ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[EPA–HQ–OAR–2015–0072; FRL–10008–31–OAR]

RIN 2060–AS50

Review of the National Ambient Air Quality Standards for Particulate Matter

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed action.

SUMMARY: Based on the Environmental Protection Agency's (EPA's) review of the air quality criteria and the national ambient air quality standards (NAAQS) for particulate matter (PM), the Administrator has reached proposed decisions on the primary and secondary PM NAAQS. With regard to the primary standards meant to protect against fine particle exposures (*i.e.,* annual and 24-hour $PM_{2.5}$ standards), the primary standard meant to protect against coarse particle exposures (*i.e.,* 24-hour PM_{10} standard), and the secondary $PM_{2.5}$ and PM_{10} standards, the EPA proposes to retain the current standards, without revision.

DATES: Comments must be received on or before June 29, 2020.

Public Hearings: The EPA will hold one or more virtual public hearings on this proposed rule. These will be announced in a separate **Federal Register** notice that provides details, including specific dates, times, and contact information for these hearings.

ADDRESSES: You may submit comments, identified by Docket ID No. EPA–HQ–OAR–2015–0072, by any of the following means:

- *Federal eRulemaking Portal: https://www.regulations.gov* (our preferred method). Follow the online instructions for submitting comments.
- *Email: a-and-r-Docket@epa.gov.* Include the Docket ID No. EPA–HQ–OAR–2015–0072 in the subject line of the message.

Instructions: All submissions received must include the Docket ID No. for this document. Comments received may be posted without change to *https://www.regulations.gov,* including any personal information provided. For detailed instructions on sending comments, see the **SUPPLEMENTARY INFORMATION** section of this document. Out of an abundance of caution for members of the public and our staff, the EPA Docket Center and Reading Room was closed to public visitors on March 31, 2020, to reduce the risk of transmitting COVID–19. Our Docket Center staff will continue to provide remote customer service via email, phone, and webform. We encourage the public to submit comments via *https://www.regulations.gov* or email, as there is a temporary suspension of mail delivery to EPA, and no hand deliveries are currently accepted. For further information of EPA Docket Center services and the current status, please visit us online at *https://www.epa.gov/dockets.*

FOR FURTHER INFORMATION CONTACT: Dr. Scott Jenkins, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Mail Code C504–06, Research Triangle Park, NC 27711; telephone: (919) 541–1167; fax: (919) 541–5315; email: *jenkins.scott@epa.gov.*

SUPPLEMENTARY INFORMATION:

General Information

Written Comments: Submit your comments, identified by Docket ID No. EPA–HQ–OAR–2015–0072, at *https://www.regulations.gov* (our preferred method), or the other methods identified in the **ADDRESSES** section. Once submitted, comments cannot be edited or removed from the docket. The EPA may publish any comment received to its public docket. Do not submit electronically any information you consider to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Multimedia submissions (audio, video, etc.) must be accompanied by a written submission. The written submission is considered the official submission and should include discussion of all points you wish to make. The EPA will generally not consider submissions or submission content located outside of the primary submission (*i.e.,* on the web, cloud, or other file sharing system). For additional submission methods, the full EPA public comment policy, information about CBI or multimedia submissions, and general guidance on making effective comments, please visit *https://www.epa.gov/dockets/commenting-epa-dockets.*

The EPA is temporarily suspending its Docket Center and Reading Room for public visitors to reduce the risk of transmitting COVID–19. Written comments submitted by mail are temporarily suspended and no hand deliveries will be accepted. Our Docket Center staff will continue to provide remote customer service via email, phone, and webform. We encourage the public to submit comments via *https://www.regulations.gov.* For further information and updates on EPA Docket Center services, please visit us online at *https://www.epa.gov/dockets.*

The EPA continues to carefully and continuously monitor information from the Centers for Disease Control and Prevention (CDC), local area health departments, and our Federal partners so that we can respond rapidly as conditions change regarding COVID–19.

Availability of Information Related to This Action

A number of the documents that are relevant to this proposed decision are available through the EPA's website at *https://www.epa.gov/naaqs/particulate-matter-pm-air-quality-standards.* These documents include the Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter (U.S. EPA, 2016), available at *https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf,* the Integrated Science Assessment for Particulate Matter (U.S. EPA, 2019), available at *https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=347534,* and the Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (U.S. EPA, 2020), available at *https://www.epa.gov/naaqs/particulate-matter-pm-standards-policy-assessments-current-review-0.* These and other related documents are also available for inspection and copying in the EPA docket identified above.

Table of Contents

The following topics are discussed in this preamble:

Executive Summary

This document presents the Administrator's proposed decisions on the primary (health-based) and secondary (welfare-based) National Ambient Air Quality Standards (NAAQS) for particulate matter (PM). In ambient air, PM is a mixture of substances suspended as small liquid and/or solid particles. Particles in the atmosphere range in size from less than 0.01 to more than 10 micrometers (μm) in diameter. Particulate matter and its precursors are emitted from both anthropogenic sources (*e.g.,* electricity generating units, cars and trucks, agricultural operations) and natural sources (*e.g.,* sea salt, wildland fires, biological aerosols).

When describing PM, subscripts are used to denote particle size. For example, $PM_{2.5}$ includes particles with diameters generally less than or equal to 2.5 μm and PM_{10} includes particles with diameters generally less than or equal to 10 μm.

The EPA has established primary (health-based) and secondary (welfare-based) NAAQS for $PM_{2.5}$ and PM_{10}. This includes two primary $PM_{2.5}$ standards, an annual average standard with a level of 12.0 $\mu g/m^3$ and a 24-hour standard with a 98th percentile form and a level of 35 $\mu g/m^3$. It also includes a primary PM_{10} standard with a 24-hour averaging time, a 1-expected exceedance form, and a level of 150 $\mu g/m^3$. Secondary PM standards are set equal to the primary standards, except that the level of the secondary annual $PM_{2.5}$ standard is 15.0 $\mu g/m^3$. In reaching proposed decisions on these PM standards in the current review, the Administrator has considered the available scientific evidence assessed in the Integrated Science Assessment (ISA), analyses in the Policy Assessment (PA), and advice from the Clean Air Scientific Advisory Committee (CASAC).

For the primary $PM_{2.5}$ standards, the Administrator proposes to conclude that there are important uncertainties in the evidence for adverse health effects below the current standards and in the potential public health impacts of reducing ambient $PM_{2.5}$ concentrations below those standards. As a result, he proposes to conclude that the available evidence and information do not call into question the adequacy of the current primary $PM_{2.5}$ standards, and he proposes to retain those standards (*i.e.,* both the annual and 24-hour standards) without revision in this review.

For the primary PM_{10} standard, the Administrator observes that, while the available health effects evidence has expanded, recent studies are subject to the same types of uncertainties that were judged important in the last review. He proposes to conclude that the newly available evidence does not call into question the adequacy of the current primary PM_{10} standard, and he proposes to retain that standard without revision in this review.

For the secondary standards, the Administrator observes that the expanded evidence for non-ecological welfare effects is consistent with the last review [1] and that updated quantitative analyses show results similar to those in the last review. Therefore, he proposes to conclude that the newly available evidence and updated analyses do not call into question the adequacy of the current secondary PM standards, and he proposes to retain those standards without revision in this review.

These proposed decisions are consistent with the CASAC's consensus advice on the primary 24-hour $PM_{2.5}$ standard, the primary PM_{10} standard, and the secondary standards. The CASAC did not reach consensus on the primary annual $PM_{2.5}$ standard, with some committee members

[1] The welfare effects considered in this review include visibility impairment, climate effects, and materials effects. Ecological effects associated with PM, and the adequacy of protection provided by the secondary PM standards for those effects, are being addressed in the separate review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM. Information on the current review of these secondary NAAQS can be found at *https://www.epa.gov/naaqs/nitrogen-dioxide-no2-and-sulfur-dioxide-so2-secondary-air-quality-standards.*

recommending that EPA retain the current standard and other members recommending revision of that standard.

I. Background

A. Legislative Requirements

Two sections of the Clean Air Act (CAA) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those pollutants ''emissions of which, in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare''; ''the presence of which in the ambient air results from numerous or diverse mobile or stationary sources''; and for which he ''plans to issue air quality criteria . . . . '' (42 U.S.C. 7408(a)(1)). Air quality criteria are intended to ''accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air . . . . '' (42 U.S.C. 7408(a)(2)).

Section 109 [42 U.S.C. 7409] directs the Administrator to propose and promulgate ''primary'' and ''secondary'' NAAQS for pollutants for which air quality criteria are issued [42 U.S.C. 7409(a)]. Section 109(b)(1) defines primary standards as ones ''the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.'' [2] 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.'' [3]

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); *accord Murray Energy Corporation* v. *EPA,* 936 F.3d 597, 623–24 (D.C. Cir. 2019).

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); *American Petroleum Institute* v. *Costle,* 665 F.2d at 1186; *Coalition of Battery Recyclers Ass'n* v. *EPA,* 604 F.3d 613, 617–18 (D.C. Cir. 2010); *Mississippi* v. *EPA,* 744 F.3d 1334, 1353 (D.C. Cir. 2013). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries Ass'n* 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 Ass'n* v. *EPA,* 647 F.2d at 1161–62; *Mississippi* v. *EPA,* 744 F.3d at 1353.

Section 109(d)(1) of the Act requires the review every five years of existing air quality criteria and, if appropriate, the revision of those 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.

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 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

[2] 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).

[3] 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.''

that are not relevant to standard setting may be relevant to implementation of the NAAQS once they are established.[4]

B. Related PM Control Programs

States are primarily responsible for ensuring attainment and maintenance of ambient air quality standards once the EPA has established them. Under section 110 and 171–190 of the CAA, and related provisions and regulations, states are to submit, for EPA's approval, state implementation plans (SIPs) that provide for the attainment and maintenance of such standards through control programs directed to sources of the pollutants involved. The states, in conjunction with the EPA, also administer the Prevention of Significant Deterioration (PSD) program (CAA sections 160 to 169). In addition, Federal programs provide for nationwide reductions in emissions of PM and other air pollutants through the Federal motor vehicle and motor vehicle fuel control program under title II of the Act (CAA sections 202 to 250), which involves controls for emissions from mobile sources and controls for the fuels used by these sources, and new source performance standards for stationary sources under section 111 of the CAA.

C. Review of the Air Quality Criteria and Standards for Particulate Matter

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).[5] 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 μm (referred to as total suspended particulates or TSP). The primary standards were set at 260 μg/m^3, 24-hour average, not to be exceeded more than once per year, and 75 μg/m^3, annual geometric mean. The secondary standards were set at 150 μg/m^3, 24-hour average, not to be exceeded more than once per year, and 60 μg/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.[6] The level of the 24-hour standards (primary and secondary) was set at 150 μg/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 μg/m^3, and the form was annual arithmetic mean, averaged over three years.

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 μg/m^3, based on the 3-year average of annual arithmetic mean $PM_{2.5}$ concentrations from single or multiple community-oriented monitors; [7] and (2) a 24-hour standard with a level of 65 μg/m^3, based on the 3-year average of the 98th percentile of 24-hour $PM_{2.5}$ concentrations at each monitor within an area. Also, the EPA established a new reference method for the measurement of $PM_{2.5}$ in the ambient air and adopted rules for determining attainment of the new standards. To continue to address the health effects of the coarse fraction of PM_{10} (referred to as thoracic coarse particles or $PM_{10-2.5}$; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 μm and less than or equal to 10 μm), the EPA retained the primary annual PM_{10} standard and revised the form of the primary 24-hour 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 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, Inc.* v. *EPA,* 175 F. 3d 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

[4] Some aspects of the CASAC's advice may not be relevant to the 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 CAA directs the 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 the 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.

[5] Prior to the review initiated in 2007 (see below), the AQCD provided the scientific foundation (*i.e.,* the air quality criteria) for the NAAQS. Beginning in that review, the Integrated Science Assessment (ISA) has replaced the AQCD.

[6] 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.

[7] The 1997 annual $PM_{2.5}$ standard was compared with measurements made at the community-oriented monitoring site recording the highest concentration or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged (*i.e.,* ''spatial averaging''). In the last review (completed in 2012) the EPA replaced the term ''community-oriented'' monitor with the term ''area-wide'' monitor. Area-wide monitors are those sited at the neighborhood scale or larger, as well as those monitors sited at micro- or middle-scales that are representative of many such locations in the same core-based statistical area (CBSA) (78 FR 3236, January 15, 2013).

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 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 D.C. Circuit 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 D.C. Circuit 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).

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 National Center for Environmental Assessment (NCEA) finalized the AQCD in October 2004 (U.S. EPA, 2004). The EPA's Office of Air Quality Planning and Standards (OAQPS) finalized a Risk Assessment and Staff Paper in December 2005 (Abt Associates, 2005, U.S. EPA, 2005).[8] 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 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.[9] The Administrator judged that the available evidence generally did not suggest a link between long-term exposure to existing ambient levels of coarse particles and health or welfare effects. In addition, a new reference method was added for the measurement of $PM_{10\text{-}2.5}$ in the ambient air in order to provide a basis for approving federal equivalent methods (FEMs) and to promote the gathering of scientific data to support future reviews of the PM NAAQS.

Several parties filed petitions for review following promulgation of the revised PM NAAQS in 2006. These petitions addressed the following issues: (1) Selecting the level of the primary annual $PM_{2.5}$ standard; (2) retaining PM_{10} as the indicator of a standard for thoracic coarse particles, retaining the level and form of the 24-hour PM_{10} standard, and revoking the PM_{10} annual standard; and (3) setting the secondary $PM_{2.5}$ standards identical to the primary standards. On February 24, 2009, the D.C. 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 had 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. *Id. at* 520–27. 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. *Id.* 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. *Id.* 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).

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 (72 FR 35462, June 28, 2007). Based on the NAAQS review process, as revised in 2008 and again in 2009,[10] 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, 2009c), REA planning documents for health and welfare (U.S. EPA, 2009b, U.S. EPA, 2009a), a quantitative health risk assessment (U.S. EPA, 2010a) and an urban-focused visibility assessment

[8] 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.

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

[10] 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.*

(U.S. EPA, 2010a), 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 [11] to 12.0 μg/m^3 and retained the 24-hour $PM_{2.5}$ standard, with its level of 35 μg/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 [12] 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).

5. Current Review

In December 2014, the EPA announced the initiation of the current periodic review of the air quality criteria for PM and of the $PM_{2.5}$ and PM_{10} NAAQS and issued a call for information (79 FR 71764, December 3, 2014). On February 9 to 11, 2015, the EPA's NCEA and OAQPS held a public workshop to inform the planning for the current review of the PM NAAQS (announced in 79 FR 71764, December 3, 2014). Workshop participants, including a wide range of external experts as well as EPA staff representing a variety of areas of expertise (*e.g.,* epidemiology, human and animal toxicology, risk/exposure analysis, atmospheric science, visibility impairment, climate effects), were asked to highlight significant new and emerging PM research, and to make recommendations to the Agency regarding the design and scope of this review. This workshop provided for a public discussion of the key science and policy-relevant issues around which the EPA has structured the current review of the PM NAAQS and of the most meaningful new scientific information that would be available in this review to inform 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 (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.

In May 2018, the Administrator issued a memorandum describing a ''back-to-basics'' process for reviewing the NAAQS (Pruitt, 2018). This memo announced the Agency's intention to conduct the current review of the PM NAAQS in such a manner as to ensure that any necessary revisions are finalized by December 2020. Following this memo, on October 10, 2018 the Administrator additionally announced that the role of reviewing the key assessments developed as part of the ongoing review of the PM NAAQS (*i.e.,* drafts of the ISA and PA) would be performed by the seven-member chartered CASAC (*i.e.,* rather than the CASAC Particulate Matter Panel that reviewed the draft IRP).[13]

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, 2019b). In that letter, the CASAC's recommendations address both the draft ISA's assessment of the science for PM-related effects and the process under which this review of the PM NAAQS is being conducted.

Regarding the assessment of the evidence, the CASAC letter states that ''the Draft ISA does not provide a sufficiently comprehensive, systematic assessment of the available science relevant to understanding the health impacts of exposure to particulate matter (PM)'' (Cox, 2019b, p. 1 of letter). The CASAC recommended that this and other limitations (*i.e.,* ''[i]nadequate evidence for altered causal determinations'' and the need for a ''[c]learer discussion of causality and causal biological mechanisms and pathways'') be remedied in a revised ISA (Cox, 2019b, p. 1 of letter).

Given the Administrator's timeline for this review, as noted above (Pruitt, 2018), the EPA did not prepare a second draft ISA. Rather, the EPA has taken steps to address the CASAC's comments in the Final PM ISA (U.S. EPA, 2019). In particular, the final ISA includes additional text and a new appendix to clarify the comprehensive and systematic process employed by the EPA to develop the PM ISA. In addition, several causality determinations were re-examined and, consistent with the CASAC advice, the final ISA reflects a revised causality determination for long-term ultrafine particle (UFP) exposures and nervous system effects (*i.e.,* from ''likely to be causal'' to ''suggestive of, but not sufficient to infer, a causal relationship'').[14] The final ISA also contains additional text to clarify the evidence for biological pathways of particular PM-related effects and the role of that evidence in causality determinations.

Among its comments on the process, the chartered CASAC recommended ''that the EPA reappoint the previous CASAC PM panel (or appoint a panel with similar expertise)'' (Cox, 2019b). The Agency's response to this advice was provided in a letter from the Administrator to the CASAC chair dated July 25, 2019.[15] In that letter, the Administrator announced his intention to identify a pool of non-member subject matter expert consultants to support the CASAC's review activities for the PM and ozone NAAQS. A **Federal Register** notice requesting the nomination of scientists from a broad range of disciplines ''with demonstrated expertise and research in the field of air pollution related to PM and ozone'' was published in August 2019 (84 FR 38625,

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

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

[13] The CASAC charter is available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/2019casaccharter/$File/CASAC%202019%20Renewal%20Charter%203.21.19%20-%20final.pdf.* The Administrator's announcement is available at: *https://archive.epa.gov/epa/newsreleases/acting-administrator-wheeler-announces-science-advisors-key-clean-air-act-committee.html.*

[14] Based on the CASAC's comments, the EPA also re-examined the causality determinations for cancer and for nervous system effects following long-term $PM_{2.5}$ exposures. The EPA's consideration of these comments in the final ISA is discussed below in sections II.B.1.d and II.B.1.e.

[15] Available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002_Response.pdf.*

August 7, 2019). The Administrator selected consultants from among those nominated, and input from members of this pool of consultants informed the CASAC's review of the draft PA.

The EPA released the draft PA in September 2019 (84 FR 47944, September 11, 2019). The draft PA drew from the assessment of the evidence in the draft ISA. It was reviewed by the chartered CASAC and discussed in October 2019 at a public meeting held in Cary, NC. Public comments were received via a separate public teleconference (84 FR 51555, September 30, 2019). A public meeting to discuss the chartered CASAC letter and response to charge questions on the draft PA was held in Cary, NC in December 2019 (84 FR 58713, November 1, 2019), and the CASAC provided its advice on the draft PA, including its advice on the current primary and secondary PM standards, in a letter to the EPA Administrator dated December 16, 2019 (Cox, 2019a).

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. The CASAC's advice on the primary and secondary PM standards, and the Administrator's consideration of that advice in reaching proposed decisions, is discussed in detail in sections II.C.2 and II.C.3 (primary $PM_{2.5}$ standards), III.C.2 and III.C.3 (primary PM_{10} standards), and IV.D.2 and IV.D.3 (secondary standards) of this document.

The CASAC additionally made a number of recommendations regarding the information and analyses presented in the draft PA. Specifically, the CASAC recommended that a revised PA include (1) additional discussion of the current CASAC and NAAQS review process; (2) additional characterization of PM-related emissions, monitoring and air quality information, including uncertainties in that information; (3) additional discussion and examination of uncertainties in the $PM_{2.5}$ health evidence and the risk assessment; (4) updates to reflect changes in the ISA's causality determinations; and (5) additional discussion of the evidence for PM-related welfare effects, including uncertainties (Cox, 2019a, pp. 2–3 in letter). In response to the CASAC's comments, the final PA [16] incorporated a number of changes, including the following (U.S. EPA, 2020):

- Text was added to Chapter 1 to clarify the process followed for this review of the PM NAAQS, including how the process has evolved since the initiation of the review.
- Text and figures were added to Chapter 2 on emissions of PM and PM precursors, and a section discussing uncertainty in emissions estimates was added. A discussion of measurement uncertainty for FRM, FEM, CSN, and IMPROVE monitors was also added.
- Chapter 3 and Appendices B and C include a number of changes, including:
  - An expanded characterization and discussion of the evidence related to exposure measurement error, the potential confounders examined by key studies, the shapes of concentration-response functions, and the results of causal inference and quasi-experimental studies.
  - An expanded and clarified discussion of uncertainties in the risk assessment, and additional air quality model performance evaluations for each of the urban study areas included in the risk assessment.
  - Additional detail on the procedure used to derive concentration-response functions used in the risk assessment.
  - Changes in the text to reflect the change in the final ISA's causality determination from ''likely to be causal'' to ''suggestive of, but not sufficient to infer, a causal relationship.''
- Throughout the document (Chapters 3, 4 and 5), summaries of the CASAC advice on the PM standards are included, and expanded discussions of data gaps and areas for future research in the health and welfare effects evidence are presented.

D. Air Quality Information

This section provides a summary of basic information related to PM ambient air quality. It summarizes information on the distribution of particle size in ambient air (I.D.1), sources and emissions contributing to PM in the ambient air (I.D.2), monitoring of ambient PM in the U.S. (I.D.3), ambient PM concentrations and trends in the U.S. (I.D.4), and background PM (I.D.5). Additional detail on PM air quality can be found in Chapter 2 of the Policy Assessment (U.S. EPA, 2020; PA).

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 (U.S. EPA, 2019, section 2.2). 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 μm in diameter (U.S. EPA, 2019, section 2.2). When describing PM, subscripts are used to denote the aerodynamic diameter [17] of the particle size range, in 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. In addition, 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, 2019, section 2.2).

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, 2019, 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, though only a minor contributor to particle number (U.S. EPA, 2019, 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

[16] Given the Administrator's timeline for this review, as noted above (Pruitt, 2018), the EPA did not prepare a second draft PA. Rather, the CASAC's advice was considered in developing the final PA (U.S. EPA, 2020).

[17] 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, 2019, section 4.1.1).

(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, 2019, 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, 2019, 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, 2019, Table 2–1).

2. Sources and Emissions Contributing to PM in the Ambient Air

PM is composed of both primary (directly emitted particles) and secondary particles. 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, 2019, section 2.3). As discussed further in the ISA (U.S. EPA, 2019, 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 sulfur dioxide (SO_2), nitrogen oxides (NO_X), and volatile organic compounds (VOC) (U.S. EPA, 2019, section 2.3.2.1). Ammonia also plays an important role in the formation of nitrate PM by neutralizing sulfuric acid and nitric acid. Sources and emissions of PM are discussed in more detail in section 2.1.1 of the PA (U.S. EPA, 2020).

Direct emissions of PM have remained relatively unchanged in recent years, while emissions of some precursor gases have declined substantially.[18] From 1990 to 2014, SO_2 emissions have undergone the largest declines while NH_3 emissions have undergone the smallest change. Declining SO_2 emissions during this time period are primarily a result of reductions at stationary sources such as EGUs, with substantial reductions also from mobile sources (U.S. EPA, 2019, section 2.3.2.1).[19]

3. Monitoring of Ambient PM

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)[20] 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 p.m. 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. More information on PM ambient monitoring networks is available in section 2.2 of the PA (U.S. EPA, 2020).

4. Ambient Concentrations and Trends

This section summarizes available information on recent ambient PM concentrations in the U.S. and on trends in PM air quality. Sections I.D.4.a and I.D.4.b summarize information on $PM_{2.5}$ mass and components, respectively. Section I.D.4.c summarizes information on PM_{10}. Sections I.D.4.d and I.D.4.e summarize the more limited information on $PM_{10-2.5}$ and UFP, respectively. Additional detail on PM air quality and trends can be found in section 2.3 of the PA (U.S. EPA, 2020).

a. $PM_{2.5}$ Mass

At monitoring sites in the U.S., annual $PM_{2.5}$ concentrations from 2015 to 2017 averaged 8.0 μg/m³ (and ranged from 3.0 to 18.2 μg/m³) and the 98th percentiles of 24-hour concentrations averaged 20.9 μg/m³ (and ranged from 9.2 to 111 μg/m³) (U.S. EPA, 2020, section 2.3.2.1). The highest ambient $PM_{2.5}$ concentrations occur in the west, particularly in California and the Pacific northwest (U.S. EPA, 2020, Figure 2–8). Much of the eastern U.S. has lower ambient concentrations, with annual average concentrations generally at or below 12.0 μg/m³ and 98th percentiles of 24-hour concentrations generally at or below 30 μg/m³ (U.S. EPA, 2020, section 2.3.2).

Recent ambient $PM_{2.5}$ concentrations reflect the substantial reductions that have occurred across much of the U.S. (U.S. EPA, 2020, section 2.3.2.1). From 2000 to 2017, national annual average $PM_{2.5}$ concentrations have declined from 13.5 μg/m³ to 8.0 μg/m³, a 41% decrease (U.S. EPA, 2020, section 2.3.2.1).[21] These declines have occurred at 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 impact of local sources in urban areas. 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 (U.S. EPA, 2020, section 2.3.2.1). 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 (U.S. EPA, 2020, section 2.3.2.1).

The recent deployment of $PM_{2.5}$ monitors near major roads in large urban areas provides information on $PM_{2.5}$ concentrations near an important emissions source. Of the 25 CBSAs with valid design values at near-road monitoring sites,[22] 52% measured the highest annual design value at the near-road site while 24% measured the highest 24-hour design value at the near-road site (U.S. EPA, 2020, section 2.3.2.2). Of the CBSAs with highest

[18] More information on these trends, including details on methods and explanations on the noted changes over time is available at *https://gispub.epa.gov/neireport/2014/*.

[19] State-specific emission trends data for 1990 to 2014 can be found at: *https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data.*

[20] 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*).

[21] See *https://www.epa.gov/air-trends/particulate-matter-pm25-trends* and *https://www.epa.gov/air-trends/particulate-matter-pm25-trends#pmnat* for more information.

[22] A design value is considered valid if it meets the data handling requirements given in 40 CFR appendix N to Part 50. Several large CBSAs such as Chicago-Naperville-Elgin, IL–IN–WI and Houston-The Woodlands-Sugar Land, TX had near-road sites that did not have valid $PM_{2.5}$ design values for the 2015–2017 period.

annual design values at near-road sites, those design values were, on average, 0.7 μg/m^3 higher than at the highest measuring non-near-road sites (range is 0.1 to 2.0 μg/m^3 higher at near-road sites). Although most near-road monitoring sites do not have sufficient data to evaluate long-term trends in near-road $PM_{2.5}$ concentrations, analyses of the data at one near-road-like site in Elizabeth, NJ,[23] show that the annual average near-road increment has generally decreased between 1999 and 2017 from about 2.0 μg/m^3 to about 1.3 μg/m^3 (U.S. EPA, 2020, section 2.3.2.2).

b. $PM_{2.5}$ Components

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. (U.S. EPA, 2020, section 2.3.2.3). 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.

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, 2019, 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, 2019, section 2.5.1.1.6; U.S. EPA, 2020, section 2.3.1).

c. PM_{10}

At monitoring sites in the U.S., the 2015–2017 average of 2nd highest 24-hour PM_{10} concentration was 56 μg/m^3 (ranging from 18 to 173 μg/m^3) (U.S. EPA, 2020, section 2.3.2.4).[24] 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, 2019, 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.

Recent ambient PM_{10} concentrations reflect reductions that have occurred across much of the U.S. (U.S. EPA, 2020, section 2.3.2.4). From 2000 to 2017, annual second highest 24-hour PM_{10} concentrations have declined by about 30% (U.S. EPA, 2020, section 2.3.2.4).[25] These 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 (U.S. EPA, 2020, section 2.3.2.4). Analyses at individual monitoring sites indicate that annual average PM_{10} concentrations have also 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.

d. $PM_{10\text{-}2.5}$

Since the last review, the availability of $PM_{10\text{-}2.5}$ ambient concentration data has greatly increased. As illustrated in the PA (U.S. EPA, 2020, section 2.3.2.5), annual average and 98th percentile $PM_{10\text{-}2.5}$ 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 $PM_{10\text{-}2.5}$ concentrations have been small in magnitude and inconsistent in direction (U.S. EPA, 2020, section 2.3.2.5).

e. UFP

Compared to $PM_{2.5}$ mass, there is relatively little data on U.S. particle number concentrations, which are dominated by UFP. Based on measurements in two urban areas (New York City, Buffalo) and at a background site (Steuben County) in New York, urban particle number counts were several times higher than at the background site (U.S. EPA, 2020, section 2.3.2.6; U.S. EPA, 2019, Figure 2–18). The highest particle number counts in an urban area with multiple sites (Buffalo) were observed at a near-road location.

Long-term trends in UFP are not routinely available at U.S. monitoring sites. At one site in Illinois with long-term data available, the annual average particle number concentration declined between 2000 and 2017, closely matching the reductions in annual $PM_{2.5}$ mass over that same period (U.S. EPA, 2020, section 2.3.2.6). In addition, a small number of published studies have examined UFP trends over time. While limited, these studies also suggest that UFP number concentrations have declined over time along with decreases in $PM_{2.5}$ (U.S. EPA, 2020, section 2.3.2.6).

5. Background PM

In this review, background PM is defined as all particles that are formed by sources or processes that cannot be influenced by actions within the jurisdiction of concern. 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. Background PM is discussed in more detail in section 2.4 of the PA (U.S. EPA, 2020).

At annual and national scales, estimated background PM concentrations in the U.S. are small compared to contributions from domestic anthropogenic emissions. For example, based on zero-out modeling in the last review of the PM NAAQS, annual background $PM_{2.5}$ concentrations were estimated to range from 0.5–3 μg/m^3 across the sites examined. In addition, speciated monitoring data from IMPROVE sites can provide some insights into how contributions from different PM sources, including sources of background PM, may have changed over time. As discussed further in the PA (U.S. EPA, 2020, section 2.4), such data suggests that estimates of background concentrations at IMPROVE monitors are around 1–3 μg/m^3, and have not changed significantly since the last PM NAAQS Review.

As discussed further in the PA (U.S. EPA, 2020, section 2.4), sources that contribute to natural background PM include dust from the wind erosion of natural surfaces, sea salt, wildland fires, primary biological aerosol particles such as bacteria and pollen, oxidation of biogenic hydrocarbons such as isoprene and terpenes to produce secondary

[23] 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.

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

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

organic aerosols (SOA), and geogenic sources such as sulfate formed from volcanic production of SO_2 and oceanic production of dimethyl-sulfide. While most of these 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, 2019, section 2.3.3).

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 exceedances of the 24-hour $PM_{2.5}$ standard (35 μg/m³) in some cases (Schweizer et al., 2017), such events are routinely screened for and usually identifiable in the monitoring data. As described further in the PA (U.S. EPA, 2020, section 2.4), 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 frequent and represent a relatively small fraction of background PM in most places.

II. Rationale for Proposed Decisions on the Primary $PM_{2.5}$ Standards

This section provides the rationale supporting the Administrator's proposed decisions on the primary $PM_{2.5}$ standards. Section II.A describes the Agency's approach to reaching decisions on the primary $PM_{2.5}$ standards in the last review and summarizes the general approach to reaching proposed decisions in this review. Section II.B summarizes the scientific evidence for $PM_{2.5}$-related health effects. Section II.C presents the Administrator's proposed conclusions regarding the adequacy of the current primary $PM_{2.5}$ standards and his proposed decision to retain those standards in this review.[26]

A. General Approach

1. Approach Used in the Last Review

The last review of the primary PM NAAQS was completed in 2012 (78 FR 3086, January 15, 2013). As noted above (section 1.3), in the last review the EPA lowered the level of the primary annual $PM_{2.5}$ standard from 15.0 to 12.0 μg/m³,[27] and retained the existing 24-hour $PM_{2.5}$ standard with its level of 35 μg/m³. The 2012 decision to strengthen the suite of primary $PM_{2.5}$ standards was based on the prior Administrator's consideration of the extensive body of scientific evidence assessed in the 2009 ISA (U.S. EPA, 2009c); the quantitative risk analyses presented in the 2010 health risk assessment (U.S. EPA, 2010a); the advice and recommendations of the CASAC (Samet, 2009; Samet, 2010c; Samet, 2010b); and public comments on the proposed rule (78 FR 3086, January 15, 2013; U.S. EPA, 2012a). She particularly noted the ''strong and generally robust body of evidence of serious health effects associated with both long- and short-term exposures to $PM_{2.5}$'' (78 FR 3120, January 15, 2013). This included epidemiologic studies reporting health effect associations based on long-term average $PM_{2.5}$ concentrations ranging from about 15.0 μg/m³ or above (*i.e.,* at or above the level of the then-existing annual standard) to concentrations ''significantly below the level of the annual standard'' (78 FR 3120, January 15, 2013). Based on her ''confidence in the association between exposure to $PM_{2.5}$ and serious public health effects, combined with evidence of such an association in areas that would meet the current standards'' (78 FR 3120, January 15, 2013), the prior Administrator concluded that revision of the suite of primary $PM_{2.5}$ standards was necessary in order to provide increased public health protection.

The prior Administrator next considered what specific revisions to the existing primary $PM_{2.5}$ standards were appropriate, given the available evidence and quantitative risk information. She considered both the annual and 24-hour $PM_{2.5}$ standards, focusing on the basic elements of those standards (*i.e.,* indicator, averaging time, form, and level). These considerations, and the prior Administrator's conclusions, are summarized in sections II.A.1.a to II.A.1.d below.

a. Indicator

In the last review, the EPA considered issues related to the appropriate indicator for fine particles, with a focus on evaluating support for the existing $PM_{2.5}$ mass-based indicator and for potential alternative indicators based on the UFP fraction or on fine particle composition (78 FR 3121, January 15, 2013).[28] With regard to $PM_{2.5}$ mass, as in the 1997 and 2006 reviews, the health studies available during the last review continued to link adverse health outcomes (*e.g.,* premature mortality, hospital admissions, emergency department visits) with long- and short-term exposures to fine particles indexed largely by $PM_{2.5}$ mass (78 FR 3121, January 15, 2013). With regard to the ultrafine fraction of ambient PM, the 2011 PA noted the limited body of health evidence assessed in the 2009 ISA (summarized in U.S. EPA, 2009c, section 2.3.5 and Table 2–6) and the limited monitoring information available to characterize ambient concentrations of UFP (U.S. EPA, 2011, section 1.3.2). With regard to PM composition, the 2009 ISA concluded that ''the evidence is not yet sufficient to allow differentiation of those constituents or sources that are more closely related to specific health outcomes'' (U.S. EPA, 2009c, pp. 2–26 and 6–212; 78 FR 3123, January 15, 2013). The 2011 PA further noted that ''many different constituents of the fine particle mixture as well as groups of components associated with specific source categories of fine particles are linked to adverse health effects'' (U.S. EPA, 2011, p. 2–55; 78 FR 3123, January 15, 2013). Consistent with the considerations and conclusions in the 2011 PA, the CASAC advised that it was appropriate to consider retaining $PM_{2.5}$ as the indicator for fine particles. In light of the evidence and the CASAC's advice, the prior Administrator concluded that it was ''appropriate to retain $PM_{2.5}$ as the indicator for fine particles'' (78 FR 3123, January 15, 2013).

[26] Sections III and IV provide the rationales supporting the Administrator's proposed decisions on the primary PM_{10} standard and secondary standards, respectively.

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

[28] In the last review, the ISA defined UFP as generally including particles with a mobility diameter less than or equal to 0.1 μm. Mobility diameter is defined as the diameter of a particle having the same diffusivity or electrical mobility in air as the particle of interest, and is often used to characterize particles of 0.5 μm or smaller (U.S. EPA, 2009c, pp. 3–2 to 3–3).

b. Averaging Time

In 1997, the EPA set an annual $PM_{2.5}$ standard to provide protection from health effects associated with long- and short-term exposures to $PM_{2.5}$, and a 24-hour standard to supplement the protection afforded by the annual standard (62 FR 38667 to 38668, July 18, 1997). In the 2006 review, the EPA retained both annual and 24-hour averaging times (71 FR 61164, October 17, 2006). In the last review, the EPA again considered issues related to the appropriate averaging times for $PM_{2.5}$ standards, with a focus on evaluating support for the existing annual and 24-hour averaging times and for potential alternative averaging times based on sub-daily or seasonal metrics.

Based on the evidence assessed in the ISA, the 2011 PA noted that the overwhelming majority of studies that had been conducted since the 2006 review continued to utilize annual (or multi-year) or 24-hour PM averaging periods (U.S. EPA, 2011, section 2.3.2). Given this, and limitations in the data for alternatives, the 2011 PA reached the overall conclusions that the available information provided strong support for considering retaining the current annual and 24-hour averaging times (U.S. EPA, 2011, p. 2–58). The CASAC agreed that these conclusions were reasonable (Samet, 2010a, p. 13). The prior Administrator concurred with the PA conclusions and with the CASAC's advice. Specifically, she judged that it was ''appropriate to retain the current annual and 24-hour averaging times for the primary $PM_{2.5}$ standards to protect against health effects associated with long- and short-term exposure periods'' (78 FR 3124, January 15, 2013).

c. Form

In 1997, the EPA established the form of the annual $PM_{2.5}$ standard as an annual arithmetic mean, averaged over 3 years, from single or multiple community-oriented monitors.[29] 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)[30] (62 FR 38671 to 38672, July 18, 1997). In the 1997 review, the EPA also established the form of the 24-hour $PM_{2.5}$ standard as the 98th percentile of 24-hour concentrations at each monitor within an area (*i.e.,* no spatial averaging), averaged over three years (62 FR at 38671 to 38674, July 18, 1997). In the 2006 review, the EPA retained these standard forms but tightened the criteria for using spatial averaging with the annual standard (71 FR 61167, October 17, 2006).[31]

In the last review, the EPA's consideration of the form of the annual $PM_{2.5}$ standard again included a focus on the issue of spatial averaging. An analysis of air quality and population demographic information indicated that the highest $PM_{2.5}$ concentrations in a given area tended to be measured at monitors in locations where the surrounding populations were more likely to live below the poverty line and to include larger percentages of racial and ethnic minorities (U.S. EPA, 2011, p. 2–60). Based on this analysis, the 2011 PA concluded that spatial averaging could result in disproportionate impacts in at-risk populations, including minority populations and populations with lower socioeconomic status (SES). Therefore, the PA concluded that it was appropriate to consider revising the form of the annual $PM_{2.5}$ standard such that it did not allow for the use of spatial averaging across monitors (U.S. EPA, 2011, p. 2–60). The CASAC agreed with the PA conclusions that it was ''reasonable'' for the EPA to eliminate the spatial averaging provisions (Samet, 2010c, p. 2).

The prior 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 last review, the EPA also considered the form of the 24-hour $PM_{2.5}$ standard. The Agency recognized that the existing 98th percentile form for the 24-hour standard was originally selected to provide a balance between limiting the occurrence of peak 24-hour $PM_{2.5}$ concentrations and identifying a stable target for risk management programs.[32] Updated air quality analyses in the last review provided additional support for the increased stability of the 98th percentile $PM_{2.5}$ concentration, compared to the 99th percentile (U.S. EPA, 2011, Figure 2–2, p. 2–62). Consistent with the PA conclusions based on this analysis, the prior 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).

d. Level

The EPA's approach to considering alternative levels of the $PM_{2.5}$ standards in the last review was based on evaluating the public health protection afforded by the annual and 24-hour standards, taken together, against mortality and morbidity effects associated with long-term or short-term $PM_{2.5}$ exposures. This approach recognized that it is appropriate to consider the protection provided by attaining the air quality needed to meet the suite of standards, and that there is no bright line clearly directing the choice of levels. Rather, the choice of what is appropriate is a public health policy judgment entrusted to the Administrator. See *Mississippi,* 744 F.3d at 1358, *Lead Industries Ass'n,* 647 F.2d at 1147.

In selecting the levels of the annual and 24-hour $PM_{2.5}$ standard, the prior Administrator placed the greatest emphasis on health endpoints for which the evidence was strongest, based on the assessment of the evidence in the ISA and on the ISA's causality determinations (U.S. EPA, 2009c, section 2.3.1). She particularly noted that the evidence was sufficient to conclude a causal relationship exists between $PM_{2.5}$ exposures and mortality and cardiovascular effects (*i.e.,* for both long- and short-term exposures) and that the evidence was sufficient to conclude a causal relationship is ''likely'' to exist between $PM_{2.5}$ exposures and respiratory effects (*i.e.,* for both long-

[29] In the last review, the EPA replaced the term ''community-oriented'' monitor with the term ''area-wide'' monitor (U.S. EPA, 2020, section 1.3). *Area-wide* monitors are those sited at the neighborhood scale or larger, as well as those monitors sited at micro- or middle scales that are representative of many such locations in the same core-based statistical area (CBSA; 78 FR 3236, January 15, 2013). CBSAs are required to have at least one area-wide monitor sited in the area of expected maximum $PM_{2.5}$ concentration.

[30] The original criteria for spatial averaging included: (1) The annual mean concentration at each site shall be within 20% of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.6 for each calendar quarter (62 FR 38671 to 38672, July 18, 1997).

[31] 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).

[32] See *ATA III,* 283 F.3d at 374–76 which concludes that it is legitimate for the EPA to consider overall stability of the standard and its resulting promotion of overall effectiveness of NAAQS control programs in setting a standard that is requisite to protect the public health.

and short-term exposures). She also noted additional, but more limited, evidence for a broader range of health endpoints, including evidence ''suggestive of a causal relationship'' between long-term exposures and developmental and reproductive effects as well as carcinogenic effects (78 FR 3158, January 15, 2013).

To inform her decisions on an appropriate level for the annual standard, the prior Administrator considered the degree to which epidemiologic studies indicate confidence in the reported health effect associations over distributions of ambient $PM_{2.5}$ concentrations. She noted that a level of 12.0 μg/m^3 was below the long-term mean $PM_{2.5}$ concentrations reported in key epidemiologic studies that provided evidence of an array of serious health effects (78 FR 3161, January 15, 2013). She further noted that 12.0 μg/m^3 generally corresponded to the lower portions (*i.e.,* about the 25th percentile) of distributions of health events in the limited number of epidemiologic studies for which population-level information was available. A level of 12.0 μg/m^3 also reflected placing some weight on studies of reproductive and developmental effects, for which the evidence was more uncertain (78 FR 3161–3162, January 15, 2013).[33]

Given the uncertainties remaining in the scientific information, the prior Administrator judged that an annual standard level below 12.0 μg/m^3 was not supported. She specifically noted uncertainties related to understanding the relative toxicity of the different components in the fine particle mixture, the role of $PM_{2.5}$ in the complex ambient mixture, exposure measurement errors in epidemiologic studies, and the nature and magnitude of estimated risks at relatively low ambient $PM_{2.5}$ concentrations. Furthermore, she noted that epidemiologic studies had reported heterogeneity in responses both within and between cities and in geographic regions across the U.S. She recognized that this heterogeneity may be attributed, in part, to differences in fine particle composition in different regions and cities. With regard to evidence for reproductive and developmental effects, the prior Administrator recognized that there were a number of limitations associated with this body of evidence, including the following: The limited number of studies evaluating such effects; uncertainties related to identifying the relevant exposure time periods of concern; and limited toxicological evidence providing little information on the mode of action(s) or biological plausibility for an association between long-term $PM_{2.5}$ exposures and adverse birth outcomes. On balance, she found that the available evidence, interpreted in light of these remaining uncertainties, did not justify an annual standard level set below 12.0 μg/m^3 as being ''requisite'' to protect public health with an adequate margin of safety (*i.e.,* a standard with a lower level would have been more stringent than necessary).

In conjunction with a revised annual standard with a level of 12.0 μg/m^3, the prior Administrator concluded that the evidence supported retaining the 35 μg/m^3 level of the 24-hour $PM_{2.5}$ standard. She noted that the existing 24-hour standard, with its 35 μg/m^3 level and 98th percentile form, would provide supplemental protection, particularly for areas with high peak-to-mean ratios possibly associated with strong seasonal sources and for areas with $PM_{2.5}$-related effects that may be associated with shorter than daily exposure periods (78 FR 3163, January 15, 2013). Thus, she concluded that the available evidence and information, interpreted in light of remaining uncertainties, supported an annual standard with a level of 12.0 μg/m^3 combined with a 24-hour standard with a level of 35 μg/m^3.

2. Approach in the Current Review

The EPA's approach to reaching proposed decisions on the primary $PM_{2.5}$ standards in the current review builds on the decisions made in the last review. Consistent with that review, the approach focuses on evaluating the public health protection afforded by the annual and 24-hour standards, taken together, against mortality and morbidity associated with long-term or short-term $PM_{2.5}$ exposures. As discussed in the PA (U.S. EPA, 2020, section 3.1.2), in adopting this approach the EPA recognizes that changes in $PM_{2.5}$ air quality designed to meet an annual standard would likely result not only in lower annual average $PM_{2.5}$ concentrations, but also in fewer and lower short-term peak $PM_{2.5}$ concentrations. Additionally, changes designed to meet a 24-hour standard, with a 98th percentile form, would result not only in fewer and lower peak 24-hour $PM_{2.5}$ concentrations, but also in lower annual average $PM_{2.5}$ concentrations. Thus, the EPA's approach recognizes that it is appropriate to consider the protection provided by attaining the air quality needed to meet the suite of standards.

This approach to reviewing the primary $PM_{2.5}$ standards is based most fundamentally on considering the available scientific evidence and technical information as assessed and discussed in the ISA (U.S. EPA, 2019) and PA (U.S. EPA, 2020), including the uncertainties inherent in that evidence and information, and on consideration of advice received from the CASAC in this review (Cox, 2019a). The EPA emphasizes the health outcomes for which the ISA determines that the evidence supports either a ''causal'' or a ''likely to be causal'' relationship with $PM_{2.5}$ exposures (U.S. EPA, 2019). This approach focuses proposed decisions on the health outcomes for which the evidence is strongest. Such a focus, which is supported by the CASAC (Cox, 2019a, p. 12 of consensus responses), recognizes that standards set based on evidence supporting ''causal'' and ''likely to be causal'' health outcomes will also provide some measure of protection against the broader range of $PM_{2.5}$-associated outcomes, including those for which the evidence is less certain.

As in past reviews, the EPA's approach recognizes that there is no bright line clearly directing the choice of standards. Rather, the choice of what is appropriate is a public health policy judgment entrusted to the Administrator. Specifically, the CAA requires primary standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety. In setting primary standards that are ''requisite'' to protect public health, the EPA's task is to establish standards that are neither more nor less stringent than necessary for this purpose. Thus, as discussed above (I.A), the CAA does not require that primary standards be set at a zero-risk level, but rather at a level that, in the judgment of the Administrator, limits risk sufficiently so as to protect public health with an adequate margin of safety. As in previous reviews, this judgment includes consideration of the strengths and limitations of the scientific and technical information, and the appropriate inferences to be drawn from that information.

[33] With respect to cancer, mutagenic, and genotoxic effects, the Administrator observed that the $PM_{2.5}$ concentrations reported in studies evaluating these effects generally included ambient concentrations that are equal to or greater than ambient concentrations observed in studies that reported mortality and cardiovascular and respiratory effects (U.S. EPA, 2009c, section 7.5). Therefore, the Administrator concluded that, in selecting a standard level that provides protection from mortality and cardiovascular and respiratory effects, it is reasonable to anticipate that protection will also be provided for carcinogenic effects (78 FR 3161–3162, January 15, 2013).

B. Health Effects Related to Fine Particle Exposures

This section draws from the EPA's synthesis and assessment of the scientific evidence presented in the ISA (U.S. EPA, 2019) and the summary of that evidence in the PA (U.S. EPA, 2020, section 3.2.1). The ISA uses a weight-of-evidence framework for characterizing the strength of the available scientific evidence for health effects attributable to PM exposures (U.S. EPA, 2015, Preamble, Section 5). As in the last review (U.S. EPA, 2009c), the ISA for this review has adopted a five-level hierarchy to classify the overall weight-of-evidence into one of the following categories: Causal relationship; a likely to be causal relationship; suggestive of, but not sufficient to infer, a causal relationship;[34] inadequate to infer the presence or absence of a causal relationship; and not likely to be a causal relationship (U.S. EPA, 2015, Preamble Table II). In using the weight-of-evidence approach to inform judgments about the likelihood that various health effects are caused by PM exposures, evidence is evaluated for major outcome categories or groups of related outcomes (*e.g.,* respiratory effects), integrating evidence from across disciplines, including epidemiologic, controlled human exposure, and animal toxicological studies and evaluating the coherence of evidence across a spectrum of related endpoints as well as biological plausibility of the effects observed (U.S. EPA, 2015, Preamble, Section 5.c.). Based on application of this approach, the EPA believes that the final ISA ''accurately reflects 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 [PM] in the ambient air, in varying quantities'' as required by the CAA (42 U.S.C. 7408(a)(2)).

In this review of the NAAQS, the EPA considers the full body of health evidence, placing the greatest emphasis on the health effects for which the evidence has been judged in the ISA to demonstrate a ''causal'' or a ''likely to be causal'' relationship with PM exposures. The ISA defines these causality determinations as follows (U.S. EPA, 2019, p. p–20):

- *Causal relationship:* The pollutant has been shown to result in health effects at relevant exposures based on studies encompassing multiple lines of evidence and chance, confounding, and other biases can be ruled out with reasonable confidence.
- *Likely to be a causal relationship:* There are studies in which results are not explained by chance, confounding, or other biases, but uncertainties remain in the health effects evidence overall. For example, the influence of co-occurring pollutants is difficult to address, or evidence across scientific disciplines may be limited or inconsistent.

The sections below briefly summarize the health effects evidence determined in the ISA to support either a ''causal'' or a ''likely to be causal'' relationship with fine particle exposures (II.B.1), the populations potentially at increased risk for PM-related effects (II.B.2), and the CASAC's advice on the draft ISA (II.B.3). Additional detail on these topics can be found in the ISA (U.S. EPA, 2019) and in the PA (U.S. EPA, 2020, section 3.2).

1. Nature of Effects

Drawing from the assessment of the evidence in the ISA (U.S. EPA, 2019), and the summaries of that assessment in the PA (U.S. EPA, 2020), the sections below summarize the evidence for relationships between long- or short-term $PM_{2.5}$ exposures and mortality (II.B.1.a), cardiovascular effects (II.B.1.b), respiratory effects (II.B.1.c), cancer (II.B.1.d), and nervous system effects (II.B.1.e). For these outcomes, the ISA concludes that the evidence supports either a ''causal'' or a ''likely to be causal'' relationship with $PM_{2.5}$ exposures.

a. Mortality

i. Long-term $PM_{2.5}$ exposures

In the last review, the 2009 PM ISA reported that the evidence was ''sufficient to conclude that the relationship between long-term $PM_{2.5}$ exposures and mortality is causal'' (U.S. EPA, 2009c, 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) cohort and the Harvard Six Cities cohort. 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, 2009c).

Recent cohort studies, which have become available since the 2009 ISA, continue to provide consistent evidence of positive associations between long-term $PM_{2.5}$ exposures and mortality. These studies add support for associations with total and non-accidental mortality,[35] as well as with specific causes of death, including cardiovascular disease and respiratory disease (U.S. EPA, 2019, section 11.2.2). Many of these recent studies have extended the follow-up periods originally evaluated in the ACS and Harvard Six Cities cohort studies 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 recent evaluations of the ACS and Six Cities cohorts, studies conducted with other cohorts also show 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 11.2.2.1, 11.2.5). This includes some of the largest cohort studies conducted to date, with analyses of the U.S. Medicare cohort that include nearly 61 million enrollees (Di et al., 2017b) and studies that control for a range of individual and ecological covariates.

A recent series of retrospective studies has additionally 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). Pope et al. (2009) conducted a cross-sectional analysis using air quality data from 51 metropolitan areas across the U.S., beginning in the 1970s through the early 2000s, and found 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, the 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 $PM_{2.5}$

[34] As noted in the 2019 p.m. ISA (U.S. EPA, 2019, p. ES–15), this causality determination language has been updated since the last review.

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

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).

The 2019 ISA specifically evaluates the degree to which recent studies that examine the relationship between long-term $PM_{2.5}$ exposure and mortality have addressed key policy-relevant issues and/or previously identified data gaps in the scientific evidence. For example, based on its assessment of the evidence, the ISA concludes that positive associations between long-term $PM_{2.5}$ exposures and mortality are robust across analyses examining a variety of study designs (*e.g.,* U.S. EPA, 2019, section 11.2.2.4), approaches to estimating $PM_{2.5}$ exposures (U.S. EPA, 2019, section 11.2.5.1), approaches to controlling for confounders (U.S. EPA, 2019, sections 11.2.3 and 11.2.5), geographic regions and populations, and temporal periods (U.S. EPA, 2019, sections 11.2.2.5 and 11.2.5.3). Recent evidence further demonstrates that associations with mortality remain robust in copollutant 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).

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. Several recent studies 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). However, interpreting the shapes of these 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, Preamble section 6.c).

The biological plausibility of $PM_{2.5}$-attributable mortality is supported by the coherence of effects across scientific disciplines (*i.e.,* animal toxicological, controlled human exposure studies, and epidemiologic), including in recent studies evaluating the morbidity effects that are the largest contributors to total (nonaccidental) mortality. The ISA outlines the available evidence for plausible pathways by which inhalation exposure to $PM_{2.5}$ could progress from initial events (*e.g.,* respiratory tract inflammation, autonomic nervous system modulation) to endpoints relevant to population outcomes, particularly those related to cardiovascular diseases such as ischemic heart disease, stroke and atherosclerosis (U.S. EPA, 2019, section 6.2.1), and to metabolic disease and diabetes (U.S. EPA, 2019, section 7.2.1). The ISA notes ''more limited evidence from respiratory morbidity'' (U.S. EPA, 2019, p. 11–101) 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 reaffirm and further strengthen the body of evidence from the 2009 ISA for the relationship between long-term $PM_{2.5}$ exposure and mortality. Recent epidemiologic studies consistently report positive associations with mortality across different geographic locations, populations, and analytic approaches. Such studies reduce key uncertainties identified in the last review, including those related to potential copollutant confounding, and provide additional information on the shape of the concentration-response curve. Recent experimental and epidemiologic evidence for cardiovascular effects, and respiratory effects to a more limited degree, supports the plausibility of mortality due to long-term $PM_{2.5}$ exposures. The 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).

ii. Short-term $PM_{2.5}$ exposures

The 2009 PM ISA concluded that ''a causal relationship exists between short-term exposure to $PM_{2.5}$ and mortality'' (U.S. EPA, 2009c). This conclusion was based on the evaluation of both multi- and single-city epidemiologic studies that consistently reported positive associations between short-term $PM_{2.5}$ exposure and non-accidental mortality. These associations were strongest, in terms of magnitude and precision, primarily at lags of 0 to 1 days. Examination of the potential confounding effects of gaseous copollutants was limited, though evidence from single-city studies indicated that gaseous copollutants have minimal effect on the $PM_{2.5}$-mortality relationship (*i.e.,* associations remain robust to inclusion of other pollutants in copollutant models). The evaluation of cause-specific mortality found that effect estimates were larger in magnitude, but also had larger confidence intervals, for respiratory mortality compared to cardiovascular mortality. Although the largest mortality risk estimates were for respiratory mortality, the interpretation of the results was complicated by the limited coherence from studies of respiratory morbidity. However, the evidence from studies of cardiovascular morbidity provided both coherence and biological plausibility for the relationship between short-term $PM_{2.5}$ exposure and cardiovascular mortality.

Recent multicity studies evaluated since the 2009 ISA continue to provide evidence of primarily positive associations between daily $PM_{2.5}$ exposures and mortality, with percent increases in total mortality ranging from 0.19% (Lippmann et al., 2013) to 2.80% (Kloog et al., 2013) [36] at lags of 0 to 1 days in single-pollutant models. Whereas most studies rely on assigning exposures using data from ambient monitors, associations are also reported in recent studies that employ hybrid modeling approaches using additional $PM_{2.5}$ data (*i.e.,* from satellites, land use information, and modeling, in addition to monitors), allowing for the inclusion of more rural locations in analyses (Kloog et al., 2013, Shi et al., 2016, Lee et al., 2015).

Some recent studies have expanded the examination of potential confounders (*e.g.,* U.S. EPA, 2019, section 11.1.5.1), including copollutants. 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).

The generally positive associations reported with mortality are supported

[36] 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).

by a small group of studies employing causal inference or quasi-experimental statistical approaches (U.S. EPA, 2019, section 11.1.2.1). For example, a recent study examines whether a specific regulatory action in Tokyo, Japan (*i.e.,* a diesel emission control ordinance) resulted in a subsequent reduction in daily mortality (Yorifuji et al., 2016). The authors report a reduction in mortality in Tokyo due to the ordinance, compared to Osaka, which did not have a similar diesel emission control ordinance in place.

The positive associations for total mortality reported across the majority of studies evaluated are further supported by analyses reporting generally consistent, positive associations with both cardiovascular and respiratory mortality (U.S. EPA, 2019, section 11.1.3). For both cardiovascular and respiratory mortality, there has been only limited assessment of potential copollutant confounding, though initial evidence indicates that associations remain positive and relatively unchanged in models with gaseous pollutants and $PM_{10-2.5}$. This evidence further supports the copollutant analyses conducted for total mortality. The 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%) (U.S. National Institutes of Health, 2013). Although there is evidence for exacerbations of chronic obstructive pulmonary disease (COPD) and asthma, the collective body of evidence, particularly from controlled human exposure studies of respiratory effects, provides only limited support for the biological plausibility of $PM_{2.5}$-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 as housing stock and commuting, as well as city-specific factors (*e.g.,* land-use, port volume, and traffic information), may explain some of the observed heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Collectively, recent studies indicate that the heterogeneity in $PM_{2.5}$-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).

A number of recent studies conducted systematic evaluations of the lag structure of associations for the $PM_{2.5}$-mortality relationship by examining either a series of single-day or multiday lags and these studies continue to support an immediate effect (*i.e.,* lag 0 to 1 days) of short-term $PM_{2.5}$ exposures on mortality (U.S. EPA, 2019, section 11.1.8.1). Recent studies also conducted analyses comparing the traditional 24-hour average exposure metric with a sub-daily metric (*i.e.,* 1-hour max). These initial studies provide evidence of a similar pattern of associations for both the 24-hour average and 1-hour max metric, with the association larger in magnitude for the 24-hour average metric.

Recent multicity studies indicate that positive and statistically significant associations with mortality persist in analyses restricted to short-term $PM_{2.5}$ exposures below 35 $\mu g/m^3$ (Lee et al., 2015),[37] below 30 $\mu g/m^3$ (Shi et al., 2016), and below 25 $\mu g/m^3$ (Di et al., 2017a). Additional studies examine the shape of the concentration-response 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 found linear relationships with no evidence of a threshold. Recent analyses provide initial evidence indicating that $PM_{2.5}$-mortality associations persist and may be stronger (*i.e.,* a steeper slope) at lower concentrations (*e.g.,* Di et al., 2017a; Figure 11–12 in U.S. EPA, 2019). However, given the limited data available at the lower end of the distribution of ambient $PM_{2.5}$ concentrations, the shape of the concentration-response curve remains uncertain at these low concentrations and, to date, studies have not conducted extensive analyses exploring alternatives to linearity when examining the shape of the $PM_{2.5}$-mortality concentration-response relationship.

Overall, recent epidemiologic studies build upon and extend the conclusions of the 2009 ISA for the relationship between short-term $PM_{2.5}$ exposures and total mortality. Supporting evidence for $PM_{2.5}$-related cardiovascular morbidity, and more limited evidence from respiratory morbidity, provides biological plausibility for mortality due to short-term $PM_{2.5}$ exposures. The primarily positive associations observed across studies conducted in diverse geographic locations is further supported by the results from co-pollutant analyses indicating robust associations, along with evidence from analyses of the concentration-response relationship. The 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).

b. Cardiovascular Effects

i. Long-Term $PM_{2.5}$ Exposures

The scientific evidence reviewed in the 2009 PM ISA was ''sufficient to infer a causal relationship between long-term $PM_{2.5}$ exposure and cardiovascular effects'' (U.S. EPA, 2009c). The strongest line of evidence comprised findings from several large epidemiologic studies of U.S. cohorts that consistently showed positive associations between long-term $PM_{2.5}$ exposure and cardiovascular mortality (Pope et al., 2004, Krewski et al., 2009, Miller et al., 2007, Laden et al., 2006). Studies of long-term $PM_{2.5}$ exposure and cardiovascular morbidity were limited in number. Biological plausibility and coherence with the epidemiologic findings were provided by studies using genetic mouse models of atherosclerosis demonstrating enhanced atherosclerotic plaque development and inflammation, as well as changes in measures of impaired heart function, following 4- to 6-month exposures to $PM_{2.5}$ concentrated ambient particles (CAPs), and by a limited number of studies reporting CAPs-induced effects on coagulation factors, vascular reactivity, and worsening of experimentally induced hypertension in mice (U.S. EPA, 2009c).

Studies conducted since the last review continue to support the relationship between long-term exposure to $PM_{2.5}$ and cardiovascular effects. As discussed above, results from recent U.S. and Canadian cohort studies consistently report positive associations between long-term $PM_{2.5}$ exposure and cardiovascular mortality (U.S. EPA, 2019, Figure 6–19) in evaluations

[37] 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 $\mu g/m^3$.

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).[38]

The body of literature examining the relationship between long-term $PM_{2.5}$ exposure and cardiovascular morbidity has greatly expanded since the 2009 PM ISA, with positive associations reported in several cohorts (U.S. EPA, 2019, section 6.2). Though results for cardiovascular morbidity are less consistent than those for cardiovascular mortality (U.S. EPA, 2019, section 6.2), recent studies provide some evidence for associations between long-term $PM_{2.5}$ exposures and the progression of cardiovascular disease. Positive associations with cardiovascular morbidity (*e.g.,* coronary heart disease, stroke) and atherosclerosis progression (*e.g.,* coronary artery calcification) are observed in several epidemiologic studies (U.S. EPA, 2019, sections 6.2.2. to 6.2.9). Associations in such studies are supported by toxicological evidence for increased plaque progression in mice following long-term exposure to $PM_{2.5}$ 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.

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

In summary, the 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. 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 an independent effect of $PM_{2.5}$ exposure on mortality. Additional evidence of the independent effect of $PM_{2.5}$ on the cardiovascular system is provided by experimental studies in animals, which support the biological plausibility of pathways by which long-term 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 ISA conclusion that ''a causal relationship exists between long-term exposure to $PM_{2.5}$ and cardiovascular effects'' (U.S. EPA, 2019, p. 6–222).

ii. Short-Term $PM_{2.5}$ Exposures

The 2009 PM ISA concluded that ''a causal relationship exists between short-term exposure to $PM_{2.5}$ and cardiovascular effects'' (U.S. EPA, 2009c). The strongest evidence in the 2009 PM ISA was from epidemiologic studies of emergency department visits and hospital admissions for ischemic heart disease (IHD) and heart failure (HF), with supporting evidence from epidemiologic studies of cardiovascular mortality (U.S. EPA, 2009c). Animal toxicological studies provided coherence and biological plausibility for the positive associations reported with myocardial ischemia, emergency department visits, 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.[39] Key uncertainties from the last review resulted from inconsistent results across disciplines with respect to the relationship between short-term exposure to $PM_{2.5}$ and changes in blood pressure, blood coagulation markers, and markers of systemic inflammation. In addition, while the 2009 PM ISA identified a growing body of evidence from controlled human exposure and animal toxicological studies, uncertainties remained with respect to biological plausibility.

A large body of recent evidence confirms and extends the evidence from the 2009 ISA supporting the 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 hospital admissions for IHD, 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). Additional multicity studies conducted in the northeast U.S. report positive associations between short-term $PM_{2.5}$ exposures and emergency department visits or hospital admissions for IHD (U.S. EPA, 2019, section 6.1.2.1) while studies conducted in the U.S. and Canada reported positive associations between short-term $PM_{2.5}$ exposures and emergency department visits for HF. Epidemiologic studies conducted in single cities contribute some support, though associations reported in 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.

In addition, a number of more recent controlled human exposure, animal toxicological, and epidemiologic panel studies provide evidence that $PM_{2.5}$

[38] As noted above for mortality, uncertainty in the shape of the concentration-response relationship increases near the upper and lower ends of the concentration distribution where the data are limited.

[39] Some animal studies included in the 2009 PM ISA examined exposures to mixtures, such as motor vehicle exhaust or woodsmoke. In these studies, it was unclear if the resulting cardiovascular effects could be attributed specifically to the particulate components of the mixture.

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 $PM_{2.5}$ CAPs report evidence for small increases in blood pressure (U.S. EPA, 2019, section 6.1.6.3). In addition, although not entirely consistent, there is also some evidence across controlled human exposure studies for conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (HRV) (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2). Thus, when taken as a whole, controlled human exposure studies are coherent with epidemiologic studies in that they provide evidence that short-term exposures to $PM_{2.5}$ may result in the types of cardiovascular endpoints that could lead to emergency department visits and hospital admissions in some people.

Animal toxicological studies published since the 2009 ISA also support a relationship between short-term $PM_{2.5}$ exposure and cardiovascular effects. A recent study demonstrating decreased cardiac contractility and left ventricular pressure in mice is coherent with the results of epidemiologic studies that report 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 supports the conclusions reported in the 2009 ISA indicating relationships between short-term $PM_{2.5}$ exposures and hospital admissions and ED visits for IHD and HF, along with cardiovascular mortality. Epidemiologic studies reporting robust associations in copollutant models are supported by direct evidence from controlled human exposure and animal toxicological studies reporting independent effects of $PM_{2.5}$ exposures on endothelial dysfunction as well as endpoints indicating impaired cardiac function, increased risk of arrhythmia, changes in HRV, increases in BP, and increases in indicators of systemic inflammation, oxidative stress, and coagulation (U.S. EPA, 2019, section 6.1.16). Epidemiologic panel studies, although not entirely consistent, provide some evidence that $PM_{2.5}$ exposures are associated with cardiovascular effects, including increased risk of arrhythmia, decreases in HRV, increases in BP, and ST segment depression. 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 reporting positive associations between short-term $PM_{2.5}$ exposures 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).

c. Respiratory Effects

i. Long-Term $PM_{2.5}$ Exposures

The 2009 PM ISA concluded that ''a causal relationship is likely to exist between long-term $PM_{2.5}$ exposure and respiratory effects'' (U.S. EPA, 2009c). 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 the nasal airways of healthy animals. Other animal studies examined exposure to mixtures, such as motor vehicle exhaust and woodsmoke, and effects were not attributed specifically to the particulate components of the mixture.

Recent cohort studies provide additional support for the relationship between long-term $PM_{2.5}$ exposure and decrements in lung function growth (as a measure of lung development), indicating a robust and consistent association across study locations, exposure assessment methods, and time periods (U.S. EPA, 2019, section 5.2.13). This relationship is further supported by a recent 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 recent prospective cohort studies reporting generally positive associations, though several are imprecise (*i.e.,* they report wide confidence intervals). Supporting evidence is provided by studies reporting associations with asthma prevalence in children, with childhood wheeze, and with exhaled nitric oxide, a marker of pulmonary inflammation (U.S. EPA, 2019, section 5.2.13). A recent animal toxicological study showing the development of an allergic phenotype and an increase in a marker of airway responsiveness supports the biological plausibility of the development of 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 recent longitudinal study found declining $PM_{2.5}$ concentrations are also associated with an improvement in chronic bronchitis symptoms in children, strengthening evidence reported in the 2009 ISA for a relationship between

increased chronic bronchitis symptoms and long-term $PM_{2.5}$ exposure (U.S. EPA, 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'' (U.S. EPA, 2019, p. 1–34). Additional epidemiologic evidence ''supports a relationship with asthma development in children, increased bronchitic symptoms in children with asthma, acceleration of lung function decline in adults, and respiratory mortality, including 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. Recent animal studies show pulmonary oxidative stress, inflammation, and morphologic changes in the upper (nasal) and lower airways. Other results show that changes are consistent with the development of allergy and asthma, and with impaired lung development. Overall, the ISA concludes that ''the collective evidence is sufficient to conclude a likely to be causal relationship between long-term $PM_{2.5}$ exposure and respiratory effects'' (U.S. EPA, 2019, p. 5–220).

ii. Short-Term $PM_{2.5}$ Exposures

The 2009 PM ISA (U.S. EPA, 2009c) 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 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 available epidemiologic evidence was inconsistent for hospital admissions or emergency department visits for asthma. Studies examining copollutant models showed that $PM_{2.5}$ associations with respiratory effects were robust to inclusion of CO or SO_2 in the model, but often were attenuated (though still positive) with inclusion of O_3 or NO_2. In addition to the copollutant models, evidence supporting an independent effect of $PM_{2.5}$ exposure on the respiratory system was provided by animal toxicological studies of $PM_{2.5}$ CAPs demonstrating changes in some pulmonary function parameters, as well as inflammation, oxidative stress, injury, enhanced allergic responses, and reduced host defenses. Many of these effects have been implicated in the pathophysiology for asthma exacerbation, COPD exacerbation, or respiratory infection. In the few controlled human exposure studies conducted in individuals with asthma or COPD, $PM_{2.5}$ exposure mostly had no effect on respiratory symptoms, lung function, or pulmonary inflammation. Available studies in healthy people also did not clearly find respiratory effects following short-term $PM_{2.5}$ exposures.

Recent epidemiologic studies provide evidence for a relationship between short-term $PM_{2.5}$ exposure and several respiratory-related endpoints, including asthma exacerbation (U.S. EPA, 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 on the studies evaluated in the 2009 ISA, recent epidemiologic studies expand the assessment of potential copollutant confounding. There is some evidence that $PM_{2.5}$ associations with asthma exacerbation, combined respiratory-related diseases, and respiratory mortality remain relatively unchanged in copollutant models with gaseous pollutants (*i.e.,* O_3, NO_2, SO_2, with more limited evidence for CO) and other particle sizes (*i.e.,* $PM_{10-2.5}$) (U.S. EPA, 2019, section 5.1.10.1).

Insight into whether there is an independent effect of $PM_{2.5}$ on respiratory health is provided by findings from animal toxicological studies. Specifically, short-term exposure to $PM_{2.5}$ has been shown to enhance asthma-related responses in an animal model of allergic airways disease and 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 and controlled human exposure studies provide limited evidence of respiratory effects (U.S. EPA, 2019, section 5.1.12).

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 ISA concludes that this evidence ''is sufficient to conclude a likely to be causal relationship between short-term $PM_{2.5}$ exposure and respiratory effects'' (U.S. EPA, 2019, p. 5–155).

d. Cancer

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, 2009c). 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). Reanalyses of the ACS cohort using different years of $PM_{2.5}$ data and follow-up, along with various exposure assignment approaches, provide consistent evidence of positive associations between long-term $PM_{2.5}$ exposure and lung cancer mortality (U.S. EPA, 2019, Figure 10–3). Additional support for positive associations with lung cancer mortality is provided by recent epidemiologic studies using individual-level data to control for smoking status, by studies of people who have never smoked (though such studies generally report wide confidence intervals due to the small number of lung cancer mortality cases within this population), and in analyses of cohorts that relied upon proxy measures to account for smoking status (U.S. EPA, 2019, section 10.2.5.1.1). Although studies that evaluate lung cancer incidence, including studies of people who have never smoked, are limited in number, recent studies generally report positive associations with long-term $PM_{2.5}$ exposures (U.S. EPA, 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 recent epidemiologic studies have addressed the potential for confounding by copollutants and the shape of the concentration-response relationship. To date, relatively few studies have evaluated the potential for copollutant confounding of the relationship between long-term $PM_{2.5}$ exposure and lung cancer mortality or incidence. The small number of such studies have generally focused on O_3 and report that $PM_{2.5}$ associations remain relatively unchanged in copollutant models (U.S. EPA, 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 (discussed above), fewer studies have examined the shape of the concentration-response curve for cause-specific mortality outcomes, including lung cancer. Several studies have reported no evidence of deviations from linearity in the shape of the concentration-response relationship (Lepeule et al., 2012; *Raaschou-Nielsen et al., 2013;* Puett et al., 2014), though authors provided only limited discussions of results (U.S. EPA, 2019, section 10.2.5.1.4).

In support of the biological plausibility of an independent effect of $PM_{2.5}$ on cancer, the 2019 ISA notes evidence from recent experimental 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).

Epidemiologic evidence for associations between $PM_{2.5}$ exposure 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 there is a likely to be causal relationship between long-term $PM_{2.5}$ exposure and cancer'' (U.S. EPA, 2019, p. 10–77).

In its letter to the Administrator on the draft ISA, the CASAC states that ''the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term $PM_{2.5}$ exposure and . . . cancer'' (Cox, 2019a, p. 1 of letter). The CASAC specifically states that this causality determination ''relies largely on epidemiology studies that . . . do not provide exposure time frames that are appropriate for cancer causation and that there are no animal studies showing direct effects of $PM_{2.5}$ on cancer formation'' (Cox, 2019a, p. 20 of consensus responses).

With respect to the latency period, it is well recognized that ''air pollution exposures experienced over an extended historical time period are likely more relevant to the etiology of lung cancer than air pollution exposures experienced in the more recent past'' (Turner et al. 2011). However, many epidemiologic studies conducted within the U.S. that examine long-term $PM_{2.5}$ exposure and lung cancer incidence and lung cancer mortality rely on more recent air quality data because routine $PM_{2.5}$ monitoring did not start until 1999–2000. An exception to this is the American Cancer Society (ACS) study that had $PM_{2.5}$ concentration data from two time periods, 1979–1983 and from 1999–2000. Turner et al. (2011), conducted a comparison of $PM_{2.5}$ concentrations between these two time periods and found that they were highly correlated ($r > 0.7$), with the relative rank order of metropolitan statistical areas (MSAs) by $PM_{2.5}$ concentrations being ''generally retained over time.'' Therefore, areas where $PM_{2.5}$ concentrations were high remained high over decades (or low remained low) relative to other locations. Long-term exposure epidemiologic studies rely on spatial contrasts between locations; therefore, if a location with high $PM_{2.5}$ concentrations continues to have high concentrations over decades relative to other locations a relationship between the $PM_{2.5}$ exposure and cancer should persist. This was confirmed in a sensitivity analysis conducted by

Turner et al. (2011), where the authors reported a similar hazard ratio (HR) for lung cancer mortality for participants assigned exposure to $PM_{2.5}$ (1979–1983) and $PM_{2.5}$ (1999–2000) in two separate analyses.

While experimental studies showing a direct effect of $PM_{2.5}$ on cancer formation were limited to an animal model of urethane-induced tumor initiation, a large number of experimental studies report that $PM_{2.5}$ exhibits several key characteristics of carcinogens, as indicated by genotoxic effects, oxidative stress, electrophilicity, and epigenetic alterations, all of which provide biological plausibility that $PM_{2.5}$ exposure can contribute to cancer development. The experimental evidence, in combination with multiple recent and previously evaluated epidemiologic studies examining the relationship between long-term $PM_{2.5}$ exposure and both lung cancer incidence and lung cancer mortality that reported generally positive associations across different cohorts, exposure assignment methods, and in analyses of never smokers further addresses uncertainties identified in the 2009 PM ISA. Therefore, upon re-evaluating the causality determination for cancer, when considering CASAC comments on the Draft PM ISA and applying the causal framework as described (U.S. EPA, 2015; U.S. EPA, 2019, section A.3.2.1), the EPA continues to conclude in the 2019 Final PM ISA that the evidence for long-term $PM_{2.5}$ exposure and cancer supports a ''likely to be causal relationship'' (U.S. EPA, 2019, p. 10–77).

e. Nervous System Effects

Reflecting the very limited evidence available in the last review, the 2009 ISA did not make a causality determination for long-term $PM_{2.5}$ exposures and nervous system effects (U.S. EPA, 2009c). Since the last review, this body of evidence has grown substantially (U.S. EPA, 2019, section 8.2). Recent studies in adult animals 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, early markers of Alzheimer's disease pathology have been reported 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, and 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 following long-term $PM_{2.5}$ exposures (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 recent studies of neurodevelopmental effects in children have also been conducted. Positive associations between long-term exposure to $PM_{2.5}$ during the prenatal period and autism spectrum disorder (ASD) are observed in multiple epidemiologic studies (U.S. EPA, 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 the strongest evidence of an effect of long-term exposure to $PM_{2.5}$ on the nervous system is provided by toxicological studies that show inflammation, oxidative stress, morphologic changes, and neurodegeneration in multiple brain regions following long-term exposure of adult animals to $PM_{2.5}$ CAPs. These findings are coherent with epidemiologic studies reporting consistent associations with cognitive decrements and with all-cause dementia. There is also initial, and limited, evidence for neurodevelopmental effects, particularly ASD. The ISA determines that ''[o]verall, the collective evidence is sufficient to conclude a likely to be causal relationship between long-term $PM_{2.5}$ exposure and nervous system effects'' (U.S. EPA, 2019, p. 8–61).

In its letter to the Administrator on the draft ISA, the CASAC states that ''the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term $PM_{2.5}$ exposure and nervous system effects'' (Cox, 2019a, p. 1 of letter). The CASAC specifically states that ''[f]or a likely causal conclusion, there would have to be evidence of health effects in studies where results are not explained by chance, confounding, and other biases, but uncertainties remain in the overall evidence'' (Cox, 2019a, p. 20 of consensus responses). These uncertainties in the eyes of CASAC reflect that animal toxicological studies ''have largely been done by a single group'' (P.20), and for epidemiologic studies that examined brain volume that ''brain volumes can vary . . . between normal people'' and the results from studies of cognitive function were ''largely non-statistically significant''.

With these concerns in mind, the EPA re-evaluated the evidence and note that animal toxicological studies were conducted in ''multiple research groups [and show a range of effects including] inflammation, oxidative stress, morphologic changes, and neurodegeneration in multiple brain regions following long-term exposure of adult animals to $PM_{2.5}$ CAPs'' (U.S. EPA, 2019, p. 8–61). The results from the animal toxicological studies ''are coherent with a number of epidemiologic studies reporting consistent associations with cognitive decrements and with all-cause dementia'' (U.S. EPA, 2019, p. 8–61). Additionally, as discussed in the Preamble to the ISAs (U.S. EPA, 2015):

> ''. . . the U.S. EPA emphasizes the importance of examining the pattern of results across various studies and does not focus solely on statistical significance or the magnitude of the direction of the association as criteria of study reliability. Statistical significance is influenced by a variety of factors including, but not limited to, the size of the study, exposure and outcome measurement error, and statistical model specifications. Statistical significance . . . is just one of the means of evaluating confidence in the observed relationship and assessing the probability of chance as an explanation. Other indicators of reliability such as the consistency and coherence of a body of studies as well as other confirming data may be used to justify reliance on the results of a body of epidemiologic studies, even if results in individual studies lack statistical significance . . . [Therefore, the U.S. EPA] . . . does not limit its focus or consideration to statistically significant results in epidemiologic studies.''

Therefore, upon re-evaluating the causality determination, when considering the CASAC comments on the Draft PM ISA and applying the causal framework as described (U.S. EPA, 2015; U.S. EPA, 2019, section A.3.2.1), the EPA continues to conclude in the 2019 Final PM ISA that the evidence for long-term $PM_{2.5}$ exposure and nervous system effects supports a ''likely to be causal relationship'' (U.S. EPA, 2019, p. 8–61).

2. Populations at Risk of $PM_{2.5}$-Related Health Effects

The NAAQS are meant to protect the population as a whole, including groups that may be at increased risk for pollutant-related health effects. In the last review, based on the evidence assessed in the 2009 ISA (U.S. EPA, 2009c), the 2011 PA focused on children, older adults, people with pre-existing heart and lung diseases, and those of lower socioeconomic status as populations that are ''likely to be at increased risk of PM-related effects'' (U.S. EPA, 2011, p. 2–31). In the current review, the 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). For example, in support of its ''causal'' and ''likely to be causal'' determinations, the ISA cites substantial evidence for:

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

The ISA additionally notes that stratified analyses (*i.e.,* analyses that directly compare PM-related health effects across groups) provide support for racial and ethnic differences in $PM_{2.5}$ exposures and in $PM_{2.5}$-related health risk (U.S. EPA, 2019, section 12.5.4). Drawing from such studies, the ISA concludes that ''[t]here is strong evidence demonstrating that black and Hispanic populations, in particular, have higher $PM_{2.5}$ exposures than non-Hispanic white populations'' and that ''there is consistent evidence across multiple studies demonstrating an increase in risk for nonwhite populations'' (U.S. EPA, 2019, p. 12–38). Stratified analyses focusing on other groups also suggest that populations with pre-existing cardiovascular or respiratory disease, populations that are overweight or obese, populations that have particular genetic variants, populations that are of low socioeconomic status, and current/former smokers could be at increased risk for $PM_{2.5}$-related adverse health effects (U.S. EPA, 2019, Chapter 12).

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

3. CASAC Advice

In its review of the draft ISA, the CASAC provided advice on the assessment of the scientific evidence for PM-related health and welfare effects and on the process under which this review of the PM NAAQS is being conducted (Cox, 2019b). With regard to the assessment of the evidence, the CASAC recommended that a revised ISA should ''provide a clearer and more complete description of the process and criteria for study quality assessment'' and that it should include a ''[c]learer discussion of causality and causal biological mechanisms and pathways'' (Cox, 2019b, p. 1 of letter). The CASAC further advised that the draft ISA ''does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term $PM_{2.5}$ exposure and nervous system effects; between long-term ultrafine particulate (UFP) exposure and nervous system effects; or between long-term $PM_{2.5}$ exposure and cancer'' (Cox, 2019b, p. 1 of letter).

As discussed above in section I.C.5, and as detailed in the final ISA, to address these comments the EPA: (1) Added text to the Preface and developed a new Appendix to more clearly articulate the process of ISA development; (2) added text to the Preface and to the health effects chapters to clarify the discussion of biological plausibility and its role in forming causality determinations; and (3) revised the determination for long-term UFP exposure and nervous system effects to *suggestive of, but not sufficient to infer, a causal relationship.* The EPA's rationales for not revising the other causality determinations questioned by the CASAC are discussed above in sections II.B.1.d (*i.e.,* for cancer) and II.B.1.e (*i.e.,* for nervous system effects).

With regard to the process for reviewing the PM NAAQS, the CASAC requested the opportunity to review a 2nd draft ISA (Cox, 2019b, p. 1 of letter) and recommended that ''the EPA reappoint the previous CASAC PM panel (or appoint a panel with similar expertise)'' (Cox, 2019b, p. 2 of letter). As discussed above in section I.C.5, the Agency's responses to these recommendations were described in a letter from the Administrator to the CASAC chair.[40]

In addition to the consensus advice noted above, the CASAC did not reach consensus on some issues related to the assessment of the $PM_{2.5}$ health effects evidence. In particular, the CASAC members ''had varying opinions on whether there is robust and convincing evidence to support the EPA's conclusion that there is a causal relationship between $PM_{2.5}$ exposure and mortality'' (Cox, 2019b, p. 3 of letter). ''Some members of the CASAC'' concluded that ''the EPA must better justify their determination that short-term or long-term exposure to $PM_{2.5}$ causes mortality'' (Cox, 2019b, p. 1 of consensus responses). These members recommended that the ISA should specifically address the biological action of PM and how exposures to low concentrations of $PM_{2.5}$ could cause mortality; the geographic heterogeneity in effect estimates between $PM_{2.5}$ exposure and mortality; concentration concordance across epidemiologic, controlled human exposure and animal toxicological studies (*i.e.,* how the continuum of effects is impacted by the concentrations at which different effects have been observed); uncertainties in the shapes of concentration-response functions and in the potential for thresholds to exist; how results compare between and within studies; and whether $PM_{2.5}$ exposures result in mortality in animal studies (Cox, 2019b, pp. 1–2).

In contrast, ''[o]ther members of the CASAC are of the opinion that, although uncertainties remain, the evidence supporting the causal relationship between $PM_{2.5}$ exposure and mortality is robust, diverse, and convincing'' (Cox, 2019b, p. 3 of consensus responses). These members noted that epidemiologic observations ''have been reproduced around the world in communities with widely varying exposures'' and that ''the findings of many of the largest studies have been repeatedly reanalyzed, with

[40] Available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/0/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002_Response.pdf.*

confirmation of the original findings'' (Cox, 2019b, p. 3). These committee members additionally stated that the ISA's causality determinations consider ''a wide range of evidence from a variety of sources, including human clinical exposure and animal toxicology studies that have provided rational biological plausibility and potential mechanisms'' (Cox, 2019b, p. 3). They highlighted the fact that there is new evidence in the current review from epidemiologic studies supporting associations between $PM_{2.5}$ and mortality and new evidence from toxicology studies informing the biological plausibility of mechanisms that could lead to mortality (Cox, 2019b, p. 3).

C. Proposed Conclusions on the Current Primary $PM_{2.5}$ Standards

This section describes the Administrator's proposed conclusions regarding the adequacy of the current primary $PM_{2.5}$ standards. His approach to reaching these proposed conclusions draws from the ISA's assessment of the scientific evidence for health effects attributable to $PM_{2.5}$ exposures (U.S. EPA, 2019) and the analyses in the PA (U.S. EPA, 2020), including uncertainties in the evidence and analyses. Section II.C.1 discusses the evidence- and risk-based considerations in the PA. Section II.C.2 summarizes CASAC advice on the current primary $PM_{2.5}$ standards, based on its review of the draft PA (Cox, 2019a). Section II.C.3 presents the Administrator's proposed decision to retain the current primary $PM_{2.5}$ standards.

1. Evidence- and Risk-Based Considerations in the Policy Assessment

The Administrator's proposed decision in this review draws from his consideration of the $PM_{2.5}$ health evidence assessed in the ISA (U.S. EPA, 2019) and the evidence- and risk-based analyses presented in the PA (U.S. EPA, 2020), including the uncertainties inherent in the evidence and analyses. The sections below summarize the consideration of the evidence-based information (II.C.1.a) and risk-based information (II.C.1.b) in the PA.

a. Evidence-Based Considerations

The PA considers the degree to which the available scientific evidence provides support for the current and potential alternative standards in terms of the basic elements of those standards (*i.e.,* indicator, averaging time, form, and level). With regard to the current indicator, averaging times, and forms, the PA concludes that the available evidence continues to support these elements in the current review. For indicator, the PA specifically concludes that available studies provide strong support for health effects following long- and short-term $PM_{2.5}$ exposures and that the evidence is too limited to support potential alternatives (U.S. EPA, 2020, section 3.5.2.1). For averaging time, the PA notes that epidemiologic studies continue to provide strong support for health effects based on annual (or multiyear) and 24-hour $PM_{2.5}$ averaging periods and concludes that the evidence does not support considering alternatives (U.S. EPA, 2020, section 3.5.2.2). For form, the PA notes that the foremost consideration is the adequacy of the public health protection provided by the combination of the form and the other elements of the standard. It concludes that (1) the form of the current annual standard (*i.e.,* arithmetic mean, averaged over three years) remains appropriate for targeting protection against the annual and daily $PM_{2.5}$ exposures around the middle portion of the $PM_{2.5}$ air quality distribution, and (2) the form of the current 24-hour standard (98th percentile, averaged over three years) continues to provide 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).

With regard to level, the considerations in the PA reflect analyses of the $PM_{2.5}$ exposures and ambient concentrations in studies reporting $PM_{2.5}$-related health effects (U.S. EPA, 2020). As noted above, the focus is on health outcomes for which the ISA concludes the evidence supports a ''causal'' or a ''likely to be causal'' relationship with PM exposures.[41] While the causality determinations in the ISA are informed by studies evaluating a wide range of $PM_{2.5}$ concentrations, the PA considers the degree to which the evidence supports the occurrence of PM-related effects at concentrations relevant to informing conclusions on the primary $PM_{2.5}$ standards. Section II.C.1.a.i below summarizes the PA's consideration of exposure concentrations that have been evaluated in experimental studies and section II.C.1.a.ii summarizes the PA's consideration of ambient concentrations in locations evaluated by epidemiologic studies.

[41] As discussed above in II.A.2, such a focus recognizes that standards set to provide protection based on evidence for ''causal'' and ''likely to be causal'' health outcomes will also provide some measure of protection against the broader range of $PM_{2.5}$-associated outcomes, including those for which the evidence is less certain.

i. $PM_{2.5}$ Exposure Concentrations Evaluated in Experimental Studies

Evidence for a particular $PM_{2.5}$-related health outcome is strengthened when results from experimental studies demonstrate biologically plausible mechanisms through which adverse human health outcomes could occur (U.S. EPA, 2015, Preamble p. 20). Two types of experimental studies are of particular importance in understanding the effects of PM exposures: Controlled human exposure and animal toxicology studies. In such studies, investigators expose human volunteers or laboratory animals, respectively, to known concentrations of air pollutants under carefully regulated environmental conditions and activity levels. Thus, controlled human exposure and animal toxicology studies can provide information on the health effects of experimentally administered pollutant exposures under well-controlled laboratory conditions (U.S. EPA, 2015, Preamble, p. 11). The sections below summarize the PA's evaluation of the $PM_{2.5}$ exposure concentrations that have been examined in controlled human exposure studies and animal toxicology studies.

Controlled Human Exposure Studies

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 (U.S. EPA, 2019, section 6.1). The most consistent evidence from these studies is for impaired vascular function (U.S. EPA, 2019, section 6.1.13.2). In addition, although less consistent, the ISA notes that studies examining $PM_{2.5}$ 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).

Table 3–2 in the PA (U.S. EPA, 2020) summarizes information from the ISA on available controlled human exposure studies that evaluate effects on markers of cardiovascular function following exposures to $PM_{2.5}$. Most of the controlled human exposure studies in Table 3–2 of the PA have evaluated average $PM_{2.5}$ exposure concentrations at or above about 100 $\mu g/m^3$, 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/m³, with less consistent evidence for effects following exposures to lower concentrations. Impaired vascular function, the effect identified in the ISA as the most consistent across studies (U.S. EPA, 2019, section 6.1.13.2), is shown following 2-hour exposures to $PM_{2.5}$ concentrations at and above 149 μg/m³. Mixed results are reported in the few studies that evaluate longer exposure durations (*i.e.,* longer than 2 hours) and lower $PM_{2.5}$ concentrations (U.S. EPA, 2020, section 3.2.3.1).

To provide some insight into what these studies may indicate regarding the primary $PM_{2.5}$ standards, analyses in the PA examine monitored 2-hour $PM_{2.5}$ concentrations at sites meeting the current standards (U.S. EPA, 2020, section 3.2.3.1). At these sites, most 2-hour concentrations are below 11 μg/m³, and they almost never exceed 32 μg/m³. Even the highest 2-hour concentrations remain well-below the exposure concentrations consistently shown to cause effects in controlled human exposure studies (*i.e.,* 99.9th percentile of 2-hour concentrations is 68 μg/m³ during the warm season). Thus, while controlled human exposure studies support the plausibility of the serious cardiovascular effects that have been linked with ambient $PM_{2.5}$ exposures (U.S. EPA, 2019, Chapter 6), the PA notes that the $PM_{2.5}$ exposures evaluated in most of these studies are well-above the ambient concentrations typically measured in locations meeting the current primary standards (U.S. EPA, 2020, section 3.2.3.2.1).

Animal Toxicology Studies

The 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.

As with controlled human exposure studies, most of the animal toxicology studies assessed in the ISA have examined effects following exposures to $PM_{2.5}$ concentrations well-above the concentrations likely to be allowed by the current $PM_{2.5}$ standards. Such studies have generally examined short-term exposures to $PM_{2.5}$ concentrations from 100 to >1,000 μg/m³ and long-term exposures to concentrations from 66 to >400 μg/m³ (*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/m³ (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/m³ (Cangerana Pereira et al., 2011). These two studies report serious effects following long-term exposures to $PM_{2.5}$ concentrations close 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. 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), but have not evaluated $PM_{2.5}$ exposures likely to occur in areas meeting the current primary standards.

ii. Ambient Concentrations in Locations of Epidemiologic Studies

As summarized above in section II.B.1, 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 ISA's ''causal'' and ''likely to be causal'' determinations for cardiovascular effects, respiratory effects, cancer, and mortality. The PA uses two approaches to consider what information from epidemiologic studies may indicate regarding primary $PM_{2.5}$ standards (U.S. EPA, 2020, section 3.2.3.2). In one approach, the PA evaluates the $PM_{2.5}$ air quality distributions reported by key epidemiologic studies, with a focus on overall mean $PM_{2.5}$ concentrations (*i.e.,* averages over the study period of the daily or annual $PM_{2.5}$ concentrations used to estimate exposures) and the concentrations somewhat below these overall means (*i.e.,* corresponding to the lower quartiles of exposure or health data) (U.S. EPA, 2020, section 3.2.3.2.1). In another approach, the PA calculates study area air quality metrics similar to $PM_{2.5}$ design values (*i.e.,* referred to as pseudo-design values) and considers the degree to which such metrics indicate that study area air quality would likely have met or violated the current standards during study periods (U.S. EPA, 2020, section 3.2.3.2.2). These approaches are discussed briefly below.

$PM_{2.5}$ Air Quality Distributions Associated With Mortality or Morbidity

The PA evaluates the $PM_{2.5}$ air quality distributions over which epidemiologic studies support health effect associations and the degree to which such distributions are likely to occur in areas meeting the current standards. As discussed further in the PA (U.S. EPA, 2020, section 3.2.3.2.1), epidemiologic studies generally provide the strongest support for reported health effect associations over the part of the air quality distribution corresponding to the bulk of the underlying data (*i.e.,* estimated exposures and/or health events), often falling in the middle part of the distribution (*i.e.,* rather than at the extreme upper or lower ends). Thus, in considering $PM_{2.5}$ air quality data from epidemiologic studies, the PA evaluates study-reported means (or medians) of daily and annual average $PM_{2.5}$ concentrations as proxies for the middle portions of the air quality distributions that support reported associations. When data are available, the PA also considers the broader $PM_{2.5}$ air quality distributions around the overall mean concentrations, with a focus on the lower quartiles of data to provide insight into the concentrations below which data supporting reported associations become relatively sparse.

Based on its evaluation of study-reported $PM_{2.5}$ concentrations, the PA notes that key epidemiologic studies conducted in the U.S. or Canada report generally positive and statistically significant associations between estimated $PM_{2.5}$ exposures (short- or long-term) and mortality or morbidity across a wide range of ambient $PM_{2.5}$ concentrations (U.S. EPA, 2020, section 3.2.3.2.1). With regard to these study-reported concentrations, the PA makes a number of observations, including the following:

- For the large majority of key studies, the $PM_{2.5}$ air quality distributions that support reported associations are characterized by overall mean (or median) $PM_{2.5}$ concentrations ranging from just above 8.0 μg/m³ to just above 16.0 μg/m³. Most of these key studies, including all but one U.S. study, report overall mean (or median) concentrations at or above 9.6 μg/m³.
- Several U.S. studies report positive and statistically significant health effect associations in analyses restricted to annual average $PM_{2.5}$ concentrations <12 μg/m³ (Lee et al. (2015); Shi et al. (2016); Di et al., 2017b). Studies also report positive and statistically significant health effect associations in analyses restricted to days with 24-hour average $PM_{2.5}$ concentrations <35 μg/m³

(Lee et al. (2015); Shi et al. (2016); Di et al. (2017a)).

- For some key studies, information on the broader distributions of $PM_{2.5}$ exposure estimates and/or health events is available. In these studies, ambient $PM_{2.5}$ concentrations corresponding to 25th percentiles of the underlying data (*i.e.,* estimated exposures or health events) are generally >6.0 μg/m³.
- A small group of studies report increased life expectancy, decreased mortality, and decreased respiratory effects following past declines in ambient $PM_{2.5}$ concentrations. These studies have examined ''starting'' annual average $PM_{2.5}$ concentrations (*i.e.,* prior to the reductions being evaluated) ranging from about 13 to >20 μg/m³ (*i.e.,* U.S. EPA, 2020, Table 3–3).

The PA concludes that the overall mean $PM_{2.5}$ concentrations reported by several of these key epidemiologic studies are likely below the long-term mean concentrations (*i.e.,* averaged across space and over time) in areas just meeting the current annual $PM_{2.5}$ standard (U.S. EPA, 2020, section 3.2.3.3). The PA also concludes that there are uncertainties in using study-reported concentrations to inform conclusions on the primary $PM_{2.5}$ standards (U.S. EPA, 2020, section 3.2.3.2.1). For example, the overall mean $PM_{2.5}$ concentrations reported by key epidemiologic studies are not the same as the ambient concentrations used by the EPA to determine whether areas meet or violate the PM NAAQS. Overall mean $PM_{2.5}$ concentrations in key studies reflect averaging of short- or long-term $PM_{2.5}$ exposure estimates across locations (*i.e.,* across multiple monitors or across modeled grid cells) and over time (*i.e.,* over several years). In contrast, to determine whether areas meet or violate the NAAQS, the EPA measures air pollution concentrations at individual monitors (*i.e.,* concentrations are not averaged across monitors) and calculates ''design values'' at monitors meeting appropriate data quality and completeness criteria. For the annual $PM_{2.5}$ standard, design values are calculated as the annual arithmetic mean $PM_{2.5}$ concentration, averaged over 3 years (described in appendix N of 40 CFR part 50). For an area to meet the NAAQS, all valid design values in that area, including the highest monitored values, must be at or below the level of the standard. Additional uncertainties associated with using the $PM_{2.5}$ concentrations reported by key epidemiologic studies to inform conclusions on the primary $PM_{2.5}$ standards result from the fact that (1) epidemiologic studies do not identify specific $PM_{2.5}$ exposures that result in health effects or exposures below which effects do not occur and (2) exposure estimates in some recent studies are based on hybrid modeling approaches for which performance depends on the availability of monitoring data and varies by location. These results and uncertainties are discussed in detail in the PA (U.S. EPA, 2020, section 3.2.3.2.1).

$PM_{2.5}$ Pseudo-Design Values in Epidemiologic Study Locations

As noted above, a key uncertainty in using study-reported $PM_{2.5}$ concentrations to inform conclusions on the primary $PM_{2.5}$ standards is that they reflect the averages of daily or annual $PM_{2.5}$ air quality concentrations or exposure estimates in the study population over the years examined by the study, and are not the same as the $PM_{2.5}$ design values used by the EPA to determine whether areas meet the NAAQS. Therefore, the PA also considers a second approach to evaluating information from epidemiologic studies. In this approach, the PA calculates study area air quality metrics similar to $PM_{2.5}$ design values (*i.e.,* referred to in the PA as pseudo-design values; U.S. EPA, 2020, section 3.2.3.2.2) and considers the degree to which such metrics indicate that study area air quality would likely have met or violated the current standards during study periods. When pseudo-design values in individual study locations are linked with the populations living in those locations, or with the number of study-specific health events recorded in those locations, these values can provide insight into the degree to which reported health effect associations are based on air quality likely to have met or violated the current (or alternative) primary $PM_{2.5}$ standards. The results of these analyses are summarized below in Table 1 (from U.S. EPA, 2020, Appendix B, Tables B–5 and B–6).

TABLE 1—SUMMARY OF RESULTS FROM ANALYSIS OF $PM_{2.5}$ PSEUDO-DESIGN VALUES IN LOCATIONS OF KEY U.S. AND CANADIAN MULTICITY STUDIES

[From U.S. EPA, 2020, Table B–5]

Percent of population/ health events in locations meeting current standards	Number of studies (of the 29 evaluated)
> 25%	17
> 50%	9
> 75%	4
< 25%	12

Given the results of these analyses, the PA concludes that several key epidemiologic studies report positive and statistically significant $PM_{2.5}$ health effect associations based largely, or entirely, on air quality likely to be allowed by the current primary $PM_{2.5}$ standards (U.S. EPA, 2020, section 3.2.3.3). The PA also concludes that there are important uncertainties to consider when using this information to inform conclusions on the primary $PM_{2.5}$ standards. For example, for most key multicity studies, some study locations would likely have met the current primary standards over study periods while others would likely have violated one or both standards, complicating the interpretation of these analyses. In addition, pseudo-design values are averaged over multiyear study periods of varying lengths, rather than reflecting the three-year averages of actual design values; analyses necessarily focus on locations with at least one $PM_{2.5}$ monitor, while unmonitored areas are not included; and recent changes to $PM_{2.5}$ monitoring requirements are not reflected in analyses of pseudo-design values. These results and uncertainties are discussed in greater detail in the PA (U.S. EPA, 2020, section 3.2.3.2.2).

b. Risk-Based Considerations

In addition to evaluating $PM_{2.5}$ concentrations in locations of key epidemiologic studies, the PA includes a risk assessment that estimates population-level health risks associated with $PM_{2.5}$ air quality that has been adjusted to simulate air quality scenarios of policy interest (*e.g.,* ''just meeting'' the current standards). The general approach to estimating $PM_{2.5}$-associated health risks combines concentration-response functions from epidemiologic studies with model-based $PM_{2.5}$ air quality surfaces, baseline health incidence data, and population demographics for forty-seven urban study areas (U.S. EPA, 2020, section 3.3, Figure 3–10 and Appendix C).

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 from about 16,000 to 17,000 long-term $PM_{2.5}$ exposure-related deaths from ischemic heart disease in a single year (*i.e.,* confidence intervals range from about 12,000 to

21,000 deaths).[42] 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 by about 7 to 9% for a level of 11.0 μg/m^3, 14 to 18% for a level of 10.0 μg/m^3, and 21 to 27% for a level of 9.0 μg/m^3.

Limitations in the underlying data and risk assessment approaches lead to uncertainty in these estimates of $PM_{2.5}$-associated risks (*e.g.,* in the size of risk estimates). Uncertainty in risk estimates results 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 in the underlying epidemiologic studies, and the methods used to adjust $PM_{2.5}$ air quality. The PA characterizes these and other sources of uncertainty in risk estimates using a combination of quantitative and qualitative approaches (U.S. EPA, 2020, Appendix C, section C.3).

2. CASAC Advice

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the public health protection afforded by the current primary $PM_{2.5}$ standards.[43] Its advice is documented in a letter sent to the EPA Administrator (Cox, 2019a). In this letter, the committee recommends retaining the current 24-hour $PM_{2.5}$ standard but does not reach consensus on whether the scientific and technical information support retaining or revising the current annual standard. In particular, though the CASAC agrees that there is a long-standing body of health evidence supporting relationships between $PM_{2.5}$ exposures and various health outcomes, including mortality and serious morbidity effects, individual CASAC members ''differ in their assessments of the causal and policy significance of these associations'' (Cox, 2019a, p. 8 of consensus responses). Drawing from this evidence, ''some CASAC members'' express support for retaining the current annual standard while ''other members'' express support for revising that standard in order to increase public health protection (Cox, 2019a, p.1 of letter). These views are summarized below.

The CASAC members who support retaining the current annual standard express the view that substantial uncertainty remains in the evidence for associations between $PM_{2.5}$ exposures and mortality or serious morbidity effects. These committee members assert that ''such associations can reasonably be explained in light of uncontrolled confounding and other potential sources of error and bias'' (Cox, 2019a, p. 8 of consensus responses). They note that associations do not necessarily reflect causal effects, and they contend that recent epidemiologic studies reporting positive associations at lower estimated exposure concentrations mainly confirm what was anticipated or already assumed in setting the 2012 NAAQS. In particular, they conclude that such studies have some of the same limitations as prior studies and do not provide new information calling into question the existing standard. They further assert that ''accountability studies provide potentially crucial information about whether and how much decreasing $PM_{2.5}$ causes decreases in future health effects'' (Cox, 2019a, p. 10), and they cite recent reviews (*i.e.,* Henneman et al., 2017; Burns et al., 2019) to support their position that in such studies, ''reductions of $PM_{2.5}$ concentrations have not clearly reduced mortality risks'' (Cox, 2019a, p. 8 of consensus responses). Thus, the committee members who support retaining the current annual standard advise that, ''while the data on associations should certainly be carefully considered, this data should not be interpreted more strongly than warranted based on its methodological limitations'' (Cox, 2019a, p. 8 of consensus responses).

These members of the CASAC further conclude that the $PM_{2.5}$ risk assessment does not provide a valid basis for revising the current standards. This conclusion is based on concerns that (1) ''the risk assessment treats regression coefficients as causal coefficients with no justification or validation provided for this decision;'' (2) the estimated regression concentration-response functions ''have not been adequately adjusted to correct for confounding, errors in exposure estimates and other covariates, model uncertainty, and heterogeneity in individual biological (causal) [concentration-response] functions;'' (3) the estimated concentration-response functions ''do not contain quantitative uncertainty bands that reflect model uncertainty or effects of exposure and covariate estimation errors;'' and (4) ''no regression diagnostics are provided justifying the use of proportional hazards . . . and other modeling assumptions'' (Cox, 2019a, p. 9 of consensus responses). These committee members also contend that details regarding the derivation of concentration-response functions, including specification of the beta values and functional forms, are not well-documented, hampering the ability of readers to evaluate these design details. Thus, these members ''think that the risk characterization does not provide useful information about whether the current standard is protective'' (Cox, 2019a, p. 11 of consensus responses).

Drawing from their evaluation of the evidence and the risk assessment, these committee members conclude that ''the Draft PM PA does not establish that new scientific evidence and data reasonably call into question the public health protection afforded by the current 2012 $PM_{2.5}$ annual standard'' (Cox, 2019a, p.1 of letter).

In contrast, ''[o]ther members of CASAC conclude that the weight of the evidence, particularly reflecting recent epidemiology studies showing positive associations between $PM_{2.5}$ and health effects at estimated annual average $PM_{2.5}$ concentrations below the current standard, does reasonably call into question the adequacy of the 2012 annual $PM_{2.5}$ [standard] to protect public health with an adequate margin of safety'' (Cox, 2019a, p.1 of letter). The committee members who support this conclusion note that the body of health evidence for $PM_{2.5}$ includes not only the repeated demonstration of associations in epidemiologic studies, but also includes support for biological plausibility established by controlled human exposure and animal toxicology studies. They point to recent studies demonstrating that the associations between $PM_{2.5}$ and health effects occur in a diversity of locations, in different time periods, with different populations, and using different exposure estimation and statistical methods. They conclude that ''the entire body of evidence for PM health effects justifies the causality determinations made in the Draft PM ISA'' (Cox, 2019a, p. 8 of consensus responses).

The members of the CASAC who support revising the current annual standard particularly emphasize recent findings of associations with $PM_{2.5}$ in areas with average long-term $PM_{2.5}$ concentrations below the level of the

[42] For the only other cause-specific mortality endpoint evaluated (*i.e.,* lung cancer), substantially fewer deaths were estimated (U.S. EPA, 2020, section 3.3.2, *e.g.,* Figure 3–5). Risk estimates were not generated for other ''likely to be causal'' outcome categories (*i.e.,* respiratory effects, nervous system effects).

[43] The CASAC also provided advice on the draft ISA's assessment of the scientific evidence (Cox, 2019b) and on the analyses and information in the draft PA (Cox, 2019a), which drew from the draft ISA. That advice, and the resulting changes made in the final ISA and final PA, are summarized above in sections I.C.5, II.B.1.d, II.B.1.e and II.B.3, and in the final ISA (U.S. EPA, 2019, ES–3 to ES–4) and the final PA (U.S. EPA, 2020, section 1.4).

annual standard and studies that show positive associations even when estimated exposures above 12 μg/m³ are excluded from analyses. They find it ''highly unlikely'' that the extensive body of evidence indicating positive associations at low estimated exposures could be fully explained by confounding or by other non-causal explanations (Cox, 2019a, p. 8 of consensus responses). They additionally conclude that ''the risk characterization does provide a useful attempt to understand the potential impacts of alternate standards on public health risks'' (Cox, 2019a, p. 11 of consensus responses). These committee members conclude that the evidence available in this review reasonably calls into question the protection provided by the current primary $PM_{2.5}$ standards and supports revising the annual standard to increase that protection (Cox, 2019a).

3. Administrator's Proposed Decision on the Current Primary $PM_{2.5}$ Standards

This section summarizes the Administrator's considerations and conclusions related to the current primary $PM_{2.5}$ standards and presents his proposed decision to retain those standards, without revision. As described above (section II.A.2), his approach to considering the adequacy of the current standards focuses on evaluating the public health protection afforded by the annual and 24-hour standards, taken together, against mortality and morbidity associated with long- or short-term $PM_{2.5}$ exposures. This approach recognizes that changes in $PM_{2.5}$ air quality designed to meet either the annual or the 24-hour standard would likely result in changes to both long-term average and short-term peak $PM_{2.5}$ concentrations and that the protection provided by the suite of standards results from the combination of all of the elements of those standards (*i.e.,* indicator, averaging time, form, level). Thus, the Administrator's consideration of the public health protection provided by the current primary $PM_{2.5}$ standards is based on his consideration of the combination of the annual and 24-hour standards, including the indicators ($PM_{2.5}$), averaging times, forms (arithmetic mean and 98th percentile, averaged over three years), and levels (12.0 μg/m³, 35 μg/m³) of those standards.

In establishing primary standards under the Act that are ''requisite'' to protect public health with an adequate margin of safety, the Administrator is seeking to establish standards that are neither more nor less stringent than necessary for this purpose. He recognizes that the requirement to provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information and to provide a reasonable degree of protection against hazards that research has not yet identified. However, the Act does not require that primary standards be set at a zero-risk level; rather, the NAAQS must be sufficiently protective, but not more stringent than necessary.

Given these requirements, the Administrator's final decision in this review will be a public health policy judgment drawing upon scientific and technical information examining the health effects of $PM_{2.5}$ exposures, including how to consider the range and magnitude of uncertainties inherent in that information. This public health policy judgment will be based on an interpretation of the scientific and technical information that neither overstates nor understates its strengths and limitations, nor the appropriate inferences to be drawn, and will be informed by the Administrator's consideration of advice from the CASAC and public comments received on this proposal document.

With regard to the CASAC, the Administrator recognizes that while the committee supports retaining the current 24-hour $PM_{2.5}$ standard, it does not reach consensus on the annual standard (Cox, 2019a, pp. 1–3 of letter). In particular, some members of the CASAC conclude that the new scientific evidence and data do not reasonably call into question the public health protection afforded by the current annual standard, while other members conclude that the weight of the evidence does reasonably call into question the adequacy of that standard (Cox, 2019a, p. 1 of letter).

As discussed above (II.C.2), the CASAC members who support retaining the annual standard emphasize their concerns with available $PM_{2.5}$ epidemiologic studies. They assert that recent studies ''mainly confirmed what had already been anticipated or assumed in setting the 2012 NAAQS'' (Cox, 2019a, p. 8 consensus responses) and do not provide a basis for revising the current standards. They also identify several key concerns regarding the associations reported in $PM_{2.5}$ epidemiologic studies and conclude 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, 2019a, p. 8 consensus responses).

One of the methodological limitations highlighted by these committee members is that associations reported in epidemiologic studies are not necessarily indicative of causal relationships and such associations ''can reasonably be explained in light of uncontrolled confounding and other potential sources of error and bias'' (Cox, 2019a, p. 8). Thus, these committee members do not think that recent epidemiologic studies reporting health effect associations at $PM_{2.5}$ air quality concentrations likely to have met the current primary standards support revising those standards.

Consistent with the views expressed by these CASAC members, the Administrator recognizes that epidemiologic studies examine associations between distributions of $PM_{2.5}$ air quality and health outcomes, and they do not identify particular $PM_{2.5}$ exposures that cause effects (U.S. EPA, 2020, section 3.1.2). In contrast, he notes that experimental studies (*i.e.,* controlled human exposure, animal toxicology) do provide evidence for health effects following particular $PM_{2.5}$ exposures under carefully controlled laboratory conditions (*e.g.,* U.S. EPA, 2015, Preamble Chapters 5 and 6). He further notes that the evidence for a given $PM_{2.5}$-related health outcome is strengthened when results from experimental studies demonstrate biologically plausible mechanisms through which such an outcome could occur (*e.g.,* U.S. EPA, 2015, Preamble p. 20). Thus, when using the $PM_{2.5}$ health evidence to inform conclusions on the adequacy of the current primary standards, the Administrator is most confident in the potential for $PM_{2.5}$ exposures to cause adverse effects at concentrations supported by multiple types of studies, including experimental studies as well as epidemiologic studies.

In light of this approach to considering the evidence, the Administrator recognizes that controlled human exposure and animal toxicology studies report a wide range of effects, many of which are plausibly linked to the serious cardiovascular and respiratory outcomes reported in epidemiologic studies (including mortality), though the $PM_{2.5}$ exposures examined in these studies are above the concentrations typically measured in areas meeting the current annual and 24-hour standards (U.S. EPA, 2020, section 3.2.3.1). In the absence of evidence from experimental studies that $PM_{2.5}$ exposures typical of areas meeting the current annual and 24-hour standards can activate biological pathways that plausibly contribute to serious health outcomes, the Administrator is cautious about placing too much weight on reported $PM_{2.5}$ health effect associations for air quality

meeting those standards. He concludes that such associations alone, without supporting experimental evidence at similar $PM_{2.5}$ concentrations, leave important questions unanswered regarding the degree to which the typical $PM_{2.5}$ exposures likely to occur in areas meeting the current standards can cause the mortality or morbidity outcomes reported in epidemiologic studies. Given this concern, the Administrator does not think that recent epidemiologic studies reporting health effect associations at $PM_{2.5}$ air quality concentrations likely to have met the current primary standards support revising those standards. Rather, he judges that the overall body of evidence, including controlled human exposure and animal toxicological studies, in addition to epidemiologic studies, indicates continuing uncertainty in the degree to which adverse effects could result from $PM_{2.5}$ exposures in areas meeting the current annual and 24-hour standards.

The Administrator additionally considers the emerging body of evidence from studies examining past reductions in ambient $PM_{2.5}$, and the degree to which those reductions have resulted in public health improvements. As an initial matter, he notes the observation from some CASAC members (*i.e.,* those who support retaining the current annual standard) that in accountability studies, ''reductions of $PM_{2.5}$ concentrations have not clearly reduced mortality risks, especially when confounding was tightly controlled'' (Cox, 2019a, p. 8). The Administrator recognizes that interpreting such studies in the context of the current primary $PM_{2.5}$ standards is also complicated by the fact that some of the available studies have 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 have 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 recognizes that the small number of available studies that do report public health improvements following past declines in ambient $PM_{2.5}$ have not examined air quality meeting the current standards (U.S. EPA, 2020, Table 3–3). This includes recent U.S. studies that report increased life expectancy, decreased mortality, and decreased respiratory effects following past declines in ambient $PM_{2.5}$ concentrations. Such studies have examined ''starting'' annual average $PM_{2.5}$ concentrations (*i.e.,* prior to the reductions being evaluated) ranging from about 13 to > 20 $\mu g/m^3$ (*i.e.,* U.S. EPA, 2020, Table 3–3). It also includes a recent study conducted in Japan that reports reduced mortality following reductions in ambient $PM_{2.5}$ due to the introduction of diesel emission controls (Yorifuji et al., 2016). As in the U.S. studies, ambient $PM_{2.5}$ concentrations in this study were above those allowed by the current primary $PM_{2.5}$ standards. Given the lack of studies reporting public health improvements attributable to reductions in ambient $PM_{2.5}$ in locations meeting the current standards, together with his broader concerns regarding the lack of experimental studies examining $PM_{2.5}$ exposures typical of areas meeting the current standards (discussed above), the Administrator judges that there is considerable uncertainty in the potential for increased public health protection from further reductions in ambient $PM_{2.5}$ concentrations beyond those achieved under the current primary $PM_{2.5}$ standards.

In addition to the evidence, the Administrator considers the potential implications of the risk assessment for his proposed decision. In doing so, he notes that all risk assessments have limitations and that, in previous reviews, these 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 3128, January 15, 2013). Such limitations in risk estimates can result from 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).

In addition to these general uncertainties with risk assessments, the Administrator notes the concerns expressed by members of the CASAC who support retaining the current standards. Their concerns largely reflect their overall views on the limitations in the $PM_{2.5}$ epidemiologic evidence, which provides key inputs to the risk assessment. These committee members assert that ''the conclusions from the risk assessment do not comprise valid empirical evidence or grounds for revising the current NAAQS'' (Cox, 2019a, p. 9 consensus responses). As discussed above, the Administrator agrees with the broad concerns expressed by these members of the CASAC regarding associations at $PM_{2.5}$ concentrations meeting the current standards. He further notes their concerns regarding the characterization of uncertainty in the risk assessment and the evaluation of modeling assumptions (Cox, 2019a). In light of these concerns, together with the more general uncertainty in risk estimates summarized above, the Administrator judges it appropriate to place little weight on quantitative estimates of $PM_{2.5}$-associated mortality risk in reaching conclusions on the primary $PM_{2.5}$ standards.

When the above considerations are taken together, the Administrator proposes to conclude that the scientific evidence that has become available since the last review of the PM NAAQS, together with the analyses in the PA based on that evidence, does not call into question the public health protection provided by the current annual and 24-hour $PM_{2.5}$ standards. In particular, the Administrator judges that there is considerable uncertainty in the potential public health impacts of reductions in ambient $PM_{2.5}$ below the concentrations achieved under the current primary standards and, therefore, that standards more stringent than the current standards (*e.g.,* with lower levels) are not supported. That is, he judges that such standards would be more than requisite to protect the public health with an adequate margin of safety. As described above, this judgment reflects his consideration of the uncertainties in the potential implications of recent 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 current standards.

For the 24-hour standard, he notes that this judgment is consistent with the consensus advice of the CASAC (Cox, 2019). For the annual standard, this judgment is consistent with the advice of some CASAC members and reflects the Administrator's disagreement with the ''[o]ther members of CASAC'' who recommend revising the current annual standard based largely on evidence from recent epidemiology studies (Cox, 2019a, p. 1 of letter).

In addition, based on the Administrator's review of the science, including experimental and accountability studies conducted at levels just above the current standard, he judges that the degree of public health protection provided by the current standard is not greater than warranted. This judgment, together with the fact that no CASAC member expressed support for a less stringent standard, leads the Administrator to conclude that standards less stringent

than the current standards (*e.g.,* with higher levels) are also not supported.

When the above information is taken together, the Administrator proposes to conclude that the available scientific evidence and technical information continue to support the current annual and 24-hour $PM_{2.5}$ standards. This proposed conclusion reflects the fact that important limitations in the evidence remain. The Administrator proposes to conclude that these limitations lead to considerable uncertainty regarding the potential public health implications of revising the existing suite of $PM_{2.5}$ standards. Given this uncertainty, and the advice from some CASAC members, he proposes to conclude that the current suite of primary standards, including the current indicators ($PM_{2.5}$), averaging times (annual and 24-hour), forms (arithmetic mean and 98th percentile, averaged over three years) and levels (12.0 μg/m³, 35 μg/m³), when taken together, remain requisite to protect the public health. Therefore, the Administrator proposes to retain the current suite of primary $PM_{2.5}$ standards, without revision, in this review. He solicits comment on this proposed decision and on the supporting rationale described above.

III. Rationale for Proposed Decisions on the Primary PM_{10} Standard

The current primary PM_{10} standard is intended to protect the public health against exposures to $PM_{10\text{-}2.5}$ (78 FR 3164, January 15, 2013). This section provides the rationale supporting the Administrator's proposed decision to retain the current primary PM_{10} standard. Section III.A summarizes the Agency's approach to reaching a decision on the primary PM_{10} standard in the last review and presents the general approach to reaching a proposed decision in this review. Section III.B summarizes the scientific evidence for $PM_{10\text{-}2.5}$-related health effects. Section III.C presents the Administrator's proposed conclusions regarding the adequacy of the current primary PM_{10} standard.

A. General Approach

1. Approach Used in the Last Review

The last review of the PM NAAQS was completed in 2012 (78 FR 3086, January 15, 2013). In that review the EPA retained the existing primary 24-hour PM_{10} standard, with its level of 150 μg/m³ and its one-expected-exceedance form on average over three years, to continue to provide public health protection against exposures to $PM_{10\text{-}2.5}$. In support of this decision, the prior Administrator emphasized her consideration of three issues: (1) The extent to which it was appropriate to maintain a standard that provides some measure of protection against all $PM_{10\text{-}2.5}$ (regardless of composition or source or origin), (2) the extent to which a standard with a PM_{10} indicator can provide protection against exposures to $PM_{10\text{-}2.5}$, and (3) the degree of public health protection provided by the existing PM_{10} standard. Her consideration of each of these issues is summarized below.

First, the prior Administrator judged that the evidence provided ''ample support for a standard that protects against exposures to all thoracic coarse particles, regardless of their location or source of origin'' (78 FR 3176, January 15, 2013). In support of this, she noted that epidemiologic studies had reported positive associations between $PM_{10\text{-}2.5}$ and mortality or morbidity in a large number of cities across North America, Europe, and Asia, encompassing a variety of environments where $PM_{10\text{-}2.5}$ sources and composition are expected to vary widely. Though most of the available studies examined associations in urban areas, she noted that some studies had also linked mortality and morbidity with relatively high ambient concentrations of particles of non-urban crustal origin. In light of this body of available evidence, and consistent with the CASAC's advice, the prior Administrator concluded that it was appropriate to maintain a standard that provides some measure of protection against exposures to all thoracic coarse particles, regardless of their location, source of origin, or composition (78 FR 3176, January 15, 2013).

In reaching the conclusion that it was appropriate to retain a PM_{10} indicator for a standard meant to protect against exposures to ambient $PM_{10\text{-}2.5}$, the prior Administrator noted that PM_{10} mass includes both coarse PM ($PM_{10\text{-}2.5}$) and fine PM ($PM_{2.5}$). As a result, the concentration of $PM_{10\text{-}2.5}$ allowed by a PM_{10} standard set at a single level declines as the concentration of $PM_{2.5}$ increases. Because $PM_{2.5}$ concentrations tend to be higher in urban areas than rural areas, she observed that a PM_{10} standard would generally allow lower $PM_{10\text{-}2.5}$ concentrations in urban areas than in rural areas. She judged it appropriate to maintain such a standard given that much of the evidence for $PM_{10\text{-}2.5}$ toxicity, particularly at relatively low particle concentrations, came from study locations where thoracic coarse particles were of urban origin, and given the possibility that $PM_{10\text{-}2.5}$ contaminants in urban areas could increase particle toxicity. Thus, in the last review the prior Administrator concluded that it remained appropriate to maintain a standard that allows lower ambient concentrations of $PM_{10\text{-}2.5}$ in urban areas, where the evidence was strongest that exposure to thoracic coarse particles was associated with morbidity and mortality, and higher concentrations in non-urban areas, where the public health concerns were less certain. The prior Administrator concluded that the varying concentrations of coarse particles that would be permitted in urban versus non-urban areas under the 24-hour PM_{10} standard, based on the varying levels of $PM_{2.5}$ present, appropriately reflected the differences in the strength of evidence regarding coarse particle health effects.

Finally, in specifically evaluating the degree of public health protection provided by the primary PM_{10} standard, with its level of 150 μg/m³ and its one-expected-exceedance form on average over three years, the prior Administrator recognized that the available health evidence and air quality information was much more limited for $PM_{10\text{-}2.5}$ than for $PM_{2.5}$. In particular, the strongest evidence for health effects attributable to $PM_{10\text{-}2.5}$ exposure was for cardiovascular effects, respiratory effects, and/or premature mortality following short-term exposures. For each of these categories of effects, the 2009 ISA concluded that the evidence was ''suggestive of a causal relationship'' (U.S. EPA, 2009c, section 2.3.3). These determinations contrasted with those for $PM_{2.5}$, as described in Chapter 3 above, which were determined in the ISA to be either ''causal'' or ''likely to be causal'' for mortality, cardiovascular effects, and respiratory effects (U.S. EPA, 2009c, Tables 2–1 and 2–2).

The prior Administrator judged that the important uncertainties and limitations associated with the $PM_{10\text{-}2.5}$ evidence and information raised questions as to whether additional public health improvements would be achieved by revising the existing PM_{10} standard. She specifically noted several uncertainties and limitations, including the following:

- The number of epidemiologic studies that have employed copollutant models to address the potential for confounding, particularly by $PM_{2.5}$, was limited. Therefore, the extent to which $PM_{10\text{-}2.5}$ itself, rather than one or more copollutants, contributes to reported health effects remained uncertain.
- Only a limited number of experimental studies provided support for the associations reported in epidemiologic studies, resulting in

further uncertainty regarding the plausibility of the associations between $PM_{10-2.5}$ and mortality and morbidity reported in epidemiologic studies.

- Limitations in $PM_{10-2.5}$ monitoring data (*i.e.,* limited data available from FRM/FEM sampling methods) and the different approaches used to estimate $PM_{10-2.5}$ concentrations across epidemiologic studies resulted in uncertainty in the ambient $PM_{10-2.5}$ concentrations at which the reported effects occur, increasing uncertainty in estimates of the extent to which changes in ambient $PM_{10-2.5}$ concentrations would likely impact public health.
- While $PM_{10-2.5}$ effect estimates reported for mortality and morbidity were generally positive, most were not statistically significant, even in single-pollutant models. This included effect estimates reported in some study locations with PM_{10} concentrations above those allowed by the current 24-hour PM_{10} standard.
- The composition of $PM_{10-2.5}$, and the effects associated with various components, were also key uncertainties in the available evidence. Without more information on the chemical speciation of $PM_{10-2.5}$, the apparent variability in associations across locations was difficult to characterize.

In considering these uncertainties and limitations, the prior Administrator particularly emphasized the considerable degree of uncertainty in the extent to which health effects reported in epidemiologic studies are due to $PM_{10-2.5}$ itself, as opposed to one or more co-occurring pollutants. This uncertainty reflected the relatively small number of $PM_{10-2.5}$ studies that had evaluated copollutant models, particularly copollutant models that included $PM_{2.5}$, and the very limited body of controlled human exposure evidence supporting the plausibility of $PM_{10-2.5}$-attributable adverse effects at ambient concentrations.

When considering the evidence as a whole, the prior Administrator concluded that the degree of public health protection provided by the current PM_{10} standard against exposures to $PM_{10-2.5}$ should be maintained (*i.e.,* neither increased nor decreased). Her judgment that protection did not need to be increased was supported by her consideration of uncertainties in the overall body of evidence. Her judgment that the degree of public health protection provided by the current standard is not greater than warranted was supported by the observation that positive and statistically significant associations with mortality were reported in some single-city U.S. study locations likely to have violated the current PM_{10} standard. Thus, the prior Administrator concluded that the existing 24-hour PM_{10} standard, with its one-expected exceedance form on average over three years and a level of 150 $\mu g/m^3$, was requisite to protect public health with an adequate margin of safety against effects that have been associated with $PM_{10-2.5}$. In light of this conclusion, the EPA retained the existing PM_{10} standard.

2. Approach in the Current Review

The approach for this review builds on the last review, taking into account the more recent scientific information now available. The approach summarized below draws from the approach taken in the PA (U.S. EPA, 2020) and is most fundamentally based on using the ISA's assessment of the current scientific evidence for health effects of $PM_{10-2.5}$ exposures (U.S. EPA, 2019).

As discussed above for $PM_{2.5}$ (II.A.2), the approach in the PA places the greatest weight on effects for which the evidence has been determined to demonstrate a ''causal'' or a ''likely to be causal'' relationship with PM exposures (U.S. EPA, 2019). This approach focuses policy considerations and conclusions on health outcomes for which the evidence is strongest. Unlike for $PM_{2.5}$, the ISA does not identify any $PM_{10-2.5}$-related health outcomes for which the evidence supports either a ''causal'' or a ''likely to be causal'' relationship. Thus, for $PM_{10-2.5}$ the PA considers the evidence determined to be ''suggestive of, but not sufficient to infer, a causal relationship,'' recognizing the greater uncertainty in such evidence.

The preamble to the ISA states that ''suggestive'' evidence is ''limited, and chance, confounding, and other biases cannot be ruled out'' (U.S. EPA, 2015, Preamble Table II). In light of the additional uncertainty in the evidence for $PM_{10-2.5}$-related health outcomes, compared to the evidence supporting ''causal'' or ''likely to be causal'' relationships for $PM_{2.5}$, the approach to evaluating the primary PM_{10} standard in this review is more limited than the approach to evaluating the primary $PM_{2.5}$ standards (discussed in II.A.2). Specifically, the approach for PM_{10} does not include evaluations of air quality distributions in locations of individual epidemiologic studies, comparisons of experimental exposures with ambient air quality, or the quantitative assessment of $PM_{10-2.5}$ health risks. The substantial uncertainty in such analyses, if they were to be conducted based on the currently available $PM_{10-2.5}$ health studies, would limit their utility for informing conclusions on the primary PM_{10} standard. Therefore, as discussed further below, the focus of the evaluation of the primary PM_{10} standard is on the overall body of evidence for $PM_{10-2.5}$-related health effects. This includes consideration of the degree to which uncertainties in the evidence from the last review have been reduced and the degree to which new uncertainties have been identified.

B. Health Effects Related to Thoracic Coarse Particle Exposures

This section briefly outlines the key evidence for health effects associated with $PM_{10-2.5}$ exposures. This evidence is discussed more fully in the ISA (U.S. EPA, 2019) and the PA (U.S. EPA, 2020, Chapter 4).

While studies conducted since the last review have strengthened support for relationships between $PM_{10-2.5}$ exposures and some health outcomes (discussed below), several key uncertainties in the evidence from the last review have, to date, ''still not been addressed'' (U.S. EPA, 2019, section 1.4.2, p. 1–41). For example, epidemiologic studies available in the last review relied on various methods to estimate $PM_{10-2.5}$ exposures, and these methods had not been systematically compared to evaluate spatial and temporal correlations in exposure estimates. Methods included (1) calculating the difference between PM_{10} and $PM_{2.5}$ concentrations at co-located monitors, (2) calculating the difference between county-wide averages of monitored PM_{10} and $PM_{2.5}$ based on monitors that are not necessarily co-located, and (3) direct measurement of $PM_{10-2.5}$ using a dichotomous sampler (U.S. EPA, 2019, section 1.4.2). In the current review, more recent epidemiologic studies continue to use these approaches to estimate $PM_{10-2.5}$ concentrations. Additionally, some recent studies estimate long-term $PM_{10-2.5}$ exposures as the difference between PM_{10} and $PM_{2.5}$ concentrations based on information from spatiotemporal or land use regression (LUR) models, in addition to monitors. As in the last review, the various methods used to estimate $PM_{10-2.5}$ concentrations have not been systematically evaluated (U.S. EPA, 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 and section 2.5.2.2.3). Given the greater spatial and temporal variability of $PM_{10-2.5}$ and fewer $PM_{10-2.5}$ monitoring sites, compared to $PM_{2.5}$,

this uncertainty is particularly important for the coarse size fraction.

Beyond uncertainty associated with $PM_{10-2.5}$ exposure estimates in epidemiologic studies, the limited information on the potential for confounding by copollutants and the limited support available for the biological plausibility of serious effects following $PM_{10-2.5}$ exposures also continue to contribute broadly to uncertainty in the $PM_{10-2.5}$ health evidence. Uncertainty related to potential confounding stems from the relatively small number of epidemiologic studies that have evaluated $PM_{10-2.5}$ health effect associations in copollutants models with both gaseous pollutants and other PM size fractions. Uncertainty related to the biological plausibility of serious effects caused by $PM_{10-2.5}$ exposures results from the small number of controlled human exposure and animal toxicology [44] studies that have evaluated the health effects of experimental $PM_{10-2.5}$ inhalation exposures. The evidence supporting the ISA's ''suggestive'' causality determinations for $PM_{10-2.5}$, including uncertainties in this evidence, is summarized below in sections III.B.1 to III.B.7.

1. Mortality

a. Long-Term Exposures

Due to the dearth of studies examining the association between long-term $PM_{10-2.5}$ exposure and mortality, the 2009 PM ISA concluded that the evidence was ''inadequate to determine if a causal relationship exists'' (U.S. EPA, 2009c). Since the completion of the 2009 ISA, some recent cohort studies conducted in the U.S. and Europe report positive associations between long-term $PM_{10-2.5}$ exposure and total (nonaccidental) mortality, though results are inconsistent across studies (U.S. EPA, 2019, Table 11–11). The examination of copollutant models in these studies remains limited and, when included, $PM_{10-2.5}$ effect estimates are often attenuated after adjusting for $PM_{2.5}$ (U.S. EPA, 2019, Table 11–11). Across studies, $PM_{10-2.5}$ exposure concentrations are estimated using a variety of approaches, including direct measurements from dichotomous samplers, calculating the difference between PM_{10} and $PM_{2.5}$ concentrations measured at collocated monitors, and calculating difference of area-wide concentrations of PM_{10} and $PM_{2.5}$. As discussed above, temporal and spatial correlations between these approaches have not been evaluated, contributing to uncertainty regarding the potential for exposure measurement error (U.S. EPA, 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 ISA additionally concludes that the evidence for long-term $PM_{10-2.5}$ exposures and cardiovascular effects, respiratory morbidity, and metabolic disease provide limited biological plausibility for $PM_{10-2.5}$-related mortality (U.S. EPA, 2019, sections 11.4.1 and 11.4). Taken together, the 2019 ISA concludes that, ''this body of evidence is suggestive, but not sufficient to infer, that a causal relationship exists between long-term $PM_{10-2.5}$ exposure and total mortality'' (U.S. EPA, 2019, p. 11–125).

b. 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, 2009c). Since the completion of the 2009 ISA, multicity epidemiologic studies conducted primarily in Europe and Asia continue to provide consistent evidence of positive associations between short-term $PM_{10-2.5}$ exposure and total (nonaccidental) mortality (U.S. EPA, 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. In addition, the 2019 ISA notes that an analysis by Adar et al. (2014) indicates ''possible evidence of publication bias, which was not observed for $PM_{2.5}$'' (U.S. EPA, 2019, section 11.3.2, p. 11–106). Recent studies expand the assessment of potential copollutant confounding of the $PM_{10-2.5}$-mortality relationship and provide evidence that $PM_{10-2.5}$ associations generally remain positive in copollutant models, though associations are attenuated in some instances (U.S. EPA, 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 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, but not sufficient to infer, that a causal relationship exists between short-term $PM_{10-2.5}$ exposure and total mortality'' (U.S. EPA, 2019, p. 11–120).

2. Cardiovascular Effects

a. Long-term Exposures

In the 2009 PM ISA, the evidence describing the relationship between long-term exposure to $PM_{10-2.5}$ and cardiovascular effects was characterized as ''inadequate to infer the presence or absence of a causal relationship.'' The limited number of epidemiologic studies reported contradictory results and experimental evidence demonstrating an effect of $PM_{10-2.5}$ on the cardiovascular system was lacking (U.S. EPA, 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 and, while results across studies are not entirely consistent, some epidemiologic studies report positive associations with IHD and myocardial infarction (MI) (U.S. EPA, 2019, Figure 6–34); stroke (U.S. EPA, 2019, Figure 6–35); atherosclerosis (U.S. EPA, 2019, section 6.4.5); venous thromboembolism (VTE) (U.S. EPA, 2019, section 6.4.7); 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 copollutants models that adjust for $PM_{2.5}$. For morbidity outcomes, associations are inconsistent in copollutant models that adjust for $PM_{2.5}$, NO_2, and chronic noise pollution (U.S. EPA, 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

[44] Compared to humans, smaller fractions of inhaled $PM_{10-2.5}$ penetrate into the thoracic regions 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.

ISA conclusion that ''evidence from experimental animal studies is of insufficient quantity to establish biological plausibility'' (U.S. EPA, 2019, p. 6–277). Based largely on the observation of positive associations in some high-quality epidemiologic studies, the ISA concludes that ''evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term $PM_{10-2.5}$ exposure and cardiovascular effects'' (U.S. EPA, 2019, p. 6–277).

b. Short-Term Exposures

The 2009 ISA found that the available evidence for short-term $PM_{10-2.5}$ exposure and cardiovascular effects was ''suggestive of a causal relationship.'' This conclusion was based on several epidemiologic studies reporting associations between short-term $PM_{10-2.5}$ exposure and cardiovascular effects, including IHD hospitalizations, supraventricular ectopy, and changes in heart rate variability (HRV). In addition, dust storm events resulting in high concentrations of crustal material were linked to increases in total cardiovascular disease emergency department visits and hospital admissions. However, the 2009 ISA noted the potential for exposure measurement error and copollutant confounding in these epidemiologic studies. In addition, there was only limited evidence of cardiovascular effects from a small number of experimental studies (*e.g.* animal toxicological studies and controlled human exposure studies) that examined short-term $PM_{10-2.5}$ exposures (U.S. EPA, 2009c, section 6.2.12.2). In the last review, key uncertainties included the potential for exposure measurement error, copollutant confounding, and limited evidence of biological plausibility for cardiovascular effects following inhalation exposure (U.S. EPA, 2019, section 6.3.13).

The evidence for short-term $PM_{10-2.5}$ exposure and cardiovascular outcomes has expanded since the last review, 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, there is limited evidence to suggest that these associations are biologically plausible, or independent of copollutant confounding. The ISA also concludes that it remains unclear how the approaches used to estimate $PM_{10-2.5}$ 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).

3. Respiratory Effects—Short-Term Exposures

Based on a small number of epidemiologic studies observing associations with some respiratory effects and limited evidence from experimental studies to support biological plausibility, the 2009 ISA (U.S. EPA, 2009c) concluded that the relationship between short-term exposure to $PM_{10-2.5}$ and respiratory effects is ''suggestive of a causal relationship.'' Epidemiologic findings were consistent for respiratory infection and combined respiratory-related diseases, but not for COPD. Studies were characterized by overall uncertainty in the exposure assignment approach and limited information regarding potential copollutant confounding. Controlled human exposure studies of short-term $PM_{10-2.5}$ exposures found no lung function decrements and inconsistent evidence for pulmonary inflammation. Animal toxicological studies were limited to those using non-inhalation (*e.g.,* intra-tracheal instillation) routes of $PM_{10-2.5}$ exposure.

Recent epidemiologic findings consistently link $PM_{10-2.5}$ exposure to asthma exacerbation and respiratory mortality, with some evidence that associations remain positive (though attenuated in some studies of mortality) in copollutant models that include $PM_{2.5}$ or gaseous pollutants. Studies provide limited evidence for positive associations with other respiratory outcomes, including COPD exacerbation, respiratory infection, and combined respiratory-related diseases (U.S. EPA, 2019, Table 5–36). As noted above for other endpoints, an uncertainty in these epidemiologic studies is the lack of a systematic evaluation of the various methods used to estimate $PM_{10-2.5}$ concentrations and the resulting uncertainty in the spatial and temporal variability in $PM_{10-2.5}$ concentrations compared to $PM_{2.5}$ (U.S. EPA, 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. Cancer—Long-Term Exposures

In the last review, little information was available from studies of cancer following inhalation exposures to $PM_{10-2.5}$. Thus, the 2009 ISA determined the evidence was ''inadequate to assess the relationship between long-term $PM_{10-2.5}$ exposures and cancer'' (U.S. EPA, 2009c). Since the 2009 ISA, the assessment of long-term $PM_{10-2.5}$ exposure and cancer remains limited, with a few recent epidemiologic studies reporting positive, but imprecise, associations with lung cancer incidence. Uncertainty remains in these studies with respect to exposure measurement error due to the use of $PM_{10-2.5}$ predictions that have not been validated by monitored $PM_{10-2.5}$ concentrations (U.S. EPA, 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).

5. Metabolic Effects—Long-Term Exposures

The 2009 ISA did not make a causality determination for $PM_{10-2.5}$-related metabolic effects. Since the last review, one epidemiologic study shows an association between long-term $PM_{10-2.5}$ exposure and incident diabetes, while additional cross-sectional studies report associations with effects on glucose or insulin homeostasis (U.S. EPA, 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 7–15 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).

6. Nervous System Effects—Long-Term Exposures

The 2009 ISA did not make a causality determination for $PM_{10-2.5}$-related nervous system effects. In the current review, newly available epidemiologic studies report associations between $PM_{10-2.5}$ and impaired cognition and anxiety in adults in longitudinal analyses (U.S. EPA, 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).

C. Proposed Conclusions on the Current Primary PM_{10} Standard

This section describes the Administrator's proposed conclusions regarding the adequacy of the current primary PM_{10} standard. The approach to reaching these proposed conclusions draws from the ISA's assessment of the scientific evidence for health effects attributable to $PM_{10-2.5}$ exposures (U.S. EPA, 2019). Section III.C.1 discusses the evidence-based considerations from the PA. Section III.C.2 summarizes CASAC advice on the current primary PM_{10} standard, based on its review of the draft PA. Section III.C.3 presents the Administrator's proposed conclusions on the current primary PM_{10} standard.

1. Evidence-Based Considerations in the Policy Assessment

In the last review, the strongest evidence for $PM_{10-2.5}$-related health effects was for cardiovascular effects, respiratory effects, and premature mortality following short-term exposures. For each of these categories of effects, the ISA concluded that the evidence was ''suggestive of a causal relationship'' (U.S. EPA, 2009c, section 2.3.3). As summarized in the sections above, key uncertainties in the evidence resulted from limitations in the approaches used to estimate ambient $PM_{10-2.5}$ concentrations in epidemiologic studies, limited examination of the potential for confounding by co-occurring pollutants, and limited support for the biological plausibility of the serious effects reported in many epidemiologic studies. Since 2009, the evidence base for several $PM_{10-2.5}$-related health effects has expanded, broadening our understanding of the range of health effects linked to $PM_{10-2.5}$ exposures (U.S. EPA, 2020, Chapter 4). This includes expanded evidence for the relationships between long-term exposures and cardiovascular effects, metabolic effects, nervous system effects, cancer, and mortality. However, key limitations in the evidence that were identified in the 2009 ISA persist in studies that have become available since the last review. As discussed in the PA, these limitations include the following:

- The use of a variety of methods to estimate $PM_{10-2.5}$ exposures in epidemiologic studies and the lack of systematic evaluation of these methods, together with the relatively high spatial and temporal variability in ambient $PM_{10-2.5}$ concentrations and the small number of monitoring sites, results in uncertainty in exposure estimates;
- The limited number of studies that evaluate $PM_{10-2.5}$ health effect associations in copollutant models, together with evidence from some studies for attenuation of associations in such models, results in uncertainty in the independence of $PM_{10-2.5}$ health effect associations from co-occurring pollutants;
- The limited number of controlled human exposure and animal toxicology studies of $PM_{10-2.5}$ inhalation contributes to uncertainty in the biological plausibility of the $PM_{10-2.5}$-related effects reported in epidemiologic studies.

Thus, while new evidence is available for a broader range of health outcomes in the current review, including an increase in the number of studies that report effects related to long-term $PM_{10-2.5}$ exposure, that evidence is subject to the same types of uncertainties that were identified in the last review of the PM NAAQS. As in the last review, these uncertainties contribute to the conclusions in the 2019 ISA that the evidence for the $PM_{10-2.5}$-related health effects discussed in this section is ''suggestive of, but not sufficient to infer'' causal relationships.

2. CASAC Advice

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the public health protection afforded by the current primary PM_{10} standard. As for $PM_{2.5}$ (section II.C.2), the CASAC's advice is documented in a letter sent to the EPA Administrator (Cox, 2019a).

In its comments on the draft PA, the CASAC concurs with the draft PA's overall preliminary conclusions that it is appropriate to consider retaining the current primary PM_{10} standard without revision. The CASAC finds the more limited approach taken for PM_{10}, compared with the approach taken for $PM_{2.5}$, to be ''reasonable and appropriate'' given the less certain evidence and the conclusion that ''key uncertainties identified in the last review remain'' (Cox, 2019a, p. 13 of consensus responses). To reduce these uncertainties in future reviews, the CASAC recommends improvements to $PM_{10-2.5}$ exposure assessment, including a more extensive network for direct monitoring of the $PM_{10-2.5}$ fraction (Cox, 2019a, p. 13 of consensus responses). The CASAC also recommends additional human clinical and animal toxicology studies of the $PM_{10-2.5}$ fraction to improve the understanding of biological causal mechanisms and pathways (Cox, 2019a, p. 13 of consensus responses). Overall, the CASAC agrees with the EPA that ''. . . the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard and that evidence supports considering of retaining the current standard in this review'' (Cox, 2019a, p. 3 of letter).

3. Administrator's Proposed Decision on the Current Primary PM_{10} Standard

This section summarizes the Administrator's considerations and proposed conclusions related to the current primary PM_{10} standard and presents his proposed decision to retain that standard, without revision. As discussed above for $PM_{2.5}$ (II.C.3), in establishing primary standards under the Act that are ''requisite'' to protect the public health with an adequate margin of safety, the Administrator is seeking to establish standards that are neither more nor less stringent than necessary for this purpose. He recognizes that the Act does not require that primary standards be set at a zero-risk level; rather, the NAAQS must be sufficiently protective, but not more stringent than necessary.

Given these requirements, and consistent with the primary $PM_{2.5}$

standards discussed above (II.C.3), the Administrator's final decision in this review will be a public health policy judgment that draws upon the scientific information examining the health effects of $PM_{10-2.5}$ exposures, including how to consider the range and magnitude of uncertainties inherent in that information. His decision will require judgments based on an interpretation of the science that neither overstates nor understates its strengths and limitations, nor the appropriate inferences to be drawn.

As an initial matter, the Administrator notes that the decision to retain the primary PM_{10} standard in the last review recognized that epidemiologic studies had reported positive associations between $PM_{10-2.5}$ and mortality or morbidity in cities across North America, Europe, and Asia. These studies encompassed a variety of environments where $PM_{10-2.5}$ sources and composition were expected to vary widely. Although most of these studies examined $PM_{10-2.5}$ health effect associations in urban areas, some studies had also linked mortality and morbidity with relatively high ambient concentrations of particles of non-urban crustal origin. Drawing from this evidence, the EPA judged it appropriate to maintain a standard that provides some measure of protection against exposures to $PM_{10-2.5}$, regardless of location, source of origin, or particle composition (78 FR 3176, January 15, 2013). The Agency further judged it appropriate to retain a PM_{10} standard to provide such protection given that the varying concentrations of $PM_{10-2.5}$ permitted in urban versus non-urban areas under a PM_{10} standard, based on the varying levels of $PM_{2.5}$ present (*i.e.,* lower $PM_{10-2.5}$ concentrations allowed in urban areas, where $PM_{2.5}$ concentrations tend to be higher), appropriately reflected differences in the strength of $PM_{10-2.5}$ health effects evidence.

Since the last review, the Administrator notes that the evidence for several $PM_{10-2.5}$-related health effects has expanded, particularly for long-term exposures. Recent epidemiologic studies continue to report positive associations with mortality and morbidity in cities across North America, Europe, and Asia, where $PM_{10-2.5}$ sources and composition are expected to vary widely. While the Administrator recognizes that important uncertainties remain, as described below, he also recognizes that the expansion in the evidence since the last review has broadened the range of effects that have been linked with $PM_{10-2.5}$ exposures. Such studies provide an important part of the body of evidence supporting the ISA's 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; U.S. EPA, 2020, section 4.2). Drawing from his consideration of this evidence, the Administrator proposes to conclude that the scientific studies that have become available since the last review do not call into question the decision to maintain a primary PM_{10} 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 in the evidence, the Administrator notes that the decision in the last review highlighted limitations in estimates of ambient $PM_{10-2.5}$ concentrations used in epidemiologic studies, the limited evaluation of copollutant models to address the potential for confounding, and the limited number of experimental studies supporting biologically plausible pathways for $PM_{10-2.5}$-related effects. These and other limitations in the $PM_{10-2.5}$ evidence raised questions as to whether additional public health improvements would be achieved by revising the existing PM_{10} standard.

In the current review, despite the expanded body of evidence for $PM_{10-2.5}$-related health effects, the Administrator recognizes that similar uncertainties remain. As summarized above (III.B), 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 support for the biological plausibility of $PM_{10-2.5}$-related effects (*e.g.,* from controlled human exposure and animal toxicology studies) (U.S. EPA, 2020, section 4.2). 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). In light of his emphasis on evidence supporting ''causal'' and ''likely to be causal'' relationships (II.A.2, III.A.2), the Administrator judges that the $PM_{10-2.5}$-related health effects evidence provides an uncertain scientific foundation for making standard-setting decisions. He further judges that, as in the last review, limitations in this evidence raise questions as to whether additional public health improvements would be achieved by revising the existing PM_{10} standard.

In reaching conclusions on the primary PM_{10} standard, the Administrator also considers advice from the CASAC. As noted above, the CASAC recognizes the uncertainties in the evidence for $PM_{10-2.5}$-related health effects, stating that ''key uncertainties identified in the last review remain'' (Cox, 2019a, p. 13 of consensus responses). Given these uncertainties, the CASAC agrees with the PA conclusion that the evidence ''does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard'' (Cox, 2019a, p. 3 of letter). The CASAC further recommends that this evidence ''supports consideration of retaining the current standard in this review'' (Cox, 2019a, p. 3 of letter).

When the above information is taken together, the Administrator proposes to conclude that the available scientific evidence continues to support a PM_{10} standard to provide some measure of protection against $PM_{10-2.5}$ exposures. This conclusion reflects the expanded evidence for $PM_{10-2.5}$-related health effects in the current review. However, important limitations in the evidence remain. Consistent with the decision in the last review, the Administrator proposes to conclude that these limitations lead to considerable uncertainty regarding the potential public health implications of revising the existing PM_{10} standard. Given this uncertainty, and consistent with the CASAC's advice, the Administrator proposes to conclude that the available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard. Therefore, he proposes to retain the primary PM_{10} standard, without revision, in the current review. The Administrator solicits comment on this proposed decision and on the supporting rationale described above.

IV. Rationale for Proposed Decisions on the Secondary PM Standards

This section presents the rationale for the Administrator's proposed decision to retain the current secondary PM standards, without revision. This rationale is based on a thorough review of the latest scientific information generally published through December 2017,[45] as presented in the ISA, on non-ecological public welfare effects

[45] In addition to the review's opening ''call for information'' (79 FR 71764, December 3, 2014), ''the current ISA identified and evaluated studies and reports that have undergone scientific peer review and were published or accepted for publication between January 1, 2009 and March 31, 2017. A limited literature update identified some additional studies that were published before December 31, 2017'' (U.S. EPA, 2019, Appendix, p. A–3). References that are cited in the ISA, the references that were considered for inclusion but not cited, and electronic links to bibliographic information and abstracts can be found at: *https://hero.epa.gov/hero/particulate-matter.*

associated with PM and pertaining to the presence of PM in ambient air. The Administrator's rationale also takes into account the PA's evaluation of the policy-relevant information in the ISA and quantitative analyses of air quality related to visibility impairment and the CASAC's advice and recommendations, as reflected in discussions of the drafts of the ISA and PA at public meetings and in the CASAC's letters to the Administrator.

In presenting the rationale for the Administrator's proposed decision and its foundations, section IV.A provides background on the general approach for review of the secondary PM standards, including a summary of the approach used in the last review (section IV.A.1) and the general approach for the current review (section IV.A.2). Section IV.B summarizes the currently available evidence for PM-related visibility impairment and section IV.C summarizes the available information for other PM-related welfare effects. Section IV.D presents the Administrator's proposed conclusions on the current secondary PM standards.

A. General Approach

In the last review of the PM NAAQS, completed in 2012, the EPA retained the secondary 24-hour $PM_{2.5}$ standard, with its level of 35 μg/m^3, and the 24-hour PM_{10} standard, with its level of 150 μg/m^3 (78 FR 3228, January 15, 2013). The EPA also retained the level, set at 15 μg/m^3, and averaging time of the secondary annual $PM_{2.5}$ standard, while revising the form. With regard to the form of the annual $PM_{2.5}$ standard, the EPA removed the option for spatial averaging (78 FR 3228, January 15, 2013). Key aspects of the Administrator's decisions on the secondary PM standards for non-visibility effects and visibility effects are described below in section IV.A.1.

1. Approach Used in the Last Review

The 2012 decision on the adequacy of the secondary PM standards was based on consideration of the protection provided by those standards for visibility and for the non-visibility effects of materials damage, climate effects and ecological effects. As noted earlier, the current review of the public welfare protection provided by the secondary PM standards against ecological effects is occurring in the separate, on-going review of the secondary NAAQS for oxides of nitrogen and oxides of sulfur (U.S. EPA, 2016, Chapter 1, section 5.2; U.S. EPA, 2020, Chapter 1, section 5.1.1). Thus, the consideration of ecological effects in the 2012 review is not discussed here. Rather, the sections below focus on the prior Administrator's consideration of climate and materials effects (section IV.A.1.a) and visibility effects (section IV.A.1.b).

a. Non-Visibility Effects

With regard to the role of PM in climate, the prior Administrator considered whether it was appropriate to establish any distinct secondary PM standards to address welfare effects associated with climate impacts. In considering the scientific evidence, she noted the 2009 ISA conclusion ''that a causal relationship exists between PM and effects on climate'' and that aerosols [46] alter climate processes directly through radiative forcing and by indirect effects on cloud brightness, changes in precipitation, and possible changes in cloud lifetimes (U.S. EPA, 2009c, section 9.3.10). Additionally, the major aerosol components with the potential to affect climate processes (*i.e.,* black carbon (BC), organic carbon (OC), sulfates, nitrates and mineral dusts) vary in their reflectivity, forcing efficiencies, and direction of climate forcing (U.S. EPA, 2009c, section 9.3.10).

Noting the strong evidence indicating that aerosols affect climate, the prior Administrator further considered what the available information indicated regarding the adequacy of protection provided by the secondary PM standards. She noted that a number of uncertainties in the scientific information affected our ability to quantitatively evaluate the standards in this regard. For example, the ISA and PA noted the spatial and temporal heterogeneity of PM components that contribute to climate forcing, uncertainties in the measurement of aerosol components, inadequate consideration of aerosol impacts in climate modeling, insufficient data on local and regional microclimate variations and heterogeneity of cloud formations. In light of these uncertainties and the lack of sufficient data, the 2011 PA concluded that it was not feasible in the last review ''to conduct a quantitative analysis for the purpose of informing revisions [to the secondary PM NAAQS] based on climate'' (U.S. EPA, 2011, pp. 5–11 to 5–12) and that there was insufficient information available to base a national ambient air quality standard on climate impacts associated with ambient air concentrations of PM or its constituents (U.S. EPA, 2011, section 5.2.3). The prior Administrator agreed with this conclusion (78 FR 3225–3226, January 15, 2013).

With regard to materials effects, the she also considered effects associated with the deposition of PM (*i.e.,* dry and wet deposition), including both physical damage (materials effects) and aesthetic qualities (soiling effects). The deposition of PM can physically affect materials, adding to the effects of natural weathering processes, by promoting or accelerating the corrosion of metals; by degrading paints; and by deteriorating building materials such as stone, concrete, and marble (U.S. EPA, 2009c, section 9.5). Additionally, the deposition of PM from ambient air can reduce the aesthetic appeal of buildings and objects through soiling. The ISA concluded that evidence was ''sufficient to conclude that a causal relationship exists between PM and effects on materials'' (U.S. EPA, 2009c, sections 2.5.4 and 9.5.4). However, the 2011 PA noted that quantitative relationships were lacking between particle size, concentrations, and frequency of repainting and repair of surfaces and that considerable uncertainty exists in the contributions of co-occurring pollutants to materials damage and soiling processes (U.S. EPA, 2011, p. 5–29). The 2011 PA concluded that none of the evidence available in the last review called into question the adequacy of the existing secondary PM standards to protect against material effects (U.S. EPA, 2011, p. 5–29). The prior Administrator agreed with this conclusion (78 FR 3225–3226, January 15, 2013).

In considering non-visibility welfare effects in the last review, as discussed above, the prior Administrator concluded that, while it is important to maintain an appropriate degree of control of fine and coarse particles to address non-visibility welfare effects, ''[i]n the absence of information that would support any different standards . . . it is appropriate to retain the existing suite of secondary standards'' (78 FR 3225–3226, January 15, 2013). Her decision was consistent with the CASAC advice related to non-visibility effects. Specifically, the CASAC agreed with the 2011 PA conclusions that, while these effects are important, ''there is not currently a strong technical basis

[46] In the climate sciences research community, PM is encompassed by what is typically referred to as aerosol. An aerosol is defined as a solid or liquid suspended in a gas, but PM refers to the solid or liquid phase of an aerosol. In this review of the secondary PM NAAQS the discussion on climate effects of PM uses the term PM throughout for consistency with the ISA (U.S. EPA, 2019) as well as to emphasize that the climate processes altered by aerosols are generally altered by the PM portion of the aerosol. Exceptions to this practice include the discussion of climate effects in the last review, when aerosol was used when discussing suspending aerosol particles, and for certain acronyms that are widely used by the climate community that include the term aerosol (*e.g.,* aerosol optical depth, or AOD).

to support revisions of the current standards to protect against these other welfare effects'' (Samet, 2010a, p. 5). Thus, the prior Administrator concluded that it was appropriate to retain all aspects of the existing 24-hour $PM_{2.5}$ and PM_{10} secondary standards. With regard to the secondary annual $PM_{2.5}$ standard, she 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 (78 FR 3225–3226, January 15, 2013).

b. Visibility Effects

Having reached the conclusion to retain the existing secondary PM standards to protect against non-visibility welfare effects, the prior Administrator next considered the level of protection that would be requisite to protect public welfare against PM-related visibility impairment and whether to adopt a distinct secondary standard to achieve this level of protection. In reaching her final decision that the existing 24-hour $PM_{2.5}$ standard provides sufficient protection against PM-related visibility impairment (78 FR 3228, January 15, 2013), she considered the evidence assessed in the 2009 ISA (U.S. EPA, 2009c) and the analyses included in the Urban-Focused Visibility Assessment (2010 UFVA; U.S. EPA, 2010b) and the 2011 PA (U.S. EPA, 2011). She also considered the degree of protection for visibility that would be provided by the existing secondary standard, focusing specifically on the secondary 24-hour $PM_{2.5}$ standard with its level of 35 $\mu g/m^3$. These considerations, and the prior Administrator's conclusions regarding visibility are discussed in more detail below.

In the last review, the ISA concluded that, ''collectively, the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment'' (U.S. EPA, 2009c, p. 2–28). Visibility impairment is caused by light scattering and absorption by suspended particles and gases, including water content of aerosols.[47] The available evidence in the last review indicated that specific components of PM have been shown to contribute to visibility impairment. For example, at sufficiently high relative humidity values, sulfate and nitrate are the PM components that scatter more light and thus contribute most efficiently to visibility impairment. Elemental carbon (EC) and organic carbon (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, 2009c, section 2.5.1).

Visibility impairment can have implications for people's enjoyment of daily activities and for their overall sense of well-being (U.S. EPA, 2009c, section 9.2). In consideration of the potential public welfare implication of various degrees of PM-related visibility impairment, the prior Administrator considered the available visibility preference studies that were part of the overall body of evidence in the 2009 ISA and reviewed as a part of the 2010 UFVA. These preference studies provided information about the potential public welfare implications of visibility impairment from surveys in which participants were asked questions about their preferences or the values they placed on various visibility conditions, as displayed to them in scenic photographs or in images with a range of known light extinction levels.[48]

In noting the relationship between PM concentrations and PM-related light extinction, the prior Administrator focused on identifying an adequate level of protection against visibility-related welfare effects. She first concluded that a standard in terms of a $PM_{2.5}$ visibility index would provide a measure of protection against PM-related light extinction that directly takes into account the factors (*i.e.,* species composition and relative humidity) that influence the relationship between $PM_{2.5}$ in ambient air and PM-related visibility impairment. A $PM_{2.5}$ visibility index standard would afford a relatively high degree of uniformity of visual air quality protection in areas across the country by directly incorporating the effects of differences of $PM_{2.5}$ composition and relative humidity. In defining a target level of protection in terms of a $PM_{2.5}$ visibility index, as discussed below, she considered specific elements of the index, including the basis for its derivation, as well as an appropriate averaging time, level, and form.

With regard to the basis for derivation of a visibility index, the prior Administrator concluded that it was appropriate to use an adjusted version of the original IMPROVE algorithm,[49] in conjunction with monthly average relative humidity data based on long-term climatological means. In so concluding, she noted the CASAC conclusion on the reasonableness of reliance on a $PM_{2.5}$ light extinction indicator calculated from $PM_{2.5}$ chemical composition and relative humidity. In considering alternative approaches for a focus on visibility, she recognized that the available mass monitoring methods did not include measurement of the full water content of ambient $PM_{2.5}$, nor did they provide information on the composition of $PM_{2.5}$, both of which contribute to visibility impacts (77 FR 38980, June 29, 2012). In addition, at the time of the proposal, she recognized that suitable equipment and performance-based verification procedures did not then exist for direct measurement of light extinction and could not be developed within the time frame of the review (77 FR 38980–38981, June 29, 2012).

With regard to the averaging time of the index, the prior Administrator concluded that a 24-hour averaging time would be appropriate for a visibility index (78 FR 3226, January 15, 2013). Although she recognized that hourly or sub-daily (4- to 6-hour) averaging times, within daylight hours and excluding hours with relatively high humidity, are more directly related to the short-term nature of the perception of PM-related visibility impairment and relevant exposure periods for segments of the viewing public than a 24-hour averaging time, she also noted that there were data quality uncertainties associated with the instruments used to provide the hourly $PM_{2.5}$ mass measurements required for an averaging time shorter than 24 hours. She also considered the results of analyses that compared 24-hour and 4-hour averaging times for calculating the index. These analyses showed good correlation between 24-hour and 4-hour

[47] 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, 2009c, section 2.5.1). Particles with hygroscopic components (*e.g.,* particulate sulfate and nitrate) contribute more to light extinction at higher relative humidity than at lower relative humidity because they change size in the atmosphere in response to relative humidity.

[48] Preference studies were available in four urban areas in the last review. Three western preference studies were available, including one in Denver, Colorado (Ely *et al.*, 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009). More details about these studies are available in Appendix D of the PA.

[49] The revised IMPROVE algorithm (Pitchford *et al.*, 2007) uses major PM chemical composition measurements and relative humidity estimates to calculate light extinction. For more information about the derivation of and input data required for the original and revised IMPROVE algorithms, see 78 FR 3168–3177, January 15, 2013.

average $PM_{2.5}$ light extinction, as evidenced by reasonably high city-specific and pooled R-squared values, generally in the range of over 0.6 to over 0.8. Based on these analyses and the 2011 PA conclusions regarding them, the prior Administrator concluded that a 24-hour averaging time would be a reasonable and appropriate surrogate for a sub-daily averaging time.

With regard to the statistical form of the index, the prior Administrator settled on a 3-year average of annual 90th percentile values. In so doing, she noted that a 3-year average form provided stability from the occasional effect of inter-annual meteorological variability that can result in unusually high pollution levels for a particular year (78 FR 3198, January 15, 2013; U.S. EPA, 2011, p. 4–58).[50] Regarding the annual statistic to be averaged, the 2010 UFVA evaluated three different statistics: 90th, 95th, and 98th percentiles (U.S. EPA, 2010b, chapter 4). In considering these alternative percentiles, the 2011 PA noted that the Regional Haze Program targets the 20 percent most impaired days for improvements in visual air quality in Federal Class I areas and that the median of the distribution of these 20 percent worst days would be the 90th percentile. The 2011 PA further noted that strategies that are implemented so that 90 percent of days would have visual air quality that is at or below the level of the standard would reasonably be expected to lead to improvements in visual air quality for the 20 percent most impaired days. Lastly, the 2011 PA recognized that the available studies on people's preferences did not address frequency of occurrence of different levels of visibility and did not identify a basis for a different target for urban areas than that for Class I areas (U.S. EPA, 2011, p. 4–59). These considerations led the prior Administrator to conclude that 90th percentile form was the most appropriate annual statistic to be averaged across three years (78 FR 3226, January 15, 2013).

With regard to the level of the index, she considered the visibility preferences studies conducted in four urban areas (U.S. EPA, 2011, p. 4–61). Based on these studies, the PA identified a range of levels from 20 to 30 deciviews (dv)[51] as being a reasonable range of ''candidate protection levels'' (CPLs).[52] In considering this range of CPLs, she noted the uncertainties and limitations in public preference studies, including the small number of stated preference studies available; the relatively small number of study participants and the extent to which the study participants may not be representative of the broader study area population in some of the studies; and the variations in the specific materials and methods used in each study. She concluded that the substantial degree of variability and uncertainty in the public preference studies should be reflected in a target protection level at the upper end of the range of CPLs. Therefore, she concluded that it was appropriate to set a target level of protection in terms of a 24-hour $PM_{2.5}$ visibility index at 30 dv (78 FR 3226–3227, January 15, 2013).

Based on her considerations and conclusions summarized above, the prior Administrator concluded that the protection provided by a secondary standard based on a 3-year visibility metric, defined in terms of a $PM_{2.5}$ visibility index with a 24-hour averaging time, a 90th percentile form averaged over 3 years, and a level of 30 dv, would be requisite to protect public welfare with regard to visual air quality (78 FR 3227, January 15, 2013). Having reached this conclusion, she next determined whether an additional distinct secondary standard in terms of a visibility index was needed given the degree of protection from visibility impairment afforded by the existing secondary standards. Specifically, she noted that the air quality analyses showed that all areas meeting the existing 24-hour $PM_{2.5}$ standard, with its level of 35 $\mu g/m^3$, had visual air quality at least as good as 30 dv, based on the visibility index defined above (Kelly et al., 2012b, Kelly et al., 2012a). Thus, the secondary 24-hour $PM_{2.5}$ standard would likely be controlling relative to a 24-hour visibility index set at a level of 30 dv. Additionally, areas would be unlikely to exceed the target level of protection for visibility of 30 dv without also exceeding the existing secondary 24-hour standard. Thus, the prior Administrator judged that the 24-hour $PM_{2.5}$ standard ''provides sufficient protection in all areas against the effects of visibility impairment—*i.e.,* that the existing 24-hour $PM_{2.5}$ standard would provide *at least* the target level of protection for visual air quality of 30 dv which [she] judges appropriate'' (78 FR 3227, January 15, 2013). She further judged that ''[s]ince sufficient protection from visibility impairment would be provided for all areas of the country without adoption of a distinct secondary standard, and adoption of a distinct secondary standard will not change the degree of over-protection for some areas of the country. . . . adoption of such a distinct secondary standard is not needed to provide requisite protection for both visibility and nonvisibility related welfare effects'' (78 FR 3228, January 15, 2013).

2. Approach for the Current Review

To evaluate whether it is appropriate to consider retaining the current secondary PM standards, or whether consideration of revision is appropriate, the EPA has adopted an approach in this review that builds upon the general approach used in the last review and reflects the body of evidence and information now available. As summarized above, past approaches have been based most fundamentally on using information from studies of PM-related visibility effects, quantitative analyses of PM-related visibility impairment, information from studies of non-visibility welfare effects, advice from the CASAC, and public comments to inform the selection of secondary PM standards that, in the Administrator's judgment, protect the public welfare from any known or anticipated effects.

Similarly, in this review, the EPA draws on the available evidence and quantitative assessments pertaining to the public welfare impacts of PM in ambient air. In considering the scientific and technical information, the Agency considers both the information available at the time of the last review and the information that is newly available in this review. This includes information on PM-related visibility and non-visibility effects. Consistent with the approach in the last review, the quantitative air quality analyses for PM-related visibility effects provide a context for interpreting the evidence of visibility impairment and the potential public welfare significance of PM concentrations in ambient air associated with recent air quality conditions.

B. PM-Related Visibility Impairment

The information summarized here is based on the EPA's scientific assessment of the latest evidence on visibility effects associated with PM; this assessment is documented in the ISA

[50] The EPA recognized that a percentile form averaged over multiple years offers greater stability to the air quality management process by reducing the possibility that statistically unusual indicator values will lead to transient violations of the standard, thus reducing the potential for disruption of programs implementing the standard and reducing the potential for disruption of the protections provided by those programs.

[51] 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.

[52] For comparison, 20 dv, 25 dv, and 30 dv are equivalent to 64, 112, and 191 megameters (Mm^{-1}), respectively.

and its policy implications are further discussed in the PA. In considering the scientific and technical information, the PA reflects upon both the information available in the last review and information that is newly available since the last review. Policy implications of the currently available evidence are discussed in the PA (as summarized in section IV.D.1). The subsections below briefly summarize the following aspects of the evidence: The nature of PM-related visibility impairment (section IV.B.1), the relationship between ambient PM and visibility (section IV.B.2), and public perception of visibility impairment (section IV.B.3).

1. Nature of PM-Related Visibility Impairment

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. The conclusions of the ISA that ''the evidence is sufficient to conclude that a causal relationship exists between PM and visibility impairment'' is consistent with conclusions of causality in the last review (U.S. EPA, 2019, section 13.2.6). These conclusions are based on strong and consistent evidence that ambient PM can impair visibility in both urban and remote areas (U.S. EPA, 2019, section 13.1; U.S. EPA, 2009c, section 9.2.5).

2. Relationship Between Ambient PM and Visibility

The fundamental relationship between light extinction and PM mass, and the EPA's understanding of this relationship, has changed little since the 2009 ISA (U.S. EPA, 2009c). 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 units 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 than PM mass-based estimates because measurements do not depend on assumptions about particle characteristics (*e.g.,* size, shape, density, component mixture, etc.) (U.S. EPA, 2019, section 13.2.2.2). Measurements of light extinction can be made with high time resolution, allowing for characterization of subdaily temporal patterns of visibility impairment. A variety of measurement methods have been used (*e.g.,* transmissometers, integrating nephelometers, teleradiometers, telephotometers, and photography and photographic modeling), each with its own strengths and limitations (U.S. EPA, 2019, Table 13–1). However, there are no common performance-based criteria to evaluate these methods and none have been deployed broadly across the U.S. for routine measurement of visibility impairment.

In the absence of a robust monitoring network for the routine measurement of light extinction across the U.S., estimation of light extinction based on existing PM monitoring can be used. 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, 2009c, sections 9.2.2.2 and 9.2.3.1). However, routine ambient air monitoring rarely includes measurements of particle size and composition information with sufficient detail for these calculations. Accordingly, a much simpler algorithm has been developed to make estimating light extinction more practical.

This algorithm, known as the IMPROVE algorithm,[53] provides for the estimation of light extinction (b_{ext}), in units of Mm^{-1}, using routinely monitored components of fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) PM. Relative humidity data are also needed to estimate the contribution by liquid water that is in solution with the hygroscopic components of PM. To estimate each component's contribution to light extinction, their concentrations are multiplied by extinction coefficients and are additionally multiplied by a water growth factor that accounts for their expansion with moisture. Both the extinction efficiency coefficients and water growth factors of the IMPROVE algorithm have been developed by a combination of empirical assessment and theoretical calculation using particle size distributions associated with each of the major aerosol components (U.S. EPA, 2019, section 13.2.3.1, section 13.2.3.3).

The *original IMPROVE algorithm,* 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* was developed (Pitchford *et al.*, 2007). Since the last review, Lowenthal and Kumar (2016) further offered a number of modifications to the revised IMPROVE equation, with a focus of the application of the IMPROVE equation in remote sites. In particular, one of the modifications was to increase the multiplier to estimate the concentration of organic matter, [OM], from the concentration of organic carbon, [OC]. This modification was based on their evaluations of monitoring data from remote IMPROVE sites, which showed that in areas further away from PM sources, PM mass is often more oxygenated and contains a larger amount of organic PM. (U.S. EPA, 2019, section 13.2.3.3). As discussed below in section IV.D.1, analyses conducted in the current review estimate PM-related visibility impairment using each of these versions of the IMPROVE equation.

[53] The algorithm is referred to as the IMPROVE algorithm as it was developed specifically to use monitoring data generated at IMPROVE network sites and with equipment specifically designed to support the IMPROVE program and was evaluated using IMPROVE optical measurements at the subset of monitoring sites that make those measurements (Malm et al., 1994).

3. Public Perception of Visibility Impairment

In the last review, visibility preference studies were available from four areas in North America.[54] 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 those 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, 2010b). Based on the results of the studies in the four cities, 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 was identified (U.S. EPA, 2010b, p. 4–24; PA, Figure 5–2). Much lower visibility (considerably more haze resulting in higher values of light extinction) was considered acceptable in Washington, DC, than was in Denver, and 30 dv reflected the highest degree of visibility impairment judged to be acceptable by at least 50 percent of study participants (78 FR 3226–3227, January 15, 2013).

Since the time of the last review, no new visibility preference studies have been conducted in the U.S. Similarly, there is little newly available information regarding acceptable levels of visibility impairment in the U.S.

C. Other PM-Related Welfare Effects

The information summarized here is based on the EPA's scientific assessment of the latest evidence on the non-visibility welfare effects associated with PM. This assessment is documented in the ISA and its policy implications are further discussed in the PA. In considering the scientific and technical information, the PA reflects consideration of both the information available in the last review and information that is newly available since the last review. The subsections below briefly summarize the evidence related to climate effects (section IV.C.1) and materials effects (section IV.C.2).

1. Climate

In this review, as in the last review, the ISA concludes that ''overall the evidence is sufficient to conclude that a causal relationship exists between PM and climate effects'' (U.S. EPA, 2019, section 13.3.9). Since the last review, climate impacts have been extensively studied and recent research reinforces and strengthens the evidence evaluated in the 2009 ISA. New evidence provides greater specificity about the details of radiative forcing effects[55] and increases the 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, and since the last review, has issued the Fifth IPCC Assessment Report (AR5; IPCC, 2013) which summarizes any key scientific advances in understanding the climate effects of PM since the previous report. As in the last review, the ISA draws substantially on the IPCC report to summarize climate effects. As discussed in more detail below, the general conclusions are similar between the IPCC AR4 and AR5 reports with regard to effects of PM on global climate.

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 effects 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 (*i.e.,* reflectance) of the surface (Ban-Weiss *et al.*, 2014). PM also perturbs incoming solar radiation 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 on climate, as opposed to the indirect effects that involve aerosol-cloud interactions (ACI). The direct effects of PM on climate result primarily from particles scattering light away from Earth and sending a fraction of solar energy back into space, decreasing the transmission of visible radiation to the surface of the Earth and resulting in a decrease in the heating rate of the surface and the lower atmosphere. The IPCC AR5, taking into account both model simulations and satellite observations, reports a radiative forcing from aerosol-radiation interactions (RFari) from anthropogenic PM of -0.35 ± 0.5 watts per square meter (Wm^{-2}) (Boucher, 2013), which is comparable to AR4 (-0.5 ± 0.4 Wm^{-2}). Estimates of effective radiative forcing[56] 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 (second indirect effect). These effects are reported as the radiative

[54] Preference studies were available in four urban areas in the last review: Denver, Colorado (Ely *et al.*, 1991), Vancouver, British Columbia, Canada (Pryor, 1996), Phoenix, Arizona (BBC Research & Consulting, 2003), and Washington, DC (Abt Associates, 2001; Smith and Howell, 2009).

[55] Radiative forcing (RF) for a given atmospheric constituent is defined as the perturbation in net radiative flux, at the tropopause (or the top of the atmosphere) caused by that constituent, in watts per square meter (Wm–2), after allowing for temperatures in the stratosphere to adjust to the perturbation but holding all other climate responses constant, including surface and tropospheric temperatures (Fiore *et al.*, 2015; Myhre *et al.*, 2013). A positive forcing indicates net energy trapped in the Earth system and suggests warming of the Earth's surface, whereas a negative forcing indicates net loss of energy and suggests cooling (U.S. EPA, 2019, section 13.3.2.2).

[56] 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).

forcing from aerosol-cloud interaction (ERFaci) (U.S. EPA, 2019, section 13.3.3.2).[57] 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). 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 effects is +0.04 Wm^{-2}, with an uncertainty range of +0.02 to +0.09 Wm^{-2} (Boucher, 2013).

A number of new studies are available since the last review that have exampled the individual climate effects associated with key PM components, including sulfate, nitrate, OC, BC, and dust, along with updated quantitative estimate of the radiative forcing 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; U.S. EPA, 2020, section 2.3.1). Sulfate particles are highly reflective. Consistent with other recent estimates (Takemura, 2012, Zelinka et al., 2014, Adams et al., 2001, described below), 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), which is the same estimate from AR4. Sulfate is also a major contributor to the influence of PM on clouds (Takemura, 2012). A total effective radiative forcing (ERFari+aci) for anthropogenic sulfate has been estimated to be nearly -1.0 Wm^{-2} (Zelinka et al., 2014, Adams et al., 2001).

Nitrate particles form through the oxidation of nitrogen oxides and occur mainly in the form of ammonium nitrate. Ammonium preferentially associates with sulfate rather than nitrate, leading to formation of ammonium sulfate at the expense of ammonium nitrate (Adams et al., 2001). As anthropogenic emissions of SO_2 decline, more ammonium will be available to react with nitrate, potentially leading to future increases in ammonium nitrate particles in the atmosphere (U.S. EPA, 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. 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). Changes in the RFari estimates for individual PM components since AR4 have generally been modest, with one exception for the estimate for primary organic PM from fossil fuel combustion and biofuel use (Myhre et al., 2013).[58] 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).

The new research available in this review expands upon the evidence available at the time of the last review. Consistent with the evidence available in the last review, the key PM components, including sulfate, nitrate, OC, BC, and dust, that contribute to climate processes vary in their reflectivity, forcing efficiencies, and direction of forcing.

Radiative forcing due to PM elicits a number of responses in the climate system that can lead to significant effects on weather and climate over a range of spatial and temporal scales, mediated by a number of feedbacks that link PM and climate. Since the last review, the evidence base has expanded with respect to the mechanisms of climate responses and feedbacks to PM radiative forcing. However, the new literature published since the last review does not reduce the considerable

[57] While the ISA includes estimates of RFaci and ERFaci from a number of studies (U.S. EPA, 2019, sections 13.3.4.2, 13.3.4.3, 13.3.3.3), this discussion focuses on the single best estimate with a range of uncertainty, as reported in the IPCC AR5 (Boucher, 2013).

[58] The estimate of RFari for SOA is new in AR5 and was not included in AR4 (Myhre et al., 2013).

uncertainties that continue to exist related to these mechanisms.

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 to this PM forcing, however, 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, and may be quite complex, affecting multiple climate variables with possible differences in the sign of the response in different regions or for different variables (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 in this review suggests that PM influenced temperature trends across the southern and eastern U.S. in the 20th century, this evidence is not conclusive and significant uncertainties continue to exist. Further research is needed to better characterize the effects of PM on regional climate in the U.S. before PM climate effects can be quantified.

While expanded since the last review, the evidence of PM-related climate effects is still limited by significant uncertainties, particularly for understanding effects at regional scales. Large spatial and temporal heterogeneities in direct and indirect PM radiative forcing, and associated climate effects, 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 radiative forcing (U.S. EPA, 2019, section 13.3). In addition to the uncertainty in characterizing radiative forcing, large uncertainty exists in quantifying changes in specific climate variables associated with PM-related radiative forcing. Moreover, studies have shown that predicting climate variables for regions within the U.S. (which is of particular interest for the review of the PM NAAQS) is more uncertain than predicting climate variables globally due to natural climate variability (*e.g.,* Deser et al., 2012) and uncertainties in the representation of key atmospheric processes in state-of-the-art climate models. Furthermore, quantifying the influence of incremental changes in U.S. anthropogenic emissions on regional climate is subject to even greater uncertainty because the signal of U.S. anthropogenic emissions is relatively small compared with the global emissions considered in the studies cited above. Overall, these limitations and uncertainties make it difficult to quantify how incremental changes in the level of PM mass in ambient air in the U.S. would result in changes to climate in the U.S. Thus, as in the last review, the PA concludes that the data remain insufficient to conduct quantitative analyses for PM effects on climate in the current review (U.S. EPA, 2020, section 5.2.2.2.1).

2. Materials

In considering the evidence available in the current review of PM-related materials effects, the current evidence continues to support the conclusion from the last review that there is a causal relationship between PM deposition and materials effects. Effects of deposited PM, particularly sulfates and nitrates, 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.[59] The newly available evidence on materials effects of PM in this review are primarily from studies conducted outside of the U.S. on buildings and other items of cultural heritage and at concentrations greater than those typically observed in the U.S.; however, they provide limited new data for consideration in this review (U.S. EPA, 2019, section 13.4).

Materials damage from PM generally involves one or both of two processes: soiling and corrosion (U.S. EPA, 2019, section 13.4.2). Soiling and corrosion are complex, interdependent processes, typically beginning with deposition of atmospheric PM or SO_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 impact the aesthetic value of a structure or result in reversible or irreversible damage to the surface. The presence of air pollution can increase the frequency and duration of cleaning and can enhance biodeterioration processes on the surface of materials. For example, deposition of carbonaceous components of PM can lead to the formation of black crusts on surfaces, and the buildup of microbial biofilms[60] can discolor surfaces by trapping PM more efficiently (U.S. EPA, 2009c, 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 and nutrient content of deposited PM may enhance microbial growth on surfaces.

Since the last review, very little new evidence has become available related to deposition of SO_2 to materials such as limestone, granite, and metal. Deposition of SO_2 onto limestone can transform the limestone into gypsum, resulting in a rougher surface, which allows for increased surface area for accumulation of deposited PM (Camuffo and Bernardi, 1993; U.S. EPA, 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

[59] As discussed in the ISA (U.S. EPA, 2019, section 13.4.1), corrosion typically involves reactions of acidic PM (i.e., acidic sulfate or nitrate) with material surfaces, but gases like SO_2 and nitric acid (HNO_3) also contribute. Because ''the impacts of gaseous and particulate N and S wet deposition cannot be clearly distinguished'' (U.S. EPA, 2019, p. 13–1), the assessment of the evidence in the ISA considers the combined impacts.

[60] 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.

carbonaceous PM (both elemental and organic carbon) (McAlister et al., 2008, Grossi et al., 2007). Ozga et al. (2011) characterized damage to two concrete buildings in Poland and Italy. Gypsum was the main damage product on surfaces of these buildings that were sheltered from rain runoff, while PM embedded in the concrete, particularly carbonaceous particles, were responsible for darkening of the building walls (Ozga et al., 2011).

Building on the evidence available in the 2009 ISA, research has progressed on the theoretical understanding of soiling of cultural heritage in a number of studies. Barca et al. (2010) developed and tested a new methodological approach for characterizing trace elements and heavy metals in black crusts on stone monuments to identify the origin of the chemicals and 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, 2009c, section 9.5.3). Since that time, advances have been made on the quantification of degradation rates and further characterization of the factors that influence damage of stone materials (U.S. EPA, 2019, section 13.4.2). Decay rates of marble grave stones were found to be greater in heavily polluted areas compared to a relatively pristine area (Mooers et al., 2016). The time of wetness and the number of dissolution/crystallization cycles were identified as hazard indicators for stone materials, with greater hazard during the spring and fall when these indicators are relatively high (Casati et al., 2015).

A study examining the corrosion of steel as a function of PM composition and particle size found that changes in the composition of resulting rust gradually changed with particle size (Lau et al., 2008). In a study of damage to metal materials under in Hong Kong, which generally has much higher PM concentrations than those observed in the U.S., Liu et al. (2015) found that iron and steel were corroded by both PM and gaseous pollutants (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 last review, other materials have been studied for damage attributable to PM, including glass and photovoltaic panels. Soiling of glass can impact its optical and thermal properties and can lead to increased cleaning costs and frequency. The development of haze [61] on modern glass has been measured and modeled, with a strong correlation between the size distribution of particles and the evolution of the mass deposited on the surface of the glass. Measurements showed that, under sheltered conditions, mass deposition accelerated regularly with time in areas closest to sources of PM (*i.e.,* near roadways) and coarse mineral particles were more prevalent compared to other sites (Alfaro et al., 2012). Model predictions were found to correctly simulate the development of haze at site locations when compared with measurements (Alfaro et al., 2012).

Soiling of photovoltaic panels can lead to decreased energy efficiency. For example, soiling by carbonaceous PM decreased solar efficiency by nearly 38%, while soil particles reduced efficiency by almost 70% (Radonjic et al., 2017). The rate of photovoltaic power output can also be degraded by soiling and has been found to be related to the rate of dust accumulation. In five sites in the U.S. representing different meteorological and climatological conditions,[62] 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).

Some progress has been made since the last review in the development of dose-response relationships for soiling of building materials, yet 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

[61] In this discussion of non-visibility welfare effects, 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 V.B above, where it is used as a qualitative description of the blockage of sunlight by dust, smoke, and pollution.

[62] 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).

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-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 time of the last review, there has also been progress in developing methods to more rapidly evaluate soiling of different materials by PM mixtures. Modern buildings typically have simpler lines, less detailed surfaces, and a greater use of glass, tile, and metal, which are easier to clean than stone. There have also been major changes in the types of materials used for buildings, including a variety of polymers available for use as coatings and sealants. New economic and environmental considerations beyond aesthetic appeal and structural damage are emerging (U.S. EPA, 2019, section 13.4.3). Changes in building materials and design, coupled with new approaches in quantifying the dose-response relationship between PM and materials effects, may reduce the amount of time needed for observations to support the development of material-specific dose-response relationships.

In addition to dose-response functions, damage functions have also been used to quantify material decay as a function of pollutant type and load. Damage can be determined from sample surveys or inspection of actual damage and a damage function can be developed to link the rate of material damage to time of replacement or maintenance. A cost function can then link the time for replacement and maintenance to a monetary cost, and an economic function links cost to the dose of pollution based on the dose-response relationship (U.S. EPA, 2019, section 13.4.3). Damage functions are difficult to assess because it depends on human perception of the level of soiling deemed to be acceptable and evidence in this area remains limited in the current review. Since the last review, damage functions for a wide range of building materials (*i.e.,* stone, aluminum, zinc, copper, plastic, paint, rubber, stone) have been developed and reviewed (Brimblecombe and Grossi, 2010). One study estimated long-term deterioration of building materials and found that damage to durable building material (such as limestone, iron, copper, and discoloration of stone) is no longer controlled by pollution as was historically documented but rather that natural weathering is a more important influence on these materials in modern times (Brimblecombe and Grossi, 2009). Even as PM-attributable damage to stone and metals has decreased over time, it has been predicted that there will be potentially higher degradation rates for polymeric materials, plastic, paint, and rubber due to increased oxidant concentrations and solar radiation (Brimblecombe and Grossi, 2009).

As at the time of the last review and described just above, sufficient evidence is not available to conduct a quantitative assessment of PM mass or component-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 to inform the EPA's understanding of the public welfare implications of soiling (U.S. EPA, 2019, section 13.4). Similarly, while some information is available with regard to microbial deterioration of surfaces and the contribution of carbonaceous PM to the formation of black crusts that contribute to soiling, the available evidence does not support quantitative analyses (U.S. EPA, 2019, section 13.4). While some new evidence is available with respect to PM-attributable materials effects, the data are insufficient to conduct quantitative analyses for PM effects on materials in the current review.

D. Proposed Conclusions on the Current Secondary PM Standards

In reaching proposed conclusions on the current secondary PM standards, the Administrator takes into account policy-relevant evidence-based and quantitative information-based considerations, as well as advice from the CASAC. Evidence-based considerations draw from the EPA's assessment and integrated synthesis of the scientific evidence of PM-related welfare effects in the ISA (U.S. EPA, 2019, section 13.2). Quantitative information-based considerations draw from the EPA's assessment of recent air quality and associated PM-related visibility impairment in the PA (U.S. EPA, 2020, Chapter 5). Section IV.D.1 below summarizes evidence- and quantitative information-based considerations and the associated conclusions reached in the PA. Section IV.D.2 describes advice received from the CASAC on the secondary standards. Section IV.D.3 presents the Administrator's proposed decision on the current secondary PM standards.

1. Evidence- and Quantitative Information-Based Considerations in the Policy Assessment

The PA considers the degree to which the available scientific evidence and quantitative information supports or calls into question the adequacy of the protection afforded by the current secondary PM standards. In doing so, the PA considers the evidence assessed in the ISA, including the extent to which the new evidence for PM-related visibility impairment, climate effects, or materials effects alters key conclusions from the last review. The PA also considers quantitative analyses of visibility impairment and the extent to which they may indicate different conclusions from those in the last review regarding the degree of protection from adverse effects provided by the current secondary standards.

With regard to visibility impairment, the PA presents updated analyses based on recent air quality information, with a focus on locations meeting the current 24-hour $PM_{2.5}$ and PM_{10} standards. In the absence of advances in the monitoring methods for directly measuring light extinction, and given the lack of a robust monitoring network for the routine measurement of light extinction across the U.S. (section IV.B.2), as in the last review, the PA analyses use calculated light extinction to estimate PM-related visibility impairment (U.S. EPA, 2020, section 5.2.1.1). Compared to the last review, updated analyses incorporate several refinements. These include (1) the evaluation of three versions of the IMPROVE equation [63] to calculate light extinction (U.S. EPA, 2020, Appendix D, Equations D–1 through D–3) in order to better understand the influence of variability in equation inputs; [64] (2) the

[63] Given the lack of new information to inform a different visibility metric, the metric used in the PA is that defined by the EPA in the last review as the target level of protection for visibility (discussed above in section IV.A.1): A $PM_{2.5}$ visibility index with a 24-hour averaging time, a 90th percentile form averaged over 3 year, and a level of 30 dv (U.S. EPA, 2020, section 5.2.1.2).

[64] While the $PM_{2.5}$ monitoring network has an increasing number of continuous FEM monitors reporting hourly $PM_{2.5}$ mass concentrations, there continue to be data quality uncertainties associated with providing hourly $PM_{2.5}$ mass and component measurements that could be input into IMPROVE equation calculations for sub-daily visibility impairment estimates. Therefore, the inputs to these light extinction calculations are based on 24-hour

Continued

use of 24-hour relative humidity data, rather than monthly average relative humidity as was used in the last review (U.S. EPA, 2020, section 5.2.1.2, Appendix D); and (3) the inclusion of the coarse fraction in the estimation of light extinction in the subset of areas with $PM_{10-2.5}$ monitoring data available for the time period of interest (U.S. EPA, 2020, section 5.2.1.2, Appendix D). The PA's updated analyses include 67 monitoring sites that measure $PM_{2.5}$, including 20 sites that measure both PM_{10} and $PM_{2.5}$, that are geographically distributed across the U.S. in both urban and rural areas (U.S. EPA, 2020, Appendix D, Figure D–1).[65]

In areas that meet the current 24-hour $PM_{2.5}$ standard for the 2015–2017 time period, all sites have light extinction estimates at or below 27 dv using the original and revised IMPROVE equations (and most areas are below 25 dv; U.S. EPA, 2020, section 5.2.1.2). In addition, the one location that exceeds the current 24-hour $PM_{2.5}$ standard also has light extinction estimates at or below 27 dv (U.S. EPA, 2020, Figure 5-3). These findings are consistent with the findings of the analysis in the last review with older air quality data from 102 sites (Kelly et al., 2012b; 78 FR 3201, January 15, 2013).

When light extinction is calculated using the updated IMPROVE equation from Lowenthal and Kumar (2016), the resulting 3-year visibility metrics are slightly higher at all sites compared to light extinction calculated using the IMPROVE equations used in previous reviews (U.S. EPA, 2020, Figure 5-4). These results are consistent with the higher OC multiplier included in the IMPROVE equation from Lowenthal and Kumar (2016), reflecting the use of data from remote areas with higher concentrations of organic PM when validating that equation. As such, it is important to note that the Lowenthal and Kumar (2016) version of the IMPROVE equation may overestimate light extinction in non-remote areas, including in the urban areas in the PA's analyses.

Nevertheless, when light extinction is calculated 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 generally at or below 30 dv. The one exception to this is a site in Fairbanks, Alaska that just meets the current 24-hour $PM_{2.5}$ standard in 2015–17 and has a 3-year visibility index value just above 30 dv, rounding to 31 dv (compared to 27 dv when light extinction is calculated with the original and revised IMPROVE equations) (U.S. EPA, 2020, Appendix D, Table D–3). However, the unique conditions at this urban site (*e.g.,* higher OC concentrations, much lower temperatures, and the complete lack of sunlight for long periods) affect quantitative relationships between OC, OM and visibility (*e.g.,* Hand et al., 2012; Hand et al., 2013), making the most appropriate approach for characterizing light extinction in this area unclear.

In the last review, the EPA noted that $PM_{2.5}$ is the size fraction of PM responsible for most of the visibility impairment in urban areas (77 FR 38980, June 29, 2012). Data available at the time of the last review suggested that $PM_{10-2.5}$ is often a minor contributor to visibility impairment (U.S. EPA, 2010b), though it may make a larger contribution in some areas in the desert southwestern region of the U.S. However, at the time of the last review, there was little data available from $PM_{10-2.5}$ monitors to quantify the contribution of coarse PM to calculated light extinction.

Since the last review, an expansion of $PM_{10-2.5}$ monitoring efforts has increased the availability of data for use in estimating light extinction with both $PM_{2.5}$ and $PM_{10-2.5}$ concentrations included as inputs in the equations. For 2015–2017, 20 of the 67 $PM_{2.5}$ sites analyzed in the PA have collocated $PM_{10-2.5}$ monitoring data available. These 20 sites meet both the 24-hour $PM_{2.5}$ standard and 24-hour PM_{10} standard. All of these sites have 3-year visibility metrics at or below 30 dv regardless of whether light extinction is calculated with or without the coarse fraction, and for all three versions of the IMPROVE equation. Generally, the contribution of the coarse fraction to light extinction at these sites is minimal, contributing less than 1 dv to the 3-year visibility metric. However, these 20 locations would be expected to have relatively low concentrations of coarse PM. If PM_{10} and $PM_{10-2.5}$ data were available in locations with higher concentrations of coarse PM, such as in the southwestern U.S., the coarse fraction may be a more important contributor to light extinction and visibility impairment than in the locations examined in the PA analyses.

In summary, the findings of these updated quantitative analyses are consistent with those in the last review. The 3-year visibility metric is generally at or below 27 dv in areas that meet the current secondary standards, with only small differences observed for the three versions of the IMPROVE equation. Though such differences are modest, the IMPROVE equation from Lowenthal and Kumar (2016) always results in higher light extinction values, which is expected given the higher OC multiplier included in the equation and its validation using data from remote areas far away from emissions sources. There is very little difference in estimates of light extinction when $PM_{10-2.5}$ is included in the equation, although a somewhat larger coarse fraction contribution to light extinction would be expected in areas with higher coarse particle concentrations. Overall, the PA finds that updated quantitative analyses indicate that the current secondary PM standards provide a degree of protection against visibility impairment similar to the target level of protection identified in the last review, defined in terms of a PM visibility index.

With regard to PM-related climate effects, the PA recognizes that while the evidence base has expanded since the last review, the new evidence has not appreciably improved the understanding of the spatial and temporal heterogeneity of PM components that contribute to climate forcing (U.S. EPA, 2020, sections 5.2.2.1.1 and 5.4). Despite continuing research, there are still significant limitations in quantifying the contributions of PM and PM components to the direct and indirect effects on climate forcing (*e.g.,* changes to the pattern of rainfall, changes to wind patterns, effects on vertical mixing in the atmosphere) (U.S. EPA, 2020, sections 5.2.2.1.1 and 5.4). In addition, while a number of improvements and refinements have been made to climate models since the last review, these models continue to exhibit variability in estimates of the PM-related climate effects on regional scales (*e.g.,* ~100 km) compared to simulations at the global scale (U.S. EPA, 2020, sections 5.2.2.1.1 and 5.4). While new research has added to the understanding of climate forcing on a global scale, there remain significant limitations to quantifying potential adverse effects from PM on climate in the U.S. and how they would vary in response to incremental changes in PM concentrations in the U.S. Overall, the PA recognizes that while new research is available on climate forcing on a global scale, the remaining uncertainties and limitations are significant, and the new global scale

---

average measurements of $PM_{2.5}$ mass and components, rather than sub-daily information.

[65] These sites are those that have a valid 24-hour $PM_{2.5}$ design value for the 2015–2017 period and met strict criteria for PM species for this analysis, based on 24-hour average $PM_{2.5}$ mass and component data that were available from monitors in the IMPROVE network, CSN, and NCore Multipollutant Monitoring Network (U.S. EPA, 2020, Appendix D). $PM_{10-2.5}$ monitoring data is available for 20 of the 67 sites examined.

research does not translate directly to use at regional spatial scales. Thus, the evidence does not provide a clear understanding at the spatial scales needed for the NAAQS of a quantitative relationship between concentrations of PM mass in ambient air and the associated climate-related effects (U.S. EPA, 2020, sections 5.2.2.2.1 and 5.4). The PA concludes that the evidence does not call into question the adequacy of the current secondary PM standards for climate effects.

With regard to materials effects, the PA notes the availability of new evidence in this review related to the soiling process and the types of materials that are affected. Such evidence provides some limited information to inform dose-response relationships and damage functions associated with PM, though most recent studies have been conducted outside the U.S. (U.S. EPA, 2020, section 5.2.2.1.2; U.S. EPA, 2019, section 13.4). The recent evidence includes studies examining PM-related effects on the energy efficiency of solar panels and passive cooling building materials, though there remains insufficient evidence to establish quantitative relationships between PM in ambient air and these or other materials effects (U.S. EPA, 2020, section 5.2.2.1.2). While new research has expanded the body of evidence for PM-related materials effects, the PA recognizes the lack of information to inform quantitative analyses assessing materials effects or the potential public welfare implications of such effects. Thus, the PA concludes that the evidence does not call into question the adequacy of the current secondary PM standards for materials effects.

Overall, the PA recognizes that the newly available welfare effects evidence, critically assessed in the ISA as part of the full body of evidence, reaffirms the conclusions on the visibility, climate, and materials effects of PM as recognized in the last review (U.S. EPA, 2020, sections 5.2.1.1., 5.2.2.1, and 5.4). Further, there is a general consistency of the currently available evidence with the evidence that was available in the last review, including with regard to key aspects of the decision to retain the standards in the last review (U.S. EPA, 2020, sections 5.2.1.1, 5.2.2.1, and 5.4). The quantitative analyses for visibility impairment for recent air quality conditions indicate a similar level of protection against visibility effects considered to be adverse in the last review (U.S. EPA, 2020, sections 5.2.1.2 and 5.4). Collectively, the PA finds that the evidence and quantitative information-based considerations support consideration of retaining the current secondary PM standards, without revision (U.S. EPA, 2020, section 5.4).

2. CASAC Advice

As part of its review of the draft PA, the CASAC has provided advice on the adequacy of the current secondary PM standards. In its comments on the draft PA, the CASAC concurs with staff's overall preliminary conclusions that it is appropriate to consider retaining the current secondary PM standards without revision (Cox, 2019a). The CASAC ''finds much of the information . . . on visibility and materials effects of $PM_{2.5}$ to be useful, while recognizing that uncertainties and controversies remain about the best ways to evaluate these effects'' (Cox, 2019a, p. 13 of consensus responses). Regarding climate, while the CASAC agrees that research on PM-related effects has expanded since the last review, it also concludes that ''there are still significant uncertainties associated with the accurate measurement of PM contributions to the direct and indirect effects of PM on climate'' (Cox, 2019a, pp. 13–14 of consensus responses). The committee recommends that the EPA summarize the ''current scientific knowledge and quantitative modeling results for effects of reducing $PM_{2.5}$'' on several climate-related outcomes (Cox, 2019a, p. 14 of consensus responses), while also recognizing that ''it is appropriate to acknowledge uncertainties in climate change impacts and resulting welfare impacts in the United States of reductions in $PM_{2.5}$ levels'' (Cox, 2019a, p. 14 of consensus responses). When considering the overall body of scientific information for PM-related effects on visibility, materials, and climate, the CASAC agrees that ''the available evidence does not call into question the protection afforded by the current secondary PM standards and concurs that they should be retained'' (Cox, 2019a, p. 3 of letter).

3. Administrator's Proposed Decision on the Current Secondary PM Standards

This section summarizes the Administrator's considerations and conclusions related to the current secondary $PM_{2.5}$ and PM_{10} standards and presents his proposed decision to retain those standards, without revision. In establishing secondary standards under the Act that are ''requisite'' to protect the public welfare from any known or anticipated adverse effects, the Administrator is seeking to establish standards that are neither more nor less stringent than necessary for this purpose. He notes that secondary standards are not meant to protect against all known or anticipated effects, but rather those that are judged to be adverse to the public welfare. Consistent with the primary standards discussed above (sections II.C.3 and III.C.3), the Act does not require standards to be set at a zero-risk level; but rather at a level that limits risk sufficiently so as to protect the public welfare, but not more stringent than necessary to do so.

Given these requirements, the Administrator's final decision in this review will be a public welfare policy judgment that draws upon the scientific and technical information examining PM-related visibility impairment, climate effects and materials effects, including how to consider the range and magnitude of uncertainties inherent in that information. The Administrator recognizes that his final decision will be based on an interpretation of the scientific evidence and technical analyses that neither overstates nor understates their strengths and limitations, nor the appropriate inferences to be drawn.

As an initial matter in considering the secondary standards, the Administrator notes the longstanding body of evidence for PM-related visibility impairment. As in the last review, this evidence continues to demonstrate a causal relationship between ambient PM and effects on visibility (U.S. EPA, 2019, section 13.2). The Administrator recognizes that visibility impairment can have implications for people's enjoyment of daily activities and for their overall sense of well-being. Therefore, as in previous reviews, he considers the degree to which the current secondary standards protect against PM-related visibility impairment.

In doing so, the Administrator adopts an approach consistent with the approach used in the last review (section IV.A.1). That is, he first defines an appropriate target level of protection in terms of a PM visibility index that accounts for the factors that influence the relationship between particles in the ambient air and visibility (*i.e.,* size fraction, species composition, and relative humidity). He then considers air quality analyses examining this PM visibility index in locations meeting the current 24-hour $PM_{2.5}$ and PM_{10} standards (U.S. EPA, 2020, section 5.2.1.2).

To identify a target level of protection, the Administrator first defines the specific characteristics of the visibility index. He notes that in the last review, the EPA used an index based on estimates of light extinction by $PM_{2.5}$

components calculated using an adjusted version of the original IMPROVE algorithm. As described above (sections IV.B and IV.D.1), this algorithm allows the estimation of light extinction using routinely monitored components of $PM_{2.5}$ and $PM_{10\text{-}2.5}$,[66] along with estimates of relative humidity. While revisions have been made to the IMPROVE algorithm since the last review (U.S. EPA, 2020, section 5.2.1.1), the Administrator recognizes that our fundamental understanding of the relationship between ambient PM and light extinction has changed little and that the various IMPROVE algorithms can appropriately reflect this relationship across the U.S. In the absence of a robust monitoring network to directly measure light extinction (sections IV.B.2 and IV.D.1), he judges that estimated light extinction, as calculated using the IMPROVE algorithms, continues to provide a reasonable basis for defining a target level of protection against PM-related visibility impairment in the current review.

In further defining the characteristics of a visibility index based on estimates of light extinction, the Administrator considers the appropriate averaging time, form, and level of the index. With regard to the averaging time and form, the Administrator judges that the decisions made in the last review remain reasonable. In that review, a 24-hour averaging time was selected and the form was defined as the 3-year average of annual 90th percentile values. The decision on averaging time recognized the relatively strong correlations between 24-hour and sub-daily (*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 relevant for visual perception. This decision also recognized that the longer averaging time may be less influenced by atypical conditions and/or atypical instrument performance (78 FR 3226, January 15, 2013). The decision to set the form as the 3-year average of annual 90th percentile values noted that (1) a 3-year average provides stability from the occasional effect of inter-annual meteorological variability (78 FR 3198, January 15, 2013; U.S. EPA, 2011, p. 4–58); (2) the 90th percentile corresponds to the median of the distribution of the 20 percent worst days for visibility, which are targeted in Class I areas by the Regional Haze Program;[67] and (3) available studies on people's visibility preferences did not identify a basis for a different target than that identified for Class I areas (U.S. EPA, 2011, p. 4–59). Given the similar information available in the current review, the Administrator judges that these decisions remain reasonable and, therefore, that it remains appropriate to define a visibility index in terms of a 24-hour averaging time and a form based on the 3-year average of annual 90th percentile values.

The level of the index was set at 30 dv in the last review, reflecting the highest degree of visibility impairment judged to be acceptable by at least 50% of study participants in the available visibility preference studies (78 FR 3226–3227, January 15, 2013). The focus on 30 dv, rather than a lower level, was supported in light of the important uncertainties and limitations in the underlying public preference studies. Consistent with the last review, the Administrator notes the following uncertainties and limitations in these studies (U.S. EPA, 2020, section 5.2.1.1):

- The available studies may not capture the full range of visibility preferences in the U.S. population, particularly given the potential for preferences to vary based on the visibility conditions commonly encountered and the types of scenes being viewed.
- The available preference studies were conducted 15 to 30 years ago and may not reflect the visibility preferences of the U.S. population today.
- The available preference studies have used a variety of methods, potentially influencing responses as to what level of visibility impairment is deemed acceptable.
- Factors that are not captured by the methods used in available preference studies may influence people's judgments on acceptable visibility, including the duration of visibility impairment, the time of day during which light extinction is greatest, and the frequency of episodes of visibility impairment.

Because no visibility preference studies have been conducted in the U.S. since the last review, the Administrator recognizes that these uncertainties and limitations persist. Therefore, in the current review his consideration of the degree of visibility impairment constituting an adverse public welfare impact is based on the same preference studies, with the same uncertainties and limitations, that were available in the last review. Drawing from this information, the Administrator judges it appropriate to again use 30 dv as the level of the visibility index.

Having concluded that it remains appropriate in this review to define the target level of protection in terms of a visibility index based on estimated light extinction as described above (*i.e.,* with a 24-hour averaging time; a 3-year, 90th percentile form; and a level of 30 dv), the Administrator next considers the degree of protection from visibility impairment afforded by the existing secondary standards. He considers the updated analyses of PM-related visibility impairment presented in the PA (U.S. EPA, 2020, section 5.2.1.2), which reflect several improvements over the previous review. Specifically, the updated analyses examine multiple versions of the IMPROVE algorithm, including the version incorporating revisions since the last review (section IV.D.1). This approach provides an improved understanding of how variation in equation inputs impacts calculated light extinction (U.S. EPA, 2020, Appendix D). In addition, for a subset of monitoring sites with available $PM_{10\text{-}2.5}$ data, updated analyses better characterize the influence of the coarse fraction on light extinction (U.S. EPA, 2020, section 5.2.1.2).

The Administrator notes that the results of these updated analyses are consistent with the results from the last review. Regardless of the IMPROVE equation used, they demonstrate that the 3-year visibility metric is at or below about 30 dv in all areas meeting the current 24-hour $PM_{2.5}$ standard,[68] and below 25 dv in most of those areas (section IV.D.1). In the locations with available $PM_{10\text{-}2.5}$ monitoring, which met both the current 24-hour $PM_{2.5}$ and PM_{10} standards, 3-year visibility metrics were at or below 30 dv regardless of whether the coarse fraction was included in the calculation (U.S. EPA, 2020, section 5.2.1.2). Given the results of these analyses, the Administrator concludes that the updated scientific

[66] In the last review, the focus was on $PM_{2.5}$ components given their prominent role in PM-related visibility impairment in urban areas and the limited data available for $PM_{10\text{-}2.5}$ (77 FR 38980, June 29, 2012; U.S. EPA, 2020, section 5.2.1.2).

[67] In the last review, 90th, 95th, and 98th percentile forms were evaluated (U.S. EPA, 2010b, section 4.3.3; 78 FR 3198, January 15, 2013), and a standard with a 90th percentile form was reasonably expected to limit the occurrence of days with peak PM-related light extinction (78 FR 3198, January 15, 2013).

[68] As discussed in the PA (U.S. EPA, 2020, section 5.2.1.2), one site in Fairbanks, Alaska just meets the current 24-hour $PM_{2.5}$ standard and has a 3-year visibility index value of 27 dv based on the original IMPROVE equation and 31 dv based on the Lowenthal and Kumar (2016) equation. At this site, use of the Lowenthal and Kumar (2016) equation may not be appropriate given that PM composition and meteorological conditions may differ considerably from those under which revisions to the equation have been validated (U.S. EPA, 2020, section 5.2.1.2).

evidence and technical information support the adequacy of the current secondary $PM_{2.5}$ and PM_{10} standards to protect against PM-related visibility impairment. While the inclusion of the coarse fraction had a relatively modest impact on calculated light extinction in these analyses, he nevertheless recognizes the continued importance of the PM_{10} standard given the potential for larger impacts in locations with higher coarse particle concentrations, such as in the southwestern U.S., which were not included in the PA's analyses due to insufficient coarse particle data (U.S. EPA, 2019, section 13.2.4.1; U.S. EPA, 2020, section 5.2.1.2).

With respect to non-visibility welfare effects, the Administrator considers the evidence for PM-related impacts on climate and on materials and concludes that it is generally appropriate to retain the existing secondary standards and that it is not appropriate to establish any distinct secondary PM standards to address non-visibility PM-related welfare effects. With regard to climate, he recognizes that a number of improvements and refinements have been made to climate models since the time of the last review. However, despite continuing research and the strong evidence supporting a causal relationship with climate effects (U.S. EPA, 2019, section 13.3.9), the Administrator notes that there are still significant limitations in quantifying the contributions of the 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 recognizes that models continue to exhibit considerable variability in estimates of PM-related climate impacts at regional scales (*e.g.,* ~100 km), compared to simulations at the global scale (U.S. EPA, 2020, sections 5.2.2.1.1 and 5.4). The resulting uncertainty leads the Administrator to conclude that the scientific information available in the current review remains insufficient to quantify, with confidence, the impacts of ambient PM on climate in the U.S. (U.S. EPA, 2020, section 5.2.2.2.1) and that there is insufficient information at this time to base a national ambient standard on climate impacts.

With respect to materials effects, the Administrator notes that the evidence available in the current review continues to support the conclusion that there is a causal relationship with PM deposition (U.S. EPA, 2019, section 13.4). He recognizes that deposition of particles in the fine or coarse fractions can result in physical damage and/or impaired aesthetic qualities. Particles can contribute to materials damage by adding to the effects of natural weathering processes and by promoting the corrosion of metals, the degradation of painted surfaces, the deterioration of building materials, and the weakening of material components. While some new evidence on materials effects of PM is available in this review, the Administrator notes that this evidence is primarily from studies conducted outside of the U.S. (U.S. EPA, 2019, section 13.4). Given the more limited amount of information on the quantitative relationships between PM and materials effects in the U.S., and uncertainties in the degree to which those effects could be adverse to the public welfare, the Administrator judges that the scientific information available in the current review remains insufficient to quantify, with confidence, the public welfare impacts of ambient PM on materials and that there is insufficient information at this time to support a distinct national ambient standard based on materials impacts.

Taken together, the Administrator concludes that the scientific and technical information for PM-related visibility impairment, climate impacts, and materials effects, with its attendant uncertainties and limitations, supports the current level of protection provided by the secondary PM standards as being requisite to protect against known and anticipated adverse effects on public welfare. For visibility impairment, this conclusion reflects his consideration of the evidence for PM-related light extinction, together with his consideration of updated analyses of the protection provided by the current secondary $PM_{2.5}$ and PM_{10} standards. For climate and materials effects, this conclusion reflects 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, it is generally appropriate to retain the existing secondary standards and that it is not appropriate to establish any distinct secondary PM standards to address non-visibility PM-related welfare effects. His conclusions on the secondary standards are consistent with advice from the CASAC, which agrees ''that the available evidence does not call into question the protection afforded by the current secondary PM standards'' and recommends that the secondary standards ''should be retained'' (Cox, 2019a, p. 3 of letter). Thus, based on his consideration of the evidence and analyses for PM-related welfare effects, as described above, and his consideration of CASAC advice on the secondary standards, the Administrator proposes to retain those standards (*i.e.,* the current 24-hour and annual $PM_{2.5}$ standards, 24-hour PM_{10} standard), without revision.

V. Statutory and Executive Order Reviews

Additional information about these statutes and Executive Orders can be found at *http://www2.epa.gov/laws-regulations/laws-and-executive-orders.*

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

The Office of Management and Budget (OMB) determined that this action is a significant regulatory action and it was submitted to OMB for review. Any changes made in response to OMB recommendations have been documented in the docket. Because this action does not propose to change the existing NAAQS for PM, it does not impose costs or benefits relative to the baseline of continuing with the current NAAQS in effect. Thus, the EPA has not prepared a Regulatory Impact Analysis for this action.

B. Executive Order 13771: Reducing Regulations and Controlling Regulatory Costs

This action is not expected to be an Executive Order 13771 regulatory action. There are no quantified cost estimates for this proposed action because EPA is proposing to retain the current standards.

C. Paperwork Reduction Act (PRA)

This action does not impose an information collection burden under the PRA. There are no information collection requirements directly associated with a decision to retain a NAAQS without any revision under section 109 of the CAA and this action proposes to retain the current PM NAAQS without any revisions.

D. Regulatory Flexibility Act (RFA)

I certify that this action will not have a significant economic impact on a substantial number of small entities under the RFA. This action will not impose any requirements on small entities. Rather, this action proposes to retain, without revision, existing national standards for allowable concentrations of PM in ambient air as required by section 109 of the CAA. See also *American Trucking Associations* v. *EPA,* 175 F.3d 1027, 1044–45 (D.C. Cir. 1999) (NAAQS do not have significant impacts upon small entities because NAAQS themselves impose no

regulations upon small entities), rev'd in part on other grounds, *Whitman* v. *American Trucking Associations,* 531 U.S. 457 (2001).

E. Unfunded Mandates Reform Act (UMRA)

This action does not contain any unfunded mandate as described in the UMRA, 2 U.S.C. 1531–1538, and does not significantly or uniquely affect small governments. This action imposes no enforceable duty on any state, local, or tribal governments or the private sector.

F. Executive Order 13132: Federalism

This action does not have federalism implications. It will not have substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government.

G. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This action does not have tribal implications, as specified in Executive Order 13175. It does not have a substantial direct effect on one or more Indian Tribes. This action does not change existing regulations; it proposes to retain the current primary NAAQS for PM, without revision. Executive Order 13175 does not apply to this action.

H. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

This action is not subject to Executive Order 13045 because it is not economically significant as defined in Executive Order 12866. The health effects evidence for this action, which includes evidence for effects in children, is summarized in section II.B above and is described in the ISA and PA, copies of which are in the public docket for this action.

I. Executive Order 13211: Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution or Use

This action is not subject to Executive Order 13211, because it is not likely to have a significant adverse effect on the supply, distribution, or use of energy. The purpose of this document is to propose to retain the current PM NAAQS. This proposal does not change existing requirements. Thus, the EPA concludes that this proposal does not constitute a significant energy action as defined in Executive Order 13211.

J. National Technology Transfer and Advancement Act (NTTAA)

This action does not involve technical standards.

K. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

The EPA believes that this action does not have disproportionately high and adverse human health or environmental effects on minority, low-income populations and/or indigenous peoples, as specified in Executive Order 12898 (59 FR 7629, February 16, 1994). The documentation related to this is contained in sections II through IV above. The action proposed in this document is to retain, without revision, the existing NAAQS for PM based on the Administrator's conclusion that the existing standards protect public health, including the health of sensitive groups, with an adequate margin of safety and protect the public welfare. As discussed in section II, the EPA expressly considered the available information regarding health effects among at-risk populations in reaching the proposed decision that the existing standard is requisite.

L. Determination Under Section 307(d)

Section 307(d)(1)(V) of the CAA provides that the provisions of section 307(d) apply to ''such other actions as the Administrator may determine.'' Pursuant to section 307(d)(1)(V), the Administrator determines that this action is subject to the provisions of section 307(d).

REFERENCES

Abt Associates, Inc. (2001). Assessing public opinions on visibility impairment due to air pollution: Summary report. Research Triangle Park, NC, U.S. Environmental Protection Agency.

Abt Associates, Inc. (2005). Particulate matter health risk assessment for selected urban areas: Draft report. Research Triangle Park, NC, U.S. Environmental Protection Agency: 164.

Adams, PJ, Seinfeld, JH, Koch, D, Mickley, L and Jacob, D (2001). General circulation model assessment of direct radiative forcing by the sulfate-nitrate-ammonium-water inorganic aerosol system. J Geophys Res 106(D1): 1097–1111.

Adar, SD, Filigrana, PA, Clements, N and Peel, JL (2014). Ambient coarse particulate matter and human health: A systematic review and meta-analysis. Current Environmental Health Reports 1: 258–274.

Alfaro, SC, Chabas, A, Lombardo, T, Verney-Carron, A and Ausset, P (2012). Predicting the soiling of modern glass in urban environments: A new physically-based model. Atmos Environ 60: 348–357.

Ban-Weiss, GA, Jin, L, Bauer, SE, Bennartz, R, Liu, X, Zhang, K, Ming, Yi, Guo, H and Jiang, JH (2014). Evaluating clouds, aerosols, and their interactions in three global climate models using satellite simulators and observations. Journal of Geophysical Research: Atmospheres 119(18): 10876–10901.

Barca, D, Belfiore, CM, Crisci, GM, La Russa, MF, Pezzino, A and Ruffolo, SA (2010). Application of laser ablation ICP–MS and traditional techniques to the study of black crusts on building stones: A new methodological approach. Environmental Science and Pollution Research 17(8): 1433–1447.

BBC Research & Consulting (2003). Phoenix area visibility survey. Denver, CO.

Beloin, NJ and Haynie, FH (1975). Soiling of building materials. J Air Waste Manage Assoc 25(4): 399–403.

Bond, TC, Doherty, SJ, Fahey, DW, Forster, PM, Berntsen, T, Deangelo, BJ, Flanner, MG, Ghan, S, Kärcher, B, Koch, D, Kinne, S, Kondo, Y, Quinn, PK, Sarofim, MC, Schultz, MG, Schulz, M, Venkataraman, C, Zhang, H, Zhang, S, Bellouin, N, Guttikunda, SK, Hopke, PK, Jacobson, MZ, Kaiser, JW, Klimont, Z, Lohmann, U, Schwarz, JP, Shindell, D, Storelvmo, T, Warren, SG and Zender, CS (2013). Bounding the role of black carbon in the climate system: A scientific assessment. Journal of Geophysical Research: Atmospheres 118(11): 5380–5552.

Boucher, O (2013). *Clouds and Aerosols. In Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change.* Cambridge, United Kingdom and New York, NY, USA, Cambridge University Press.

Boyle, L, Burton, PD, Danner, V, Hannigan, MP and King, B (2017). Regional and national scale spatial variability of photovoltaic cover plate soiling and subsequent solar transmission losses. 7(5): 1354–1361.

Brimblecombe, P and Grossi, CM (2009). Millennium-long damage to building materials in London. Sci Total Environ 407(4): 1354–1361.

Brimblecombe, P and Grossi, CM (2010). Potential damage to modern building materials from 21st century air pollution. ScientificWorldJournal 10: 116–125.

Burns, J, Boogaard, H, Polus, S, Pfadenhauer, LM, Rohwer, AC, van Erp, AM, Turley, R and Rehfuess, E (2019). Interventions to reduce ambient particulate matter air pollution and their effect on health. Cochrane Database of Systematic Reviews(5).

Camuffo, D and Bernardi, A (1993). Microclimatic factors affecting the trajan column. Sci Total Environ 128(2–3): 227–255.

Cangerana Pereira, FA, Lemos, M, Mauad, T, de Assuncao, JV and Nascimento Saldiva, PH (2011). Urban, traffic-related particles and lung tumors in urethane treated mice. Clinics 66(6): 1051–1054.

Casati, M, Rovelli, G, D'Angelo, L, Perrone, MG, Sangiorgi, G, Bolzacchini, E and

Ferrero, L (2015). Experimental measurements of particulate matter deliquescence and crystallization relative humidity: Application in heritage climatology. Aerosol and Air Quality Research 15(2): 399–409.

Chan, EAW, Gantt, B and McDow, S (2018). The reduction of summer sulfate and switch from summertime to wintertime $PM_{2.5}$ concentration maxima in the United States. Atmos Environ 175: 25–32.

Correia, AW, Pope, CA, III, Dockery, DW, Wang, Y, un, Ezzati, M and Dominici, F (2013). Effect of air pollution control on life expectancy in the United States: An analysis of 545 U.S. counties for the period from 2000 to 2007. Epidemiology 24(1): 23–31.

Cox, LA. (2019a). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's *Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter (External Review Draft—September 2019).* December 16, 2019. EPA–CASAC–20–001. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf.*

Cox, LA. (2019b). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's *Integrated Science Assessment for Particulate Matter (External Review Draft—October 2018).* April 11, 2019. EPA–CASAC–19–002. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/932D1DF8C2A9043F852581000048170D?OpenDocument&TableRow=2.3#2.*

de Oliveira, BP, de la Rosa, JM, Miller, AZ, Saiz-Jimenez, C, Gomez-Bolea, A, Braga, MAS and Dionisio, A (2011). An integrated approach to assess the origins of black films on a granite monument. Environmental Earth Sciences 63(6–7 SI): 1677–1690.

Deser, C, Knutti, R, Solomon, S, and Phillips, AS (2012). Communication of the role of natural variability in future North American climate. Nature Climate Change 2: 775–779.

Di, Q, Dai, L, Wang, Y, Zanobetti, A, Choirat, C, Schwartz, JD and Dominici, F (2017a). Association of short-term exposure to air pollution with mortality in older adults. J Am Med Assoc 318(24): 2446–2456.

Di, Q, Wang, Y, Zanobetti, A, Wang, Y, Koutrakis, P, Choirat, C, Dominici, F and Schwartz, JD (2017b). Air pollution and mortality in the Medicare population. New Engl J Med 376(26): 2513–2522.

Ely, DW, Leary, JT, Stewart, TR and Ross, DM (1991). *The establishment of the Denver Visibility Standard.* Denver, Colorado, Colorado Department of Health.

Fiore, AM, Naik, V and Leibensperger, EM (2015). Air quality and climate connections. J Air Waste Manage Assoc 65(6): 645–685.

Grossi, CM, Brimblecombe, P, Esbert, RM and Javier Alonso, F (2007). Color changes in architectural limestones from pollution and cleaning. Color Research and Application 32(4): 320–331.

Hand, JL, Copeland, SA, Day, DA, Dillner, AM, Indresand, H, Malm, WC, McDade, CE, Moore, CT, Jr., Pitchford, ML, Schichtel, BA and Watson, JG (2011). Spatial and seasonal patterns and temporal variability of haze and its constituents in the United States, IMPROVE Report V. Fort Collins, CO, Colorado State University.

Hand, JL, Schichtel, BA, Pitchford, M, Malm, WC and Frank, NH (2012). Seasonal composition of remote and urban fine particulate matter in the United States. Journal of Geophysical Research: Atmospheres 117(D5).

Hand, JL, Schichtel, BA, Malm, WC and Frank, NH (2013). Spatial and Temporal Trends in $PM_{2.5}$ Organic and Elemental Carbon across the United States. Advances in Meteorology.

Hauglustaine, DA, Balkanski, Y and Schulz, M (2014). A global model simulation of present and future nitrate aerosols and their direct radiative forcing of climate. Atmos Chem Phys 14(20): 11031–11063.

Heald, CL, Ridley, DA, Kreidenweis, SM and Drury, EE (2010). Satellite observations cap the atmospheric organic aerosol budget. Geophys Res Lett 37.

Henneman, LR, Liu, C, Mulholland, JA and Russell, AG (2017). Evaluating the effectiveness of air quality regulations: A review of accountability studies and frameworks. Journal of the Air Waste Management Association 67(2): 144–172.

IPCC (2013). Climate change 2013: The physical science basis. Contribution of working group I to the fifth assessment report of the Intergovernmental Panel on Climate Change. T. F. Stocker, D. Qin, G. K. Plattner et al. Cambridge, UK, Cambridge University Press.

Jimenez, JL, Canagaratna, MR, Donahue, NM, Prevot, AS, Zhang, Q, Kroll, JH, Decarlo, PF, Allan, JD, Coe, H, Ng, NL, Aiken, AC, Docherty, KS, Ulbrich, IM, Grieshop, AP, Robinson, AL, Duplissy, J, Smith, JD, Wilson, KR, Lanz, VA, Hueglin, C, Sun, YL, Tian, J, Laaksonen, A, Raatikainen, T, Rautiainen, J, Vaattovaara, P, Ehn, M, Kulmala, M, Tomlinson, JM, Collins, DR, Cubison, MJ, Dunlea, EJ, Huffman, JA, Onasch, TB, Alfarra, MR, Williams, PI, Bower, K, Kondo, Y, Schneider, J, Drewnick, F, Borrmann, S, Weimer, S, Demerjian, K, Salcedo, D, Cottrell, L, Griffin, R, Takami, A, Miyoshi, T, Hatakeyama, S, Shimono, A, Sun, JY, Zhang, YM, Dzepina, K, Kimmel, JR, Sueper, D, Jayne, JT, Herndon, SC, Trimborn, AM, Williams, LR, Wood, EC, Middlebrook, AM, Kolb, CE, Baltensperger, U and Worsnop, DR (2009). Evolution of organic aerosols in the atmosphere. Science 326(5959): 1525–1529.

Kelly, J, Schmidt, M and Frank, N. (2012a). Memorandum to PM NAAQS Review Docket (EPA–HQ–OAR–2007–0492). Updated comparison of 24-hour $PM_{2.5}$ design values and visibility index design values. December 14, 2012. Docket ID No. EPA–HQ–OAR–2007–0492. Research Triangle Park, NC. Office of Air Quality Planning and Standards. Available at: *https://www3.epa.gov/ttn/naaqs/standards/pm/data/20121214kelly.pdf.*

Kelly, J, Schmidt, M, Frank, N, Timin, B, Solomon, D and Venkatesh, R. (2012b). Memorandum to PM NAAQS Review Docket (EPA–HQ–OAR–2007–0492). Technical Analyses to Support Surrogacy Policy for Proposed Secondary $PM_{2.5}$ NAAQS under NSR/PSD Programs. June 14, 2012. Docket ID No. EPA–HQ–OAR–2007–0492. Research Triangle Park, NC. Office of Air Quality Planning and Standards. Available at: *https://www3.epa.gov/ttn/naaqs/standards/pm/data/20120614Kelly.pdf.*

Kloog, I, Ridgway, B, Koutrakis, P, Coull, BA and Schwartz, JD (2013). Long- and short-term exposure to $PM_{2.5}$ and mortality: Using novel exposure models. Epidemiology 24(4): 555–561.

Kloppmann, W, Bromblet, P, Vallet, JM, Verges-Belmin, V, Rolland, O, Guerrot, C and Gosselin, C (2011). Building materials as intrinsic sources of sulphate: A hidden face of salt weathering of historical monuments investigated through multi-isotope tracing (B, O, S). Sci Total Environ 409(9): 1658–1669.

Kok, JF, Ridley, DA, Zhou, Q, Miller, RL, Zhao, C, Heald, CL, Ward, DS, Albani, S and Haustein, K (2017). Smaller desert dust cooling effect estimated from analysis of dust size and abundance. Nature Geoscience 10(4): 274–278.

Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, III, Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, Deluca, P, Finkelstein, N, Ito, K, Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H and Tempalski, B (2009). Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Boston, MA, Health Effects Institute. 140: 5–114; discussion 115–136.

Laden, F, Schwartz, J, Speizer, FE and Dockery, DW (2006). Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard Six Cities study. Am J Respir Crit Care Med 173(6): 667–672.

Lanzon, M and Garcia-Ruiz, PA (2010). Deterioration and damage evaluation of rendering mortars exposed to sulphuric acid. Mater Struct 43(3): 417–427.

Lau, NT, Chan, CK, Chan, L and Fang, M (2008). A microscopic study of the effects of particle size and composition of atmospheric aerosols on the corrosion of mild steel. Corros Sci 50(10): 2927–2933.

Lee, M, Koutrakis, P, Coull, B, Kloog, I and Schwartz, J (2015). Acute effect of fine particulate matter on mortality in three Southeastern states from 2007–2011. Journal of Exposure Science and Environmental Epidemiology 26(2): 173–179.

Lee, YH, Lamarque, JF, Flanner, MG, Jiao, C, Shindell, DT, Berntsen, T, Bisiaux, MM, Cao, J, Collins, WJ, Curran, M, Edwards, R, Faluvegi, G, Ghan, S, Horowitz, LW, McConnell, JR, Ming, J, Myhre, G, Nagashima, T, Naik, V, Rumbold, ST, Skeie, RB, Sudo, K, Takemura, T, Thevenon, F, Xu, B and Yoon, JH (2013). Evaluation of preindustrial to present-day black carbon and its albedo forcing from Atmospheric Chemistry and Climate Model Intercomparison Project (ACCMIP). Atmos Chem Phys 13(5): 2607–2634.

Leibensperger, EM, Mickley, LJ, Jacob, DJ, Chen, WT, Seinfeld, JH, Nenes, A, Adams, PJ, Streets, DG, Kumar, N and Rind, D (2012). Climatic effects of 1950–2050 changes in US anthropogenic aerosols—Part 2: Climate response. Atmos Chem Phys 12(7): 3349–3362.

Lepeule, J, Laden, F, Dockery, D and Schwartz, J (2012). Chronic exposure to fine particles and mortality: An extended follow-up of the Harvard Six Cities study from 1974 to 2009. Environ Health Perspect 120(7): 965–970.

Levy, H, Horowitz, LW, Schwarzkopf, MD, Ming, Yi, Golaz, JC, Naik, V and Ramaswamy, V (2013). The roles of aerosol direct and indirect effects in past and future climate change. Journal of Geophysical Research: Atmospheres 118(10): 4521–4532.

Lippmann, M, Chen, LC, Gordon, T, Ito, K and Thurston, GD (2013). National Particle Component Toxicity (NPACT) Initiative: Integrated epidemiologic and toxicologic studies of the health effects of particulate matter components: Investigators' Report. Boston, MA, Health Effects Institute: 5–13.

Liu, B, Wang, DW, Guo, H, Ling, ZH and Cheung, K (2015). Metallic corrosion in the polluted urban atmosphere of Hong Kong. Environ Monit Assess 187(1): 4112.

Lombardo, T, Ionescu, A, Chabas, A, Lefevre, RA, Ausset, P and Candau, Y (2010). Dose-response function for the soiling of silica-soda-lime glass due to dry deposition. Sci Total Environ 408(4): 976–984.

Lowenthal, DH and Kumar, N (2004). Variation of mass scattering efficiencies in IMPROVE. Journal of the Air and Waste Management Association (1990–1992) 54(8): 926–934.

Lowenthal, DH and Kumar, N (2016). Evaluation of the IMPROVE Equation for estimating aerosol light extinction. J Air Waste Manage Assoc 66(7): 726–737.

Malm, WC, Sisler, JF, Huffman, D, Eldred, RA and Cahill, TA (1994). Spatial and seasonal trends in particle concentration and optical extinction in the United States. J Geophys Res 99(D1): 1347–1370.

Malm, WC and Hand, JL (2007). An examination of the physical and optical properties of aerosols collected in the IMPROVE program. Atmos Environ 41(16): 3407–3427.

Mauad, T, Rivero, DH, de Oliveira, RC, Lichtenfels, AJ, Guimaraes, ET, de Andre, PA, Kasahara, DI, Bueno, HM and Saldiva, PH (2008). Chronic exposure to ambient levels of urban particles affects mouse lung development. Am J Respir Crit Care Med 178(7): 721–728.

McAlister, J, Smith, BJ and Torok, A (2008). Transition metals and water-soluble ions in deposits on a building and their potential catalysis of stone decay. Atmos Environ 42(33): 7657–7668.

McNeill, VF, Woo, JL, Kim, DD, Schwier, AN, Wannell, NJ, Sumner, AJ and Barakat, JM (2012). Aqueous-phase secondary organic aerosol and organosulfate formation in atmospheric aerosols: A modeling study. Environ Sci Technol 46(15): 8075–8081.

Mie, G (1908). Beitrage zur Optik truber Medien, speziell kolloidaler Metallosungen [Optics of cloudy media, especially colloidal metal solutions]. Annalen der Physik 25(3): 377–445.

Miller, KA, Siscovick, DS, Sheppard, L, Shepherd, K, Sullivan, JH, Anderson, GL and Kaufman, JD (2007). Long-term exposure to air pollution and incidence of cardiovascular events in women. New Engl J Med 356(5): 447–458.

Mooers, HD, Cota-Guertin, AR, Regal, RR, Sames, AR, Dekan, AJ and Henkels, LM (2016). A 120-year record of the spatial and temporal distribution of gravestone decay and acid deposition. Atmos Environ 127: 139–154.

Myhre, G, Shindell, D, Bréon, FM, Collins, W, Fuglestvedt, J, Huang, J, Koch, D, Lamarque, JF, Lee, D, Mendoza, B, Nakajima, T, Robock, A, Stephens, G, Takemura, T and Zhang, H, Eds. (2013). Anthropogenic and natural radiative forcing. Cambridge, UK, Cambridge University Press.

Ozga, I, Bonazza, A, Bernardi, E, Tittarelli, F, Favoni, O, Ghedini, N, Morselli, L and Sabbioni, C (2011). Diagnosis of surface damage induced by air pollution on 20th-century concrete buildings. Atmos Environ 45(28): 4986–4995.

Pitchford, M, Maim, W, Schichtel, B, Kumar, N, Lowenthal, D and Hand, J (2007). Revised algorithm for estimating light extinction from IMPROVE particle speciation data. J Air Waste Manage Assoc 57(11): 1326–1336.

Pope, CA, III, I, Burnett, RT, Thurston, GD, Thun, MJ, Calle, EE, Krewski, D and Godleski, JJ (2004). Cardiovascular mortality and long-term exposure to particulate air pollution: Epidemiological evidence of general pathophysiological pathways of disease. Circulation 109(1): 71–77.

Pope, CA, III, Ezzati, M and Dockery, DW (2009). Fine-particulate air pollution and life expectancy in the United States. New Engl J Med 360(4): 376–386.

Pruitt, E. (2018). Memorandum from E. Scott Pruitt, Administrator, U.S. EPA to Assistant Administrators. Back-to-Basics Process for Reviewing National Ambient Air Quality Standards. May 9, 2018. U.S. EPA HQ, Washington DC. Office of the Administrator. Available at: *https://www.epa.gov/criteria-air-pollutants/back-basics-process-reviewing-national-ambient-air-quality-standards.*

Pryor, SC (1996). Assessing public perception of visibility for standard setting exercises. Atmos Environ 30(15): 2705–2716.

Puett, RC, Hart, JE, Yanosky, JD, Spiegelman, D, Wang, M, Fisher, JA, Hong, B and Laden, F (2014). Particulate matter air pollution exposure, distance to road, and incident lung cancer in the Nurses' Health Study cohort. Environ Health Perspect 122(9): 926–932.

Raaschou-Nielsen, O, Andersen, ZJ, Beelen, R, Samoli, E, Stafoggia, M, Weinmayr, G, Hoffmann, B, Fischer, P, Nieuwenhuijsen, MJ, Brunekreef, B, Xun, WW, Katsouyanni, K, Dimakopoulou, K, Sommar, J, Forsberg, B, Modig, L, Oudin, A, Oftedal, B, Schwarze, PE, Nafstad, P, De Faire, U, Pedersen, NL, Östenson, CG, Fratiglioni, L, Penell, J, Korek, M, Pershagen, G, Eriksen, KT, Sørensen, M, Tjønneland, A, Ellermann, T, Eeftens, M, Peeters, PH, Meliefste, K, Wang, M, Bueno-De-mesquita, B, Key, TJ, De Hoogh, K, Concin, H, Nagel, G, Vilier, A, Grioni, S, Krogh, V, Tsai, MY, Ricceri, F, Sacerdote, C, Galassi, C, Migliore, E, Ranzi, A, Cesaroni, G, Badaloni, C, Forastiere, F, Tamayo, I, Amiano, P, Dorronsoro, M, Trichopoulou, A, Bamia, C, Vineis, P and Hoek, G (2013). Air pollution and lung cancer incidence in 17 European cohorts: Prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE). The Lancet Oncology 14(9): 813–822.

Radonjic, IS, Pavlovic, TM, Mirjanic, DLj, Radovic, MK, Milosavljevic, DD and Pantic, LS (2017). Investigation of the impact of atmospheric pollutants on solar module energy efficiency. Thermal Science 21(5): 2021–2030.

Rosso, F, Pisello, AL, Jin, WH, Ghandehari, M, Cotana, F and Ferrero, M (2016). Cool marble building envelopes: The effect of aging on energy performance and aesthetics. Sustainability 8(8): Article #753.

Ryan, PA, Lowenthal, D and Kumar, N (2005). Improved light extinction reconstruction in interagency monitoring of protected visual environments. J Air Waste Manage Assoc 55(11): 1751–1759.

Saha, PK, Robinson, ES, Shah, RU, Zimmerman, N, Apte, JS, Robinson, AL and Presto, AA (2018). Reduced ultrafine particle concentration in urban air: Changes in nucleation and anthropogenic emissions. Environ Sci Technol 52(12): 6798–6806.

Samet, J. (2009). Letter from Jonathan Samet, Chair, Clean Air Scientific Advisory Committee, to Administrator Lisa Jackson. Re: CASAC Particulate Matter Review of Integrated Science Assessment for Particulate Matter (Second External Review Draft, July 2009). November 24, 2009. EPA–CASAC–10–001. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *http://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1005PH9.txt.*

Samet, J. (2010a). Letter from Jonathan Samet, Chair, Clean Air Scientific Advisory Committee, to Administrator Lisa Jackson. Re: CASAC Review of Policy Assessment for the Review of the

PM NAAQS—First External Review Draft (March 2010). May 17, 2010. EPA–CASAC–10–011. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=9101XOXQ.txt.*

Samet, J. (2010b). Letter from Jonathan Samet, Chair, Clean Air Scientific Advisory Committee, to Administrator Lisa Jackson. Re: CASAC Review of Quantitative Health Risk Assessment for Particulate Matter—Second External Review Draft (February 2010). April 15, 2010. EPA–CASAC–10–008. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1007CVB.txt.*

Samet, J. (2010c). Letter from Jonathan Samet, Chair, Clean Air Scientific Advisory Committee, to Administrator Lisa Jackson. Re: CASAC Review of Policy Assessment for the Review of the PM NAAQS—Second External Review Draft (June 2010). September 10, 2010. EPA–CASAC–10–015. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: *https://yosemite.epa.gov/sab/sabproduct.nsf/CCF9F4C0500C500F8525779D0073C593/$File/EPA-CASAC-10-015-unsigned.pdf.*

Schweizer, D, Cisneros, R, Traina, S, Ghezzehei, TA and Shaw, G (2017). Using National Ambient Air Quality Standards for fine particulate matter to assess regional wildland fire smoke and air quality management. J Environ Manage 201: 345–356.

Scott, CE, Rap, A, Spracklen, DV, Forster, PM, Carslaw, KS, Mann, GW, Pringle, KJ, Kivekas, N, Kulmala, M, Lihavainen, H and Tunved, P (2014). The direct and indirect radiative effects of biogenic secondary organic aerosol. Atmos Chem Phys 14(1): 447–470.

Shi, L, Zanobetti, A, Kloog, I, Coull, BA, Koutrakis, P, Melly, SJ and Schwartz, JD (2016). Low-concentration $PM_{2.5}$ and mortality: Estimating acute and chronic effects in a population-based study. Environ Health Perspect 124(1): 46–52.

Shindell, DT, Lamarque, JF, Schulz, M, Flanner, M, Jiao, C, Chin, M, Young, PJ, Lee, YH, Rotstayn, L, Mahowald, N, Milly, G, Faluvegi, G, Balkanski, Y, Collins, WJ, Conley, AJ, Dalsoren, S, Easter, R, Ghan, S, Horowitz, L, Liu, X, Myhre, G, Nagashima, T, Naik, V, Rumbold, ST, Skeie, R, Sudo, K, Szopa, S, Takemura, T, Voulgarakis, A, Yoon, JH and Lo, F (2013). Radiative forcing in the ACCMIP historical and future climate simulations. Atmos Chem Phys 13(6): 2939–2974.

Sleiman, M, Kirchstetter, TW, Berdahl, P, Gilbert, HE, Quelen, S, Marlot, L, Preble, CV, Chen, S, Montalbano, A, Rosseler, O, Akbari, H, Levinson, R and Destaillats, H (2014). Soiling of building envelope surfaces and its effect on solar reflectance—Part II: Development of an accelerated aging method for roofing materials. Sol Energy Mater Sol Cells 122: 271–281.

Smith, AE and Howell, S (2009). An assessment of the robustness of visual air quality preference study results. Washington, DC, CRA International.

Tai, APK, Mickley, LJ and Jacob, DJ (2010). Correlations between fine particulate matter ($PM_{2.5}$) and meteorological variables in the United States: Implications for the sensitivity of $PM_{2.5}$ to climate change. Atmos Environ 44(32): 3976–3984.

Takemura, T (2012). Distributions and climate effects of atmospheric aerosols from the preindustrial era to 2100 along Representative Concentration Pathways (RCPs) simulated using the global aerosol model SPRINTARS. Atmos Chem Phys 12(23): 11555–11572.

Twomey, S (1977). The influence of pollution on the shortwave albedo of clouds. Journal of the Atmospheric Sciences 34(7): 1149–1152.

U.S. EPA. (2004). Air Quality Criteria for Particulate Matter. (Vol I and II). Research Triangle Park, NC. Office of Research and Development. U.S. EPA. EPA–600/P–99–002aF and EPA–600/P–99–002bF. October 2004. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100LFIQ.txt.*

U.S. EPA. (2005). Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Research Triangle Park, NC. Office of Air Quality Planning and Standards. U.S. EPA. EPA–452/R–05–005a. December 2005. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1009MZM.txt.*

U.S. EPA. (2008). Integrated Review Plan for the National Ambient Air Quality Standards for Particulate Matter Research Triangle Park, NC. Office of Research and Development, National Center for Environmental Assessment; Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA 452/R–08–004. March 2008. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1001FB9.txt.*

U.S. EPA. (2009a). Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment Research Triangle Park, NC. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA–452/P–09–002. February 2009. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FLWP.txt.*

U.S. EPA. (2009b). Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Urban Visibility Impact Assessment Research Triangle Park, NC. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA–452/P–09–001. February 2009. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FLUX.txt.*

U.S. EPA. (2009c). Integrated Science Assessment for Particulate Matter (Final Report). Research Triangle Park, NC. Office of Research and Development, National Center for Environmental Assessment. U.S. EPA. EPA–600/R–08–139F. December 2009. Available at: *https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546.*

U.S. EPA. (2010a). Quantitative Health Risk Assessment for Particulate Matter (Final Report). Research Triangle Park, NC. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA–452/R–10–005. June 2010. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1007RFC.txt.*

U.S. EPA. (2010b). Particulate Matter Urban-Focused Visibility Assessment (Final Document). Research Triangle Park, NC. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA–452/R–10–004 July 2010. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FO5D.txt.*

U.S. EPA. (2011). Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards Research Triangle Park, NC. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division. U.S. EPA. EPA–452/R–11–003 April 2011. Available at: *https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100AUMY.txt.*

U.S. EPA. (2012a). Responses to Significant Comments on the 2012 Proposed Rule on the National Ambient Air Quality Standards for Particulate Matter (June 29, 2012; 77 FR 38890). Research Triangle Park, NC. U.S. EPA. Docket ID No. EPA–HQ–OAR–2007–0492. Available at: *https://www3.epa.gov/ttn/naaqs/standards/pm/data/20121214rtc.pdf.*

U.S. EPA. (2012b). Report to Congress on Black Carbon. Washington, DC. U.S. Environmental Protection Agency, Office of Air and Radition. U.S. EPA. EPA–450/R–12–001. March 2012. Availble at: *http://www.epa.gov/blackcarbon/2012report/fullreport.pdf.*

U.S. EPA. (2015). Preamble to the integrated science assessments. Research Triangle Park, NC. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, RTP Division. U.S. EPA. EPA/600/R–15/067. November 2015. Available at: *https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=310244.*

U.S. EPA. (2016). Integrated review plan for the national ambient air quality standards for particulate matter. Research Triangle Park, NC. Office of Air Quality Planning and Standards. U.S. EPA. EPA–452/R–16–005. December 2016. Available at: *https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf.*

U.S. EPA. (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report). Washington, DC. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. U.S. EPA. EPA/600/R–19/188. December 2019. Available at: *https://www.epa.gov/*

naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review.

U.S. EPA. (2020). Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter. Research Triangle Park, NC. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Heath and Environmental Impacts Division. U.S. EPA. EPA–452/R–20–002. January 2020. Available at: *https://www.epa.gov/naaqs/particulate-matter-pm-standards-policy-assessments-current-review-0.*

U.S. National Institutes of Health. (2013). NHLBI fact book, fiscal year 2012: Disease statistics. Bethesda, MD. U.S. National Institutes of Health, National Heart, Lung, and Blood Institute. U.S. National Institutes of Health, NH, Lung, and Blood Institute,. February 2013. Available at: *https://www.nhlbi.nih.gov/files/docs/factbook/FactBook2012.pdf.*

Van de Hulst, H (1981). *Light scattering by small particles.* New York, Dover Publications, Inc.

Vu, TV, Delgado-Saborit, JM and Harrison, RM (2015). Review: Particle number size distributions from seven major sources and implications for source apportionment studies. Atmos Environ 122: 114–132.

Walwil, HM, Mukhaimer, A, Al-Sulaiman, FA and Said, SAM (2017). Comparative studies of encapsulation and glass surface modification impacts on PV performance in a desert climate. Solar Energy 142: 288–298.

Wang, Y, Hopke, PK, Chalupa, DC and Utell, MJ (2011). Long-term study of urban ultrafine particles and other pollutants. Atmos Environ 45(40): 7672–7680.

Watt, J, Jarrett, D and Hamilton, R (2008). Dose-response functions for the soiling of heritage materials due to air pollution exposure. Sci Total Environ 400(1–3): 415–424.

Whitby, KT, Husar, RB and Liu, BYH (1972). The aerosol size distribution of Los Angeles smog. J Colloid Interface Sci 39: 177–204.

Yorifuji, T, Kashima, S and Doi, H (2016). Fine-particulate air pollution from diesel emission control and mortality rates in Tokyo: A quasi-experimental study. Epidemiology 27(6): 769–778.

Zelinka, MD, Andrews, T, Forster, PM and Taylor, KE (2014). Quantifying components of aerosol-cloud-radiation interactions in climate models. Journal of Geophysical Research: Atmospheres 119(12): 7599–7615.

List of Subjects in 40 CFR Part 50

Environmental protection, Air pollution control, Carbon monoxide, Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

Andrew Wheeler,

Administrator.

[FR Doc. 2020–08143 Filed 4–29–20; 8:45 am]

BILLING CODE 6560–50–P