_{2.5}$ estimates from the postal code was assigned to study participants based on the postal code for residence and used to calculate 3-year moving average based on the location and year of follow-up for years 1998 – 2012.	Cox survival models. Also estimated Shape Constrained Health Impact Functions (a concentration-response function) for selected causes of death. Adjusted for individual demographic and socioeconomic variables at baseline (on Census day): Aboriginal identity, visible minority status, marital status, educational attainment, income adequacy quintile, and labor force status, and contextual variables at the census division scale.	The annual 3-year moving average $PM_{2.5}$ concentrations for study participants were used to calculate overall mean $PM_{2.5}$ concentration for the study period

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Pinault et al., 2018	LT	CVD mortality	Canada Nationwide	Cohort study (CanCHEC, mCHHS)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). $PM_{2.5}$ concentrations extended back to 1998 by applying interannual variation of a publishing $PM_{2.5}$ dataset (Boys, 2014). Annual $PM_{2.5}$ estimates from the postal code was assigned to study participants based on the postal code for residence and used to calculate 3-year moving average based on the location and year of follow-up for years 1998 – 2012.	Cox proportional hazard models. Considered co-occurring diabetes with and without other contributing causes of death: hypertension, dementia or Alzheimer's disease, and chronic kidney disease, as these comorbidities are medically related to diabetes. Also considered diabetes status at baseline as effect modifier using CCHS-mortality cohort. Adjusted model for individual-level variables (aboriginal identity, visible minority status, education, labor force status and income adequacy), and neighborhood-level variables.	The annual 3-year moving average $PM_{2.5}$ concentrations for study participants were used to calculate overall mean $PM_{2.5}$ concentration for the study period

Pope et al., 2015b	LT	All-cause, IHD mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Monthly exposure to $PM_{2.5}$ was estimated by linking geocoded home addresses of the study participants to ambient $PM_{2.5}$ concentrations derived using a national-level hybrid land use regression (LUR) and Bayesian Maximum Entropy (BME) interpolation model (LUR-BME) that incorporated data from ground-based monitors for the study period of 1999-2004.	Cox proportional hazards models The individual-level covariates incorporated in the models included 13 variables that characterized current and former smoking habits (including smoking status of never, former, or current smoker, linear and squared terms for years smoked, and cigarettes smoked per day, indicator for starting smoking at aged <18 years, and pipe/cigar smoker). 1 continuous variable that assessed exposure to second-hand cigarette smoke (hours/d exposed); 7 variables that reflected workplace $PM_{2.5}$ exposure in each subject's main lifetime occupation; a variable that indicated self-reported exposure to dust and fumes in the workplace; variables that represented marital status (separated/divorced/widowed or single versus married); variables that characterized the level	Monthly mean $PM_{2.5}$ concentration for study participants were used to calculate overall mean concentration for the study period.

						of education (high school, more than high school versus less than high school); 2 body mass index variables (linear and squared terms for body mass index); variables that characterized the consumption of alcohol (beer, missing beer, wine, missing wine, liquor, and missing liquor); and variables that indicated quartile ranges of dietary fat index and quartile ranges of a dietary vegetable/fruit/fiber index. Ecological covariates included median household income; percentage of people with <125% of poverty-level income; percentage of unemployed individual aged ≥16 years; percentage of adults with <12th grade education; and percentage of the population who were black or Hispanic. These ecological covariates were included in the models using both zip code level data and zip code deviations from	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
						the county means.	
Pope et al., 2019	LT	All-cause and cause-specific mortality	Nationwide	Cohort study	$PM_{2.5}$ concentration estimated for census block using regulatory monitoring data from 1999-2015 within a universal kriging framework employing land-use regression methods and other variables (Kim 2018). Pop-weighted annual averages were calculated for all 17 years for each 2000 and 2010 census tract. Individual were assigned air pollution conc based on their census tract of residency at the time of survey, e.g.: using year-2000 census tract for individuals surveyed 1986-2010 and using year-2010 census tract for individuals surveyed 2011-2014. For primary analysis: $PM_{2.5}$ exposure is an average concentration over the 17 yrs.	Cox Hazard models. Ran 2 models: one accounting for complex survey design and sampling strategy including sample weights (SURVEYPHREG) and another without accounting for complex survey design (PHREG). Ran model using full-cohort and sub-cohort with additional data on BMI and smoking. The shape of the $PM_{2.5}$–mortality relationship was also explored using an integrated modeling approach. Adjusted for age, sex, and race-ethnicity, income inflation-adjusted to 2015, education levels, marital status, urban vs rural, US regions, survey years, smoking status.	Annual $PM_{2.5}$ average concentrations over the 17-years (1999-2015) were used to calculate overall $PM_{2.5}$ concentration.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Shi et al., 2016	ST and LT	Total mortality (65+)	New England Area with 6 U.S. States	Open Cohort study (MEDICARE enrollees)	Daily $PM_{2.5}$ for the New England area was predicted at 1-km2 spatial resolution from novel 3-stage statistical models for the period of 2003-2008. 365-day moving average (for long-term exposure) and average lag0-1 (for short-term exposure) were calculated for each grid cell. The long-term and short-term averages at grid-cells were matched to ZIP codes by linking the ZIP code centroid to the nearest $PM_{2.5}$ grid. Participants were assigned $PM_{2.5}$ concentrations based on the ZIP codes of residence. Used lag0-1 average for short-term exposure analysis in model.	Chronic effects of air pollution assessed using Cox proportional hazard models. Acute effects of air pollution assessed using Poisson log-linear models. Both acute and chronic effects were assessed using Poisson survival analysis. Analysis performed in full-cohort as well as low exposure cohorts. Poisson survival models were adjusted for smooth function of time, temporal covariates such as temperatures and day of the week, spatial covariates such as zip code-level socio-economic variables.	Long-term average: Average annual $PM_{2.5}$ concentrations of all grid cells in the study area were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period. Short-term average: Lag01 $PM_{2.5}$ concentrations of all grid cells in the study area were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Shin et al., 2019	LT	AF and Stroke (1st HA)	Ontario, Canada	Cohort study (ONPHEC)	$PM_{2.5}$ concentrations estimated using AOD and $PM_{2.5}$ simulated by the GEOS-Chem chemical transport model (i.e. individual's exposure in 2001 was estimated as mean exposure from 1996-2000). Final surface with 1 × 1 km resolution was generated for Ontario. Annual $PM_{2.5}$ estimates was calculated for the postal code and assigned to study participants based on the postal code for residence. $PM_{2.5}$ concentration was used to calculate 5-year moving average based on the location and year of follow-up.	Cox proportional hazards models. Adjusted for individual-level variables (age and sex), neighborhood-level SES variables, and geographic indicators.	The 5-year moving average $PM_{2.5}$ concentrations for study participants were used to calculate overall mean $PM_{2.5}$ concentration for the study period

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Shin et al., 2021	ST	All-cause hospitalization and all-cause mortality	22 Canadian cities	Time series study (Statistics Canada)	Daily (24-hour) average $PM_{2.5}$ concentrations were calculated for each study city using ambient monitoring data available from Canada's NAPS for the period 2001-2012. Daily $PM_{2.5}$ concentrations were averaged across monitors within a city when multiple monitoring sites were present.	Generalized additive Poisson model and Bayesian hierarchical model. Static approach to estimate the nationwide overall associations between air pollution and health outcomes for all years combined. A two-stage hierarchical model was employed: firstly, a generalized additive Poisson model for city-specific associations between individual health outcomes and individual air pollutants, respectively, and secondly a Bayesian random effects model to combine the city-specific associations to obtain nationwide associations.	Daily $PM_{2.5}$ concentrations of 22 Canadian cities were used to calculate overall mean OM2.5 concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Stieb et al., 2009	ST	Cardiac and Respiratory ED visits	Seven Canadian Cities	Time series study (Hospital cases)	PM data obtained from National Air Pollution Surveillance (NAPS) system for the period of the 1990s and early 2000s. City averages of the $PM_{2.5}$ exposure were calculated by averaging all monitoring stations within the city. Used lag 0, 1and 2, in model.	Generalized Linear Models with natural spline functions of time to adjust for seasonal cycles in air pollution and health Confounders included: Mean daily temperature and relative humidity at lag 0,1, and 2 days, day of the week and holidays.	Daily $PM_{2.5}$ concentrations of the cities were used to calculate the overall mean $PM_{2.5}$ exposure for the study location (by site) and study period.
Sun et al., 2019	ST	Incident stroke: Total, HS and IS (self-reported)	Nationwide	Time-stratified case-crossover (WHI)	$PM_{2.5}$ concentration obtained from log-normal ordinary kriging model as previously described (Liao et al., 2006). This model estimates daily air pollutants at each address based on weighted average of measurement from nearby monitors (Legendre and Fortin, 1989). Daily mean $PM_{2.5}$ concentrations were estimated at geocoded participant address for the period of 1993-2012.	Conditional logistic regression. Adjusted for time-varying variables (daily mean ambient temperature, dew point temperature, and relative humidity) Various lags assessed (1-day moving average to 6-day moving average)	Daily $PM_{2.5}$ concentrations on the case days for the participants were used to calculate the overall mean $PM_{2.5}$ concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Szyszkowicz, 2009	ST	Angina ED	Seven Canadian Cities	Time series study (Hospital cases)	PM data obtained from National Air Pollution Surveillance (NAPS) system. City averages of the exposure were calculated by averaging stations within the city. Used lag 0, 1 and 2, in model.	Generalized Linear Mixed models Models adjusted for meteorological variables such as relative humidity, temperature, and atmospheric pressure (a daily 24-hr average measurements were calculated). Temperature and relative humidity in models were represented by natural splines. Stratified analysis by season as well as combined for the whole period.	Daily $PM_{2.5}$ concentrations of the cities were used to calculate the overall mean $PM_{2.5}$ exposure for the study location (all and by cities) and study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Thurston et al., 2016a	LT	All-cause, CVD and respiratory mortality	6 U.S. States and 2 MSAs	Cohort study (NIH_AARP cohort)	PM data obtained from US EPA AQS for the period of 2000-2008. Census-tract estimates were generated using hybrid LUR and BME models, which were combined to generate monthly estimates of $PM_{2.5}$. Participants exposure was estimated at census-tract of residence and included annual mean concentration in prior year of mortality.	Cox proportional hazard models Stratified analysis by age, sex, regions (6 states and 2 MSAs). Confounders adjusted included: race, education, marital status, BMI, alcohol consumption, smoking history, contextual variables such as median household income and % pop with less than high school education. Several interactions between $PM_{2.5}$ and socio-demographics were also tested.	Average annual $PM_{2.5}$ concentrations of census tract estimates were used to calculate overall mean $PM_{2.5}$ exposure for the study location and period.

Turner et al., 2016	LT	Lung cancer mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Estimated $PM_{2.5}$ concentrations were obtained using a national-level hybrid land use regression (LUR) and Bayesian maximum entropy (BME) interpolation model. Monthly $PM_{2.5}$ monitoring data were collected from 1,464 sites from 1999 through 2008, with 10% reserved for cross-validation. The base LUR model that predicted $PM_{2.5}$ concentrations included traffic within 1 km and green space within 100 m^3. Residual spatiotemporal variation in $PM_{2.5}$ concentrations was interpolated with a BME interpolation model. The two estimates were then combined. The cross validation R2 was approximately 0.79. Mean $PM_{2.5}$ (1999–2004) concentrations were used here.	Cox proportional hazards model Models were adjusted for education; marital status; BMI and BMI squared; cigarette smoking status; cigarettes per day and cigarettes per day squared; years smoked, and years smoked squared; started smoking at younger than 18 years of age; passive smoking (hours); vegetable, fruit, fiber, and fat intake; beer, wine, and liquor consumption; occupational exposures; an occupational dirtiness index; and six sociodemographic ecological covariates at both the postal code and postal code minus county-level mean derived from the 1990 U.S. Census (median household income and percentage of African American residents, Hispanic residents, adults with postsecondary education, unemployment, and poverty). Potential confounding examined by elevation, MSA size, annual average daily maximum air temperature, mean county-level residential radon	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
						concentrations, and 1980 percentage of air conditioning.	
Urman et al., 2014	LT	Lung-function decline	8 Southern CA communities/ counties	Cohort study (CHS)	Central monitors in each community provided data on air pollutants. Each child was assigned exposure based on the child's resident community.	Linear Regression model Models were adjusted for demographic, socio-economic and anthropometric variables (BMI, height), study community.	
Wang et al., 2017	LT	Total mortality (65+)	7 U.S. Southeast States	Cohort study (MEDICARE enrollees)	Three stage Hybrid model to predict daily $PM_{2.5}$ concentration at 1km X 1km resolution for the period 2000-2013.Annual average of $PM_{2.5}$ for each grid cell calculated and took arithmetic mean of the annual average $PM_{2.5}$ across all grids in each of the zip code tabulation area (ZCTA). Participants were assigned annual averages of $PM_{2.5}$ based on their ZCTA of residence.	Cox Proportional hazard models Models were stratified by age groups, sex, race. Adjusted for variables: year of enrollment, previous admission due to CHF, COPD, MI and diabetes, numbers of days spent in ICU and CCU, state, ZCTA level socio-demographic variables such as % pop below poverty, urbanicity, lower education, median income and median home value, and behavioral variables such as % smokers and obesity at county level. Further model also included yearly mean summer temperature at ZCTA level.	Average annual $PM_{2.5}$ concentrations of ZCTAs were used to calculate overall median $PM_{2.5}$ exposure for the study location (overall and by state), and period (overall and by year).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Wang et al., 2020	LT	Non-accidental cause-specific mortality (Resp, CVD, cancer)	Nationwide	Cohort study (MEDICARE)	Daily $PM_{2.5}$ was estimated on a 6-km grid using a spatio-temporal model described in (Yanosky, 2014) for the period of 2000-2008. Model inputs included monitored $PM_{2.5}$, meteorological and geospatial covariates, and traffic-related PM estimated using a Gaussian line-source dispersion model. Medicare beneficiaries were matched to the grid point closest to their ZIP code centroid and $PM_{2.5}$ concentrations were averaged for the 12-month period prior to death.	Cox hazard models. Also fit models using restricted cubic splines (RCS) with three knots to characterize non-linearity. Effect-modification assess for age, sex, race and urbanicity. Adjusted for SES variables.	Annual average $PM_{2.5}$ concentration for participants were used to calculate overall annual mean $PM_{2.5}$ exposure for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Weichenthal et al., 2016c	ST	Asthma and COPD ED	15 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	Daily average concentration of $PM_{2.5}$ collected from fixed-monitoring stations for the period of 2004-2011 in Ontario, which is part of Canada's National Air Pollution Data. PM data obtained from 19 sites located in 15 cities. 2 years of data available for 3 cities and remaining had 5-8 years of daily air pollution data. Case and control days of study participants were assigned $PM_{2.5}$ concentration based on the city of residence and based on monitoring station closest to the population-weighted centroid of each subject's 3-digit postal code (if multiple monitors available in participants city such as Toronto and Hamilton). Various lags assessed: lag0, lag1, lag2 and mean of lag0-2.	Conditional logistic regression models Models adjusted for 3-day mean temperature and relative humidity using cubic splines.	Daily $PM_{2.5}$ concentrations in Ontario over the period of 2004-2011 were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Weichenthal et al., 2016b	ST	MI ED	16 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	Daily average concentration of $PM_{2.5}$ collected from fixed-monitoring stations for the period of 2004-2011 in Ontario, which is part of Canada's National Air Pollution Data. PM data obtained from 20 provincial monitoring sites located in 16 cities. Case and control days of study participants were assigned $PM_{2.5}$ concentration based on the monitoring station closest to the population-weighted centroid of each subject's 3-digit postal code. Various lags assessed: lag0, lag1, lag2 and mean of lag0-2.	Conditional logistic regression models Models adjusted for 3-day mean temperature and relative humidity using cubic splines.	Daily $PM_{2.5}$ concentrations in Ontario over the period of 2004-2011 were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Wu et al., 2020	LT	All-cause mortality (65+)	US Nationwide	Cohort study (MEDICARE)	Annual $PM_{2.5}$ exposure. Modeled $PM_{2.5}$ exposure at 1km2 grid cells across the US using well-validated ensemble models (Di et al. 2019a, Di 2019b) for the period of 2000-2016. Daily concentration in grid cells were then averaged to estimate annual concentration at ZIP code and then assigned to individual based on ZIP code of residence.	Five statistical approaches: 2 regression approach (Cox Hazard, Poisson reg); 3 causal inference approach (GPSs) Stratified by individual-level characteristics. Further adjusted for community-level factors such as smoking and BMI, zip code-level census variables and meteorological variables, geographic regions, and calendar years (2000-2016).	Annual average $PM_{2.5}$ concentration for participants were used to calculate overall mean $PM_{2.5}$ concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Wyatt et al., 2020	ST	All-cause, CVD, RD 30-day hospital readmissions	530 US counties	Case-crossover and Cohort study designs (USRDS hemodialysis patients)	$PM_{2.5}$ concentration estimates from AOD integrated with chemical transport model predictions, meteorology, land use variables for 1 km grid cells (Di,2016). Gridded $PM_{2.5}$ estimates were subsequently converted to population-weighted county-level estimates using 2010 Census tract population values. Daily $PM_{2.5}$ was linked to patient hospitalizations based on the county of their last dialysis visit. Examined Lag 0 and unconstrained distributive lag model.	The relative risks of hospital admissions associated with daily $PM_{2.5}$ were estimated with conditional Poisson models for each of the three health outcomes separately. Cox proportional hazards models were used to assess the relative risk of early (1–7 days post discharge) and late (8–30 days post discharge) readmission associated with daily $PM_{2.5}$ following all-cause and cause-specific index hospitalizations. Cox model adjusted for time-dependent (daily $PM_{2.5}$, daily temperature, daily RH, and day of the week) and time-independent (patient-specific hospitalization event and county SES) risk factors.	Daily estimates at county-level were used to calculate overall $PM_{2.5}$ concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Yap et al., 2013	ST	Asthma HA	12 CA counties	Time Series study (Hospital admissions)	$PM_{2.5}$ data for the period of 2000-2005 obtained from California Air Resources Board that maintains information from the National Air Monitoring Stations. $PM_{2.5}$ reported was 24-hr average mass concentration based on measurements taken every 1, 3, or 6 days. For counties with more than 1 monitoring site, daily average $PM_{2.5}$ was calculated by taking the average across monitors within the county. Missing values were computed based on data from other monitoring stations. PM at various lags lag0-lag6 were assessed.	Generalized Additive Poisson Regression analysis were run at county-level Models adjusted for: long-term time trends and seasonality, day of the week and smoothing splines within different lags for temperature. Effect modification by single or composite area-based SES assessed.	Daily $PM_{2.5}$ concentrations in 12 CA counties over the period of 2000-2005 were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Yazdi et al., 2019	LT	First HA: Stroke, COPD, pneumonia, MI, lung cancer, and HF	7 Southeastern states: FL, AL, MS, GA, NC, SC, and TN	Cohort study (MEDICARE)	$PM_{2.5}$ concentration estimated from spatio-temporal prediction model at 1-km2 grid cell (Di et al. 2017) for the period of 2000-2012. Daily $PM_{2.5}$ concentrations for grid cells were averaged to create annual $PM_{2.5}$ concentration at zip code level and assigned to study participants based on the zip code of residence	Marginal structural Cox proportional hazards models which was weighted with stabilized IPWs (to approximate a causal model). Adjusted for individual-level variables (sex, race, year, state, Medicaid eligibility), as well as census SES.	NR

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Zanobetti et al., 2009	ST	Heart Failure and MI HA 65+	26 US communities	Time Series study (MEDICARE enrollees data)	$PM_{2.5}$ data obtained from US EPA AQS for the period of 2000-2003. For majority of cities, metropolitan counties encompassed the city and its suburbs but some cities like Boston, Minneapolis-St Paul included multiple counties. Daily $PM_{2.5}$ data available for various monitors were averaged over the county and community (Monitors ranged from 1-4). Before averaging, however, monitors were tested for correlations and those with correlation <0.8 with 2 or more monitor pairs within a county were excluded considering it does not represent exposure for general population. Generated 2-day moving average (lag01) concentration	Poisson regression analysis Models stratified by season. Controlled for long-term trend with natural cubic spline for each season and year, day of the week, three-day average temperature and dew point temperature.	

Zanobetti and Schwartz, 2009	ST	All-cause, CVD and respiratory mortality	112 US cities	Time Series study (NCHS data)	$PM_{2.5}$ data obtained from US EPA AQS (NAMS and SLAMS providing daily $PM_{2.5}$ concentration) for the period of 1999-2005. For majority of cities, counties encompassed the city but some cities like Boston, Atlanta, Washington DC, the city included multiple counties. Daily $PM_{2.5}$ (24-hr) data available for various monitors were averaged over the county and city. Before averaging, however, monitors were tested for correlations and those with correlation <0.8 with 2 or more monitor pairs within a county were excluded considering it does not represent exposure for general population. Used standardized method to fill in the missing data in some monitors with at least 265 days of data in at least one year. Generated 2-day moving average (lag 01) concentration	Poisson regression analysis First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach in total by season and region. Controlled for long-term trend with natural cubic spline for each season and year, day of the week, same day, and previous day temperature.	Daily $PM_{2.5}$ concentrations in 112 US cities over the period of 1999-2005 were used to calculate the overall mean $PM_{2.5}$ exposure for the study location and period???

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Zanobetti et al., 2014	ST	All-cause mortality 65+	121 US communities/ cities	Case-Crossover Design (MEDICARE enrollees)	$PM_{2.5}$ data obtained from US EPA AQS. Daily $PM_{2.5}$ data available for various monitors were averaged over the communities. Participants were assigned 2-day moving average (lag 0 and 1) based on community of residence.	Conditional logistic regression models at community level. In a second stage of analysis, the community specific results were combined using the multivariate meta-analysis techniques Conditional logistic regression controlled for confounders such as average temp for the same and previous day. Temperature was modelled using spline to account for nonlinear relationship. Effect modification tested for cause of prior admission due to neurological disorders or diabetes, primary or secondary hospitalization for other disease conditions. Stratified analysis by sex, age, or race.	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Zeger et al., 2008	LT	All-cause mortality 65+	668 U.S Urban counties	Retrospective Cohort Study of MEDICARE enrollees (MCAPS)	$PM_{2.5}$ data (every 6th day at many locations) available from US EPA's AirData Database for the period of 2000-2005. Calculated mean annual $PM_{2.5}$ concentration for all 4,568 ZIP code centroids within 6 miles of a monitor with >10 months of data per year. Given the focus of study on long-term exposure, ZIP code 6-year average of $PM_{2.5}$ was calculated and assigned to study participants living within a zip code both during the 6 years of follow-up and some time before cohort enrollment.	Log-linear Regression model ran for specific US regions separately Models adjusted for individual socio-demographic variables and ZIP code level SES variables (education, income, poverty etc.). Also included standardized mortality ratio for COPD as a surrogate indicator of long-term smoking pattern of its residents.	Average annual $PM_{2.5}$ concentrations of ZIP codes were used to calculate overall mean $PM_{2.5}$ exposure for the study location (all and by region) for the study period 2000-2005.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean $PM_{2.5}$ concentrations
Zhang et al., 2021	LT	Non-accidental, CVD and respiratory mortality	Ontario, Canada	Cohort study (Ontario Health Study)	$PM_{2.5}$ exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). $PM_{2.5}$ estimates at 1 km2 were used to estimate annual $PM_{2.5}$ average and then 3-year and 5-year moving averages. These annual estimates were then assigned to participants based on postal code of residence (updated annually to account for residential mobility).	Cox proportional hazard models. Basic model stratified by age, sex, ethnicity, enrollment year to control for baseline risks. Models were adjusted for born in Canada, education, marital status, household income, BMI, fruits, and vegetable intake, smoking and drinking, physical activity, urban/rural, and various neighborhood level SES indicators.	The 5-year average $PM_{2.5}$ concentrations were used to calculate overall mean $PM_{2.5}$ concentration for the baseline year.

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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 3.4 of this draft PA for the reconsideration of the 2020 final decision on the particulate matter (PM) National Ambient Air Quality Standards (NAAQS), 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 sections 3.4.2.1-3.4.2.3.
- Additional technical detail on the at-risk analytic approach, including sources and derivation of key inputs to the risk modeling process (section C.3).
- Supplemental at-risk analytics (section C.3.4.2) intended to provide additional context for the summary risk estimates presented in section 3.4.2.4.
- Characterization of variability and uncertainty related to the risk assessment (section C.5) intended to provide additional context for section 3.4.2.5.

C.1 ADDITIONAL TECHNICAL DETAIL ON THE RISK ASSESSMENT APPROACH

As discussed in section 3.4, our general approach to estimating $PM_{2.5}$-associated human health risks in this reconsideration utilizes concentration-response (CR) functions obtained from epidemiologic studies to link ambient $PM_{2.5}$ exposure to risk in the form of mortality incidence (counts). 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 section C.1.1 of this appendix. Inputs required to model risk using CR functions are identified below (Figure C-1) and include

(1) the CR functions themselves, obtained from epidemiologic studies (section C.1.1 and C.3.2),

(2) baseline health incidence data and information on population demographics (section 0 and C.3.4),

(3) study areas (section C.1.3), and

(4) modeled ambient $PM_{2.5}$ concentrations corresponding to air quality scenarios of interest (section C.1.4).

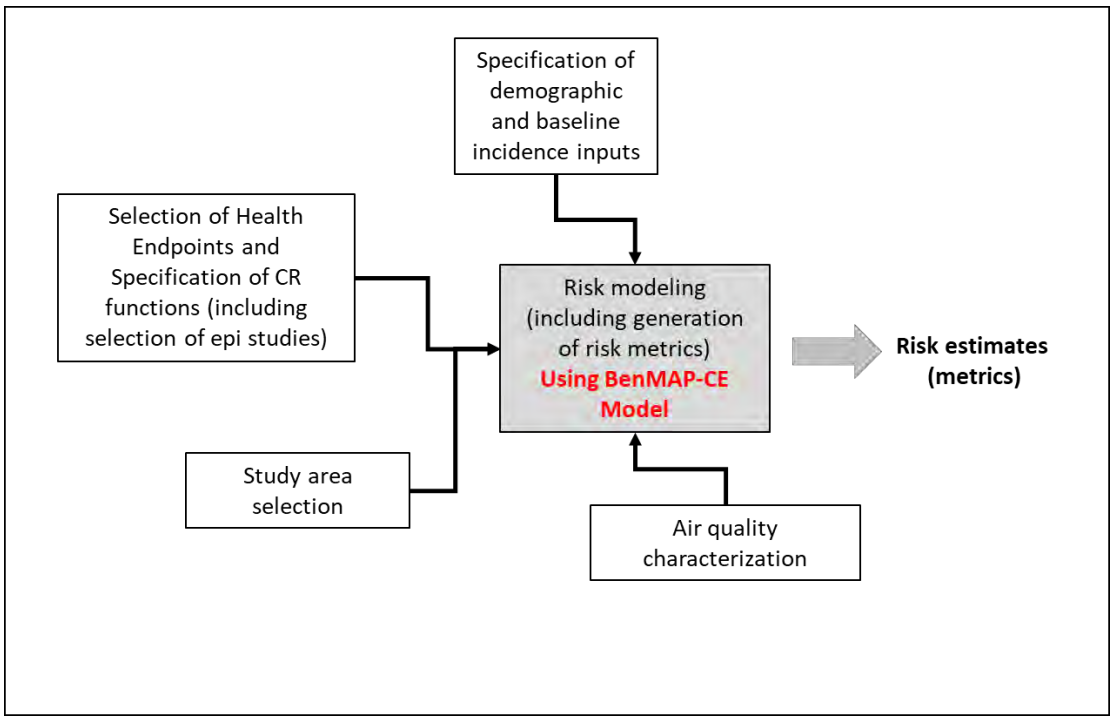


Figure C-1. Key inputs to the risk assessment.

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 began by considering health outcomes for which the 2019 Integrated Science Assessment (ISA) determined 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, 2019). As discussed in Chapter 3 (Table 3-1), these outcomes include the following:

- mortality (resulting from long- and short-term exposure),
- cardiovascular effects (resulting from long- and short-term exposure),
- respiratory effects (resulting from long- and short-term exposure),
- cancer (resulting from long-term exposure), and
- nervous system effects (resulting from long-term exposure).

We focused the risk assessment 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

- *Short-term PM exposure-related mortality*: all-cause and non-accidental

To identify specific epidemiologic studies for potential inclusion in the risk assessment, we focus on U.S. multicity studies assessed in the 2019 ISA. These studies are identified in section 3.4.1.5 of this draft PA. Of these, we used the following criteria to identify the specific set of studies for inclusion in the risk assessment:

- *National-scale geographic coverage:* We focus on epidemiologic studies reporting national-level CR functions. Epidemiologic studies that focus on individual cities or regions were excluded. Focusing on national-level epidemiologic 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 CR functions generated.
- *Evaluation of relatively lower ambient PM concentrations*: In selecting epidemiology studies, to the extent possible, we focus on 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 focus on those epidemiology studies providing CR functions 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 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 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 CR functions 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:* To the extent possible, preference was given to 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 co-pollutant models have advantages and disadvantages in characterizing the ambient PM-mortality relationship, to the extent possible, we include epidemiology studies (and associated CR functions) based on either single- or co-pollutant models that include ozone. Additional information available in the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits TSD* associated with the 2021 Revised Cross-State Air Pollution Rule Update (RCU) (U.S. EPA, 2021b).

- *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. The procedure used to derive CR functions (including specification of beta values and mathematical forms for those functions) is described below.

The remainder of this section describes the method used in specifying the CR functions used in the PM NAAQS HHRA. Information presented in this section is drawn from the EPA's Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE) Manual, Appendix C.[1] CR functions translate changes in ambient $PM_{2.5}$ into changes in baseline incidence rates for specific disease endpoints utilizing beta (β) values obtained from epidemiology studies studying the association between ambient $PM_{2.5}$ exposure and specific health endpoints. β values (and associated standard errors) are based on effect estimates obtained from the underlying epidemiology studies. In addition, the mathematical forms for the health impact functions specified for use in this risk assessment reflect the models used in the epidemiology studies providing those effect estimates. Consequently, derivation of the β values based on effect estimates from underlying epidemiology studies (and specification of the form of the health impact functions) represents a key step in the design of the HHRA.

The majority of the epidemiology studies providing effect estimates for this PM HHRA utilized either Poisson or Cox proportional hazard models which result in exponential (or log-linear) forms for the CR functions, where the natural logarithm of mortality incidence is a linear

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

function of $PM_{2.5}$.[2] If we let x_0 denote the baseline (starting) $PM_{2.5}$ level, and x_1 denote the control (ending) $PM_{2.5}$ level, y_0 denote the baseline incidences rate of the health effect, and Pop the underlying population count for the applicable demographic group in the spatial unit of analysis[3] we can derive the following CR function specifying the relationship between the change in x, $\Delta x = (x_0 - x_1)$ and the corresponding change in y, Δy (mortality incidence):

$$\Delta y = y_0[1 - e^{-\beta \Delta x}] * \text{Pop}$$

Given that the epidemiology studies providing effect estimates for long-term exposure-related mortality and short-term exposure-related mortality in the context of the current PM HHRA (Table C-1) use different categories of models (Cox proportional hazard and Poisson/Logistic, respectively) we describe the process of deriving the betas and specifying CR functional forms separately for each of these endpoint categories. As noted earlier, the logit model utilized in Zanobetti et al., 2014, is discussed at the end of the section covering short-term $PM_{2.5}$-related mortality.

Derivation of betas for long-term $PM_{2.5}$ exposure-related mortality

Cox proportional hazard models used to evaluate mortality associated with long-term $PM_{2.5}$ exposure are designed to model effects on population survival. This class of epidemiology model is based on a hazard function, defined as the probability that an individual dies at time t, conditional on that individual having survived up to time t. As such, the hazard function represents a time-specific snapshot of the rate of mortality (events per unit time) within a study population. While the risk can vary over time, in the case of the Cox proportional hazard model, it is assumed that the hazard ratio is constant. The proportional hazard model takes the form:

$$h(X,t) = h_0(t)e^{X \bullet \beta}$$

Where X is a vector of explanatory variables, β is a vector of associated coefficients and ho(t) is the baseline hazard (the risk when all covariates (X) are set to zero).

Epidemiology studies utilizing the Cox proportional hazard model in characterizing ambient $PM_{2.5}$-health effects typically report hazard ratios (HRs) as the effect estimate. HRs represent the ratio of hazard functions for the baseline and control scenarios reflecting a specific

[2] One study. Zanobetti et al., 2014, supporting the modeling of short-term $PM_{2.5}$ exposure-related mortality provided a logistic-based model form, which is discussed at the end of this section.

[3] Spatial unit of analysis refers to the geographic scale at which the CR function is applied in generating a risk (incidence) estimate (e.g., zip code, county, 12km grid cell). Typically, the spatial unit of analysis used in a REA is based on the spatial scale reflected in the epidemiology study(s) supplying the effect estimates. For this REA, the spatial unit of analysis is the 12km grid cell.

difference in ambient $PM_{2.5}$ exposure (often a 10 μg/m^3 increment). The HR simplifies as shown (with the baseline hazard ratio dropping out), allowing us to readily derive the β value from this effect estimate:

$$HR = \frac{h(X_0,t)}{h(X_C,t)} = \frac{h_0(t)e^{X_0 \bullet \beta}}{h_0(t)e^{X_C \bullet \beta}} = e^{\Delta PM \bullet \beta}$$

It is then possible to calculate the beta as follows:

$$\beta = \frac{In(HR)}{\Delta PM}$$

As noted in Sutradhar and Austin, 2018, the HR associated with a Cox-proportional hazard model may approximate the RR when the effect estimate (and consequently the β) is relatively small. This is the case with the effect on mortality modeled for long-term exposure to ambient $PM_{2.5}$ (i.e., the size of the effect estimate supports an assumed equivalency between HR and RR). The near equivalency between the HR and RR, allows us to utilize the β derived above in a CR function based on a log-linear functional form of the type presented earlier, to model changes in mortality related to changes in ambient PM.

Derivation of betas for short-term $PM_{2.5}$ exposure-related mortality

The epidemiology studies selected for use in modeling short-term $PM_{2.5}$ exposure-related mortality utilize both the Poisson (log-linear) model form (Baxter et al., 2017) and the logit model form (Zanobetti et al., 2014).[4] In both cases, the epidemiology studies provide effects in terms of *percent increase* in mortality.

The log-linear (Poisson) model is used to evaluate effects associated with continuous (count) events. With the log-linear (Poisson) model, the relative risk is simply the ratio of the two risks:

$$RR = \frac{y_0}{y_c} = e^{\beta \bullet \Delta PM}$$

The derivation of the beta with a Poisson model specified RR is as follows. Taking the natural log of both sides, the beta coefficient in the CR function underlying the relative risk can be derived as:

$$\beta = \frac{In(RR)}{\Delta PM}$$

[4] Note that the Ito et al., 2013 study also utilizes a Poisson model. However, that study provides beta values (including standard errors) and for that reason the results of this study are directly applicable in modeling changes in mortality without any of the derivations presented here for the other studies.

The beta derived in this fashion can then be used with a log-linear functional form (as presented earlier) to model changes in mortality related to changes in ambient PM.

The logistic model form is used to model dichotomous events. With the logistic model form, when we are provided with a RR value, as is the case here, we can make a similar assumption to that used above with the Cox proportional hazard function (i.e., that the OR and RR approach equivalency under conditions of relatively small effect levels). That observation in turn allows us to assume that

$$RR = \frac{y_0}{y_c} = (1 - y_0) \times e^{-\Delta PM \bullet \beta} + y_0$$

Then, assuming (based on the relatively small size of the baseline incidence) that:

$$e^{-\Delta PM \bullet \beta} \cong (1 - y_0) \times e^{-\Delta PM \bullet \beta} + y_0$$

$$\Rightarrow RR \cong e^{-\Delta PM \bullet \beta}$$

It is then possible to calculate the underlying beta coefficient as follows:

$$\frac{In(RR)}{-\Delta PM} \cong \beta$$

Since the derivation of the beta is based on the assumption of a log linear functional form, we can apply the beta in a log-liner CR function of the form described earlier:

$$\Delta y = y_0[1 - e^{-\beta \Delta x}] * \text{Pop}$$

Table C-1. Details regarding selection of epidemiology studies and specification of concentration-response functions for the risk assessment.

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemio-logic 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 µg/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 µg/m^3 applicable to only low-ozone days)[5]	Hazard ratio (95 percent CI)	All-cause	1.073 (1.071, 1.075)	7.0E-03	1E-04
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)	All-cause	1.06 (1.04-1.08)	5.8E-03	9.6E-04
Short-term exposure-related mortality studies										

[5] We note that Di et al., 2017 does include a copollutant model-based effect estimate (HR 1.073, 95th%CI 1.071-1.075). Had this effect estimate been used in risk modeling (which would translate into a beta value of 7.05E-3), we would anticipate the risk estimates for all-cause mortality to be slightly less (`13% lower based on comparison of calculated betas) than those estimated based on the single-pollutant model used in this risk assessment.

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemio-logic 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

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemio-logic Statistic	Mortality Endpoint	Selected Effect Estimate	Selected Beta	Selected Beta Standard Error (SE)
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 µg/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

1

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 for the year 2015 since the hybrid surfaces included in the analyses are based on a 2015 model year.[6] The BenMAP-CE model[7] 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 and the *Estimating $PM_{2.5}$ and Ozone- Attributable Health Benefits TSD* associated with the 2021 RCU for additional detail (U.S. EPA, 2021b).
- *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.

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 have greater confidence in estimating and simulating air quality concentrations over areas with relatively dense ambient monitoring networks, as the modeled air quality surfaces can be compared with monitored concentrations (air quality adjustments are described below in section C.1.4).
- *Geographical Diversity*: Risk assessments including areas that represent a variety of regions across the U.S. and a substantial portion of the U.S. population can be more representative.

[6] The 2015 model year was the most recent CMAQ modeling platform available at the time of the design of the risk assessment and represents the central year of the 2014-2016 design value (DV) period. A single modeling year was used in the risk assessment, rather than modeling risk for the full three-year design value period, because model inputs for the 2016 period were not available at the time of the study (section C.1.4.3).

[7] https://www.epa.gov/benmap

- *Ambient $PM_{2.5}$ Air Quality Concentrations*: Based on 2014-2016 design values, 16 CBSA[8] areas exceeded either or both the current annual and 24-hr $PM_{2.5}$ NAAQS. To include a larger portion of the U.S. in this risk assessment, we also identified CBSA areas with ambient $PM_{2.5}$ concentrations below, but near, the current annual and/or 24-hr $PM_{2.5}$ NAAQS. 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 requiring 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). Areas that appeared to be strongly influenced by exceptional events were also excluded (section C.1.4). Using these criteria, 47 urban study areas were identified (PA Figure 3-16 and Appendix section C.1.3), including 30 study areas where just meeting the current standards is controlled by the annual standard,[9] 11 study areas where just meeting the current standards is controlled by the daily standard,[10] and 6 areas where the controlling standard differed depending on the air quality adjustment approach (PA Figure 3-16). [11]

Applying these criteria resulted in the inclusion of 47 core-based statistical areas (CBSAs). These 47 study areas are identified in Figure C-2, with colors indicating whether they meet either or both the design value cutoffs. Please note, meeting the criteria for inclusion does not mean the areas exceed the current annual and/or 24-hr PM NAAQS standards. 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 the 24-hr and annual design values.

[8] 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.

[9] 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.

[10] 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.

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

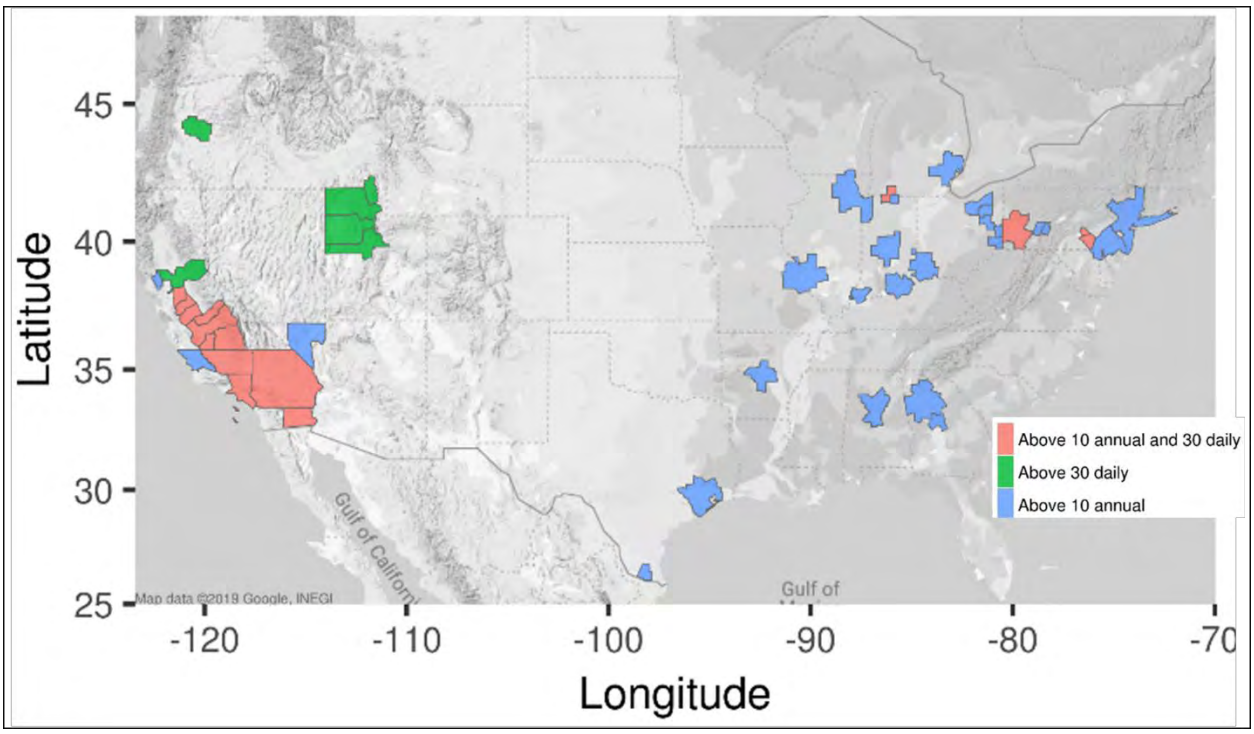


Figure C-2. Map of the areas modeled in the risk assessment, colored by 2014-2016 $PM_{2.5}$ design values (DV).

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 30% of the total U.S. population above the age of 30. Additional age-specific population information corresponding to each identified mortality study can be found in Table C-2.

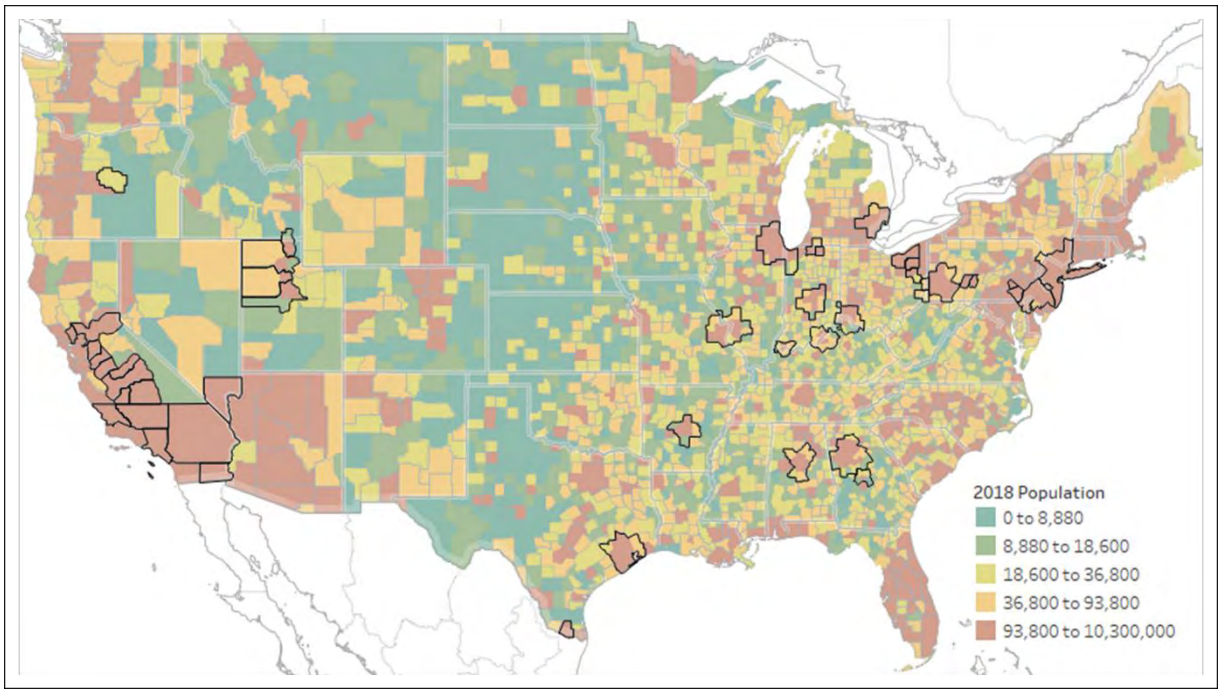


Figure C-3. Map of the 2018 U.S. population by CBSA, with the selected urban study areas outlined.

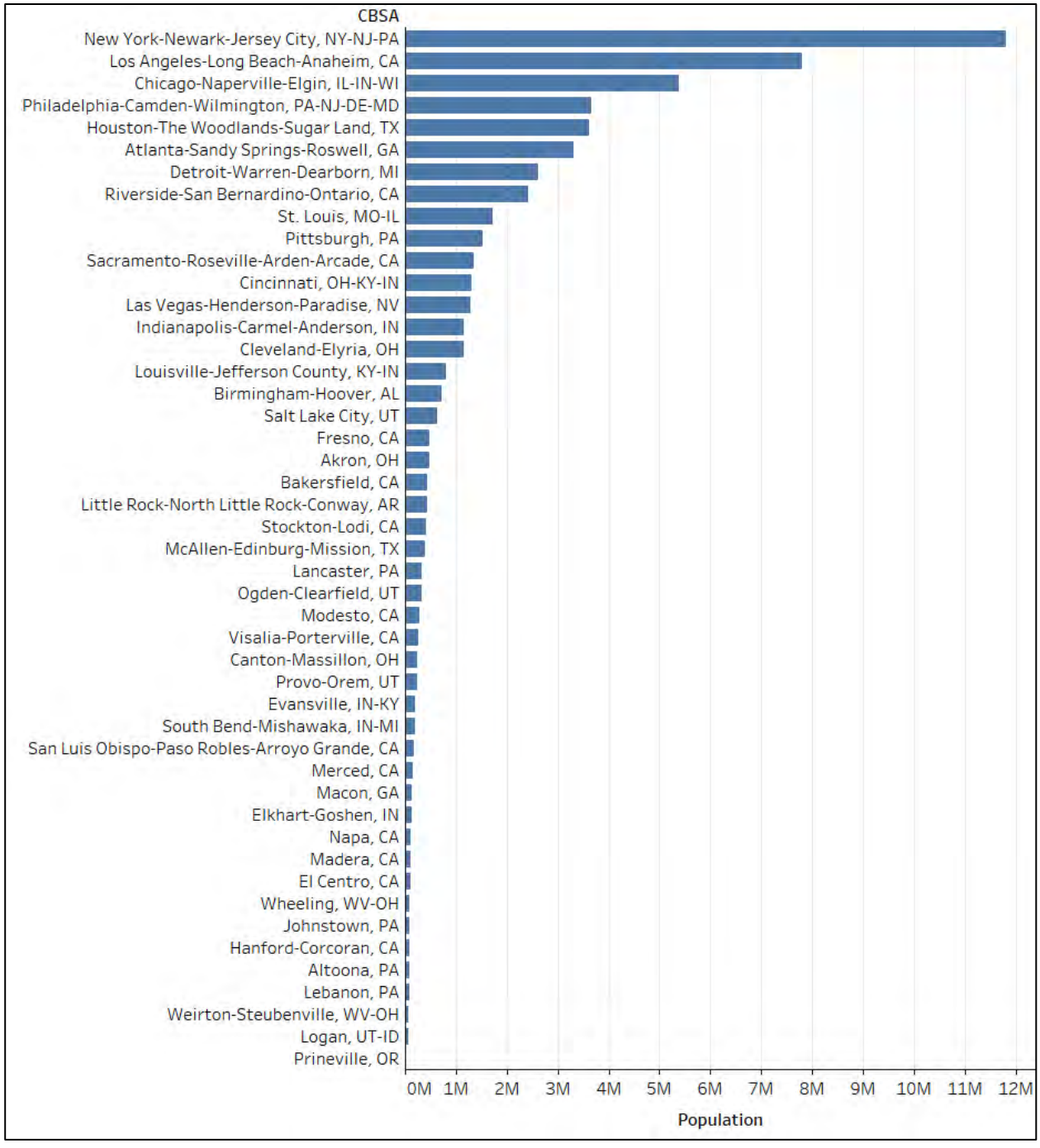
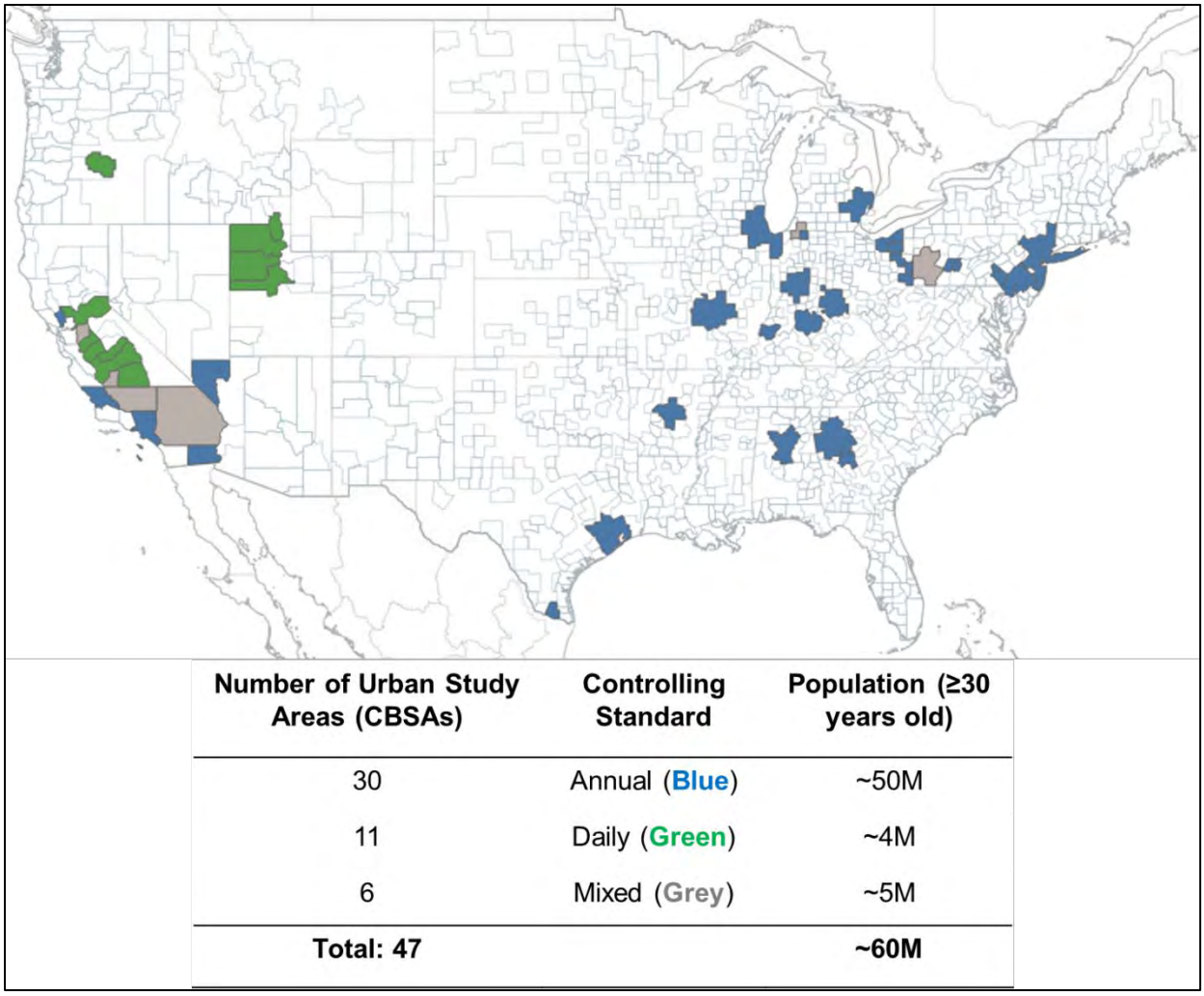


Figure C-4. Population counts for ages 30 and above from each of the 47 CBSAs included in the risk assessment.

Table C-2. Population of the 47 urban study areas by age range.

Population Age Range (Years)	Studies Using Age Range	Study Area Groupings (Millions)		
		47	30 (Annual-Controlled)	11 (24-hr-Controlled)
0-99	Baxter et al., 2017 and Ito et al., 2013	98.5	82.5	7.2
30-99	Turner et al., 2016	58.4	49.5	3.9
65-99	Di et al., 2017 and Zanobetti et al., 2014	13.2	11.1	0.8

As noted in section 3.4 of the 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 (12.0 $\mu g/m^3$), 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). 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, 9.0, and 8.0 $\mu g/m^3$, respectively (section 3.4.1.3 of the draft PA, Figure 3-15). 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 3.4.1.3 of the draft PA, Figure 3-15). 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 and 3.2.3.2 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.6.

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[12] 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)[13] and simulating $PM_{2.5}$ concentrations with the Community Multiscale Air Quality (CMAQ)[14] 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)[15] 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[16] 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.

[12] 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).

[13] www.epa.gov/aqs

[14] www.epa.gov/cmaq

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

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

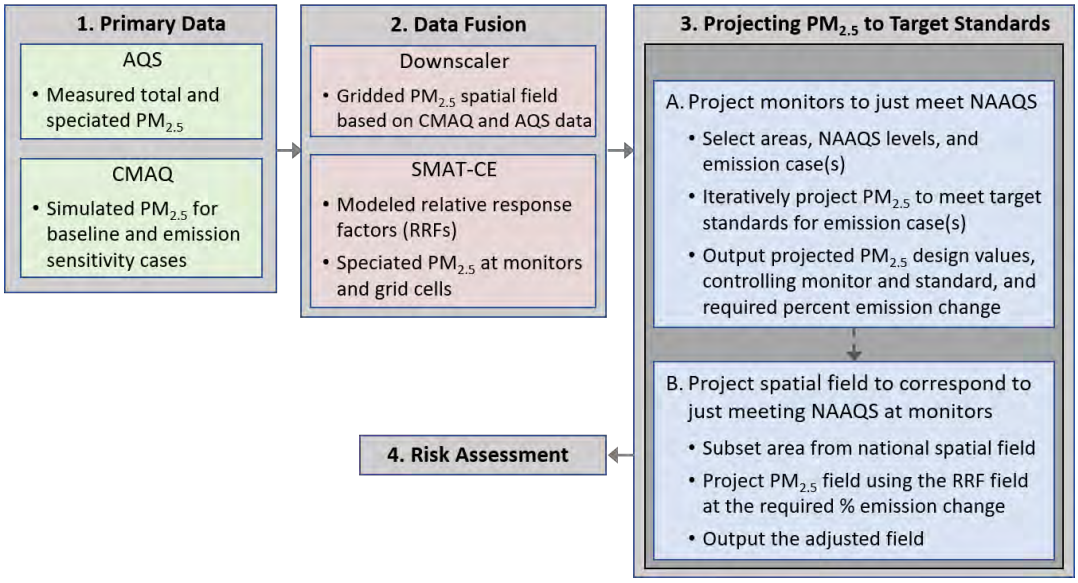


Figure C-6. Overview of the system for projecting $PM_{2.5}$ concentrations to correspond to just meeting NAAQS. See section C.1.4.6 and Kelly et al., 2019a for more details.

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[17], DVs for seven areas[18] appeared to meet the threshold due to the influence of wildfires. The influence of

[17] "10/30" indicates an annual standard level of 10 μg/ m^3 and a 24-hr standard level of 3 μg m^{-3}

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

wildfires on DVs for these areas was estimated in part by recalculating 2014-2016 DVs with days removed that were clearly associated with summertime wildfires in the northwest. Since wildfire influence is often excluded when judging NAAQS attainment, these seven areas were excluded from further consideration. Additionally, the Eugene, OR CBSA was excluded. One monitor in the Eugene CBSA has a 24-hr 2014-2016 DV slightly above the 10/30 selection threshold[19], 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[20], 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[21]) and provides a precise target for the iterative projection calculations.

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

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

[21] 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

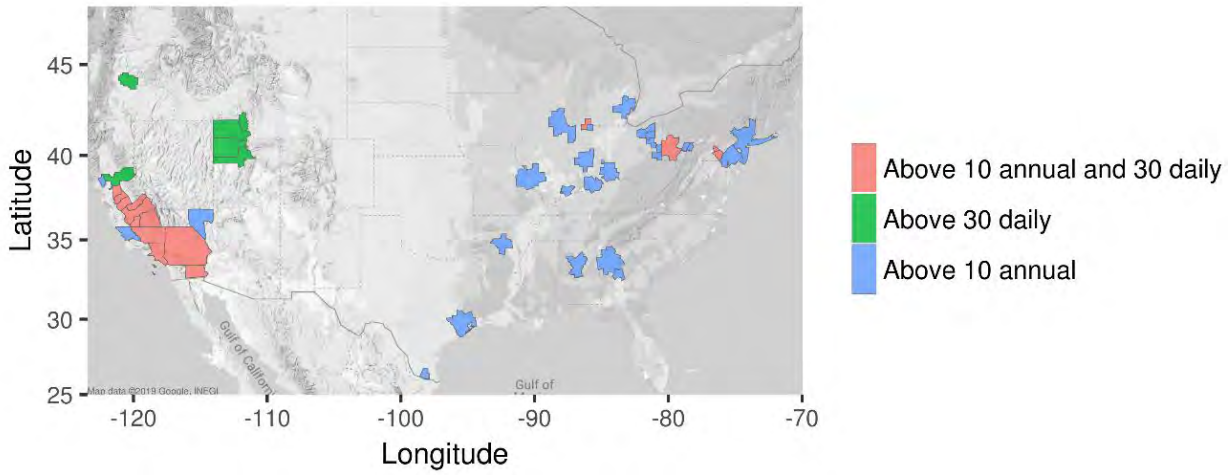


Figure C-7. CBSAs selected for the risk assessment. Colors indicate whether the maximum 2014-2016 DVs in the CBSA are above the annual (10 µg/m^3) and/or 24-hr (30 µg/m^3) selection criteria.

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

CBSA Name	# of Sites	Annual Max Site	Annual Max 14-16 DV	24-hr Max Site	24-hr Max 14-16 DV
South Bend-Mishawaka, IN-MI	1	181410015	10.45	181410015	32.5
St. Louis, MO-IL	6	290990019	10.12	295100007	23.7
Stockton-Lodi, CA	2	060771002	12.23	060771002	38.7
Visalia-Porterville, CA	1	061072002	16.23	061072002	54.0
Weirton-Steubenville, WV-OH	4	390810017	11.75	390810017	27.2
Wheeling, WV-OH	2	540511002	10.24	540511002	22.5
[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

CBSA Name	Associated Counties
Logan, UT-ID	Franklin, Cache
Los Angeles-Long Beach-Anaheim, CA	Los Angeles and Orange
Louisville/Jefferson County, KY-IN	Clark, Floyd, Harrison, Scott, Washington, Bullitt, Henry, Jefferson, Oldham, Shelby, Spencer, and Trimble
Macon, GA	Bibb, Crawford, Jones, Monroe, and Twiggs
Madera, CA	Madera
McAllen-Edinburg-Mission, TX	Hidalgo
Merced, CA	Merced
Modesto, CA	Stanislaus
Napa, CA	Napa
New York-Newark-Jersey City, NY-NJ-PA	Bergen, Essex, Hudson, Hunterdon, Middlesex, Monmouth, Morris, Ocean, Passaic, Somerset, Sussex, Union, Bronx, Dutchess, Kings, Nassau, New York, Orange, Putnam, Queens, Richmond, Rockland, Suffolk, Westchester, and Pike
Ogden-Clearfield, UT	Box Elder, Davis, Morgan, and Weber
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	New Castle, Cecil, Burlington, Camden, Gloucester, Salem, Bucks, Chester, Delaware, Montgomery, and Philadelphia
Pittsburgh, PA	Allegheny, Armstrong, Beaver, Butler, Fayette, Washington, and Westmoreland
Prineville, OR	Crook
Provo-Orem, UT	Juab and Utah
Riverside-San Bernardino-Ontario, CA	Riverside and San Bernardino
Sacramento--Roseville--Arden-Arcade, CA	El Dorado, Placer, Sacramento, and Yolo
Salt Lake City, UT	Salt Lake, and Tooele
San Luis Obispo-Paso Robles-Arroyo Grande, CA	San Luis Obispo
South Bend-Mishawaka, IN-MI	St. Joseph and Cass
St. Louis, MO-IL	Bond, Calhoun, Clinton, Jersey, Macoupin, Madison, Monroe, St. Clair, Franklin, Jefferson, Lincoln, St. Charles, St. Louis, Warren, and St. Louis city
Stockton-Lodi, CA	San Joaquin
Visalia-Porterville, CA	Tulare
Weirton-Steubenville, WV-OH	Jefferson, Brooke, and Hancock
Wheeling, WV-OH	Belmont, Marshall, and Ohio

C.1.4.3 Air Quality Modeling

Air quality modeling was conducted using version 5.2.1 of the CMAQ modeling system (Appel, 2018) 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.

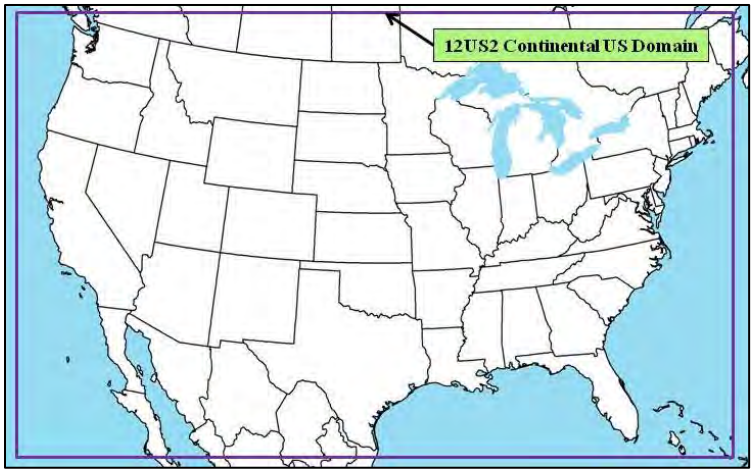


Figure C-9. CMAQ modeling domain.

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[22,23] for $PM_{2.5}$ at AQS sites for the 2015 base case.

Region[23]	Season	N	Avg. Obs. (µg m^{-3})	Avg. Mod. (µg m^{-3})	MB[22] (µg m^{-3})	NMB[22] (%)	RMSE[22] (µg m^{-3})	NME[22] (%)	r[22]
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

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

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

Region[23]	Season	N	Avg. Obs. (µg m^{-3})	Avg. Mod. (µg m^{-3})	MB[22] (µg m^{-3})	NMB[22] (%)	RMSE[22] (µg m^{-3})	NME[22] (%)	r[22]
	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
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 = $\frac{\sum_{i=1}^{n}(P_i - \bar{P})(O_i - \bar{O})}{\sqrt{\sum_{i=1}^{n}(P_i - \bar{P})^2}\sqrt{\sum_{i=1}^{n}(O_i - \bar{O})^2}}$	Pearson correlation coefficient

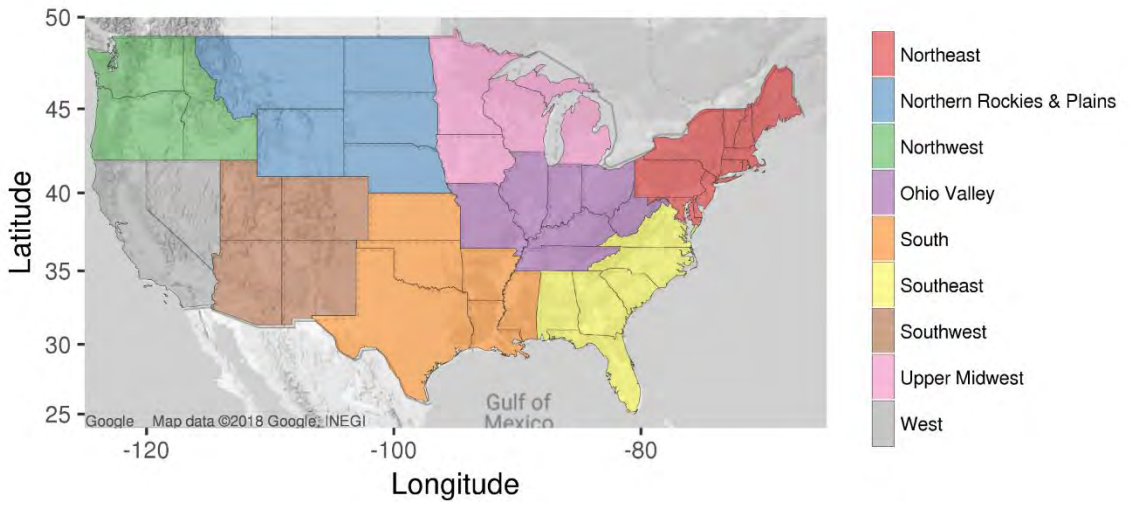


Figure C-10. U.S. climate regions[24] used in the CMAQ model performance evaluation.

In addition to the national model performance evaluation just described, CMAQ predictions of $PM_{2.5}$ concentrations were evaluated specifically for the CBSAs considered in the risk assessment. In Table C-8, model performance statistics are provided for predictions at monitors in the 47 CBSAs in 2015. Predictions generally agree well with observations over the full set of areas, with NMBs less than 10% in all seasons except Fall (NMB: 23.6%) and correlation coefficients greater than 0.60 in all seasons except Summer (r: 0.56). Model predictions are compared with observations by CBSA in Figure C-11, and NMBs at individual sites in the CBSAs are shown in Figure C-12. Predictions generally agree well with observations in the individual CBSAs, although underpredictions occurred in the Chicago-Naperville-Elgin

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

CBSA when observed $PM_{2.5}$ concentrations were > 40 μg m^{-3}. The high observed values in Chicago were associated with the 4th of July holiday, and the underpredictions on July 4th and 5th have small influence on the annual $PM_{2.5}$ projections in the risk assessment. The NMB is highest for model predictions in the Birmingham-Hoover CBSA (NMB: 66%). As mentioned above, the effects of model bias are mitigated in part by use of relative response factors (i.e., the ratio model predictions from a base and emission control simulation is used in projecting $PM_{2.5}$ concentrations, and some model bias likely cancels in the ratio). For the risk assessment projections, the key aspect of the CMAQ modeling is the spatial of pattern of $PM_{2.5}$ response to changes in emissions. The spatial response pattern was examined in the 47 CBSAs and found to be reasonable even in areas with relatively high bias, such as Birmingham. In Figure C-13, the spatial response pattern associated with the 10/30 projection case for the Birmingham-Hoover CBSA is compared for the proportional projection method and the primary PM projection case based on CMAQ modeling. Relatively high $PM_{2.5}$ responsiveness occurred in the urban part of Birmingham and along arterial roads in the CMAQ-based approach. This spatial pattern is consistent with the location of $PM_{2.5}$ emission sources in Birmingham and provides a realistic spatial response pattern despite the relatively high bias in the concentration predictions. Overall, both the national model performance evaluation and the evaluation for the 47 CBSAs of the risk assessment support use of the CMAQ modeling in this application.

To inform $PM_{2.5}$ 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.

Table C-8. Performance statistics for CMAQ predictions at monitoring sites in the 47 CBSAs considered in the risk assessment.

Season	Average Observed ($\mu g\ m^{-3}$)	Average Modeled ($\mu g\ m^{-3}$)	MB ($\mu g\ m^{-3}$)	NMB (%)	RMSE ($\mu g\ m^{-3}$)	NME (%)	*r*
Winter	12.40	13.45	1.05	8.5	8.03	42.4	0.61
Spring	9.17	9.94	0.77	8.4	5.15	38.6	0.62
Summer	10.35	10.08	-0.27	-2.6	5.51	34.6	0.56
Fall	9.00	11.11	2.12	23.6	6.26	45.6	0.67

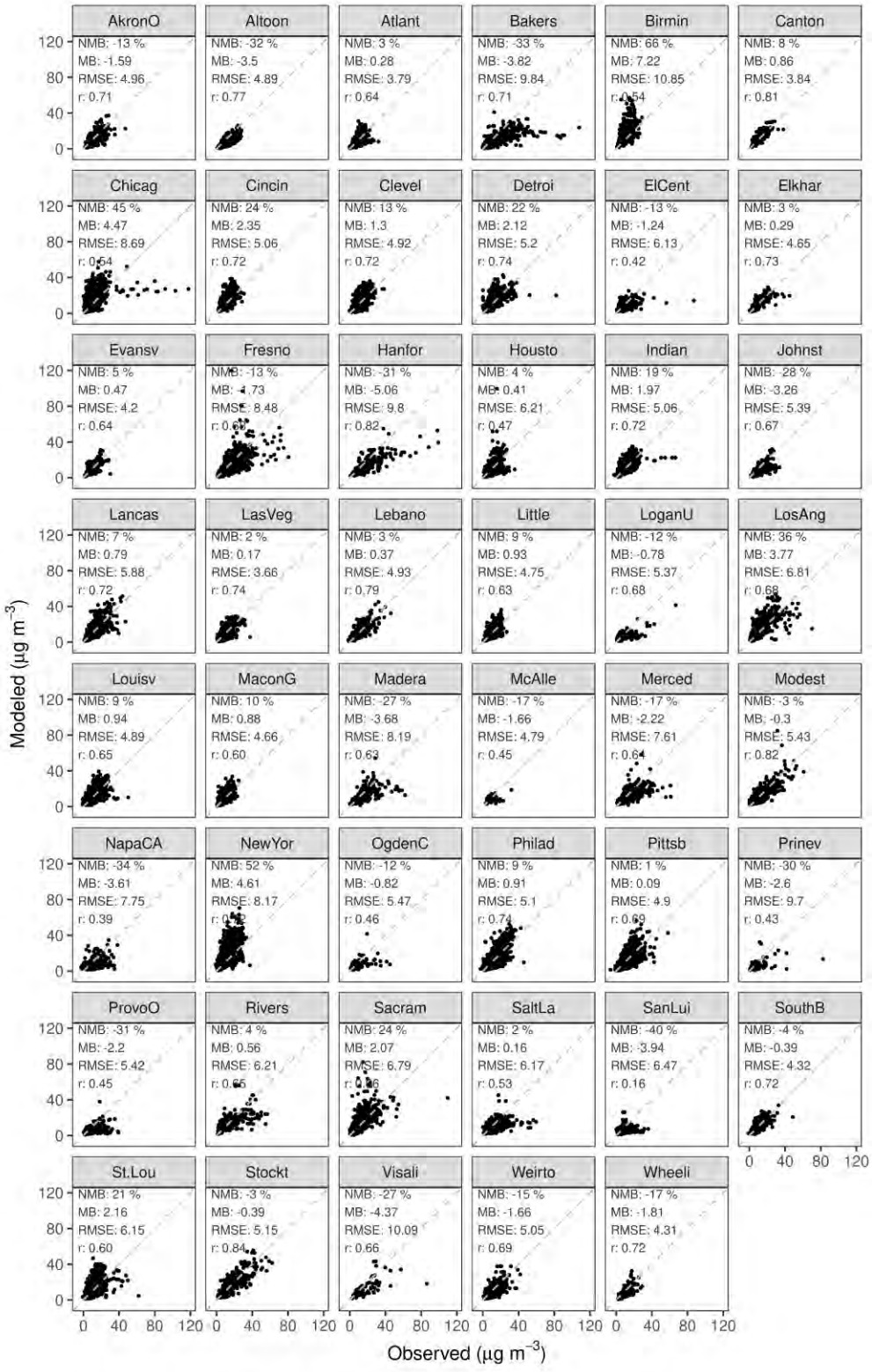


Figure C-11. Comparison of CMAQ predictions and observations at monitoring sites in the 47 CBSAs considered in the risk assessment.

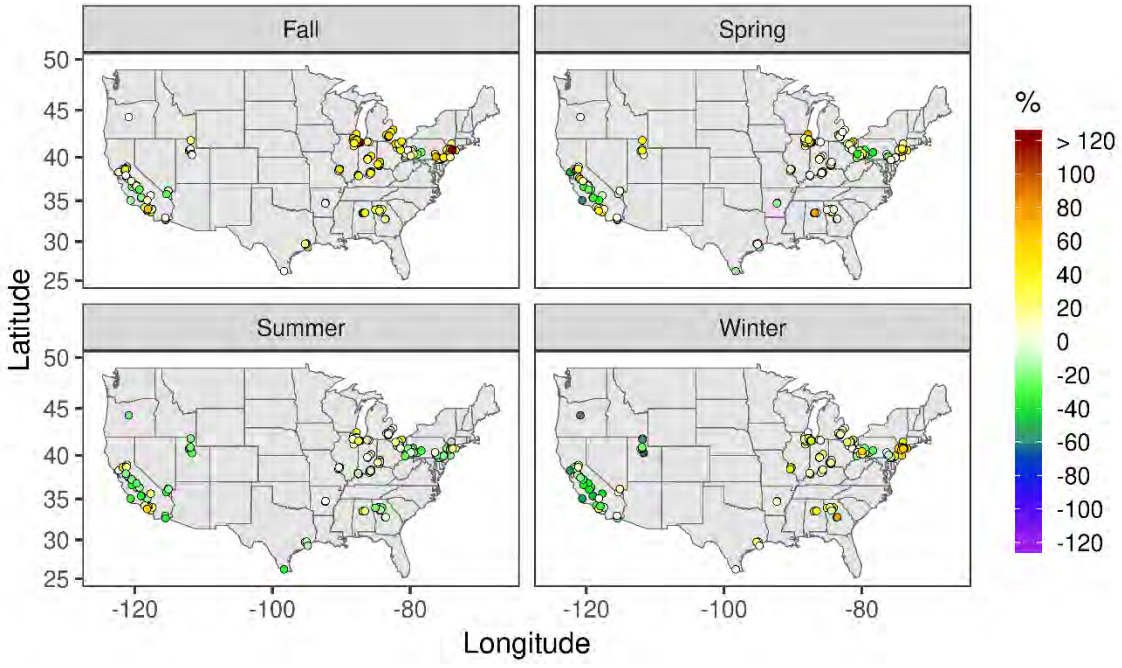


Figure C-12. NMB for CMAQ $PM_{2.5}$ predictions at monitoring sites in the 47 CBSAs by season in 2015.

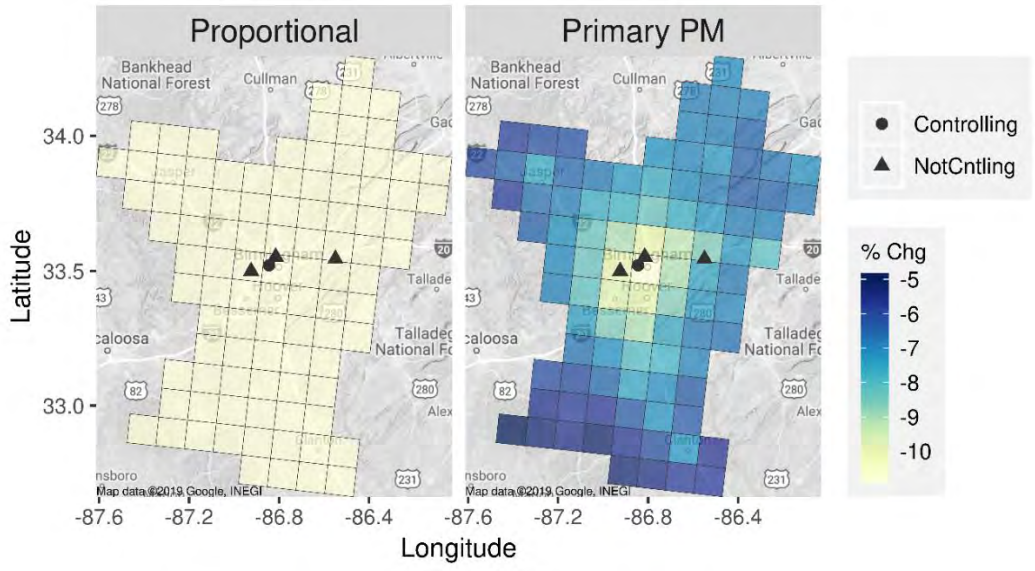


Figure C-13. Percent change in 2015 annual average $PM_{2.5}$ over the Birmingham CBSA associated with projecting 2014–2016 DVs at monitors to just meet an alternative NAAQS of 10/30 using the proportional projection method and the primary $PM_{2.5}$, CMAQ-based projection method.

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.

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)[25] and the Interagency Monitoring of Protected Visual Environments (IMPROVE)[26] 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[27] throughout 2014 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[28], 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[29]. Also, RRFs were set to one[30] in the third quarter (July-September) for select counties in the San Joaquin Valley and Utah[31] 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-14 and Figure C-15 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

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

[26] http://vista.cira.colostate.edu/Improve/

[27] Site identification number: 060290014

[28] 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.

[29] 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.

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

[31] 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.

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-16.

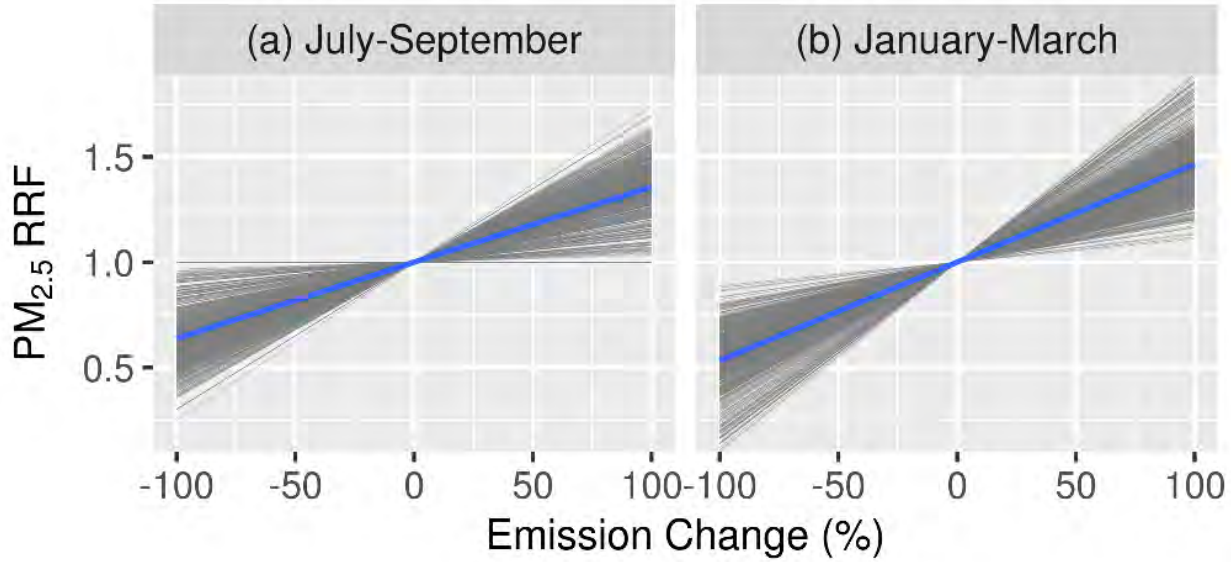


Figure C-14. Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic primary $PM_{2.5}$ emissions for monitoring sites in the contiguous U.S.

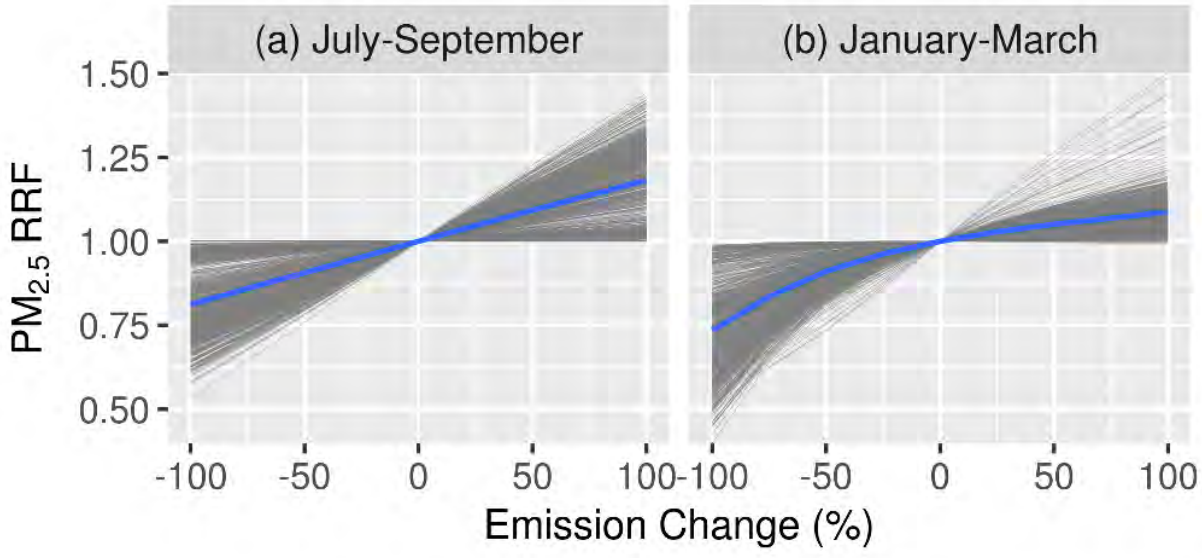


Figure C-15. Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent change in anthropogenic NOx and SO_2 emissions for monitoring sites in the contiguous U.S.

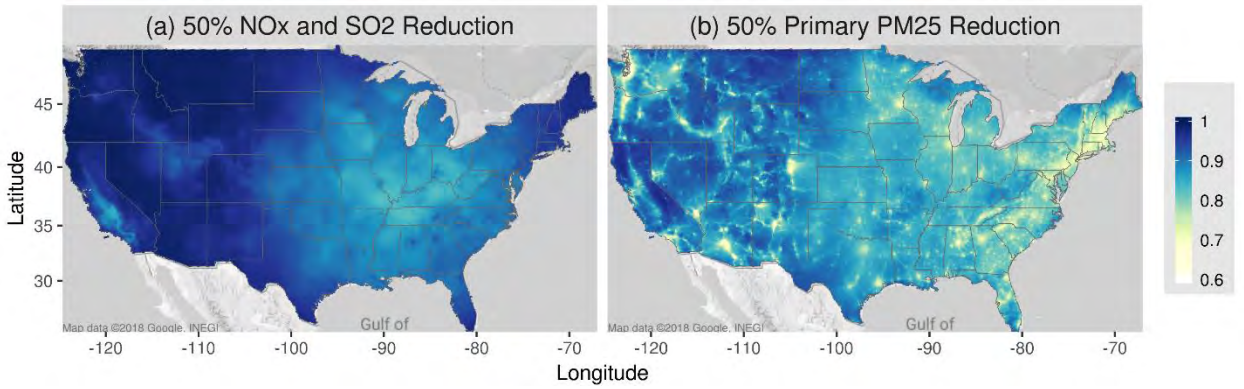


Figure C-16. Annual average $PM_{2.5}$ RRFs at CMAQ grid-cell centers for 50% reductions in anthropogenic (a) NOx and SO_2 and (b) primary $PM_{2.5}$ emissions.

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, 2018a). 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-17 along with the resulting Downscaler predictions. Cross-validation statistics are provided in Table C-9 based on comparisons of Downscaler predictions against the 10% of the observations that were randomly withheld from model fitting.

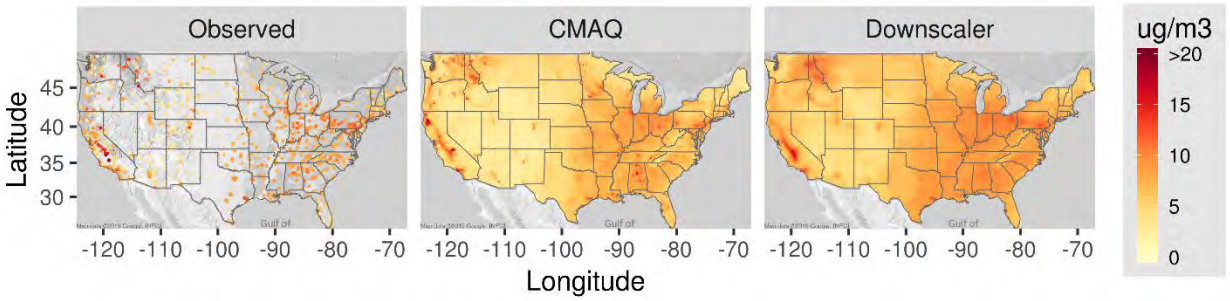


Figure C-17. Annual average of the 2015 $PM_{2.5}$ observations and CMAQ predictions used in the Downscaler model, and the annual average of the Downscaler $PM_{2.5}$ predictions.

Table C-9. 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)[32] and alternative (10/30)[33] 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 (-100 to 100%) were performed using bisection iteration until the difference between the

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

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

maximum projected DV in the CBSA and the standard level was zero or within the difference associated with a 1% emission change. Iterative projections of annual and 24-hr DVs were performed separately, and the controlling standard was determined as the standard requiring the greater percent emission change[34]. 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[35], 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[36] (Figure C-18).

[34] 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).

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

[36] 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

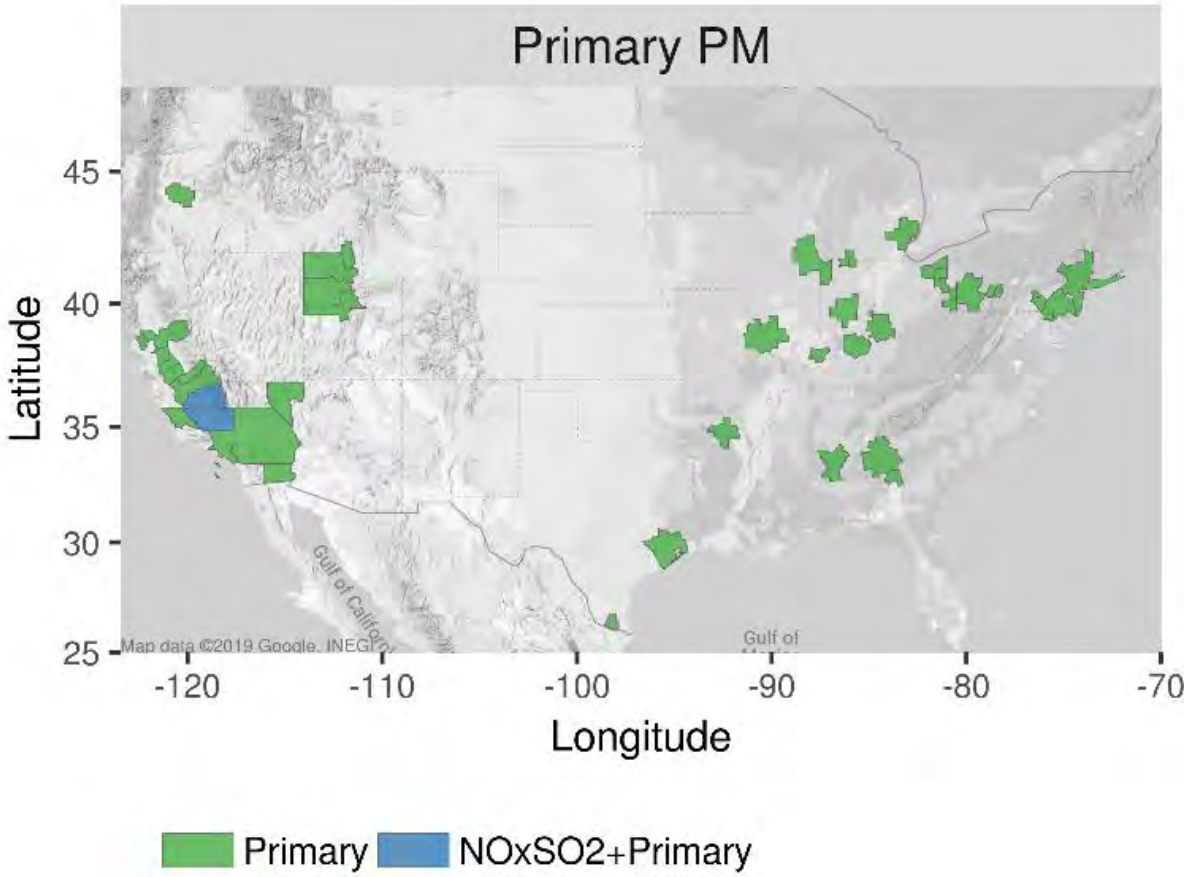


Figure C-18. Projection method used for each CBSA in the "Primary PM" projection case. See text for details.

The second scenario is referred to as "Secondary PM" or Sec-PM because projections were largely based on RRFs developed using CMAQ modeling with NOx and SO_2 emission changes, which affect concentrations of secondary PM components such as ammonium nitrate and ammonium sulfate. For 22 CBSAs[37], standards could not be just met using NOx and SO_2 emission changes alone. These areas were projected using the proportional scaling method[38] (Figure C-19). The proportional method was selected to gap-fill the Secondary PM case because

[37] 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

[38] 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).

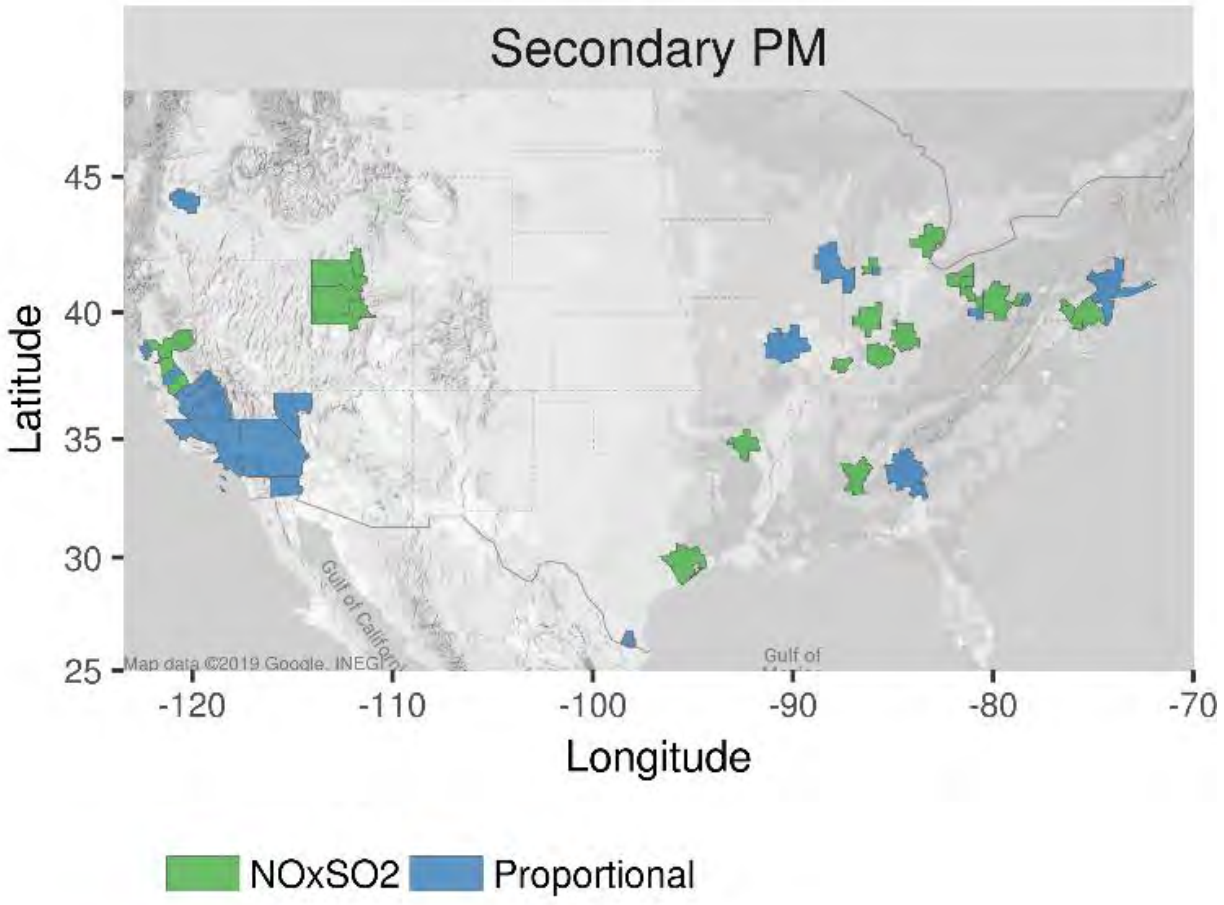


Figure C-19. Projection method used for each CBSA in the "Secondary PM" projection case.

The baseline 2015 concentration in the 47 CBSAs is shown in Figure C-20. These concentrations are the same as those in Figure C-17 but are shown only for the CBSAs included in the projections. In Figure C-21, the difference in annual concentration projected for the 12/35 case and the 2015 baseline concentration is shown. The positive and negative differences reflect areas where concentrations were projected to higher and lower levels to just meet the standard, respectively. In Figure C-22, the difference between the annual concentration projected for the 10/30 case and the 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-23. Baseline and projected $PM_{2.5}$ DVs for monitors in the 47 CBSAs are provided in Table C-19, Table C-20, Table C-21, and Table C-22 in section C.6.[39]

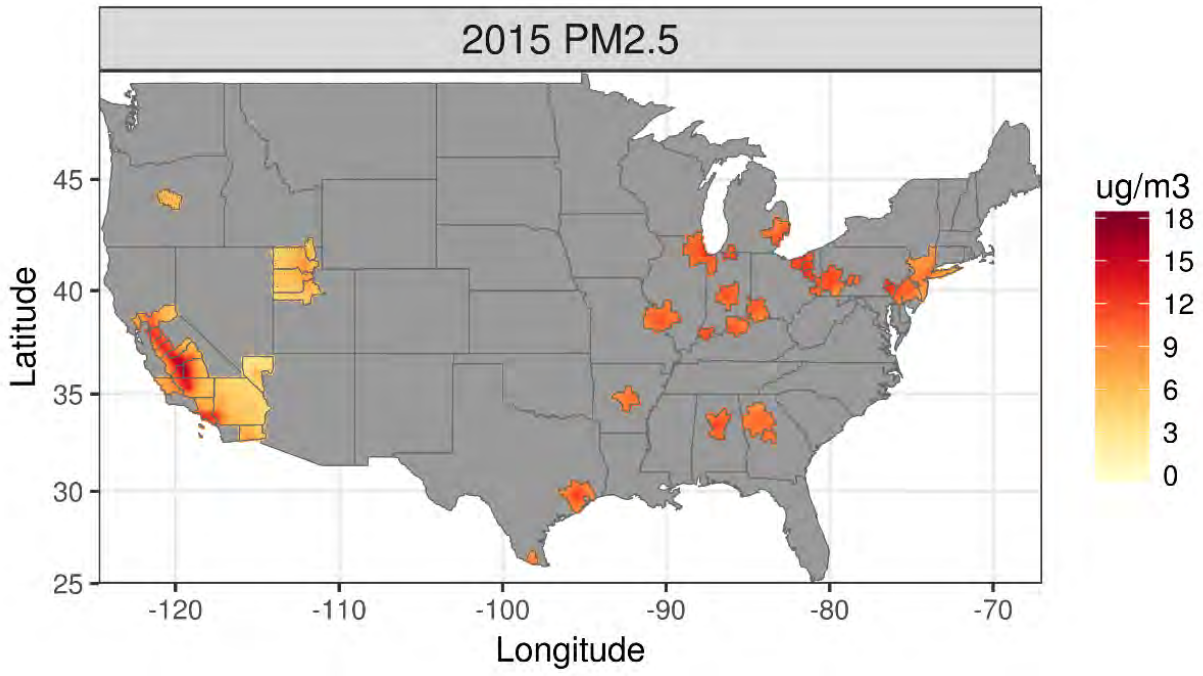


Figure C-20. Annual average 2015 $PM_{2.5}$ concentrations in the 47 CBSAs based on Downscaler modeling.

[39] 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.

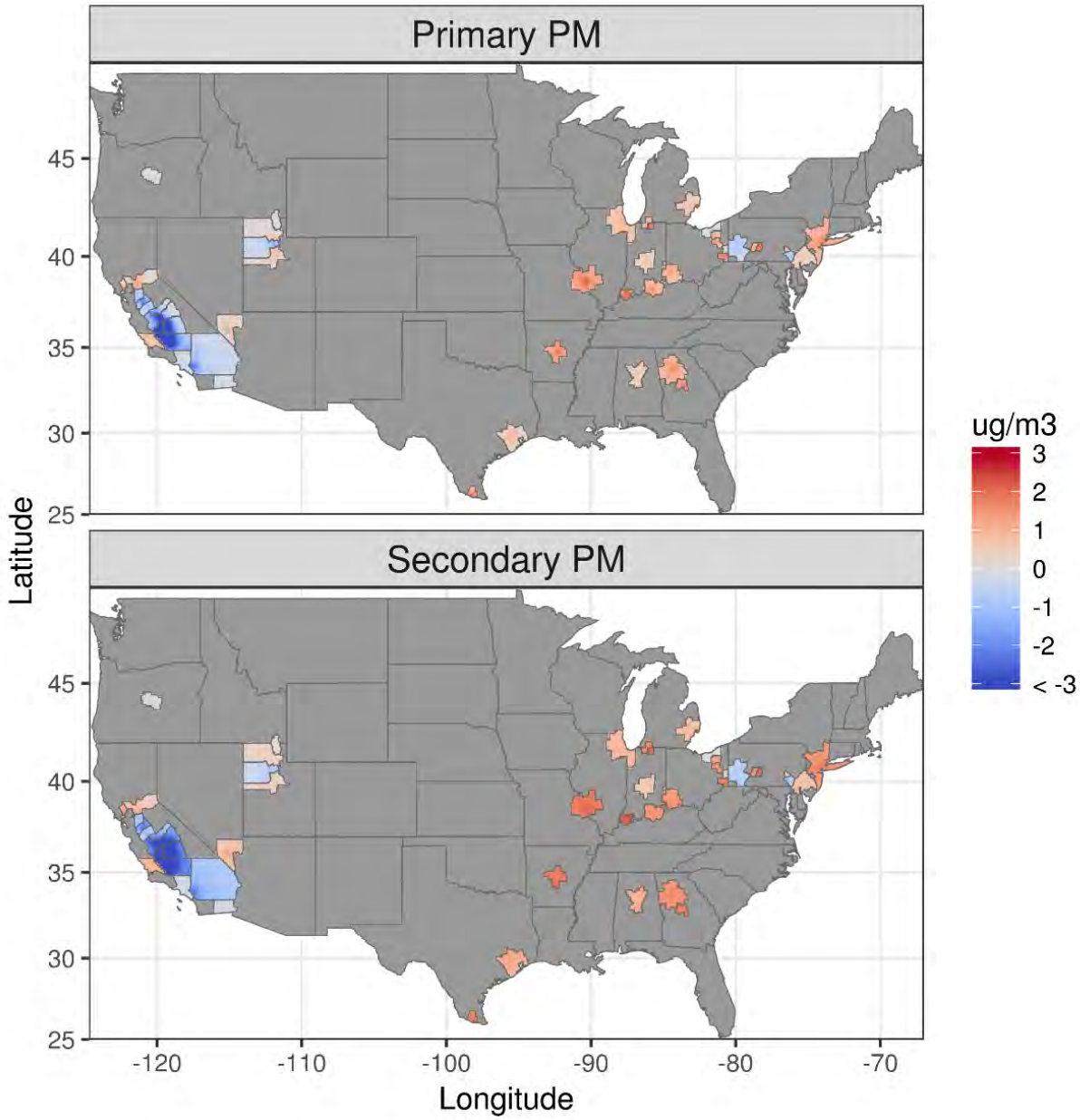


Figure C-21. Difference between the annual average projected $PM_{2.5}$ concentrations and the 2015 baseline concentrations for the 12/35 projection cases (i.e., 12/35 – baseline).

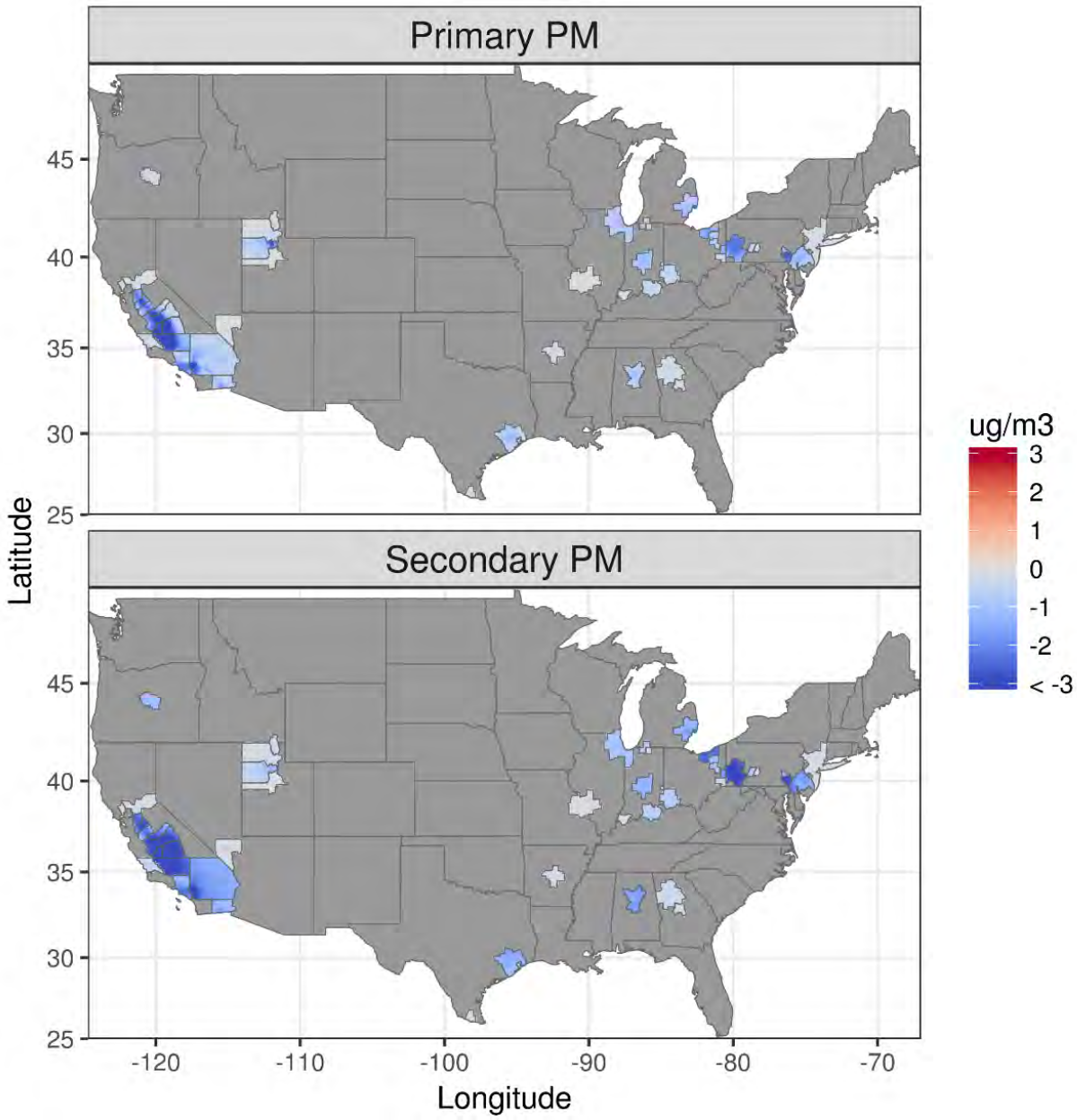


Figure C-22. Difference between the annual average projected $PM_{2.5}$ concentrations and the 2015 baseline concentrations for the 10/30 projection cases (i.e., 10/30 – baseline).

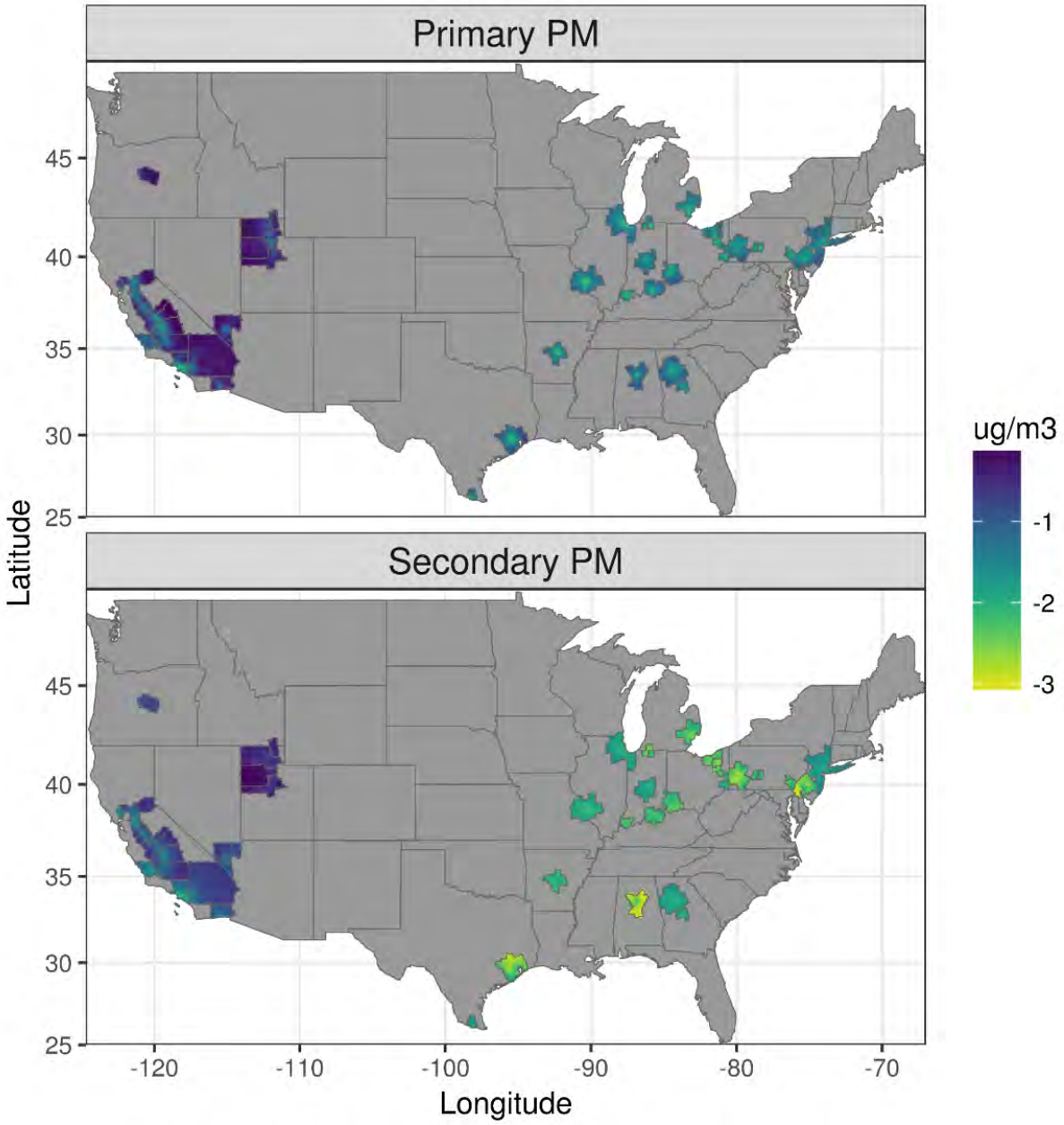


Figure C-23. Difference between the annual average projected $PM_{2.5}$ concentrations in the 10/30 and 12/35 cases (i.e., 10/30 – 12/35) for the Primary PM and Secondary PM projection cases.

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 BenMAP-CE version 1.5.[40] 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 CR 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. 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.1.2C.1.3, and C.1.4.

An overall flow diagram of the risk assessment approach is provided in Figure C-24. Application of this approach resulted in separate sets of risk estimates being generated for the following three groupings of urban study areas:

- the full set of 47,
- the 30 areas controlled by the annual standard, and
- the 11 areas controlled by the 24-hr standard.

Available air quality modeling surfaces for each of the three study area groupings are summarized in Table C-10.

[40] BenMAP-CE is a free program which can be downloaded from: https://www.epa.gov/benmap.

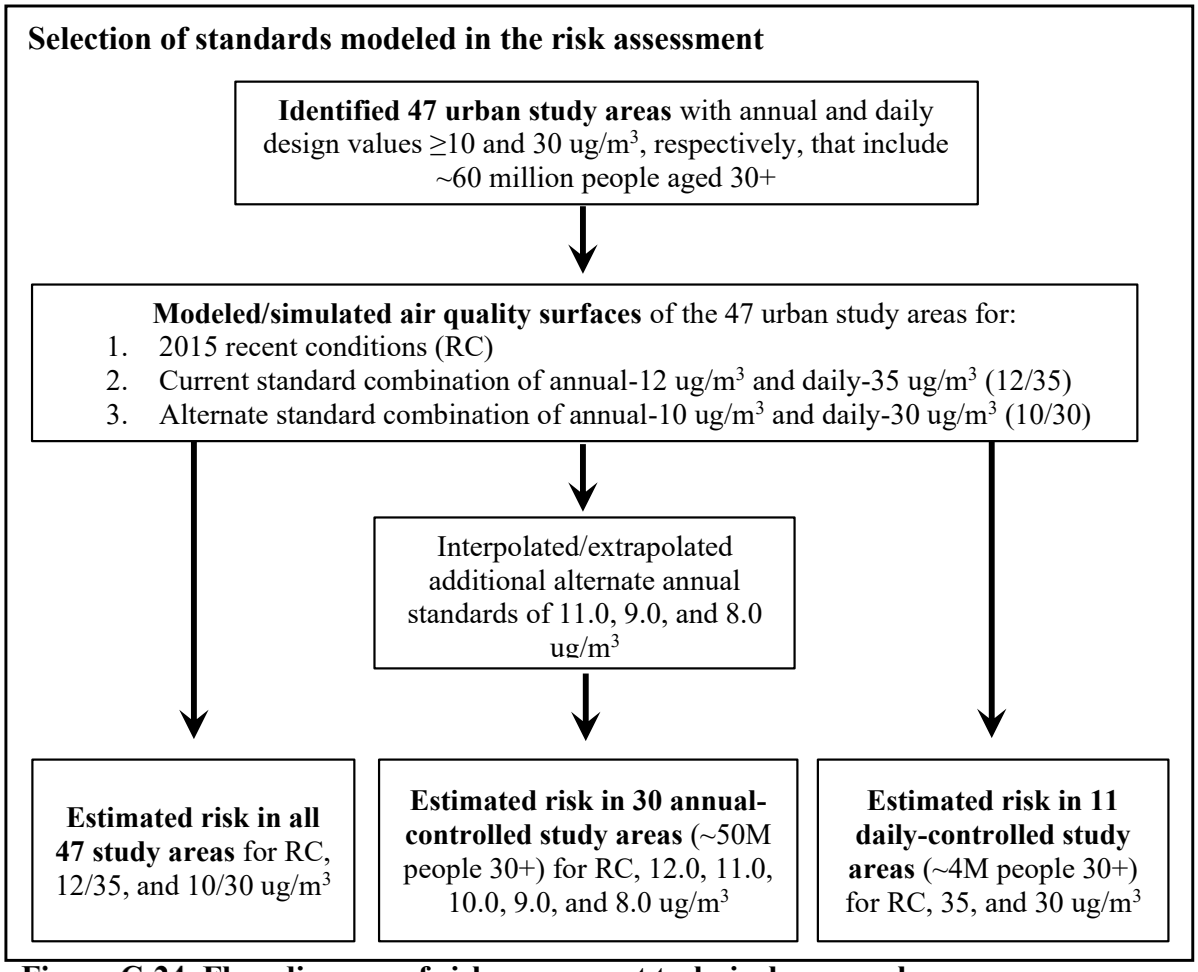


Figure C-24. Flow diagram of risk assessment technical approach.

Table C-10. Summary of available air quality scenarios for each study area set

	47 Study Areas (full set)	30 Study Areas (annually controlled)	11 Study Areas (daily controlled)
Recent Conditions (2015)	X	X	X
Just meeting 12/35 μg/m^3	X	X	X
11 μg/m^3 (interpolated)		X	
Just meeting 10/30 μg/m^3	X	X	X
9 μg/m^3 (extrapolated)		X	
8 μg/m^3 (extrapolated)		X	

Risk estimates are presented and discussed for each of these groupings in draft PA section 3.4.2, with greater emphasis being placed on results generated for the full set of 47 urban study areas and 30 annual-controlled study areas, given interest in national representation and on

those study areas where we could also consider the alternative annual standards of 8.0, 9.0 and 11.0 $\mu g/m^3$.

C.2 SUPPLEMENTAL RISK RESULTS

As noted earlier, this appendix also presents additional granular risk results that supplement the aggregated risk estimates presented and discussed in section 3.4.2 of the draft PA. The supplemental results are intended to provide additional context for the interpretation of summary risk estimates presented in draft PA section 3.4.2 and include additional line plots, maps and scatter plots illustrating the distribution of the grid-level risk estimates across ambient $PM_{2.5}$ concentrations (section C.2). Graphics provide insight into various aspects of the grid-level data underlying the summary tables presented in the draft PA, such as the spatial distribution of risk across the cities included in the risk assessment and how the distribution of grid-cell level risk estimates shifts as lower alternative standards are considered.

It can be challenging to understand how patterns of risk are changing under air quality simulated to just meet the current or alternative standards, 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). 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.

As the pattern of risk and risk reduction is similar across mortality endpoints, we focus on a single CR function to illustrate the changes graphically. Consequently, as with the graphics presented in draft PA section 3.4.2, the graphics presented in this section are also based on long-term exposure-related all-cause mortality modeled using a CR function obtained from Turner et al., 2016. The first set of graphics presented in this section (Figure C-25, Figure C-26, Figure C-27, Figure C-28, and Figure C-29) include results for the full set of 47 urban study areas and the second set (Figure C-30 and Figure C-31) include results for the 30 annual-controlled study areas. Graphical plots include:

- Histograms showing the distribution of 12 km gridded risk estimates across annual-averaged $PM_{2.5}$ concentrations (Figure C-25 and Figure C-30). These figures allow consideration of how the distribution of risk shifts when simulating air quality that just meets the current standards (12/35 $\mu g/m^3$) relative to 2015 recent 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 recent conditions simulation (Figure C-26),
(b) simulation of the current standards (12/35 $\mu g/m^3$) (Figure C-27), and
(c) simulation of the change (delta) in risk between the current and alternative standards (10/30 $\mu g/m^3$) (Figure C-28).

- Scatter plots depicting the distribution of modeled risk by annual-average $PM_{2.5}$ concentration (Figure C-29 and Figure C-31). While these scatter plots present similar distributional information as the line graphs, the scatter plots allow for a more detailed consideration of the nature of the risk distribution in relation to ambient $PM_{2.5}$ 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.[41] 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 are presented below the figures.

[41] 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 $\mu g/m^3$.

C.2.1 Results from Full Set of 47 Study Areas

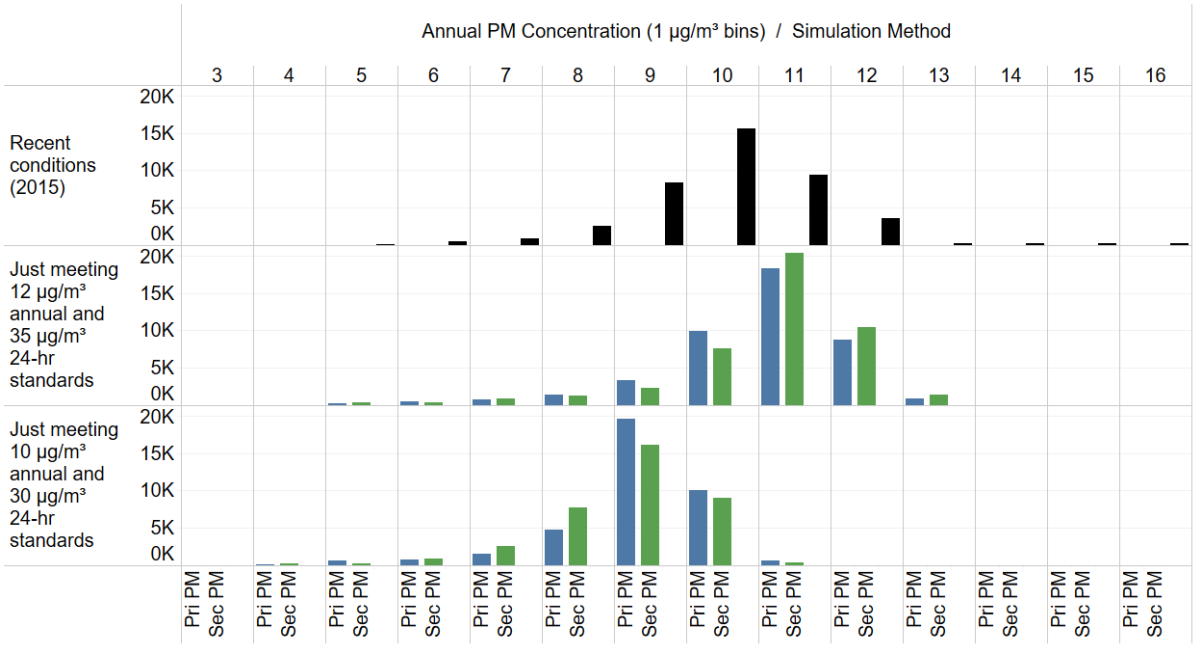


Figure C-25. Distribution of estimated $PM_{2.5}$-associated mortality for recent conditions (2015), current standards (12/35 µg/m^3), and alternative standards (10/30 µg/m^3) simulated for all 47 urban study areas.[42]

[42] Risk is rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m^3 includes risk occurring 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 recent conditions risk estimates.

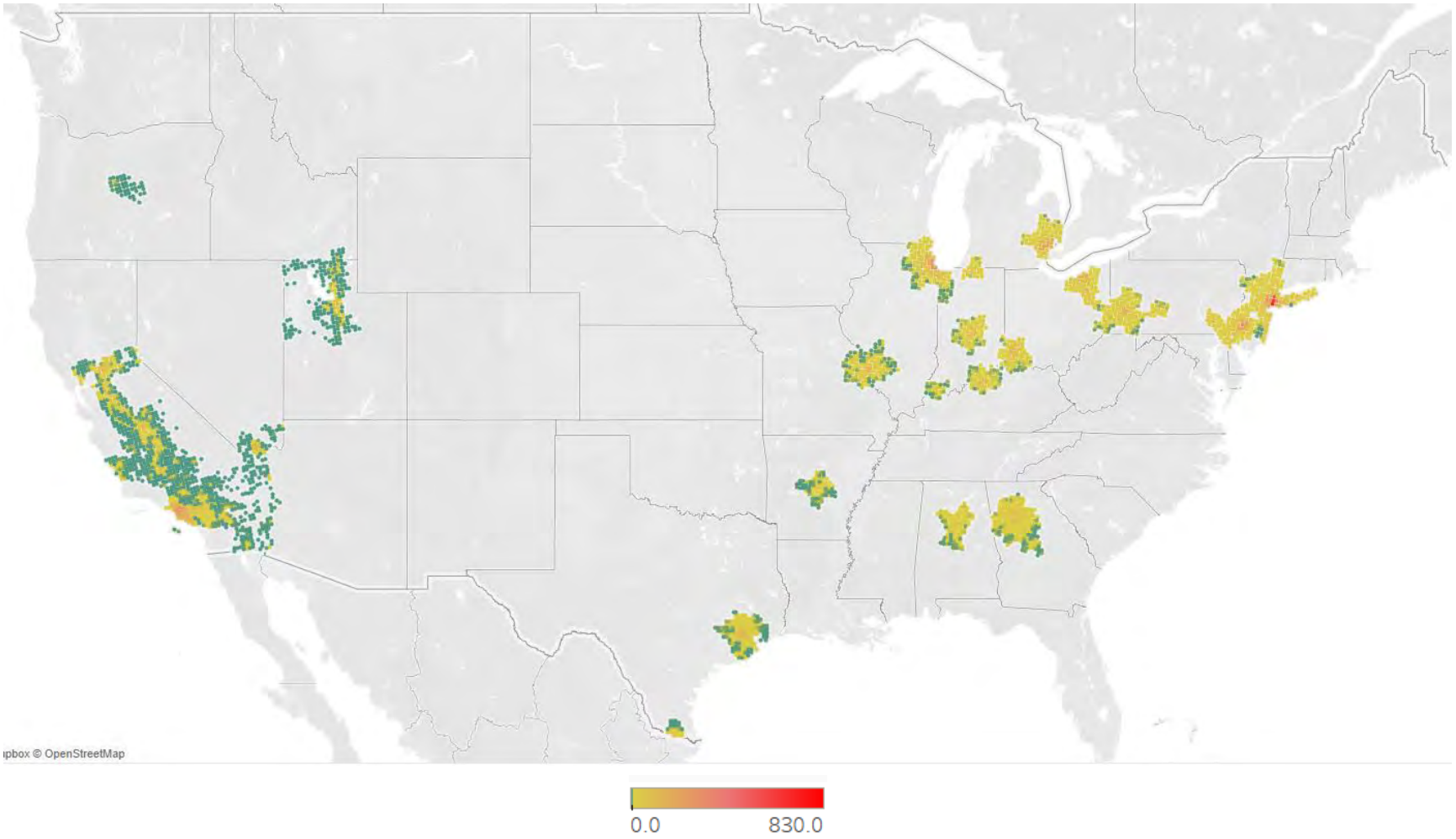


Figure C-26. Estimated number of premature deaths (by 12 km grid cell) under 2015 recent conditions in all 47 study areas.

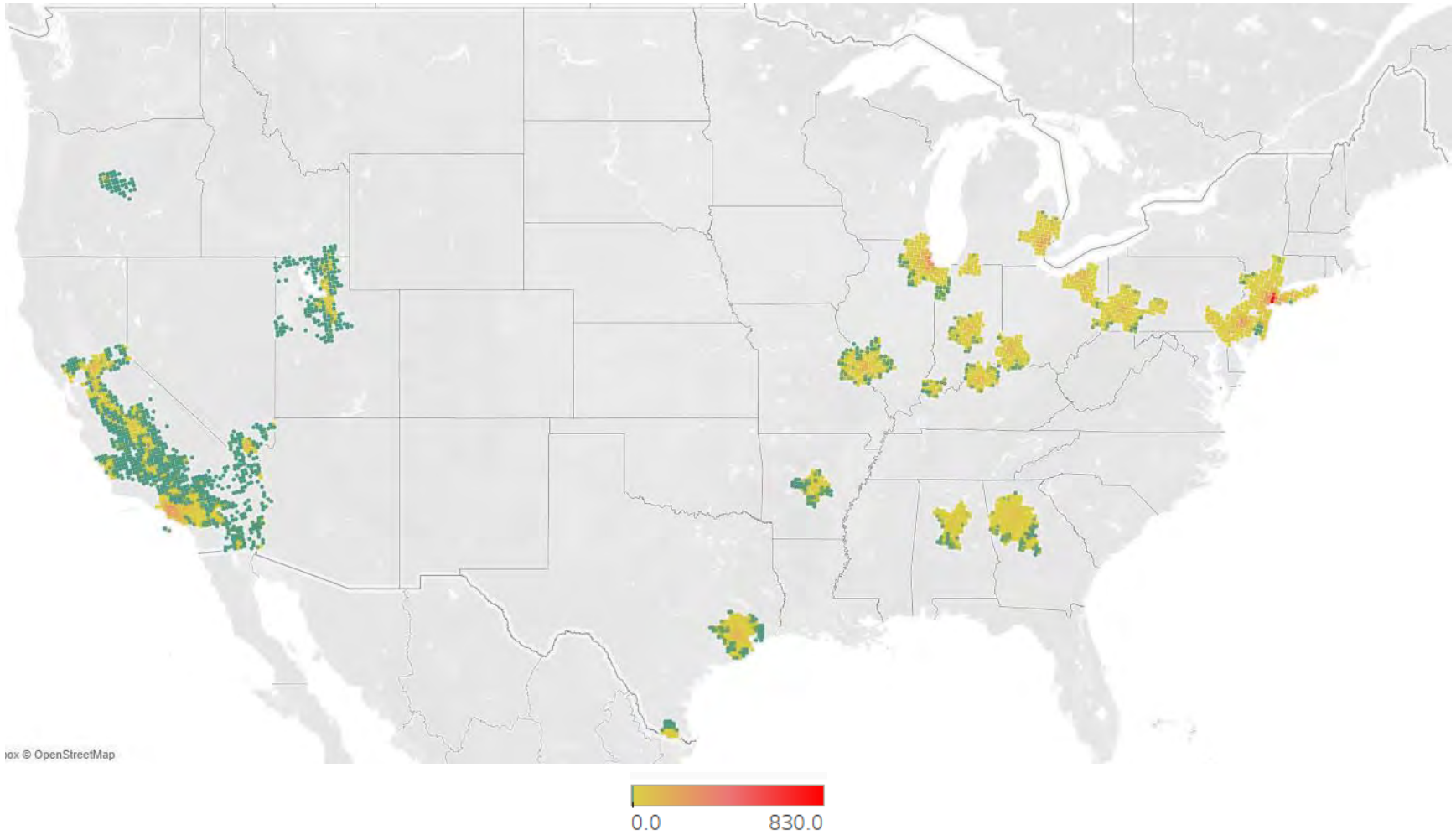


Figure C-27. Estimated number of premature deaths (by 12 km grid cell) when just meeting the current PM standards (12/35) in all 47 study areas (Pri-PM simulation).

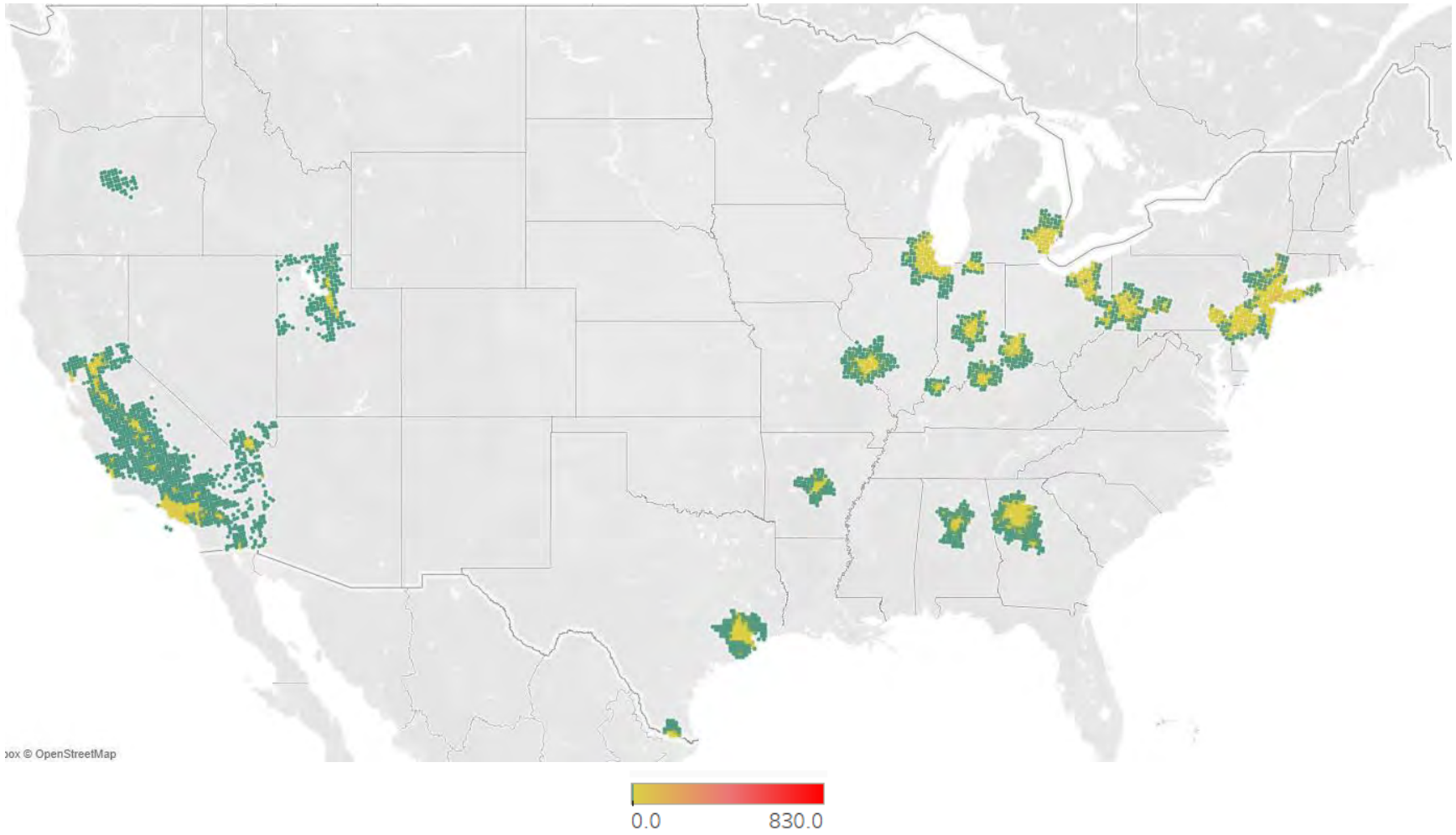


Figure C-28. Estimated reduction in the number of premature deaths (by 12 km grid cell) when going from just meeting the current standards (12/35) to just meeting the alternative standards (10/30) in all 47 study areas (Pri-PM simulation).

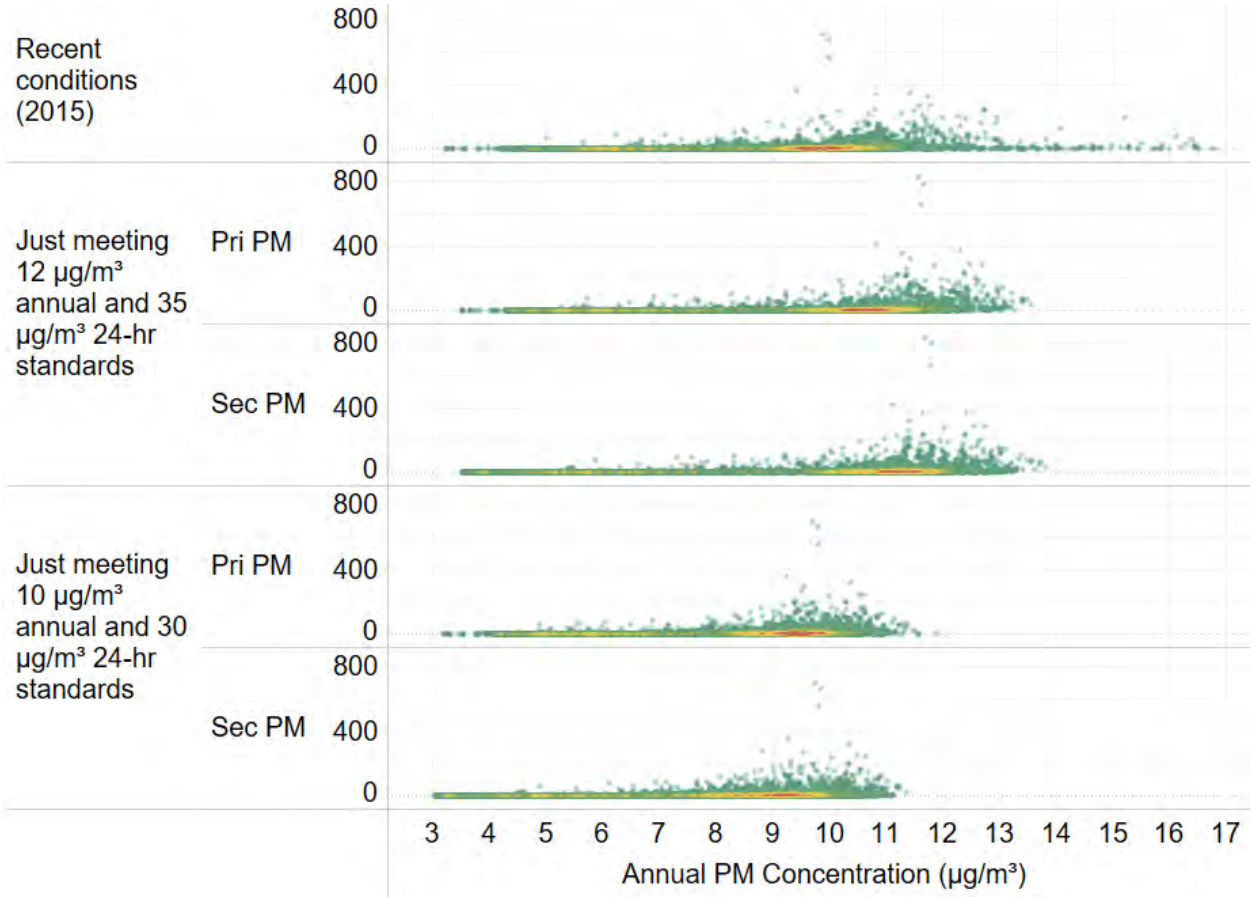


Figure C-29. Distribution of estimated premature death (by 12 km grid cell) for the current standards (12/35 µg/m^3), alternative standards (10/30 µg/m^3), and recent conditions (2015) for all 47 urban study areas (Pri-PM simulation).

C.2.2 Results from Set of 30 Study Areas controlled by the Annual Standard

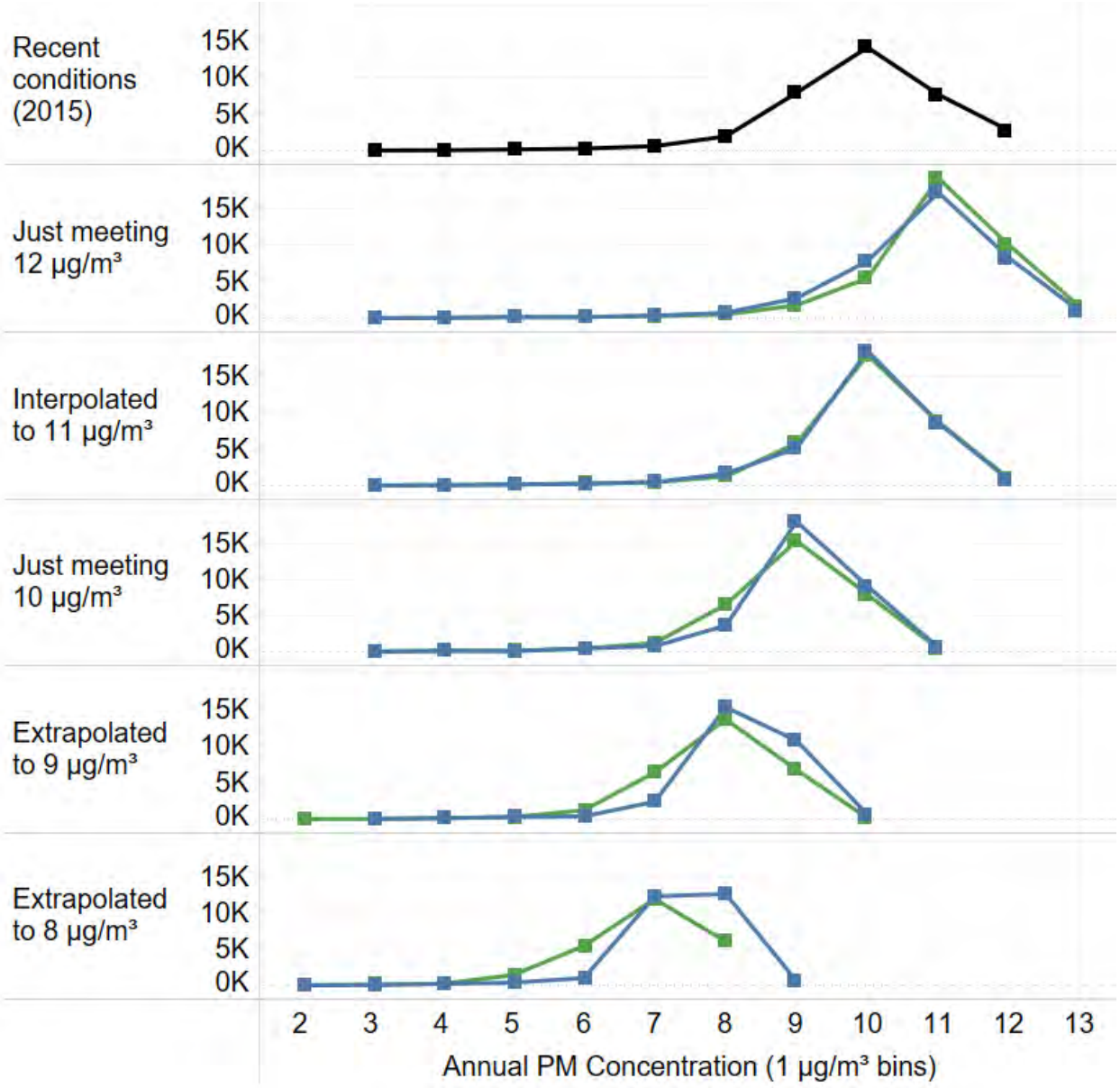


Figure C-30. Distribution of estimated $PM_{2.5}$-associated mortality for recent conditions (2015), the current annual standard (12/35 µg/m^3), and alternative standards (8.0, 9.0, 10.0, and 11.0 µg/m^3) simulated for the 30 annual-controlled urban study areas (blue and green bars represent the Pri-$PM_{2.5}$ and Sec-$PM_{2.5}$ estimates, respectively).[43]

[43] Risk is rounded toward zero into whole $PM_{2.5}$ concentration values (e.g., risk estimate at 10 µg/m^3 includes risk occurring 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 recent conditions risk estimates.

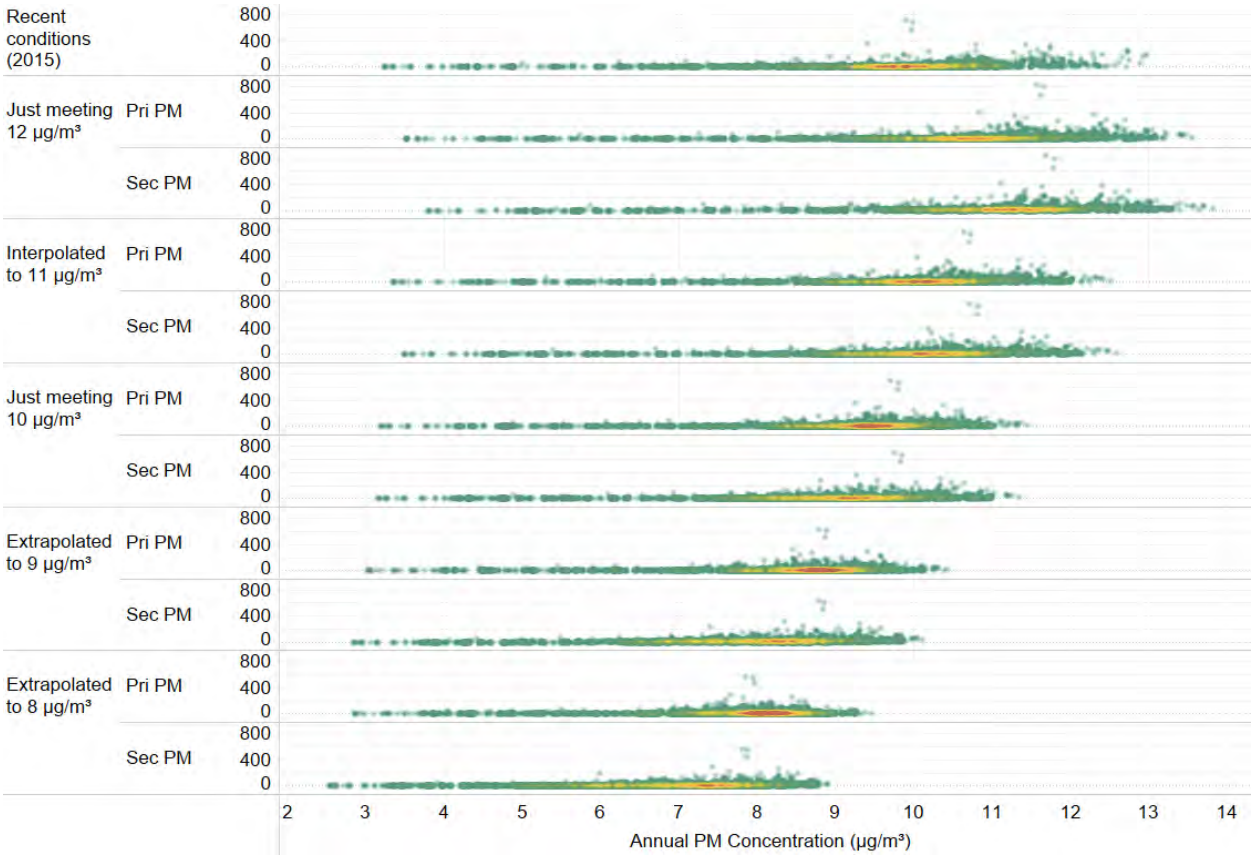


Figure C-31. Distribution of estimated premature death (by 12 km grid cell) 47 urban study areas (Pri-PM simulation) for recent conditions (2015), the current annual standard (12.0 µg/m^3), alternative annual standards (8.0, 9.0, 10.0, 11.0 µg/m^3).

C.2.3 Key Observations from the Suppmental Risk Results

Review of the distributional risk estimates presented in section C.2 further support the key observations presented in draft PA section 3.4.2. Briefly, these observations include:

- Across the full set of alternative annual standards modeled including 11.0, 10.0, 9.0, and 8.0 µg/m^3 (each evaluated for the 30 annually-controlled study areas), we see a consistent reduction in mortality (Figure C-30 and Figure C-31). 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 (8.0 µg/m^3) the majority of remaining risk occurs in grid cells with ambient $PM_{2.5}$ concentrations between 6 and 9 µg/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 µg/m^3 (Figure C-29).
- Patterns of risk reduction seen in the summary (aggregated) risk results tables presented both in draft PA section 3.4 and this appendix are driven by considerable underlying

variability across both CBSAs and across the 12km grid-level risk estimates. Specifically, if we consider the maps and scatter plots presented in section C.2, we see considerable spread (i.e., variability) in the grid-level risk estimates. We note that this underlying variability in risk 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-29 and Figure C-31), 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 occurring) concentrations (orange and red dots) as well as cells with concentrations that occur relatively infrequently (green dots). 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 ADDITIONAL TECHNICAL DETAIL ON THE AT-RISK ANALYSIS

Our consideration of estimated risks among potentially at-risk populations in the draft PA focuses on addressing the following policy-relevant questions:

- **How does $PM_{2.5}$ exposure and risk compare between demographic groups when air quality just meets the current and potential alternative primary $PM_{2.5}$ annual standards?**
- **To what extent are risks estimated to decline within each demographic group when air quality is adjusted to just meet potential alternative annual standards with lower levels?**

Estimating $PM_{2.5}$ exposure and risk within various demographic populations when just meeting the current or alternative annual standard or moving from the current annual standard to an alternative annual standard requires multiple input parameters and several simplifying assumptions. An overall summary of the analytical components is provided in Table C-11 and below we discuss in detail the various data inputs and assumptions associated with the at-risk analysis presented in the draft PA.

Table C-11. Summary of At-Risk Analysis Variables[a]

Race/Ethnicity	Concentration-Response Function	Baseline Incidence Rate
1. White 2. Black 3. Asian 4. Hispanic 5. (Non-Hispanic) 6. (All)	1. (Overall function) 2. Race/Ethnicity-stratified functions	1. (Overall baseline incidence rate) 2. Race/ethnicity-stratified baseline incidence rates

[a] Parentheses indicate the variable was used in sensitivity analyses only.

C.3.1 Race/Ethnicity

As the 2019 ISA and the draft ISA Supplement noted strong support for non-White populations, and particularly Black/African American populations, being at increased risk from $PM_{2.5}$-related health effects, in part due to disparities in exposure, we focused on comparing exposure and risk in Black and White populations. We also included exposure and risk information from Asians, Native Americans, and Hispanics, although there is less evidence in the PM ISAs that those demographic groups are at increased risk of $PM_{2.5}$ -related health effects or experience disparities in $PM_{2.5}$ exposure (U.S. EPA, 2019, U.S. EPA, 2021a).

Population information for each demographic group from both the at-risk assessment population and the original cohort population can be found in Table C-12. In general, the proportions of White, Black, and Native American people in the Di et al., 2017 study were comparable to the proportions in the 47 urban study areas, though a slightly higher proportion of the population in the 47 areas was White. In contrast, the Asian and Hispanic subpopulations represented a smaller proportion of the Di et al., 2017 cohort than the respective population proportions in the 47 areas. Importantly, the 0.3% of Native Americans assessed by Di et al., 2017 equates to approximately 180,000 individuals, which is nearly a third of the ACS cohort (Turner et al., 2016).

Table C-12. Demographic populations aged 65 and over residing in the full set of 47 study areas, the subset of 30 study areas controlled by the annual standard, and the original cohort.

Ethnicity & Race	Population in 47 Areas	Percent of Population in 47 Areas	Population in 30 Areas	Percent of Population in 30 Areas	Percent of Population in Di et al., 2017 cohort
White	10,560,891	80.0	8,756,815	78.6	85.4
Black	1,655,695	12.6	1,551,743	13.9	8.7
Asian	927,966	7.0	801,487	7.2	1.8
Native American	51,263	0.4	36,477	0.3	0.3
Non-Hispanic	11,647,164	88.3	9,897,164	88.8	-
Hispanic	1,548,639	11.7	1,249,353	11.2	1.9

C.3.2 Concentration-Response Functions

The following eight epidemiologic long-term exposure studies of $PM_{2.5}$ exposure and all-cause/nonaccidental/total mortality in nonwhite populations were identified in the 2019 ISA and draft ISA Supplement, met the minimum criteria discussed in the *Estimating $PM_{2.5}$ and Ozone-Attributable Health Benefits TSD* (U.S. EPA, 2019, U.S. EPA, 2021a, U.S. EPA, 2021b), and were considered for inclusion in the at-risk assessment: Awad et al., 2019, Di et al., 2017, Kioumourtzoglou et al., 2016, Parker et al., 2018, Lipfert and Wyzga, 2020, Son et al., 2020, Wang et al., 2017, and Wang et al., 2020. Summary information regarding these eight studies is available in Table C-13. Consistent with the main risk assessment, we focused on long-term exposure studies so as to not double-count effects of short-term exposures. No mortality studies for the at-risk group of children met the initial screening criteria.

Table C-13. Summary information for available epidemiology studies of nonwhite populations considered for the at-risk assessment.

Study	Cohort	Study Location	Health Outcome	Study Size	Health Years	Air Quality Years	Ages	Exposure Method
Awad et al., 2019	Medicare enrollees	National US	All-cause mortality	12,095,504 movers	2000-2012	2000-2012	>64	Hybrid
Di et al., 2017	Medicare enrollees	National US	All-cause mortality	60,925,443 persons; 22,567,924 deaths	2000-2012	2000-2012	>64	Hybrid or Monitor
Kioumourtzoglou et al., 2016	Medicare enrollees	National US (207 US cities)	All-cause mortality	35,295,005 subjects; 11,411,282 deaths	2000–2010	2000–2010	>64	Monitor
Lipfert and Wyzga, 2020	Veterans	31 VA clinics across 27 states	Mortality risk	Approximately 700,000 males	1976-2001	1999-2001	Average age at entry approximately 52	Hybrid or Monitor
Parker et al., 2018	NHIS	National US	All-cause mortality	657,238 adults	1997-2009	2004	>24	Hybrid
Son et al., 2020	North Carolina residents	North Carolina	Total mortality	775,338 cases (i.e., total deaths) with 3,410,015 control days	2002-2013	2002-2013	All	Hybrid or Monitor
Wang et al., 2017	Medicare enrollees	7 U.S. southeast states: AL, FL, GA, MS, NC, SC, TN	All-cause mortality	13.1 million Medicare beneficiaries; 4.7 million deaths	2000-2013	2000-2013	>64	Hybrid
Wang et al., 2020	Medicare enrollees	National US	Non-accidental mortality	52,954,845 Medicare beneficiaries; 15,324,059 deaths	2000-2008	2000-2008	>64	Hybrid

We evaluated the available studies and concentration-response functions to determine if sufficient information exists for use in a quantitative analysis and to determine which study or studies best characterizes at-risk populations across the U.S. Of the available studies from the 2019 ISA, Di et al., 2017 was the largest nationwide study, covered one of the most recent and longest time spans, used a sophisticated exposure estimation technique, and provided sufficient information to apply risk models quantifying increased risks to the following demographic groups: White, Black, Asian, Native American, and Hispanic (Table C-14). Although effect estimates from Di et al., 2017 were derived from a cohort aged 65 and older and the study did not provide a non-Hispanic concentration-response function to directly compare to the Hispanic concentration-response function, it was identified as best characterizing populations potentially at increased risk of long-term $PM_{2.5}$-attributable all-cause mortality. Health impact functions, including beta parameters and standard errors (SE), were developed for each at-risk population demographic described by Di et al., 2017 and are available in Table C-14.

Table C-14. At-risk hazard ratios, beta coefficients, and standard errors from Di et al., 2017 used in this at-risk assessment.

Demographic Population	Risk of Death Associated with 10 $\mu g/m^3$ Increase in $PM_{2.5}$	Beta Coefficient (SE)
White	1.063 (1.060, 1.065)	0.0061 (0.0001)
All	1.073 (1.071, 1.075)	0.0070 (0.0001)
Hispanic	1.116 (1.100, 1.133)	0.0110 (0.0008)
Black	1.208 (1.199, 1.217)	0.0189 (0.0004)
Asian	1.096 (1.075, 1.117)	0.0092 (0.0010)
Native American	1.100 (1.060, 1.140)	0.0095 (0.0019)

C.3.3 Age

Concentration-response functions stratified by race and ethnicity from Di et al., 2017 were only available for ages 65-99. Therefore, this at-risk analysis only evaluated a single age range group of 65-99 years.

C.3.4 Baseline Incidence Rates

BenMAP-CE includes baseline incidence rates at the most geographically- and age-specific levels available for each health endpoint assessed. For many locations within the U.S., these data are resolved at the county- or state-level, providing a better characterization of the geographic distribution of mortality rates than the national-level rates. Race- and ethnicity-stratified baseline incidence rates from 2007-2016 Census data were recently improved for the all-cause mortality health endpoint, by adding the geographic level option of rural/urban state

between county-level and state-level (sections C.3.4.1 and C.3.4.2). Both overall and race/ethnicity-stratified baseline rates are used in this at-risk analysis (section C.3.4.2).

C.3.4.1 Race-Stratified Baseline Incidence Rates

To estimate race-stratified and age-stratified incidence rates at the county level, we downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC WONDER mortality database.[44] Race-stratified incidence rates were calculated for the following age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, 75-84 years, and 85+ years. To address the frequent county-level data suppression for race-specific death counts, we stratified the county-level data into two broad race categories, White and Non-White populations. In a later step, we stratified the non-White incidence rates by race (Black, Asian, Native American) using the relative magnitudes of incidence values by race at the regional level, described in more detail below.

We followed methods outlined in Section D.1.1 of the BenMAP User Manual with one notable difference in methodology; we included an intermediate spatial scale between county and state for imputation purposes.[45] We designated urban and rural counties within each state using CDC WONDER and, where possible, imputed missing data using the state-urban and state-rural classifications before relying on broader statewide data. We followed methods for dealing with suppressed and unreliable data at each spatial scale as described in Section D.1.1.

A pooled non-White incidence rate masks important differences in mortality risks by race. To estimate county-level mortality rates by individual race (Black, Asian, Native American), we applied regional race-specific incidence relationships to the county-level pooled non-White incidence rates. We calculated a weighted average of race-specific incidence rates using regional incidence rates for each region/age/race group normalized to one reference population (the Asian race group) and county population proportions based on race-specific county populations from CDC WONDER where available. In cases of population suppression across two or more races per county, we replaced all three race-specific population proportions derived from CDC WONDER with population proportions derived from 2010 Census data in BenMAP-CE (e.g., 50 percent Black, 30 percent Asian, 20 percent Native American).

C.3.4.2 Ethnicity-Stratified Baseline Incidence Rates

To estimate ethnicity-stratified and age-stratified incidence rates at the county level, we downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC

[44] https://wonder.cdc.gov/

[45] https://www.epa.gov/sites/default/files/2015-04/documents/benmap-ce_user_manual_march_2015.pdf

WONDER mortality database.[46] Ethnicity-stratified incidence rates were calculated for the following age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, 75-84 years, and 85+ years. We stratified county-level data by Hispanic origin (Hispanic and non-Hispanic). We followed the methods outlined in Section D.1.1 to deal with suppressed and unreliable data. We also included an intermediate spatial scale between county and state designating urban and rural counties for imputation purposes, described in detail in Section D.1.3 of the BenMAP User Manual. [47]

C.3.5 Selection of Air Quality Simulation Approach

Concentration fields associated with just meeting the current and alternative standards in the 47 urban study areas were based on adjusting 2015 modeled concentrations using CMAQ sensitivity modeling with emission reductions applied throughout the modeling domain. This approach was applied to develop realistic concentration fields that correspond to just meeting standards in the 47 areas. Two distinctly different emission cases were used (Pri-PM and Sec-PM) to examine the sensitivity of results to the air quality adjustment approach. For characterizing risk in at-risk populations, we used air quality fields from the Pri-PM adjustment case alone. In the Pri-PM case, the air quality adjustments for a given area are largely associated with emission reductions within that area due to the local nature of air quality impacts from primary PM sources. For the Sec-PM case, the air quality adjustments may be strongly associated with sources located outside of the area. Since the at-risk calculations are performed for population groups within the 47 urban study areas alone, the Pri-PM adjustment case (in which air quality adjustments are primarily associated with emission sources within the 47 areas) is most appropriate for the at-risk analysis.

C.4 SUPPLEMENTAL AT-RISK RESULTS

Absolute numbers of all-cause premature mortality cases within each racial and ethnic population demographic are available in Table C-15 for total attributable burden under either the current or alternative standards and Table C-16 for the change in risk estimates when moving from the current to a potential alternative annual standard for both the full set of 47 urban study areas and the subset of 30 annually-controlled areas.

[46] https://wonder.cdc.gov/

[47] https://www.epa.gov/sites/default/files/2015-04/documents/benmap-ce_user_manual_march_2015.pdf

Table C-15. Estimates of total $PM_{2.5}$-associated mortality by demographic population for air quality adjusted to just meet the current or alternative standards.

Study Areas	Modeling Scenario	Ethnicity & Race				
		White	Black	Hispanic	Asian	Native American
47 areas	Just meeting 12/35 µg/m³	29,400 (28,200 to 30,400)	13,600 (13,100 to 14,100)	4,850 (4,220 to 5,460)	1,930 (1,530 to 2,300)	125 (77.9 to 169)
	Just meeting 10/30 µg/m³	25,200 (24,300 to 26,200)	11,700 (11,300 to 12,100)	4,160 (3,610 to 4,680)	1,650 (1,310 to 1,970)	108 (66.9 to 146)
30 areas	Just meeting 12/35 µg/m³	24,900 (23,900 to 25,800)	12,800 (12,400 to 13,300)	3,970 (3,450 to 4,460)	1,640 (1,300 to 1,960)	87.9 (54.6 to 119)
	Interpolated to 11 µg/m³	23,100 (22,200 to 24,000)	11,900 (11,500 to 12,400)	3,680 (3,200 to 4,140)	1,520 (1,210 to 1,820)	81.5 (50.6 to 110)
	Just meeting 10/30 µg/m³	21,300 (20,500 to 22,100)	11,000 (10,600 to 11,400)	3,380 (2,940 to 3,810)	1,400 (1,110 to 1,670)	75.1 (46.5 to 102)
	Extrapolated to 9 µg/m³	19,600 (18,800 to 20,300)	10,100 (9,740 to 10,500)	3,090 (2,680 to 3,480)	1,280 (1,010 to 1,530)	68.6 (42.4 to 93.0)
	Extrapolated to 8 µg/m³	17,800 (17,100 to 18,400)	9,180 (8,840 to 9,510)	2,790 (2,420 to 3,140)	1,150 (913 to 1,380)	62.0 (38.3 to 84.3)

Table C-16. Change in $PM_{2.5}$-associated mortality by demographic population for air quality adjusted to just meet the current or alternative standards.

Study Areas	Modeling Scenario	Ethnicity & Race				
		White	Black	Hispanic	Asian	Native American
47 areas	12/35-10/30 µg/m³	4,380 (4,200 to 4,540)	2,280 (2,190 to 2,370)	771 (665 to 872)	302 (238 to 364)	18.9 (11.6 to 26.0)
30 areas	12/35-11 (interpolated) µg/m³	1,890 (1,810 to 1,960)	1,090 (1,050 to 1,130)	327 (282 to 371)	133 (104 to 160)	7.04 (4.29 to 9.68)
	12/35-10/30 µg/m³	3,760 (3,610 to 3,900)	2,170 (2,080 to 2,250)	652 (563 to 737)	264 (208 to 319)	14.0 (8.57 to 19.3)
	12/35-9 (extrapolated) µg/m³	5,630 (5,410 to 5,840)	3,220 (3,100 to 3,340)	973 (840 to 1,100)	395 (311 to 476)	21.0 (12.8 to 28.7)
	12/35-8 (extrapolated) µg/m³	7,490 (7,190 to 7,770)	4,260 (4,090 to 4,420)	1,290 (1,120 to 1,460)	525 (414 to 631)	27.8 (17.0 to 38.1)

For visual purposes only the central risk estimates are included in the at-risk results presented in chapter 3 of the draft PA (section 3.4.2), but an example of the 95th percentile confidence interval (CI) risk estimate spans resulting from the epidemiologic concentration-response functions are provided in Figure C-32. The lower open circle represents the 2.5th percentile and the higher open circle represents the 97.5th percentile CI for each population demographic. CIs are derived from the concentration-response relationships presented in Di et al., 2017 (Figure C-13). While the Hispanic and Native American risk rate CIs often overlap, the Black risk rate estimates are consistently higher than the White risk rates, and the Asian risk rates are consistently lower than the White risk rates (Figure C-32).

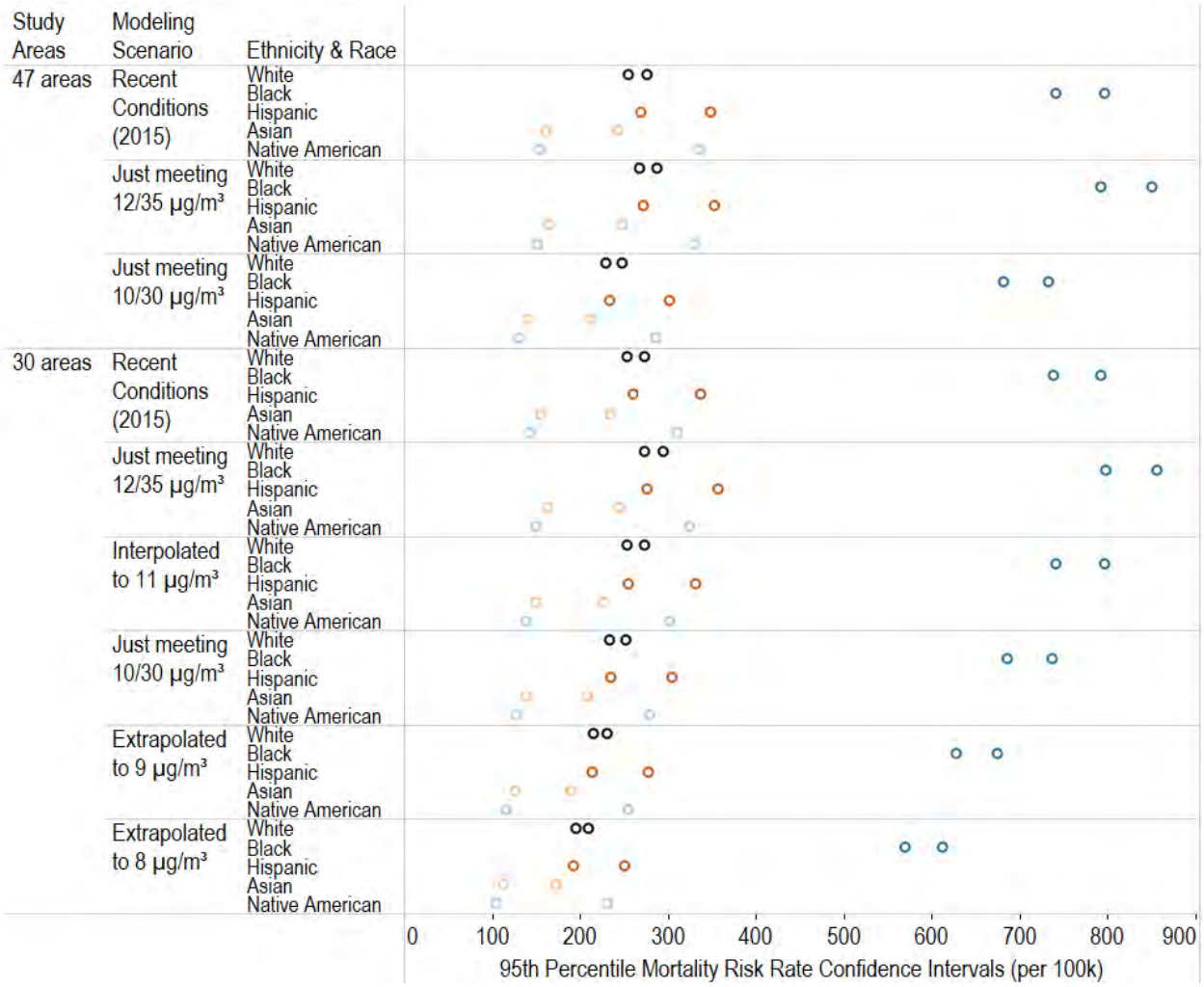


Figure C-32. Race- and ethnicity-stratified 95th percentile (2.5th percentile to 97.5th percentile) confidence interval risk estimates for recent conditions (2015), the current standard, and potential alternative standard air quality surfaces.

As the risk rate calculation integrates both population-specific baseline incidence rates and concentration-response relationships with exposure information, we wanted to separate the impacts of each data input. To distinguish the impacts of race-stratified concentration-response functions from baseline incidence rates on the results, we provide the average $PM_{2.5}$-attributable risk by demographic population in the full set of 47 urban study areas for the current standards, potential alternative standards, and recent condition (2015) air quality surfaces within each demographic group. Figure C-33 and Figure C-34 provide this information when just meeting current and alternative standards or shifting between the current and potential alternative annual standards, respectively.

Generally, race-stratified concentration-response functions increased the population-normalized risk estimated in nonwhite populations, with the greatest magnitude increase occuring in Black populations, followed by Hispanic populations, and decreased risk estimated in White populations. Di et al., 2017 did not provide a concentration-response function for the non-Hispanic population, so only the overall concentration-response function was applied to non-Hispanics in these supplemental analyses.

Many factors effect race/ethnicity-stratified baseline incidence rates, such as access to medical care, socioeconomic status, and underlying health issues. As such, race/ethnicity-stratified baseline incidence rates impacted by each race and ethnicity differently. Race/ethnicity-stratified baseline incidence rates increased risk estimates substantially in Black populations and slightly in White and non-Hispanic populations. In contrast, race/ethnicity-stratified baseline incidence rates decreased risk rates estimated in Hispanic, Asian, and Native American populations.

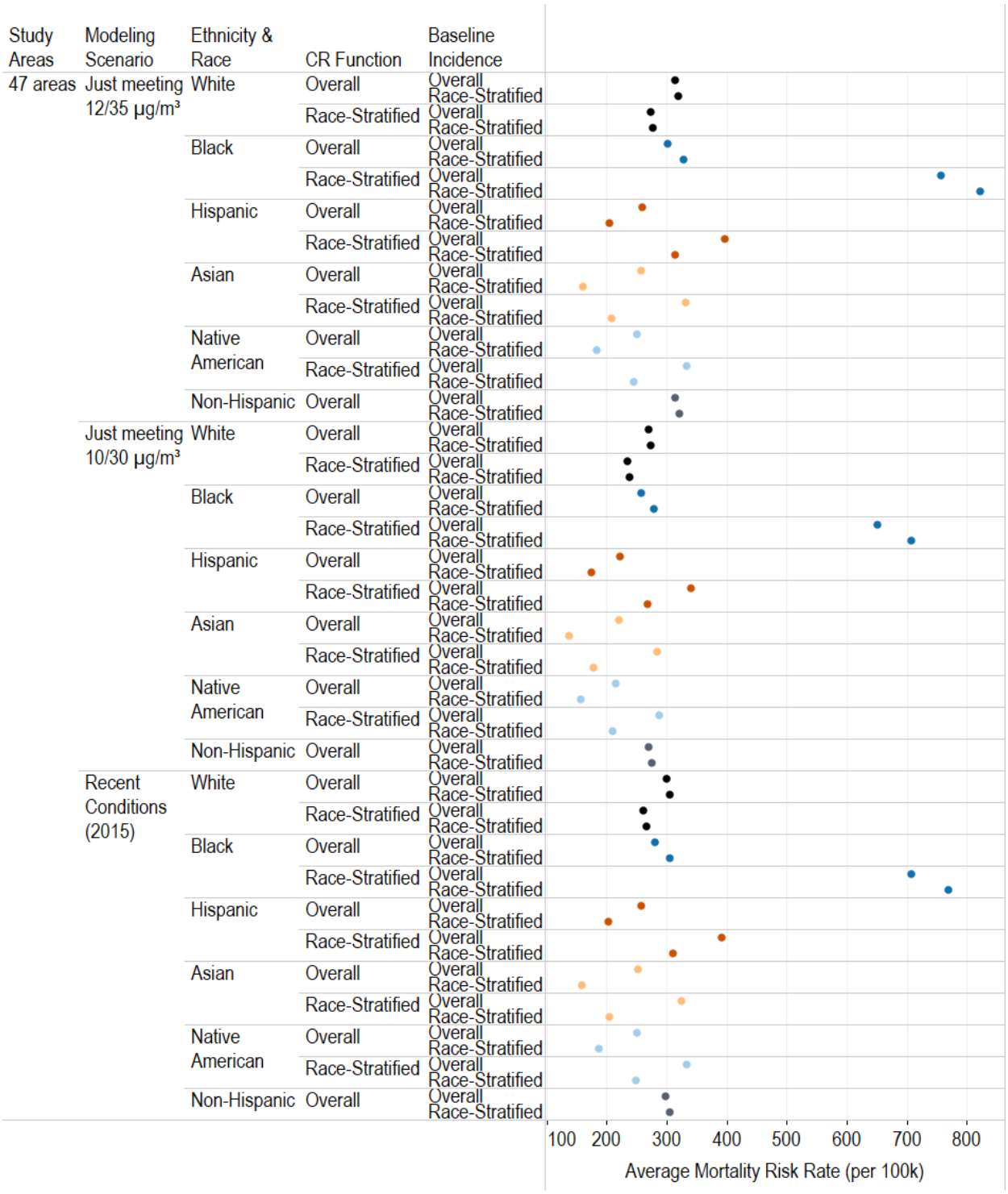


Figure C-33. Effect of race-stratified concentration-response (CR) functions and baseline incidence rates on the average $PM_{2.5}$-attributable risk by demographic population in the 47 study areas for the current standard, potential alternative standard, and recent conditions (2015) air quality surfaces within each demographic group.

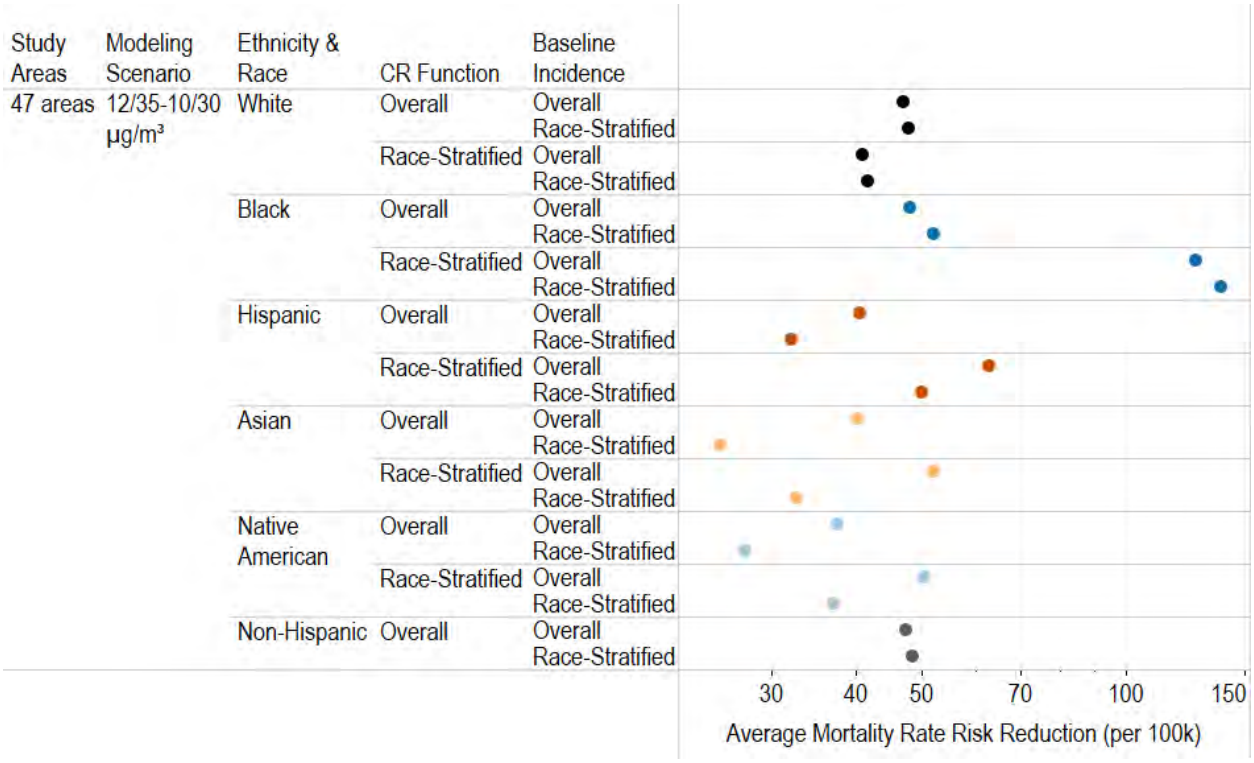


Figure C-34. Effect of race-stratified CR functions and baseline incidence rates on the average $PM_{2.5}$-attributable risk reductions by demographic population in the 47 study areas when shifting from the current to the potential alternative standards within each demographic group.

As the annual design values for many study areas required rolling up to just meet the current standard (section C.1.4.6), for informational purposes we provide cumulative distribution plots of $PM_{2.5}$ exposure and $PM_{2.5}$-attributable mortality risk per 100,000 people by demographic group for the recent condition year 2015, along with the plots for just meeting the current standards for direct comparison (Figure C-35). Several caveats should be noted when comparing the recent conditions air quality surface to those adjusted to just meet current or recent air quality conditions. Importantly, the at-risk analysis focuses on the Pri-PM adjustment approach (section C.3.4.2), in which emission increases in areas below the current standard occur predominately at and around the urban cores of the study areas. This could lead to a simulated increase of disproportionate $PM_{2.5}$ exposures in demographic populations that frequently reside at and around the urban core. Conversely, disproportionate $PM_{2.5}$ concentrations in demographic populations residing in areas above the current standards may be obscured when concentrations are adjusted downward to just meet the current standard.

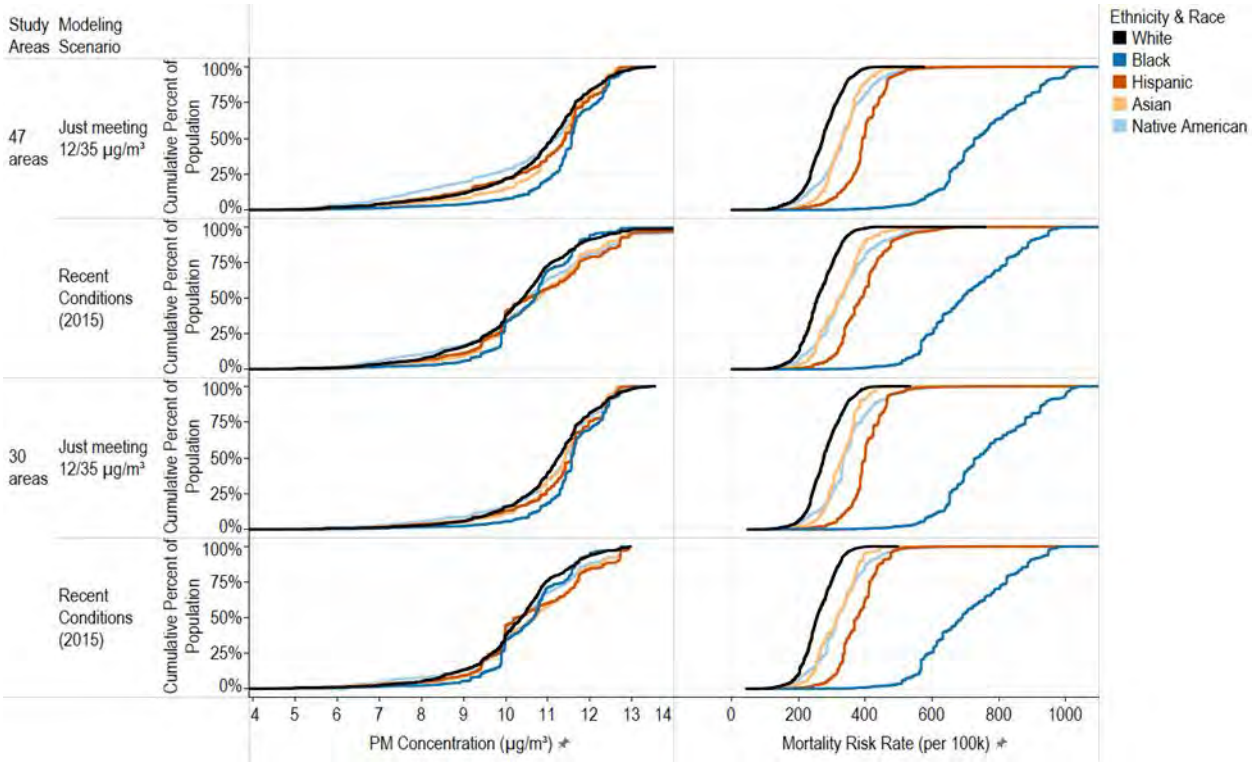


Figure C-35. $PM_{2.5}$ concentrations and $PM_{2.5}$-attributable risk by demographic population for recent air quality conditions (2015) and air quality simulated to just meet the current PM standards.

Another aspect of lowering the annual $PM_{2.5}$ standard is the percent of overall risk attributable to $PM_{2.5}$ exposure. Table C-17 shows that the percent of baseline risk is higher in racial/ethnic minority demographics in all scenarios analyzed. Additionally, some minority populations may experience a greater decrease in the percent of baseline $PM_{2.5}$-attributable risk.

Table C-17. Percent of mortality baseline incidence attributable to $PM_{2.5}$ under the current and potential alternative standards.

Ethnicity & Race	% of Baseline $PM_{2.5}$-Attributable Risk Under the Current Standard (12/35)		% of Baseline $PM_{2.5}$-Attributable Risk Under an Alternative Standard (11)	% of Baseline $PM_{2.5}$-Attributable Risk Under an Alternative Standard (10/30)		% of Baseline $PM_{2.5}$-Attributable Risk Under an Alternative Standard (9)	% of Baseline $PM_{2.5}$-Attributable Risk Under an Alternative Standard (8)
	47 areas	30 areas	30 areas	47 areas	30 areas	30 areas	30 areas
White	6	7	6	5	6	5	5
Black	19	20	18	17	17	15	14
Hispanic	11	12	11	10	10	9	8
Asian	10	10	9	8	8	8	7
Native American	9	10	9	8	9	8	7

C.5 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 and 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 characterize the remaining risk estimates uncertainty more explicitly. 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 the 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 reconsideration 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.5.1. Those sources of uncertainty addressed qualitatively in the risk assessment are discussed in section C.5.2. 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.5.1 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 associated with the underlying effect estimate (i.e., a measure of the statistical precision of the effect estimate). There is variation in the range of 95 percent CIs associated with the point estimates generated for this analysis, with some CR functions displaying substantially greater variability than others (e.g., Ito et al., 2013, tables in section 3.4.2 of the 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 multiple mortality estimates reflecting variation in CR functions across studies*: For mortality endpoints, we include risk estimates reflecting multiple epidemiology studies and associated study designs (e.g., age ranges, methods for controlling potential confounders). In some instances, we find that the CR function used

has only a small impact on risk estimates(e.g., Turner et al., 2016 and Di et al., 2017). 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. In most instances in this risk assessment, the CR function used has only a small impact on risk estimates (e.g., Di et al., 2017). 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:* Two methods are employed to adjust air quality in order to simulate just meeting the current and alternative standards, which could represent potential bounding scenarios of $PM_{2.5}$ concentrations changes across the study area (i.e., the Pri-PM-based method and the Sec-PM based method). Our evaluation of these methods reflects the fact that there is both 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 3.4.2 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% (draft PA section 3.4.2).

C.5.2 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-18).

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 reconsideration. 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-18. Qualitative analysis of sources of uncertainty and assessment of potential impact on risk assessment.

Source of Uncertainty	Description	Direction	Magnitude	Comments
Shape and corresponding statistical uncertainty around the CR function for long-term and short-term exposure-related mortality (especially at lower ambient PM levels)	Interpreting the shapes of concentration-response relationships, particularly at $PM_{2.5}$ concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and "linearize" population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015, section 6.c).	Both	Medium-High	With regard to long-term exposure-related (nonaccidental) mortality, the ISA concludes that the majority of evidence supports a linear, no-threshold concentration-response relationship, though there is initial evidence indicating that the slope of the concentration-response curve may be steeper at lower concentrations for cardiovascular mortality (U.S. EPA, 2019, section 1.5.3.2). For long-term exposure-related mortality, the ISA notes that there is less certainty in the shape of the concentration-response curve at mean annual $PM_{2.5}$ concentrations generally below 8 μg/m^3 because data density is reduced below this concentration (2019 ISA, section 11.2.4). 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 μg/m^3.
Representing population-level exposure with 12 km grid	As with long-term exposure-related mortality, short-term exposure-related mortality endpoints were also modeled	Both	Medium-High	Three studies providing effect estimates for short-term exposure-related mortality in the risk assessment all utilized some form of urban-level

Source of Uncertainty	Description	Direction	Magnitude	Comments
cell spatial framework (in context of modeling short-term exposure-related mortality)	using the same 12 km grid cell template. The disconnect between the spatial template used in the underlying short-term epidemiology studies and the 12 km grid template used in the risk assessment introduces uncertainty into risk estimates.			spatial unit in characterizing exposure (e.g., Baxter et al. (2017) utilizes the CBSA, Ito et al. (2013), utilizes the MSA), which are larger (less spatially differentiated) in general than the 12 km grid cells used in modeling risk. This means that we are generally modeling short-term exposure-related mortality at a finer level of spatial resolution in the risk assessment than reflected in the epidemiology studies supplying the effect estimates, which does introduce uncertainty into the analysis.
Representing population-level exposure with 12 km grid cell spatial framework (in context of modeling long-term exposure-related mortality)	The risk assessment utilizes a 12 km grid structure in modeling risk. A source of uncertainty associated with this approach is the mismatch between the 12 km grid cell framework and the exposure estimation approaches used in the epidemiology studies providing effect estimates for the risk assessment. This mismatch can introduce additional exposure error to risk estimates, beyond the error inherent to the underlying epidemiologic study.	Both	Medium	There are a variety of spatial templates used across the epidemiology studies providing CR functions used in the risk assessment and none of them are an exact match with the 12 km grid cell template used in the risk assessment. Jerrett et al. (2013), Pope et al. (2015)Differences between the exposure metric used in the risk assessment and those used in the underlying epidemiologic studies introduce uncertainty into risk estimates.
Simulating just meeting alternative annual standards with levels of 8.0, 9.0, and 11.0 ug/m^3 using linear extrapolation/ interpolation	The use of extrapolation/ interpolation in simulating just meeting annual standards introduces uncertainty into the risk assessment since this approach does not fully capture potential non-linearities associated with the formation of secondary $PM_{2.5}$.	Both	Medium	Extrapolation to generate the surfaces for 9.0 and 8.0 μg/m^3 are subject to greater uncertainty than interpolation to 11.0 μg/m^3 (i.e., since the former estimates concentrations below those in modeled surfaces, while the latter estimates a surface between two sets of modeled results). In addition, linear extrapolation/interpolation based on the primary-PM modeled surfaces (for current standard and 10.0 μg/m^3) is likely subject to less uncertainty than extrapolation/interpolation based on the secondary-PM modeled surfaces since the latter focus on secondary formation which could involve a higher degree of non-linearity.

Source of Uncertainty	Description	Direction	Magnitude	Comments
Simulating just meeting current and alternative standards using model-based (Downscaler) methods	The baseline and adjusted concentration fields were developed using modeling to fill spatial and temporal gaps in monitoring and to explore air quality scenarios of policy interest. State-of-the-science modeling methods were used, but model-related biases and errors can introduce uncertainty into the $PM_{2.5}$ 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 adjust $PM_{2.5}$ concentrations to just meet standards.	This source of uncertainty could bias results in either direction.	Medium	Use of state-of-the-science modeling systems with the relative response factor adjustment approach provides confidence in the broad features of the simulated national $PM_{2.5}$ 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 relatively uncertain compared with broad features of the national $PM_{2.5}$ distributions.
Potential confounding of the $PM_{2.5}$-mortalty effect	Factors are considered potential confounders if demonstrated in the scientific literature to be related to health effects and correlated with PM. Omitting potential confounders from analyses could either increase or decrease the magnitude of $PM_{2.5}$ effect estimates (e.g., Di et al., 2017, Figure S2 in Supplementary Materials). Thus, not accounting for	Both	Medium	Long-term $PM_{2.5}$ exposure and mortality studies: For studies of long-term exposures, potential confounders are those that vary spatially or temporally. These may include socioeconomic status, race, age, medication use, smoking status, stress, noise, occupational exposures, and copollutant concentrations. Cohort studies used to characterize the $PM_{2.5}$-mortality relationship used a variety of approaches to account for these and other

Source of Uncertainty	Description	Direction	Magnitude	Comments
	confounders can introduce uncertainty into effect estimates and, consequently, into the risk estimates generated using those effect estimates. Confounders vary according to study design, exposure duration, and health effect. While a range of approaches to control for potential confounders have been adopted across the studies used in the risk assessment, and across the broader body of $PM_{2.5}$ epidemiologic studies assessed in the ISA, no individual study adjusts for all potential confounders.			potential confounders (e.g., see Appendix B). Across studies, a variety of study designs and statistical approaches have been used to account for potential confounding in the $PM_{2.5}$-mortality relationship. The fact that across this diverse body of evidence epidemiologic studies continue to report consistently positive associations that are often similar in magnitude, adds support the conclusion that the $PM_{2.5}$-mortality association is robust. Specifically regarding copollutants, the final PM ISA notes that, overall, associations remained relatively unchanged in copollutant models for total (nonaccidental) mortality, cardiovascular, and respiratory adjusted for ozone. 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. Short-term $PM_{2.5}$ exposure and mortality studies: For studies of short-term exposures, potential confounders are those that vary temporally. These may include meteorology (e.g., temperature, humidity), day of week, season, medication use, allergen exposure, copollutant concentrations, and long-term temporal trends. Some recent studies have expanded the examination of potential confounders, including long-term temporal trends, weather, and copollutants. Overall, the ISA concludes that alternative approaches to controlling for long-term temporal trends and for the potential confounding effects of weather may influence the magnitude of the association between $PM_{2.5}$ exposures and mortality, but have not been found to influence the direction of the observed association (U.S. EPA, 2019, section 11.1.5.1). With regard to

Source of Uncertainty	Description	Direction	Magnitude	Comments
				copollutants, recent studies conducted outside the U.S. provide additional evidence 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, 2019, Section 11.1.4).
Lag structure in short-term exposure-related mortality epidemiology studies	It can be challenging 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, we focus here on lags associated with all-cause mortality.
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 lead to differences in public health impacts (per unit concentration in ambient air) for $PM_{2.5}$, then uncertainty may be introduced into risk estimates that are based on concentration-response relationships for $PM_{2.5}$ mass.	Both	Low	The Integrated Synthesis chapter of the final ISA (Chapter 1, U.S. EPA, 2019) states that, the assessment of PM sources and components confirms and continues to support the conclusion from the 2009 PM ISA: Many $PM_{2.5}$ components and sources are associated with health effects, and the evidence does not indicate that any one source or component is more strongly related with health effects than $PM_{2.5}$ mass.
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 Jerrett et al. (2016), Pope et al. (2015).	Both	Low	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)), Thus, 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

Source of Uncertainty	Description	Direction	Magnitude	Comments
				temporal disconnect between the time-period associated with mortality and the ambient $PM_{2.5}$ level used in characterizing exposure would not be expected to introduce significant uncertainty into the epidemiology studies and associated effect estimates.
Exposure measurement error in epidemiologic studies assessing the relationship between mortality and exposure to ambient $PM_{2.5}$	Epidemiologic studies have employed a variety of approaches to estimate population-level $PM_{2.5}$ exposures (e.g., stationary monitors, hybrid modeling approaches). These approaches are based on using measured or predicted ambient $PM_{2.5}$ concentrations as surrogates for population exposures. As such, exposure estimates in epidemiologic studies are subject to exposure error. This error in the underlying epidemiologic studies contributes to uncertainty in the risk estimates that are based on concentration-response relationships in those studies.	Both	Low	Available studies indicate that $PM_{2.5}$ health effect associations are robust across various approaches to estimating $PM_{2.5}$ exposures. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates the potential for exposure error in epidemiologic studies, such error does not call into question the findings of key $PM_{2.5}$ epidemiologic studies. The ISA notes that, while bias in either direction can occur, exposure error tends to result in underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study Hart et al. (2015) reports that correction for $PM_{2.5}$ exposure error using personal exposure information results in a moderately larger effect estimate for long-term $PM_{2.5}$ exposure and mortality (though with wider confidence intervals). While most $PM_{2.5}$ epidemiologic studies have not employed similar corrections for exposure error, several studies report that restricting analyses to populations in close proximity to a monitor (i.e., in order to reduce exposure error) result in larger $PM_{2.5}$ effect estimates (e.g., Willis et al., 2003; Kloog et al., 2013). Thus, to the extent key $PM_{2.5}$ epidemiologic studies are subject to exposure error, correction for that error

Source of Uncertainty	Description	Direction	Magnitude	Comments
				would likely result in larger effect estimates, and thus larger estimates of $PM_{2.5}$-associated mortality incidence in the risk assessment.
Use of associations reported in epidemiologic studies to estimate how mortality incidence may change with changing $PM_{2.5}$ air quality.	The ISA's determination that the evidence supports a causal relationship between $PM_{2.5}$ exposure and mortality is based on assessing a broad body of evidence from epidemiologic and experimental studies. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate how mortality incidence may change with changing $PM_{2.5}$ air quality is subject to uncertainty.	Both	Low	The ISA assesses a longstanding body of health evidence supporting relationships between $PM_{2.5}$ exposures (short- and long-term) and mortality. Much of this evidence comes from epidemiologic studies conducted in North America, Europe, or Asia that demonstrate generally positive, and often statistically significant, associations between $PM_{2.5}$ exposures and total or cause-specific mortality. In addition, recent experimental evidence, as well as evidence from panel studies, strengthens support for potential biological pathways through which $PM_{2.5}$ exposures could lead to serious health outcomes, including mortality. While this broad body of evidence from across disciplines provides the foundation for the ISA's conclusions, the risk assessment necessarily focuses on a small number of individual studies. Although the studies selected for the risk assessment are part of the evidence base supporting the ISA's causality determinations for mortality, the concentration-response relationship in any given study reflects the particular time period, locations, air quality distribution and populations evaluated in that study. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate mortality incidence across the U.S. for populations, locations and $PM_{2.5}$ air quality distributions different from those present during the study period is subject to uncertainty.

1

C.5.3 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. 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.6 $PM_{2.5}$ DESIGN VALUES FOR THE AIR QUALITY PROJECTIONS

Table C-19. $PM_{2.5}$ 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
Chicag	170434002	Annual	No	0	-15	8.87	9.48	19.9	20.7
Chicag	170316005	Annual	No	0	-15	10.79	11.66	24.1	26.1

CBSA [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	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
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

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	060250007	Annual	No	0	12	8.37	7.99	21.5	20.8
Elkhar	180390008	Annual	Yes	0	-47	10.24	12.01	28.6	33.2
Evansv	181630023	Annual	Yes	0	-44	10.11	12.03	21.5	24.0
Evansv	211010014	Annual	No	0	-44	9.64	11.32	20.7	22.3
Evansv	181630021	Annual	No	0	-44	9.84	11.68	21.6	23.3
Evansv	181630016	Annual	No	0	-44	10.02	11.91	22.0	24.0
Fresno	060195001	24-hr	Yes	0	70	14.08	10.87	49.3	35.4
Fresno	060195025	24-hr	No	0	70	13.63	9.98	47.9	31.7
Fresno	060192009	24-hr	No	0	70	8.47	7.26	31.3	25.1
Fresno	060190011	24-hr	No	0	70	14.07	10.01	53.8	34.4
Hanfor	060310004	24-hr	Yes	65	79	21.98	11.79	72.0	35.4
Hanfor	060311004	24-hr	No	65	79	16.49	9.68	58.9	30.7
Housto	482011035	Annual	Yes	0	-14	11.19	12.04	22.4	24.0
Housto	482011039	Annual	No	0	-14	9.22	9.82	21.7	23.1
Housto	482010058	Annual	No	0	-14	9.67	10.37	22.3	23.8
Housto	481671034	Annual	No	0	-14	7.36	7.57	20.3	20.8
Indian	180970087	Annual	Yes	0	-10	11.44	12.01	25.9	26.8
Indian	180970083	Annual	No	0	-10	11.06	11.59	23.9	24.9
Indian	180970081	Annual	No	0	-10	11.07	11.61	25.0	26.0
Indian	180970078	Annual	No	0	-10	10.14	10.60	24.4	24.9
Indian	180970043	Annual	No	0	-10	-	-	26.0	26.4
Indian	180950011	Annual	No	0	-10	9.05	9.40	21.8	22.3
Indian	180570007	Annual	No	0	-10	9.02	9.39	21.4	22.1
Johnst	420210011	Annual	Yes	0	-25	10.68	12.03	25.8	30.3
Lancas	420710012	Annual	Yes	0	12	12.83	12.00	32.7	30.4
Lancas	420710007	Annual	No	0	12	10.57	9.88	29.8	27.4
LasVeg	320030561	Annual	Yes	0	-22	10.28	11.98	24.5	29.4
LasVeg	320032002	Annual	No	0	-22	9.79	11.38	19.8	23.4
LasVeg	320031019	Annual	No	0	-22	5.18	5.70	11.5	12.2
LasVeg	320030540	Annual	No	0	-22	8.80	10.21	21.7	25.9
Lebano	420750100	Annual	Yes	0	-15	11.20	12.02	31.4	33.9
Little	051191008	Annual	Yes	0	-41	10.27	12.03	21.7	24.7
Little	051190007	Annual	No	0	-41	9.78	11.76	20.5	24.0
LoganU	490050007	24-hr	Yes	0	-7	6.95	7.15	34.0	35.4
LosAng	060371103	Annual	Yes	0	5	12.38	12.03	32.8	32.1
LosAng	060592022	Annual	No	0	5	7.48	7.33	15.3	15.0
LosAng	060590007	Annual	No	0	5	9.63	9.37	-	-
LosAng	060374004	Annual	No	0	5	10.25	9.97	27.3	26.7

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	060374002	Annual	No	0	5	11.06	10.76	29.2	28.6
LosAng	060371602	Annual	No	0	5	11.86	11.52	32.3	31.5
LosAng	060371302	Annual	No	0	5	11.99	11.64	31.5	30.8
LosAng	060371201	Annual	No	0	5	9.46	9.24	25.6	25.0
LosAng	060370002	Annual	No	0	5	10.52	10.27	29.2	28.6
Louisv	180190006	Annual	Yes	0	-27	10.64	12.04	23.9	26.2
Louisv	211110075	Annual	No	0	-27	10.42	11.84	22.3	24.3
Louisv	211110067	Annual	No	0	-27	9.55	10.78	21.4	23.6
Louisv	211110051	Annual	No	0	-27	10.29	11.48	21.8	23.7
Louisv	211110043	Annual	No	0	-27	10.37	11.72	22.0	24.1
Louisv	180431004	Annual	No	0	-27	9.96	11.20	22.0	24.2
Louisv	180190008	Annual	No	0	-27	8.72	9.69	20.1	21.5
MaconG	130210007	Annual	Yes	0	-39	10.13	12.01	21.2	24.8
MaconG	130210012	Annual	No	0	-39	7.68	8.90	16.6	18.6
Madera	060392010	24-hr	Yes	0	56	13.30	11.03	45.1	35.3
McAlle	482150043	Annual	Yes	0	-67	10.09	12.02	25.0	27.4
Merced	060470003	24-hr	Yes	0	28	11.81	10.97	39.0	35.4
Merced	060472510	24-hr	No	0	28	11.68	10.57	39.8	35.1
Modest	060990006	24-hr	Yes	0	51	13.02	10.70	45.7	35.3
Modest	060990005	24-hr	No	0	51	-	-	38.8	32.5
NapaCA	060550003	Annual	Yes	0	-47	10.36	12.03	25.1	29.1
NewYor	360610128	Annual	Yes	0	-26	10.20	12.00	23.9	27.8
NewYor	361030002	Annual	No	0	-26	7.18	8.10	18.8	21.0
NewYor	360810124	Annual	No	0	-26	7.52	8.65	19.5	22.4
NewYor	360710002	Annual	No	0	-26	6.95	7.81	17.5	19.6
NewYor	360610134	Annual	No	0	-26	9.70	11.38	21.6	25.0
NewYor	360610079	Annual	No	0	-26	8.42	9.82	22.8	25.6
NewYor	360470122	Annual	No	0	-26	8.66	10.10	20.5	23.7
NewYor	360050133	Annual	No	0	-26	9.05	10.53	24.0	28.0
NewYor	360050110	Annual	No	0	-26	7.39	8.56	19.4	22.8
NewYor	340392003	Annual	No	0	-26	8.59	9.87	23.6	26.3
NewYor	340390004	Annual	No	0	-26	9.87	11.40	24.2	27.3
NewYor	340310005	Annual	No	0	-26	8.42	9.63	22.2	24.7
NewYor	340292002	Annual	No	0	-26	7.23	8.04	18.1	19.8
NewYor	340273001	Annual	No	0	-26	6.78	7.56	17.1	18.8
NewYor	340171003	Annual	No	0	-26	8.79	10.15	23.4	26.9
NewYor	340130003	Annual	No	0	-26	8.89	10.21	23.8	27.3
NewYor	340030003	Annual	No	0	-26	8.90	10.22	24.5	27.4

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	490110004	24-hr	Yes	0	-18	7.28	7.77	32.6	35.4
OgdenC	490570002	24-hr	No	0	-18	8.99	9.73	-	-
OgdenC	490030003	24-hr	No	0	-18	6.35	6.76	-	-
Philad	420450002	Annual	Yes	0	-8	11.46	12.04	26.0	27.2
Philad	421010057	Annual	No	0	-8	10.86	11.37	27.0	28.4
Philad	421010055	Annual	No	0	-8	11.43	12.03	27.5	29.0
Philad	421010048	Annual	No	0	-8	10.27	10.77	25.6	27.0
Philad	420290100	Annual	No	0	-8	9.64	10.03	23.9	25.1
Philad	340150004	Annual	No	0	-8	8.33	8.69	20.6	21.5
Philad	340071007	Annual	No	0	-8	8.84	9.23	21.0	22.0
Philad	340070002	Annual	No	0	-8	10.19	10.61	23.5	24.6
Philad	240150003	Annual	No	0	-8	8.70	9.02	22.6	23.4
Philad	100031012	Annual	No	0	-8	9.04	9.40	23.0	23.8
Pittsb	420030064	Annual	Yes	0	13	12.82	12.00	35.8	32.8
Pittsb	421290008	Annual	No	0	13	8.65	8.15	19.6	18.9
Pittsb	421255001	Annual	No	0	13	8.35	7.89	17.8	17.2
Pittsb	421250200	Annual	No	0	13	8.95	8.44	19.3	18.2
Pittsb	421250005	Annual	No	0	13	11.02	10.38	22.7	21.2
Pittsb	420070014	Annual	No	0	13	10.11	9.48	21.9	20.5
Pittsb	420050001	Annual	No	0	13	11.03	10.30	21.9	20.5
Pittsb	420031301	Annual	No	0	13	11.00	10.30	24.8	23.0
Pittsb	420031008	Annual	No	0	13	9.78	9.16	20.5	19.3
Pittsb	420030008	Annual	No	0	13	9.50	8.85	20.5	19.0
Prinev	410130100	24-hr	Yes	0	10	8.60	8.17	37.6	35.3
ProvoO	490494001	24-hr	Yes	0	-30	7.74	8.57	30.9	35.3
ProvoO	490495010	24-hr	No	0	-30	6.73	7.52	-	-
ProvoO	490490002	24-hr	No	0	-30	7.41	8.31	28.9	33.2
Rivers	060658005	24-hr	Yes	0	36	14.48	11.51	43.2	35.3
Rivers	060658001	24-hr	No	0	36	-	-	36.5	29.6
Sacram	060670006	24-hr	Yes	0	-23	9.31	10.40	31.4	35.4
Sacram	061131003	24-hr	No	0	-23	6.62	7.19	15.8	17.3
Sacram	060670012	24-hr	No	0	-23	7.30	8.01	19.8	21.2
Sacram	060670010	24-hr	No	0	-23	8.67	9.65	26.5	29.9
Sacram	060610006	24-hr	No	0	-23	7.58	8.47	20.3	22.3
Sacram	060610003	24-hr	No	0	-23	6.71	7.26	19.3	20.2
SaltLa	490353010	24-hr	Yes	0	44	-	-	41.5	35.3
SaltLa	490353006	24-hr	No	0	44	7.62	6.19	36.8	30.2
SaltLa	490351001	24-hr	No	0	44	7.07	5.85	32.1	25.8

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	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-20. $PM_{2.5}$ 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	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	11.25	23.5	25.5

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 ($\mu g\ m^{-3}$)
Chicag	170310076	Annual	No	N/A	N/A	10.18	11.04	22.5	24.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	11.96	26.8	29.1
Chicag	170310052	Annual	No	N/A	N/A	10.00	10.85	23.3	25.3
Chicag	170310022	Annual	No	N/A	N/A	10.38	11.26	22.4	24.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	10.99	21.7	23.5
Cincin	390610014	Annual	Yes	-72	0	10.70	12.04	22.9	26.1
Cincin	390610042	Annual	No	-72	0	10.29	11.66	22.6	26.2
Cincin	390610040	Annual	No	-72	0	9.45	10.79	21.0	25.4
Cincin	390610010	Annual	No	-72	0	9.43	10.75	21.3	24.4
Cincin	390610006	Annual	No	-72	0	9.46	10.75	20.3	24.3
Cincin	390170020	Annual	No	-72	0	-	-	24.2	27.8
Cincin	390170019	Annual	No	-72	0	10.24	11.40	22.0	24.5
Cincin	390170016	Annual	No	-72	0	9.79	11.06	22.1	25.1
Cincin	210373002	Annual	No	-72	0	9.06	10.42	20.9	25.1
Clevel	390350065	Annual	Yes	6	0	12.17	12.04	24.9	24.7
Clevel	391030004	Annual	No	6	0	8.73	8.61	19.6	19.2
Clevel	390933002	Annual	No	6	0	8.10	7.99	20.2	19.9
Clevel	390850007	Annual	No	6	0	7.88	7.78	17.4	17.1
Clevel	390351002	Annual	No	6	0	8.86	8.74	19.5	19.2
Clevel	390350045	Annual	No	6	0	10.61	10.49	22.9	22.6
Clevel	390350038	Annual	No	6	0	11.38	11.26	25.0	24.7
Clevel	390350034	Annual	No	6	0	8.87	8.75	20.4	20.1
Detroi	261630033	Annual	Yes	-56	0	11.30	12.04	26.8	30.2
Detroi	261630039	Annual	No	-56	0	9.11	9.88	22.3	24.8
Detroi	261630036	Annual	No	-56	0	8.68	9.39	21.8	23.4
Detroi	261630025	Annual	No	-56	0	8.98	9.75	24.1	26.5
Detroi	261630019	Annual	No	-56	0	9.18	9.97	22.4	24.1
Detroi	261630016	Annual	No	-56	0	9.62	10.38	24.4	27.4
Detroi	261630015	Annual	No	-56	0	11.19	11.97	25.5	28.2
Detroi	261630001	Annual	No	-56	0	9.50	10.20	23.3	25.0
Detroi	261470005	Annual	No	-56	0	8.89	9.50	24.3	26.1
Detroi	261250001	Annual	No	-56	0	8.86	9.65	24.2	26.7
Detroi	260990009	Annual	No	-56	0	8.80	9.48	26.2	28.4
ElCent	060250005	Annual	Yes	N/A	N/A	12.63	12.04	33.5	31.9
ElCent	060251003	Annual	No	N/A	N/A	7.44	7.09	19.8	18.9
ElCent	060250007	Annual	No	N/A	N/A	8.37	7.98	21.5	20.5
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	12.04	28.6	33.6
Evansv	181630023	Annual	Yes	-89	0	10.11	12.03	21.5	32.5

CBSA [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	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
LosAng	060371302	Annual	No	N/A	N/A	11.99	11.66	31.5	30.6

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 ($\mu g\ m^{-3}$)
LosAng	060371201	Annual	No	N/A	N/A	9.46	9.20	25.6	24.9
LosAng	060370002	Annual	No	N/A	N/A	10.52	10.23	29.2	28.4
Louisv	180190006	Annual	Yes	-65	0	10.64	12.04	23.9	28.4
Louisv	211110075	Annual	No	-65	0	10.42	11.76	22.3	26.4
Louisv	211110067	Annual	No	-65	0	9.55	10.84	21.4	25.4
Louisv	211110051	Annual	No	-65	0	10.29	11.67	21.8	25.9
Louisv	211110043	Annual	No	-65	0	10.37	11.71	22.0	26.1
Louisv	180431004	Annual	No	-65	0	9.96	11.32	22.0	25.8
Louisv	180190008	Annual	No	-65	0	8.72	10.07	20.1	24.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	12.04	21.2	25.2
MaconG	130210012	Annual	No	N/A	N/A	7.68	9.13	16.6	19.7
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	11.15	45.1	35.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	12.04	25.0	29.8
Merced	060472510	24-hr	Yes	32	0	11.68	10.79	39.8	35.4
Merced	060470003	24-hr	No	32	0	11.81	10.89	39.0	34.1
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	10.82	45.7	35.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	30.1
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	12.04	25.1	29.2
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	12.04	23.9	28.2
NewYor	361030002	Annual	No	N/A	N/A	7.18	8.48	18.8	22.2
NewYor	360810124	Annual	No	N/A	N/A	7.52	8.88	19.5	23.0
NewYor	360710002	Annual	No	N/A	N/A	6.95	8.20	17.5	20.7
NewYor	360610134	Annual	No	N/A	N/A	9.70	11.45	21.6	25.5
NewYor	360610079	Annual	No	N/A	N/A	8.42	9.94	22.8	26.9
NewYor	360470122	Annual	No	N/A	N/A	8.66	10.22	20.5	24.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	10.68	24.0	28.3
NewYor	360050110	Annual	No	N/A	N/A	7.39	8.72	19.4	22.9
NewYor	340392003	Annual	No	N/A	N/A	8.59	10.14	23.6	27.9
NewYor	340390004	Annual	No	N/A	N/A	9.87	11.65	24.2	28.6
NewYor	340310005	Annual	No	N/A	N/A	8.42	9.94	22.2	26.2
NewYor	340292002	Annual	No	N/A	N/A	7.23	8.53	18.1	21.4
NewYor	340273001	Annual	No	N/A	N/A	6.78	8.00	17.1	20.2
NewYor	340171003	Annual	No	N/A	N/A	8.79	10.38	23.4	27.6
NewYor	340130003	Annual	No	N/A	N/A	8.89	10.49	23.8	28.1
NewYor	340030003	Annual	No	N/A	N/A	8.90	10.51	24.5	28.9
OgdenC	490110004	24-hr	Yes	-53	0	7.28	7.65	32.6	35.4
OgdenC	490570002	24-hr	No	-53	0	8.99	9.37	-	-
OgdenC	490030003	24-hr	No	-53	0	6.35	6.70	-	-

CBSA [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 ($\mu g\ m^{-3}$)
Philad	420450002	Annual	Yes	-75	0	11.46	12.04	26.0	27.4
Philad	421010057	Annual	No	-75	0	10.86	11.54	27.0	28.1
Philad	421010055	Annual	No	-75	0	11.43	12.03	27.5	28.8
Philad	421010048	Annual	No	-75	0	10.27	10.91	25.6	27.4
Philad	420290100	Annual	No	-75	0	9.64	10.38	23.9	25.2
Philad	340150004	Annual	No	-75	0	8.33	8.94	20.6	23.2
Philad	340071007	Annual	No	-75	0	8.84	9.51	21.0	21.9
Philad	340070002	Annual	No	-75	0	10.19	10.95	23.5	24.6
Philad	240150003	Annual	No	-75	0	8.70	9.47	22.6	23.7
Philad	100031012	Annual	No	-75	0	9.04	9.81	23.0	23.6
Pittsb	420030064	Annual	Yes	30	0	12.82	12.02	35.8	34.8
Pittsb	421290008	Annual	No	30	0	8.65	8.06	19.6	18.0
Pittsb	421255001	Annual	No	30	0	8.35	7.78	17.8	16.4
Pittsb	421250200	Annual	No	30	0	8.95	8.32	19.3	18.2
Pittsb	421250005	Annual	No	30	0	11.02	10.30	22.7	21.7
Pittsb	420070014	Annual	No	30	0	10.11	9.52	21.9	20.6
Pittsb	420050001	Annual	No	30	0	11.03	10.45	21.9	20.4
Pittsb	420031301	Annual	No	30	0	11.00	10.28	24.8	23.6
Pittsb	420031008	Annual	No	30	0	9.78	9.20	20.5	19.0
Pittsb	420030008	Annual	No	30	0	9.50	8.89	20.5	19.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	8.10	37.6	35.4
ProvoO	490494001	24-hr	Yes	-76	0	7.74	8.29	30.9	35.4
ProvoO	490495010	24-hr	No	-76	0	6.73	7.21	-	-
ProvoO	490490002	24-hr	No	-76	0	7.41	7.95	28.9	33.2
Rivers	060658005	24-hr	Yes	N/A	N/A	14.48	11.87	43.2	35.4
Rivers	060658001	24-hr	No	N/A	N/A	-	-	36.5	29.9
Sacram	060670006	24-hr	Yes	-99	0	9.31	10.04	31.4	35.3
Sacram	061131003	24-hr	No	-99	0	6.62	7.08	15.8	19.0
Sacram	060670012	24-hr	No	-99	0	7.30	7.85	19.8	21.3
Sacram	060670010	24-hr	No	-99	0	8.67	9.30	26.5	30.2
Sacram	060610006	24-hr	No	-99	0	7.58	8.08	20.3	22.2
Sacram	060610003	24-hr	No	-99	0	6.71	7.04	19.3	20.7
SaltLa	490353010	24-hr	Yes	58	0	-	-	41.5	35.4
SaltLa	490353006	24-hr	No	58	0	7.62	6.91	36.8	31.5
SaltLa	490351001	24-hr	No	58	0	7.07	6.30	32.1	25.8
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	12.04	25.9	29.1
SanLui	060798002	Annual	No	N/A	N/A	5.71	6.43	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	9.28	19.8	22.3

CBSA [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 ($\mu g\ m^{-3}$)
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.

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Table C-21. $PM_{2.5}$ 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	-	-
Chicag	170313301	Annual	No	0	18	10.37	9.38	23.5	21.3

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 ($\mu g\ m^{-3}$)
Chicag	170310076	Annual	No	0	18	10.18	9.24	22.5	20.7
Chicag	170310057	Annual	No	0	18	11.03	9.99	26.8	25.1
Chicag	170310052	Annual	No	0	18	10.00	9.06	23.3	21.4
Chicag	170310022	Annual	No	0	18	10.38	9.28	22.4	20.9
Chicag	170310001	Annual	No	0	18	10.13	9.22	21.7	19.7
Cincin	390610014	Annual	Yes	0	12	10.70	10.04	22.9	21.8
Cincin	390610042	Annual	No	0	12	10.29	9.69	22.6	21.6
Cincin	390610040	Annual	No	0	12	9.45	8.91	21.0	20.0
Cincin	390610010	Annual	No	0	12	9.43	8.93	21.3	20.5
Cincin	390610006	Annual	No	0	12	9.46	8.91	20.3	19.5
Cincin	390170020	Annual	No	0	12	-	-	24.2	23.3
Cincin	390170019	Annual	No	0	12	10.24	9.60	22.0	21.1
Cincin	390170016	Annual	No	0	12	9.79	9.22	22.1	21.2
Cincin	210373002	Annual	No	0	12	9.06	8.58	20.9	20.0
Clevel	390350065	Annual	Yes	0	33	12.17	10.00	24.9	21.3
Clevel	391030004	Annual	No	0	33	8.73	7.57	19.6	17.8
Clevel	390933002	Annual	No	0	33	8.10	6.95	20.2	18.7
Clevel	390850007	Annual	No	0	33	7.88	6.84	17.4	15.4
Clevel	390351002	Annual	No	0	33	8.86	7.64	19.5	17.5
Clevel	390350045	Annual	No	0	33	10.61	8.84	22.9	20.1
Clevel	390350038	Annual	No	0	33	11.38	9.37	25.0	22.0
Clevel	390350034	Annual	No	0	33	8.87	7.58	20.4	18.2
Detroi	261630033	Annual	Yes	0	26	11.30	10.00	26.8	24.9
Detroi	261630039	Annual	No	0	26	9.11	8.21	22.3	20.3
Detroi	261630036	Annual	No	0	26	8.68	7.88	21.8	19.8
Detroi	261630025	Annual	No	0	26	8.98	7.99	24.1	21.7
Detroi	261630019	Annual	No	0	26	9.18	8.18	22.4	19.7
Detroi	261630016	Annual	No	0	26	9.62	8.63	24.4	22.6
Detroi	261630015	Annual	No	0	26	11.19	9.94	25.5	22.8
Detroi	261630001	Annual	No	0	26	9.50	8.39	23.3	20.4
Detroi	261470005	Annual	No	0	26	8.89	8.11	24.3	22.4
Detroi	261250001	Annual	No	0	26	8.86	7.90	24.2	22.2
Detroi	260990009	Annual	No	0	26	8.80	7.94	26.2	23.8
ElCent	060250005	Annual	Yes	0	50	12.63	10.01	33.5	25.0
ElCent	060251003	Annual	No	0	50	7.44	5.67	19.8	14.6
ElCent	060250007	Annual	No	0	50	8.37	6.80	21.5	18.5
Elkhar	180390008	Annual	Yes	0	6	10.24	10.01	28.6	27.8
Evansv	181630023	Annual	Yes	0	2	10.11	10.02	21.5	21.5

CBSA [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	211010014	Annual	No	0	2	9.64	9.56	20.7	20.7
Evansv	181630021	Annual	No	0	2	9.84	9.76	21.6	21.5
Evansv	181630016	Annual	No	0	2	10.02	9.94	22.0	21.9
Fresno	060195001	24-hr	Yes	0	100	14.08	9.49	49.3	30.3
Fresno	060195025	24-hr	No	0	100	13.63	8.41	47.9	26.4
Fresno	060192009	24-hr	No	0	100	8.47	6.74	31.3	22.2
Fresno	060190011	24-hr	No	0	100	14.07	8.27	53.8	27.1
Hanfor	060310004	Annual	Yes	82	98	21.98	10.00	72.0	29.5
Hanfor	060311004	Annual	No	82	98	16.49	8.36	58.9	25.2
Housto	482011035	Annual	Yes	0	19	11.19	10.01	22.4	20.2
Housto	482011039	Annual	No	0	19	9.22	8.40	21.7	19.6
Housto	482010058	Annual	No	0	19	9.67	8.70	22.3	20.3
Housto	481671034	Annual	No	0	19	7.36	7.07	20.3	19.6
Indian	180970087	Annual	Yes	0	25	11.44	10.01	25.9	24.2
Indian	180970083	Annual	No	0	25	11.06	9.72	23.9	22.5
Indian	180970081	Annual	No	0	25	11.07	9.71	25.0	23.4
Indian	180970078	Annual	No	0	25	10.14	8.97	24.4	22.8
Indian	180970043	Annual	No	0	25	-	-	26.0	24.6
Indian	180950011	Annual	No	0	25	9.05	8.17	21.8	20.7
Indian	180570007	Annual	No	0	25	9.02	8.07	21.4	20.0
Johnst	420210011	Annual	Yes	0	12	10.68	10.02	25.8	23.5
Lancas	420710012	Annual	Yes	0	41	12.83	9.98	32.7	25.5
Lancas	420710007	Annual	No	0	41	10.57	8.20	29.8	22.0
LasVeg	320030561	Annual	Yes	0	4	10.28	9.97	24.5	23.6
LasVeg	320032002	Annual	No	0	4	9.79	9.50	19.8	19.2
LasVeg	320031019	Annual	No	0	4	5.18	5.08	11.5	11.3
LasVeg	320030540	Annual	No	0	4	8.80	8.55	21.7	20.9
Lebano	420750100	Annual	Yes	0	21	11.20	10.04	31.4	28.0
Little	051191008	Annual	Yes	0	6	10.27	10.00	21.7	21.3
Little	051190007	Annual	No	0	6	9.78	9.48	20.5	20.1
LoganU	490050007	24-hr	Yes	0	19	6.95	6.40	34.0	30.3
LosAng	060371103	Annual	Yes	0	34	12.38	9.99	32.8	27.8
LosAng	060592022	Annual	No	0	34	7.48	6.43	15.3	13.3
LosAng	060590007	Annual	No	0	34	9.63	7.84	-	-
LosAng	060374004	Annual	No	0	34	10.25	8.36	27.3	23.7
LosAng	060374002	Annual	No	0	34	11.06	9.02	29.2	24.9
LosAng	060371602	Annual	No	0	34	11.86	9.55	32.3	26.5
LosAng	060371302	Annual	No	0	34	11.99	9.64	31.5	27.0

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	060371201	Annual	No	0	34	9.46	7.93	25.6	21.6
LosAng	060370002	Annual	No	0	34	10.52	8.81	29.2	25.0
Louisv	180190006	Annual	Yes	0	12	10.64	10.01	23.9	22.8
Louisv	211110075	Annual	No	0	12	10.42	9.79	22.3	21.4
Louisv	211110067	Annual	No	0	12	9.55	8.99	21.4	20.5
Louisv	211110051	Annual	No	0	12	10.29	9.76	21.8	21.2
Louisv	211110043	Annual	No	0	12	10.37	9.77	22.0	21.2
Louisv	180431004	Annual	No	0	12	9.96	9.41	22.0	21.0
Louisv	180190008	Annual	No	0	12	8.72	8.29	20.1	19.5
MaconG	130210007	Annual	Yes	0	2	10.13	10.03	21.2	21.0
MaconG	130210012	Annual	No	0	2	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	0	84	13.30	9.89	45.1	30.4
McAlle	482150043	Annual	Yes	0	2	10.09	10.03	25.0	24.9
Merced	060470003	24-hr	Yes	0	65	11.81	9.87	39.0	30.4
Merced	060472510	24-hr	No	0	65	11.68	9.11	39.8	28.8
Modest	060990006	24-hr	Yes	0	77	13.02	9.52	45.7	30.3
Modest	060990005	24-hr	No	0	77	-	-	38.8	29.2
NapaCA	060550003	Annual	Yes	0	9	10.36	10.04	25.1	24.6
NewYor	360610128	Annual	Yes	0	3	10.20	9.99	23.9	23.5
NewYor	361030002	Annual	No	0	3	7.18	7.07	18.8	18.6
NewYor	360810124	Annual	No	0	3	7.52	7.39	19.5	19.1
NewYor	360710002	Annual	No	0	3	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	0	3	9.70	9.51	21.6	21.2
NewYor	360610079	Annual	No	0	3	8.42	8.26	22.8	22.5
NewYor	360470122	Annual	No	0	3	8.66	8.49	20.5	20.2
NewYor	360050133	Annual	No	0	3	9.05	8.87	24.0	23.6
NewYor	360050110	Annual	No	0	3	7.39	7.25	19.4	19.1
NewYor	340392003	Annual	No	0	3	8.59	8.44	23.6	23.2
NewYor	340390004	Annual	No	0	3	9.87	9.69	24.2	23.8
NewYor	340310005	Annual	No	0	3	8.42	8.28	22.2	21.9
NewYor	340292002	Annual	No	0	3	7.23	7.13	18.1	17.9
NewYor	340273001	Annual	No	0	3	6.78	6.69	17.1	16.9
NewYor	340171003	Annual	No	0	3	8.79	8.64	23.4	22.9
NewYor	340130003	Annual	No	0	3	8.89	8.73	23.8	23.4
NewYor	340030003	Annual	No	0	3	8.90	8.75	24.5	24.1
OgdenC	490110004	24-hr	Yes	0	15	7.28	6.89	32.6	30.3
OgdenC	490570002	24-hr	No	0	15	8.99	8.39	-	-
OgdenC	490030003	24-hr	No	0	15	6.35	6.02	-	-

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 ($\mu g\ m^{-3}$)
Philad	420450002	Annual	Yes	0	20	11.46	9.99	26.0	22.9
Philad	421010057	Annual	No	0	20	10.86	9.56	27.0	23.4
Philad	421010055	Annual	No	0	20	11.43	9.94	27.5	24.2
Philad	421010048	Annual	No	0	20	10.27	9.00	25.6	22.7
Philad	420290100	Annual	No	0	20	9.64	8.66	23.9	21.2
Philad	340150004	Annual	No	0	20	8.33	7.43	20.6	18.2
Philad	340071007	Annual	No	0	20	8.84	7.86	21.0	18.8
Philad	340070002	Annual	No	0	20	10.19	9.11	23.5	20.6
Philad	240150003	Annual	No	0	20	8.70	7.90	22.6	20.5
Philad	100031012	Annual	No	0	20	9.04	8.15	23.0	21.1
Pittsb	420030064	Annual	Yes	0	44	12.82	10.04	35.8	26.2
Pittsb	421290008	Annual	No	0	44	8.65	6.96	19.6	16.9
Pittsb	421255001	Annual	No	0	44	8.35	6.78	17.8	15.7
Pittsb	421250200	Annual	No	0	44	8.95	7.22	19.3	15.7
Pittsb	421250005	Annual	No	0	44	11.02	8.85	22.7	18.0
Pittsb	420070014	Annual	No	0	44	10.11	7.98	21.9	17.5
Pittsb	420050001	Annual	No	0	44	11.03	8.58	21.9	17.8
Pittsb	420031301	Annual	No	0	44	11.00	8.64	24.8	18.7
Pittsb	420031008	Annual	No	0	44	9.78	7.68	20.5	16.1
Pittsb	420030008	Annual	No	0	44	9.50	7.30	20.5	16.3
Prinev	410130100	24-hr	Yes	0	33	8.60	7.19	37.6	30.4
ProvoO	490494001	24-hr	Yes	0	3	7.74	7.65	30.9	30.4
ProvoO	490495010	24-hr	No	0	3	6.73	6.65	-	-
ProvoO	490490002	24-hr	No	0	3	7.41	7.32	28.9	28.4
Rivers	060658005	24-hr	Yes	0	58	14.48	9.69	43.2	30.4
Rivers	060658001	24-hr	No	0	58	-	-	36.5	25.4
Sacram	060670006	24-hr	Yes	0	6	9.31	9.02	31.4	30.4
Sacram	061131003	24-hr	No	0	6	6.62	6.47	15.8	15.4
Sacram	060670012	24-hr	No	0	6	7.30	7.11	19.8	19.4
Sacram	060670010	24-hr	No	0	6	8.67	8.41	26.5	25.7
Sacram	060610006	24-hr	No	0	6	7.58	7.34	20.3	19.9
Sacram	060610003	24-hr	No	0	6	6.71	6.56	19.3	19.0
SaltLa	490353010	24-hr	Yes	0	85	-	-	41.5	30.4
SaltLa	490353006	24-hr	No	0	85	7.62	4.85	36.8	23.8
SaltLa	490351001	24-hr	No	0	85	7.07	4.72	32.1	21.0
SanLui	060792007	Annual	Yes	0	22	10.70	10.04	25.9	24.9
SanLui	060798002	Annual	No	0	22	5.71	5.42	-	-
SanLui	060792004	Annual	No	0	22	8.25	7.76	19.8	19.2

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 ($\mu g\ m^{-3}$)
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-22. $PM_{2.5}$ 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	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	9.38	23.5	21.3

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 ($\mu g\ m^{-3}$)
Chicag	170310076	Annual	No	N/A	N/A	10.18	9.21	22.5	20.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	9.98	26.8	24.2
Chicag	170310052	Annual	No	N/A	N/A	10.00	9.05	23.3	21.1
Chicag	170310022	Annual	No	N/A	N/A	10.38	9.39	22.4	20.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	9.16	21.7	19.6
Cincin	390610014	Annual	Yes	28	0	10.70	10.03	22.9	21.2
Cincin	390610042	Annual	No	28	0	10.29	9.61	22.6	20.8
Cincin	390610040	Annual	No	28	0	9.45	8.78	21.0	19.0
Cincin	390610010	Annual	No	28	0	9.43	8.78	21.3	19.6
Cincin	390610006	Annual	No	28	0	9.46	8.82	20.3	18.4
Cincin	390170020	Annual	No	28	0	-	-	24.2	22.5
Cincin	390170019	Annual	No	28	0	10.24	9.66	22.0	20.6
Cincin	390170016	Annual	No	28	0	9.79	9.16	22.1	20.1
Cincin	210373002	Annual	No	28	0	9.06	8.38	20.9	18.9
Clevel	390350065	Annual	Yes	79	0	12.17	10.04	24.9	20.5
Clevel	391030004	Annual	No	79	0	8.73	6.75	19.6	13.9
Clevel	390933002	Annual	No	79	0	8.10	6.28	20.2	13.8
Clevel	390850007	Annual	No	79	0	7.88	6.10	17.4	12.9
Clevel	390351002	Annual	No	79	0	8.86	6.81	19.5	14.4
Clevel	390350045	Annual	No	79	0	10.61	8.50	22.9	17.0
Clevel	390350038	Annual	No	79	0	11.38	9.33	25.0	19.7
Clevel	390350034	Annual	No	79	0	8.87	6.90	20.4	15.4
Detroi	261630033	Annual	Yes	60	0	11.30	10.03	26.8	24.3
Detroi	261630039	Annual	No	60	0	9.11	7.82	22.3	18.8
Detroi	261630036	Annual	No	60	0	8.68	7.43	21.8	19.1
Detroi	261630025	Annual	No	60	0	8.98	7.63	24.1	19.1
Detroi	261630019	Annual	No	60	0	9.18	7.83	22.4	20.3
Detroi	261630016	Annual	No	60	0	9.62	8.33	24.4	21.3
Detroi	261630015	Annual	No	60	0	11.19	9.88	25.5	22.0
Detroi	261630001	Annual	No	60	0	9.50	8.26	23.3	20.1
Detroi	261470005	Annual	No	60	0	8.89	7.81	24.3	20.6
Detroi	261250001	Annual	No	60	0	8.86	7.49	24.2	20.5
Detroi	260990009	Annual	No	60	0	8.80	7.57	26.2	21.8
ElCent	060250005	Annual	Yes	N/A	N/A	12.63	10.04	33.5	26.6
ElCent	060251003	Annual	No	N/A	N/A	7.44	5.91	19.8	15.7
ElCent	060250007	Annual	No	N/A	N/A	8.37	6.65	21.5	17.1
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	10.04	28.6	28.0
Evansv	181630023	Annual	Yes	3	0	10.11	10.03	21.5	21.2

CBSA [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	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
LosAng	060371302	Annual	No	N/A	N/A	11.99	9.72	31.5	25.5

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	060371201	Annual	No	N/A	N/A	9.46	7.67	25.6	20.8
LosAng	060370002	Annual	No	N/A	N/A	10.52	8.53	29.2	23.7
Louisv	180190006	Annual	Yes	24	0	10.64	10.02	23.9	22.0
Louisv	211110075	Annual	No	24	0	10.42	9.83	22.3	20.3
Louisv	211110067	Annual	No	24	0	9.55	8.96	21.4	19.9
Louisv	211110051	Annual	No	24	0	10.29	9.68	21.8	20.2
Louisv	211110043	Annual	No	24	0	10.37	9.77	22.0	20.2
Louisv	180431004	Annual	No	24	0	9.96	9.37	22.0	20.4
Louisv	180190008	Annual	No	24	0	8.72	8.13	20.1	18.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	10.04	21.2	21.0
MaconG	130210012	Annual	No	N/A	N/A	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	10.04	45.1	30.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	10.04	25.0	24.9
Merced	060472510	24-hr	Yes	68	0	11.68	9.74	39.8	30.4
Merced	060470003	24-hr	No	68	0	11.81	9.82	39.0	29.8
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	9.75	45.7	30.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	25.8
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	10.04	25.1	24.3
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	10.04	23.9	23.5
NewYor	361030002	Annual	No	N/A	N/A	7.18	7.07	18.8	18.5
NewYor	360810124	Annual	No	N/A	N/A	7.52	7.40	19.5	19.2
NewYor	360710002	Annual	No	N/A	N/A	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	N/A	N/A	9.70	9.55	21.6	21.3
NewYor	360610079	Annual	No	N/A	N/A	8.42	8.29	22.8	22.4
NewYor	360470122	Annual	No	N/A	N/A	8.66	8.52	20.5	20.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	8.91	24.0	23.6
NewYor	360050110	Annual	No	N/A	N/A	7.39	7.27	19.4	19.1
NewYor	340392003	Annual	No	N/A	N/A	8.59	8.46	23.6	23.2
NewYor	340390004	Annual	No	N/A	N/A	9.87	9.72	24.2	23.8
NewYor	340310005	Annual	No	N/A	N/A	8.42	8.29	22.2	21.9
NewYor	340292002	Annual	No	N/A	N/A	7.23	7.12	18.1	17.8
NewYor	340273001	Annual	No	N/A	N/A	6.78	6.67	17.1	16.8
NewYor	340171003	Annual	No	N/A	N/A	8.79	8.65	23.4	23.0
NewYor	340130003	Annual	No	N/A	N/A	8.89	8.75	23.8	23.4
NewYor	340030003	Annual	No	N/A	N/A	8.90	8.76	24.5	24.1
OgdenC	490110004	24-hr	Yes	29	0	7.28	7.01	32.6	30.4
OgdenC	490570002	24-hr	No	29	0	8.99	8.71	-	-
OgdenC	490030003	24-hr	No	29	0	6.35	6.10	-	-

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Philad	420450002	Annual	Yes	86	0	11.46	10.04	26.0	22.3
Philad	421010057	Annual	No	86	0	10.86	9.12	27.0	22.5
Philad	421010055	Annual	No	86	0	11.43	9.95	27.5	23.9
Philad	421010048	Annual	No	86	0	10.27	8.70	25.6	21.1
Philad	420290100	Annual	No	86	0	9.64	7.87	23.9	19.5
Philad	340150004	Annual	No	86	0	8.33	6.99	20.6	16.9
Philad	340071007	Annual	No	86	0	8.84	7.23	21.0	17.1
Philad	340070002	Annual	No	86	0	10.19	8.40	23.5	20.2
Philad	240150003	Annual	No	86	0	8.70	6.90	22.6	17.5
Philad	100031012	Annual	No	86	0	9.04	7.21	23.0	17.7
Pittsb	420030064	24-hr	Yes	100	0	12.82	9.22	35.8	30.4
Pittsb	421290008	24-hr	No	100	0	8.65	6.04	19.6	12.9
Pittsb	421255001	24-hr	No	100	0	8.35	5.90	17.8	11.1
Pittsb	421250200	24-hr	No	100	0	8.95	6.10	19.3	13.7
Pittsb	421250005	24-hr	No	100	0	11.02	7.78	22.7	18.1
Pittsb	420070014	24-hr	No	100	0	10.11	7.38	21.9	15.2
Pittsb	420050001	24-hr	No	100	0	11.03	8.39	21.9	15.5
Pittsb	420031301	24-hr	No	100	0	11.00	7.79	24.8	19.7
Pittsb	420031008	24-hr	No	100	0	9.78	7.11	20.5	14.7
Pittsb	420030008	24-hr	No	100	0	9.50	6.81	20.5	14.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	6.95	37.6	30.4
ProvoO	490494001	24-hr	Yes	6	0	7.74	7.68	30.9	30.4
ProvoO	490495010	24-hr	No	6	0	6.73	6.68	-	-
ProvoO	490490002	24-hr	No	6	0	7.41	7.36	28.9	28.4
Rivers	060658005	Annual	Yes	N/A	N/A	14.48	10.04	43.2	30.0
Rivers	060658001	Annual	No	N/A	N/A	-	-	36.5	25.3
Sacram	060670006	24-hr	Yes	18	0	9.31	9.11	31.4	30.4
Sacram	061131003	24-hr	No	18	0	6.62	6.50	15.8	15.1
Sacram	060670012	24-hr	No	18	0	7.30	7.17	19.8	19.3
Sacram	060670010	24-hr	No	18	0	8.67	8.50	26.5	25.5
Sacram	060610006	24-hr	No	18	0	7.58	7.45	20.3	19.9
Sacram	060610003	24-hr	No	18	0	6.71	6.63	19.3	18.9
SaltLa	490353010	24-hr	Yes	79	0	-	-	41.5	30.3
SaltLa	490353006	24-hr	No	79	0	7.62	6.46	36.8	29.3
SaltLa	490351001	24-hr	No	79	0	7.07	5.88	32.1	23.2
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	10.04	25.9	24.3
SanLui	060798002	Annual	No	N/A	N/A	5.71	5.36	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	7.74	19.8	18.6

CBSA [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 ($\mu g\ m^{-3}$)
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 2012 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 2012 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 2012 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 2012 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 2012 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 2012 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 2012 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 2012 review described above and the 2020 review described in the 2020 PA ({U.S. EPA, 2020 #285}, section 5.2.1.2), 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 60 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 2017-2019 period that supported a valid 24-hour $PM_{2.5}$ design value[7] and met strict criteria for PM species. Light extinction calculations at these 60 monitoring sites also included the coarse fraction in the IMPROVE equations.[8] Results for these 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 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 2012 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.

[8] In the 2020 analyses, PM_{10} data were available for only a subset of 20 of the 67 monitoring sites included in the analysis ({U.S. EPA, 2020 #285}, section 5.2.1.2).

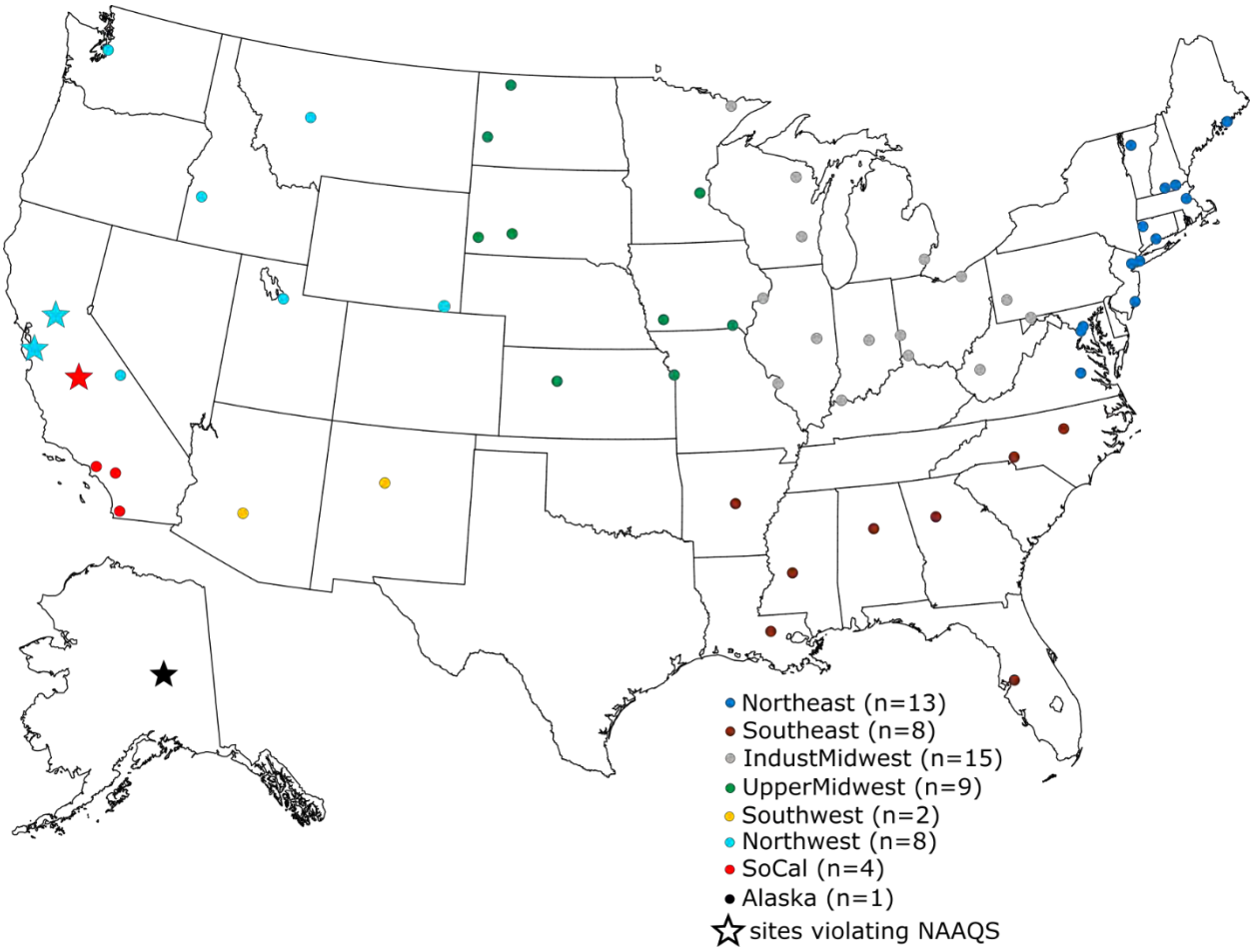

Figure D-1. Locations of monitoring sites with data for 2017-2019 with a valid $PM_{2.5}$ design value and meeting completeness criteria for PM species.

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 2017-2019 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)[9] 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 2017-2019. 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[10] 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 2017-2019.

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 2017-2019.

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

[10] 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 2017-2019 are located at https://www.epa.gov/air-trends/air-quality-design-values. Data handling of the 2017-2019 $PM_{2.5}$ 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 2017-2019 are located at https://www.epa.gov/air-trends/air-quality-design-values. Data handling of the 2017-2019 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 2017-2019 are located at https://www.epa.gov/air-trends/air-quality-design-values. Data handling of the 2017-2019 $PM_{2.5}$ 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[11] 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).

[11] 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 2017, 2018, and 2019, 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 2017-2019 $PM_{2.5}$ 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 $PM_{2.5}$ design values, 24-hour PM_{10} design values, annual $PM_{2.5}$ 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 included in the analyses. 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 60 monitoring sites in the analyses where light extinction was calculated using the original IMPROVE equation[12] and the Lowenthal and Kumar IMPROVE equation. Figure D-2 below presents the 3-year visibility metric and the 24-hour $PM_{2.5}$ design values for the 60 monitoring sites with light extinction calculated using the revised IMPROVE equation.[13]

[12] For this analysis, the original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM from the light extinction calculation, consistent with the modifications in the 2012 and 2020t review.

[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, consistent with the modifications in the 2012 and 2020 reviews.

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 60 monitoring sites (2017-2019).

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
010730023	Alabama	Southeast	21	0	10.0	23	23	26
020900034	Alaska	Alaska	40	1.4	8.9	24	25	27
040139997	Arizona	Southwest	21	0.7	7.4	21	21	24
051190007	Arkansas	Southeast	19	0	9.3	21	21	24
060270002	California	Northwest	23	3	5.6	13	14	15
060190011	California	SoCal	56	1	14.1	29	27	32
060371103	California	SoCal	31		11.9	26	25	28
060658001	California	SoCal	31	0	12.1	26	25	28
060670006	California	Northwest	37	4.1	10.2	25	25	30
060731022	California	SoCal	19	0	9.3	21	21	24
060850005	California	Northwest	43	0	10.5	22	22	26
090050005	Connecticut	Northeast	12		4.1	15	16	18
090090027	Connecticut	Northeast	18	0	6.9	23	23	26
110010043	District Of Columbia	Northeast	20	0	8.9	23	23	25
120573002	Florida	Southeast	18		7.9	18	19	21
130890002	Georgia	Southeast	19	0	8.4	20	20	24
160010010	Idaho	Northwest	29		7.4	23	22	26
170191001	Illinois	IndustrialMidwest	18		7.8	22	22	23
180970078	Indiana	IndustrialMidwest	20	0	9.0	24	24	26
181630021	Indiana	IndustrialMidwest	17	0	8.2	22	22	24
191370002	Iowa	UpperMidwest	16		6.6	21	22	22

191630015	Iowa	IndustrialMidwest	20	0	8.0	23	23	25
191770006	Iowa	UpperMidwest	16	0	7.0	21	22	23
201950001	Kansas	UpperMidwest	14		5.0	16	17	18
202090021	Kansas	UpperMidwest	26		9.4	23	23	26
220330009	Louisiana	Southeast	21	0	8.8	22	23	25
230090103	Maine	Northeast	11	0	3.2	16	18	18
240230002	Maryland	IndustrialMidwest	13		5.7	16	17	18
240330030	Maryland	Northeast	15	0	6.7	19	20	23
250250042	Massachusetts	Northeast	18		7.4	19	20	21
261630001	Michigan	IndustrialMidwest	22		8.8	23	23	25
270031002	Minnesota	UpperMidwest	20	0	7.3	23	23	25
270750005	Minnesota	IndustrialMidwest	13		3.8	16	16	17
280490020	Mississippi	Southeast	17		9.1	20	20	24
295100085	Missouri	IndustrialMidwest	21		8.7	24	24	26
300490004	Montana	Northwest	23		3.9	19	18	22
330115001	New Hampshire	Northeast	10		3.0	13	14	15
330150018	New Hampshire	Northeast	12		4.9	17	17	19
340010006	New Jersey	Northeast	15		6.6	18	19	20
340130003	New Jersey	Northeast	20	0	8.4	23	23	25
350010023	New Mexico	Southwest	15	0	5.6	16	17	19
360810124	New York	Northeast	18	0	7.0	21	22	24
371190041	North Carolina	Southeast	16		8.1	19	20	23
371830014	North Carolina	Southeast	13	0	7.7	19	19	23
380070002	North Dakota	UpperMidwest	15		3.9	18	18	20
380130004	North Dakota	UpperMidwest	16	0	3.6	20	20	21
390350060	Ohio	IndustrialMidwest	24	0	9.9	25	25	27
390610040	Ohio	IndustrialMidwest	20	0	9.4	22	23	24
391351001	Ohio	IndustrialMidwest	18		8.1	22	22	23
420030008	Pennsylvania	IndustrialMidwest	20		9.1	23	23	25
460330132	South Dakota	UpperMidwest	14	0	3.8	13	14	15
460710001	South Dakota	UpperMidwest	14	0	4.1	14	15	17

490353006	Utah	Northwest	30		7.5	26	26	28
500070007	Vermont	Northeast	12	0	4.3	15	16	17
510870014	Virginia	Northeast	15	0	7.1	19	20	23
530330080	Washington	Northwest	26		6.3	21	22	24
540390020	West Virginia	IndustrialMidwest	15		7.9	21	21	24
550270001	Wisconsin	IndustrialMidwest	21	0	7.0	24	24	26
550410007	Wisconsin	IndustrialMidwest	15		4.7	19	19	21
560210100	Wyoming	Northwest	11	0	3.2	13	14	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 $\mu g/m^3$.

[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 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 90th percentile of daily light extinction. In the 2012 and 2020 reviews, 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 from the light extinction calculation, consistent with the modifications in the 2012 and 2020 reviews.

[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 2012 and 2020 reviews.

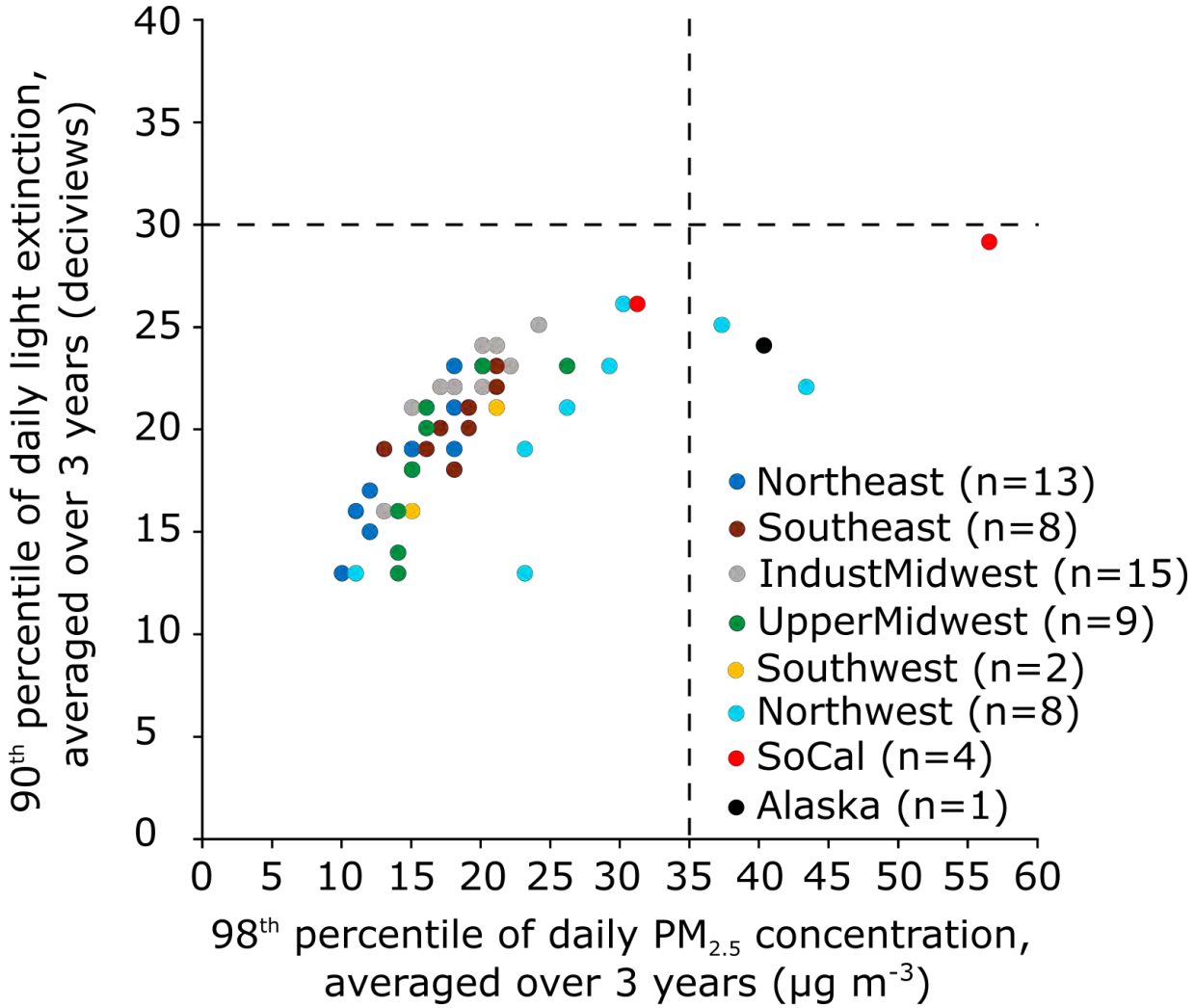

Figure D-2. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily $PM_{2.5}$ concentrations, averaged over three years, for 2017-2019 using the revised IMPROVE equation. (Note: Dashed lines indicate the level of current 24-hour $PM_{2.5}$ standard (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 2012 and 2020 reviews were conducted in four urban areas. Three western preference studies were available, including one in Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009).[14] Study specific details for these preference studies are shown in Table D-8.

[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-8 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-8. 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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