



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

EPA-452/P-21-001
October 2021

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

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

DISCLAIMER

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

TABLE OF CONTENTS

1	Introduction.....	1-1
1.1	Purpose.....	1-2
1.2	Legislative Requirements.....	1-3
1.3	History of Reviews of the PM NAAQS.....	1-6
1.3.1	Reviews Completed in 1971 and 1987.....	1-8
1.3.2	Review Completed in 1997.....	1-8
1.3.3	Review Completed in 2006.....	1-10
1.3.4	Review Completed in 2012.....	1-12
1.3.5	Review Completed in 2020.....	1-12
1.4	Reconsideration of the 2020 PM NAAQS Final Action.....	1-14
1.4.1	Decision to Initiate a Reconsideration	1-14
1.4.2	Process for Reconsideration of the 2020 PM NAAQS Decision.....	1-15
1.4.3	Ongoing Litigation.....	1-18
	References	1-19
2	PM Air Quality	2-1
2.1	Distribution of Particle Size in Ambient Air	2-1
2.1.1	Sources of PM Emissions	2-3
2.2	Ambient PM Monitoring Methods and Networks	2-14
2.2.1	Total Suspended Particulates (TSP) Sampling	2-15
2.2.2	PM ₁₀ Monitoring.....	2-16
2.2.3	PM _{2.5} Monitoring	2-17
2.2.4	PM _{10-2.5} Monitoring.....	2-22
2.2.5	Additional PM Measurements and Metrics	2-23
2.3	Ambient Air Concentrations.....	2-25
2.3.1	Trends in Emissions of PM and Precursor Gases	2-25
2.3.2	Trends in Monitored Ambient Concentrations	2-26
2.3.3	Predicted Ambient PM _{2.5} Based on Hybrid Modeling Approaches.....	2-43
2.4	Background PM	2-53
2.4.1	Natural Sources	2-55
2.4.2	International Transport.....	2-57
2.4.3	Estimating Background PM with Recent Data	2-59

	References	2-62
3	Reconsideration of the Primary Standards for PM _{2.5}	3-1
3.1	Background on the Current Standards	3-2
3.1.1	Considerations Regarding the Adequacy of the Existing Standards in the 2020 Review.....	3-4
3.2	General Approach and Key Issues in this Reconsideration of the 2020 Final Decision	3-12
3.3	Health Effects Evidence.....	3-15
3.3.1	Nature of Effects	3-18
3.3.2	Public Health Implications and At-Risk Populations.....	3-50
3.3.3	PM _{2.5} Concentrations in Key Studies Reporting Health Effects	3-57
3.3.4	Uncertainties in the Health Effects Evidence.....	3-125
3.4	Risk Information	3-128
3.4.1	Risk Assessment Overview.....	3-129
3.4.2	Results of the Risk Assessment	3-138
3.4.3	Conclusions of the Risk Assessment.....	3-156
3.5	Key Considerations Regarding the Adequacy of the Primary PM _{2.5} Standards	3-157
3.5.1	Evidence-based Considerations	3-157
3.5.2	Risk-based Considerations	3-171
3.5.3	Preliminary Conclusions	3-181
3.6	Areas for Future Research and Data Collection	3-203
	References	3-206
4	Reconsideration of the Primary Standard for PM ₁₀	4-1
4.1	Background on the Current Standard.....	4-2
4.1.1	Considerations Regarding the Adequacy of the Existing Standards in the 2020 Review.....	4-4
4.2	General Approach and Key Issues in this Reconsideration of the 2020 Final Decision	4-5
4.3	Health Effects Evidence.....	4-8
4.3.1	Nature of Effects	4-9
4.4	Preliminary Conclusions on the Adequacy of the Current Primary PM ₁₀ Standard	4-16
4.5	Areas for Future Research and Data Collection	4-18
	References	4-20
5	Reconsideration of the Secondary Standards for PM	5-1

5.1	Background on the Current Standards	5-2
5.1.1	Non-Visibility Effects	5-2
5.1.2	Visibility Effects	5-5
5.2	General Approach and Key Issues in this Reconsideration of the 2020 Final Decision	5-11
5.3	Welfare Effects and Quantitative Information.....	5-14
5.3.1	Visibility Effects	5-16
5.3.2	Non-Visibility Effects	5-33
5.4	Preliminary Conclusions Regarding the Adequacy of the Secondary PM Standards	5-45
5.5	Areas for Future Research and Data Collection	5-49
	References	5-51

LIST OF APPENDICES

Appendix A. Supplemental Information on PM Air Quality Analyses

Appendix B. Supplemental Study Information: Selection Criteria, Study Methods and Details

Appendix C. Supplemental Information Related to the Human Health Risk Assessment

Appendix D. Quantitative Analyses for Visibility Impairment

LIST OF TABLES

Table 1-1. Summary of NAAQS promulgated for particulate matter 1971-2012.	1-7
Table 2-1. Percent Changes in PM and PM precursor emissions in the NEI for the time periods 1990-2017 and 2002-2017.....	2-26
Table 2-2. Nationwide averages of ratios of maximum annual PM _{2.5} design values to average composite monitor PM _{2.5} concentrations across CBSAs.	2-45
Table 2-3. Mean 2011 PM _{2.5} concentration by region for predictions in Figure 2-29.....	2-52
Table 2-4. Average Annual PM _{2.5} Concentration (µg/m ³) by Year.....	2-58
Table 2-5. Three-Year Average of the Average Annual PM _{2.5} Concentrations (µg/m ³).....	2-59
Table 2-6. Average Annual PM _{2.5} Concentrations and Ratios to Regulatory Design Values.	2-60
Table 3-1. Key causality determinations for PM _{2.5} and UFP exposures.....	3-19
Table 3-2. National demographic information, 2019.....	3-53
Table 3-3. Prevalence of cardiovascular and respiratory diseases among adults by age, race, and ethnicity in the U.S. in 2018.	3-56
Table 3-4. Summary of information from PM _{2.5} controlled human exposure studies.	3-59
Table 3-5. Key U.S. Epidemiologic Studies: Monitor-Based Exposure.....	3-82
Table 3-6. Key Canadian Epidemiologic Studies: Monitor-Based Exposure.....	3-86
Table 3-7. Key U.S. Epidemiologic Studies: Model-Based Exposure	3-89
Table 3-8. Key Canadian Epidemiologic Studies: Model-Based Exposure	3-95
Table 3-9. PM _{2.5} Concentrations Metrics from Monitor and Modeled Data	13-00
Table 3-10. Epidemiologic studies examining the health impacts associated with ambient PM _{2.5} concentrations when studies are conducted with restricted air quality exposures.	3-115
Table 3-11. Summary of information from studies that use causal modeling statistical methods.	3-119
Table 3-12. Epidemiologic studies examining the health impacts of long-term reductions in ambient PM _{2.5} concentrations.....	3-122
Table 3-13. Epidemiologic studies used to estimate PM _{2.5} -associated risk.....	3-130
Table 3-14. Estimates of PM _{2.5} -associated mortality for air quality adjusted to just meet the current or alternative standards (47 urban study areas).....	3-140
Table 3-15. Estimated reduction in PM _{2.5} -associated mortality for alternative annual and 24-hour standards (47 urban study areas).....	3-141

Table 3-16. Estimates of PM _{2.5} -associated mortality for the current and potential alternative annual standards in the 30 study areas where the annual standard is controlling	3-143
Table 3-17. Estimated delta and percent reduction in PM _{2.5} -associated mortality for the current and potential alternative annual standards in the 30 study areas where the annual standard is controlling.....	3-144
Table 3-18. Estimates of PM _{2.5} -associated mortality for the current 24-hour standard, and an alternative, in the 11 study areas where the 24-hour standard is controlling.	3-147
Table 3-19. Average national percent PM _{2.5} reduction in demographic populations aged 65 and over residing in the full set of 47 study areas and subset of 30 study areas controlled by the annual standard.....	3-151
Table 3-20. Average national percent PM _{2.5} risk reduction in demographic populations aged 65 and over residing in the full set of 47 study areas and subset of 30 study areas controlled by the annual standard.	3-151
Table 4-1. Key Causality Determinations for PM _{10-2.5} Exposures.....	4-9
Table 5-1. Key causality determinations for PM-related welfare effects.	5-15

LIST OF FIGURES

Figure 2-1. Comparisons of PM _{2.5} and PM ₁₀ diameters to human hair and beach sand.....	2-2
Figure 2-2. Percent contribution of PM _{2.5} national emissions by source sectors.....	2-5
Figure 2-3. 2017 NEI PM _{2.5} Emissions Density Map, tons per square mile	2-6
Figure 2-4. Percent contribution of PM ₁₀ emissions by national source sectors.	2-7
Figure 2-5. 2017 NEI PM ₁₀ Emissions Density Map, tons per square mile	2-7
Figure 2-6. Percent contribution to organic carbon (top panel) and elemental carbon (bottom panel) national emissions by source sectors.....	2-9
Figure 2-7. 2017 NEI Elemental Carbon Emissions Density Map, tons per square mile....	2-10
Figure 2-8. Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B), ammonia (panel C), and volatile organic compounds (panel D) national emissions by source sectors. (Source: 2017 NEI). All graphics only show anthropogenic contributions.....	2-11
Figure 2-9. SO ₂ Emissions Density Map, tons per square mile.....	2-12
Figure 2-10. NO _x Emissions Density Map, tons per square mile.....	2-12
Figure 2-11. NH ₃ Emissions Density Map, tons per square mile.	2-12
Figure 2-12. Anthropogenic (including wildfires) VOC Emissions Density Map, tons per square mile.	2-13
Figure 2-13. PM Monitoring stations reporting to EPA’s AQS database by PM size fraction, 1970-2020.....	2-15
Figure 2-14. National emission trends of PM _{2.5} , PM ₁₀ , and precursor gases from 1990 to 2017.	2-26
Figure 2-15. Annual average and 98 th percentile of 24-hour PM _{2.5} concentrations (in µg/m ³) from 2017-2019 (top) and linear trends and their associated significance (based on p-values) in PM _{2.5} concentrations from 2000-2019 (bottom).	2-28
Figure 2-16. Seasonally-weighted annual average PM _{2.5} concentrations in the U.S. from 2000 to 2019 (406 sites).	2-29
Figure 2-17. Pearson’s correlation coefficient between annual average and 98 th percentile of 24-hour PM _{2.5} concentrations from 2000-2019.....	2-30
Figure 2-18. Scatterplot of CBSA maximum annual versus daily design values (2017-2019) with the solid black line representing the ratio of daily and annual NAAQS values.....	2-31
Figure 2-19. Frequency distribution of 2017-2019 2-hour averages for sites meeting both or violating either PM _{2.5} NAAQS for October to March (blue) and April to September (red)	2-33

Figure 2-20. Annual average PM _{2.5} sulfate, nitrate, organic carbon, and elemental carbon concentrations (in $\mu\text{g}/\text{m}^3$) from 2017-2019.....	2-34
Figure 2-21. Annual average and 2 nd highest PM ₁₀ concentrations (in $\mu\text{g}/\text{m}^3$) from 2017-2019 (top) and linear trends and their associated significance in PM ₁₀ concentrations from 2000-2019 (bottom).....	2-36
Figure 2-22. National trends in Annual 2 nd Highest 24-Hour PM ₁₀ concentrations from 2000 to 2019 (262 sites).	2-37
Figure 2-23. Annual average PM _{2.5} /PM ₁₀ ratio for 2017-2019.....	2-38
Figure 2-24. PM _{2.5} /PM ₁₀ ratio on the date of the second highest PM ₁₀ concentrations for 2017-2019.....	2-38
Figure 2-25. Annual average and 98 th percentile PM _{10-2.5} concentrations ($\mu\text{g}/\text{m}^3$) from 2017-2019 (top) and linear trends and their associated significance in PM _{10-2.5} concentrations from 2000-2019 (bottom).	2-40
Figure 2-26. Average hourly particle number concentrations from three locations in the State of New York for 2014 to 2015..	2-41
Figure 2-27. Time series of annual average mass and number concentrations (left) and scatterplot of mass vs. number concentration (right) between 2000-2019 in Bondville, IL. 2-42	
Figure 2-28. Comparison of CBSA average annual design values and CBSA maximum annual design values for 2017-2019.....	2-44
Figure 2-29. Comparison of CBSA average annual design values and CBSA maximum daily design values for 2017-2019.	2-46
Figure 2-30. R ² for ten-fold cross-validation of daily PM _{2.5} predictions in 2015 from three methods for individual sites as a function of observed concentration.	2-50
Figure 2-31. Comparison of 2011 annual average PM _{2.5} concentrations from four methods. ... 2-51	
Figure 2-32. Comparison of 2011 annual average PM _{2.5} concentrations from four methods for regions centered on the (a) California (b) New Jersey, and (c) Arizona.....	2-53
Figure 2-33. (a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in percentage units for the four models in Figure 2-29. (b) Boxplot distributions of CV for grid cells binned by the average PM _{2.5} concentration for the four models.	2-54
Figure 2-34. Distance from the center of the 12-km grid cells to the nearest PM _{2.5} monitoring site for PM _{2.5} measurements from the AQS database and IMPROVE network.	2-54
Figure 2-35. Location of PM _{2.5} predictions by range in annual average concentration for the four prediction methods at their native resolution.	2-55

Figure 2-36. Annual mean PM _{2.5} from the VD2019 method (van Donkelaar et al., 2019) for 2001, 2006, 2011, and 2016.	2-56
Figure 2-37. Spatial distribution of the annual average PM _{2.5} concentrations for 2015 using the DI2019 surface nationwide (panel A) and for CBSAs only (panel B).....	2-57
Figure 2-38. Smoke and fire detections observed by the MODIS instrument onboard the Aqua satellite on August 4 th , 2017 accessed through NASA Worldview.	2-65
Figure 2-39. Fine PM mass time series during 2017 from the North Cascades IMPROVE site in north central Washington state.	2-65
Figure 2-40. Speciated annual average IMPROVE PM _{2.5} in µg/m ³ at select remote monitors during 2004 and 2016.	2-69
Figure 2-41. Site locations for the IMPROVE monitors in Figure 2-40.	2-70
Figure 3-1. Overview of general approach for the reconsideration of the 2020 final decision on the primary PM _{2.5} standards.	3-14
Figure 3-2. Estimated concentration-response function and 95% confidence intervals between PM _{2.5} and cardiovascular mortality in the Six Cities Study (1974-2009).	3-66
Figure 3-3. Epidemiologic studies examining associations between long-term PM _{2.5} exposures and mortality.	3-73
Figure 3-4. Epidemiologic studies examining associations between long-term PM _{2.5} exposures and morbidity.	3-74
Figure 3-5. Epidemiologic studies examining associations between short-term PM _{2.5} exposures and mortality.	3-76
Figure 3-6. Epidemiologic studies examining associations between short-term PM _{2.5} exposures and morbidity.	3-78
Figure 3-7. Estimated PM _{2.5} concentrations using the DI2019 hybrid approach and monitoring locations and design values for the state of Georgia and the Atlanta-Sandy Springs-Roswell, Georgia CBSA.	3-99
Figure 3-8. Monitor-based PM _{2.5} concentrations in key U.S. epidemiologic studies.....	3-104
Figure 3-9. Monitor-based PM _{2.5} concentrations in key Canadian epidemiologic studies.	3-105
Figure 3-10. Hybrid model-predicted PM _{2.5} concentrations in key U.S. epidemiologic studies.....	3-106
Figure 3-11. Hybrid model-predicted PM _{2.5} concentrations in key Canadian epidemiologic studies.....	3-107
Figure 3-12. Hybrid model-predicted PM _{2.5} concentrations in key U.S. epidemiologic studies, subset by spatial scale.	3-109
Figure 3-13. Hybrid model-predicted PM _{2.5} concentrations in key U.S. epidemiologic studies, subset by method used to average grid cells in study-reported long-term mean PM _{2.5} concentrations.	3-110

Figure 3-14. Hybrid model-predicted PM _{2.5} concentrations in key U.S. epidemiologic studies, subset by spatial scale and method used to average grid cells in study-reported long-term mean PM _{2.5} concentrations.	3-111
Figure 3-15. Illustration of approach to adjusting air quality to simulate just meeting annual standards with levels of 11.0, 9.0, and 8.0 µg/m ³	3-131
Figure 3-16. Map of 47 urban study areas included in risk modeling.	3-134
Figure 3-17. Available epidemiologic associations between long-term PM _{2.5} exposure and mortality outcomes in demographic populations.	3-136
Figure 3-18. Distribution of total risk estimates (PM _{2.5} -attributable mortality) for the current and alternative annual standards for the subset of 30 urban study areas where the annual standard is controlling (blue and green bars represent the Pri-PM _{2.5} and Sec-PM _{2.5} estimates, respectively).	3-145
Figure 3-19. Distribution of the difference in risk estimates between the current annual standard (level of 12.0 µg/m ³) and alternative annual standards with levels of 11.0, 10.0, 9.0, and 8.0 µg/m ³ for the subset of 30 urban study areas where the annual standard is controlling.	3-146
Figure 3-20. Average PM _{2.5} exposure concentration and PM _{2.5} -attributable risk estimates by demographic population when just meeting current or alternative PM _{2.5} standards.	3-149
Figure 3-21. Average change in PM _{2.5} exposure concentration and PM _{2.5} -attributable mortality risk estimates by demographic population when moving from the current to alternative PM _{2.5} standards.	3-150
Figure 3-22. PM _{2.5} exposure concentrations and PM _{2.5} -attributable mortality risk estimates by demographic population when just meeting current or alternative PM _{2.5} standards.	3-152
Figure 3-23. Change in PM _{2.5} exposure concentrations and PM _{2.5} -attributable mortality risk estimates by demographic population when moving from the current to alternative PM _{2.5} standards.	3-153
Figure 5-1. Overview of general approach for the reconsideration of the 2020 final decision on the secondary PM standards.	5-13
Figure 5-2. Relationship of viewer acceptability ratings to light extinction.	5-24
Figure 5-3. Comparison of 90 th percentile of daily light extinction, averaged over three years, and 98 th percentile of daily PM _{2.5} concentrations, averaged over three years, for 2017-2019 using the original IMPROVE equation.	5-29
Figure 5-4. Comparison of 90 th percentile of daily light extinction, averaged over three years, and 98 th percentile of daily PM _{2.5} concentrations, averaged over three years, for 2015-2017 using the Lowenthal and Kumar equation.	5-31

1 INTRODUCTION

2 This document, *Policy Assessment for the Reconsideration of the National Ambient Air*
3 *Quality Standards for Particulate Matter, External Review Draft* (hereafter referred to as the
4 draft PA), presents the draft policy assessment for the U.S. Environmental Protection Agency’s
5 (EPA’s) reconsideration of the review of the national ambient air quality standards (NAAQS) for
6 particulate matter (PM) completed in 2020.¹ The overall plan for the 2020 review was presented
7 in the *Integrated Review Plan for the National Ambient Air Quality Standards for Particulate*
8 *Matter* (IRP; U.S. EPA, 2016). The IRP also identified key policy-relevant issues to be addressed
9 in the 2020 review and discussed the key documents that generally inform NAAQS reviews,
10 including an Integrated Science Assessment (ISA) and a Policy Assessment (PA). The key
11 considerations presented in this draft PA are intended to provide updates to the policy
12 information to support the reconsideration of the 2020 PM NAAQS final action, which retained
13 the primary and secondary PM_{2.5} and PM₁₀ standards without revision (85 FR 82684, December
14 18, 2020). In reconsidering the 2020 final action, the EPA will consider the scientific and
15 technical analyses on which the December 2020 PM NAAQS final action was based, as well as
16 the newly available scientific information evaluated in the *Supplement to the 2019 Integrated*
17 *Science Assessment for Particulate Matter (External Review Draft)* (hereafter referred to as the
18 draft ISA Supplement; U.S. EPA, 2021) and the policy implications of the new scientific
19 evidence and updated quantitative analyses presented in this draft PA. Much of the information
20 in this draft PA is drawn directly from information included in the 2019 ISA (U.S. EPA, 2019)
21 and the 2020 PA (U.S. EPA, 2020).

22 This document is organized into five chapters. Chapter 1 presents introductory
23 information on the purpose of the PA, legislative requirements for reviews of the NAAQS, an
24 overview of the history of the PM NAAQS, including background information on prior reviews,
25 and a summary of the progress to date for the reconsideration of the 2020 final decision. Chapter
26 2 provides an overview of the available information on PM-related emissions, atmospheric
27 chemistry, monitoring and air quality. Chapter 3 focuses on policy-relevant aspects of the
28 currently available health effects evidence as presented in the 2019 ISA and draft ISA
29 Supplement, as well as updated exposure/risk information, and identifies and summarizes the key
30 considerations related to this reconsideration of the primary PM_{2.5} standards. Chapter 4 draws
31 substantially from the information presented in the 2020 PA on the policy-relevant aspects of the

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

1 health effects evidence presented in the 2019 ISA and identifies and summarizes the key
2 considerations related to this reconsideration of the primary standard PM₁₀. Chapter 5 focuses on
3 policy-relevant aspects of the currently available welfare effects evidence as presented in the
4 2019 ISA and draft ISA Supplement, as well as updated quantitative analyses for visibility
5 effects, and identifies and summarizes the key considerations related to this reconsideration of
6 the secondary PM standards.² More detail about the process for this reconsideration is described
7 in section 1.4.2 below, and the approach for considering the available information for this
8 reconsideration is presented within Chapters 3, 4, and 5 of this draft PA.

9 **1.1 PURPOSE**

10 The PA evaluates the potential policy implications of the available scientific evidence, as
11 assessed in the ISA, and the potential implications of the available air quality, exposure or risk
12 analyses. The role of the PA is to help “bridge the gap” between the Agency’s scientific
13 assessments and quantitative technical analyses, and the judgments required of the Administrator
14 in determining whether it is appropriate to retain or revise the NAAQS.

15 In evaluating the question of adequacy of the current standards, and whether it may be
16 appropriate to consider alternative standards, the PA focuses on information that is most
17 pertinent to evaluating the standards and their basic elements: indicator, averaging time, form,
18 and level.³ These elements, which together serve to define each standard, must be considered
19 collectively in evaluating the health and welfare protection the standards afford.

20 The PA is also intended to facilitate advice to the Agency and recommendations to the
21 Administrator from an independent scientific review committee, the Clean Air Scientific
22 Advisory Committee (CASAC), as provided for in the Clean Air Act (CAA). As discussed below
23 in section 1.2, the CASAC is to advise on subjects including the Agency’s assessment of the
24 relevant scientific information and on the adequacy of the current standards, and to make

² The welfare effects considered in this review include visibility impairment, climate effects, and materials effects (i.e., damage and soiling). Ecological effects associated with PM, and the adequacy of protection provided by the secondary PM standards for them, are being addressed in the separate review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM in recognition of the linkages between oxides of nitrogen, oxides of sulfur, and PM with respect to atmospheric chemistry and deposition, and with respect to ecological effects. Information on the current review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM can be found at <https://www.epa.gov/naaqs/nitrogen-dioxide-no2-and-sulfur-dioxide-so2-secondary-air-quality-standards>.

³ The indicator defines the chemical species or mixture to be measured in the ambient air for the purpose of determining whether an area attains the standard. The averaging time defines the period over which air quality measurements are to be averaged or otherwise analyzed. The form of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. For example, the form of the annual NAAQS for fine particulate matter is the average of annual mean concentrations for three consecutive years, while the form of the 8-hour NAAQS for carbon monoxide is the second-highest 8-hour average in a year. The level of the standard defines the air quality concentration used for that purpose.

1 recommendations as to any revisions of the standards that may be appropriate. The EPA
2 generally makes available to the CASAC and the public one or more drafts of the PA for
3 CASAC review and public comment.

4 In this draft PA, we⁴ take into account the available scientific evidence, as assessed in the
5 *Integrated Science Assessment for Particulate Matter (Final Report)* (2019 ISA [U.S. EPA,
6 2019]) and in the draft ISA Supplement (U.S. EPA, 2021), as well as additional policy-relevant
7 analyses of air quality and risks. The evaluation and preliminary conclusions presented in this
8 draft PA have been informed by the scientific evidence presented in the 2019 ISA and the draft
9 ISA Supplement, as well as the policy-relevant considerations and conclusions reached in the
10 2020 PA, along with updated quantitative analyses of air quality, risk, and exposure, where
11 available. Review and comments from the CASAC, as well as public comment, on this draft PA
12 will inform the final evaluation and conclusions in the final PA. The final PA is intended to help
13 the Administrator in considering the scientific and technical information, and in formulating
14 judgments regarding the adequacy of the current standards and regarding alternative standards,
15 as appropriate.

16 Beyond informing the Administrator and facilitating the advice and recommendations of
17 the CASAC, the PA is also intended to be a useful reference to all parties interested in the review
18 of the PM NAAQS. In these roles, it is intended to serve as a source of policy-relevant
19 information that informs the Agency’s review of the NAAQS for PM, and it is written to be
20 understandable to a broad audience.

21 **1.2 LEGISLATIVE REQUIREMENTS**

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

⁴ The terms “we,” “our,” and “staff” throughout this document refer to the staff in the EPA’s Office of Air Quality Planning and Standards (OAQPS).

1 Section 109 [42 U.S.C. 7409] directs the Administrator to propose and promulgate
2 “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued [42
3 U.S.C. § 7409(a)]. Section 109(b)(1) defines primary standards as ones “the attainment and
4 maintenance of which in the judgment of the Administrator, based on such criteria and allowing
5 an adequate margin of safety, are requisite to protect the public health.”⁵ Under section
6 109(b)(2), a secondary standard must “specify a level of air quality the attainment and
7 maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite
8 to protect the public welfare from any known or anticipated adverse effects associated with the
9 presence of [the] pollutant in the ambient air.”⁶

10 In setting primary and secondary standards that are “requisite” to protect public health
11 and welfare, respectively, as provided in section 109(b), the EPA’s task is to establish standards
12 that are neither more nor less stringent than necessary. In so doing, the EPA may not consider the
13 costs of implementing the standards. See generally, *Whitman v. American Trucking Associations*,
14 531 U.S. 457, 465-472, 475-76 (2001). Likewise, “[a]ttainability and technological feasibility are
15 not relevant considerations in the promulgation of national ambient air quality standards.”
16 *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1185 (D.C. Cir. 1981). At the same time,
17 courts have clarified the EPA may consider “relative proximity to peak background ...
18 concentrations” as a factor in deciding how to revise the NAAQS in the context of considering
19 standard levels within the range of reasonable values supported by the air quality criteria and
20 judgments of the Administrator. *American Trucking Associations, Inc. v. EPA*, 283 F.3d 355, 379
21 (D.C. Cir. 2002).

22 The requirement that primary standards provide an adequate margin of safety was
23 intended to address uncertainties associated with inconclusive scientific and technical
24 information available at the time of standard setting. It was also intended to provide a reasonable
25 degree of protection against hazards that research has not yet identified. See *Lead Industries*
26 *Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980);
27 *American Petroleum Institute v. Costle*, 665 F.2d at 1186 (D.C. Cir. 1981), *cert. denied*, 455 U.S.
28 1034 (1982); *Coalition of Battery Recyclers Ass’n v. EPA*, 604 F.3d 613, 617-18 (D.C. Cir.
29 2010); *Mississippi v. EPA*, 744 F.3d 1334, 1353 (D.C. Cir. 2013). Both kinds of uncertainties are

⁵ The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

⁶ Under CAA section 302(h) (42 U.S.C. § 7602(h)), effects on welfare include, but are not limited to, “effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”

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

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

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

21 Section 109(d)(2) addresses the appointment and advisory functions of an independent
22 scientific review committee. Section 109(d)(2)(A) requires the Administrator to appoint this
23 committee, which is to be composed of “seven members including at least one member of the
24 National Academy of Sciences, one physician, and one person representing State air pollution
25 control agencies.” Section 109(d)(2)(B) provides that the independent scientific review
26 committee “shall complete a review of the criteria...and the national primary and secondary
27 ambient air quality standards...and shall recommend to the Administrator any new...standards
28 and revisions of existing criteria and standards as may be appropriate....” Since the early 1980s,
29 this independent review function has been performed by the Clean Air Scientific Advisory
30 Committee (CASAC) of the EPA’s Science Advisory Board. A number of other advisory
31 functions are also identified for the committee by section 109(d)(2)(C), which reads:

32 Such committee shall also (i) advise the Administrator of areas in which
33 additional knowledge is required to appraise the adequacy and basis of existing,
34 new, or revised national ambient air quality standards, (ii) describe the research

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

1 efforts necessary to provide the required information, (iii) advise the
2 Administrator on the relative contribution to air pollution concentrations of
3 natural as well as anthropogenic activity, and (iv) advise the Administrator of any
4 adverse public health, welfare, social, economic, or energy effects which may
5 result from various strategies for attainment and maintenance of such national
6 ambient air quality standards.

7 As previously noted, the Supreme Court has held that section 109(b) “unambiguously bars cost
8 considerations from the NAAQS-setting process” (*Whitman v. Am. Trucking Associations*, 531
9 U.S. 457, 471 [2001]). Accordingly, while some of these issues regarding which Congress has
10 directed the CASAC to advise the Administrator are ones that are relevant to the standard setting
11 process, others are not. Issues that are not relevant to standard setting may be relevant to
12 implementation of the NAAQS once they are established.⁸

13 **1.3 HISTORY OF REVIEWS OF THE PM NAAQS**

14 This section summarizes the PM NAAQS that have been promulgated in past reviews
15 (Table 1-1). Each of these reviews is discussed briefly below.
16

⁸ Some aspects of CASAC advice may not be relevant to EPA’s process of setting primary and secondary standards that are requisite to protect public health and welfare. Indeed, were EPA to consider costs of implementation when reviewing and revising the standards “it would be grounds for vacating the NAAQS.” *Whitman*, 531 U.S. at 471 n.4. At the same time, the Clean Air Act directs CASAC to provide advice on “any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance” of the NAAQS to the Administrator under section 109(d)(2)(C)(iv). In *Whitman*, the Court clarified that most of that advice would be relevant to implementation but not standard setting, as it “enable[s] the Administrator to assist the States in carrying out their statutory role as primary *implementers* of the NAAQS.” *Id.* at 470 (emphasis in original). However, the Court also noted that CASAC’s “advice concerning certain aspects of ‘adverse public health ... effects’ from various attainment strategies is unquestionably pertinent” to the NAAQS rulemaking record and relevant to the standard setting process. *Id.* at 470 n.2.

1 **Table 1-1. Summary of NAAQS promulgated for particulate matter 1971-2012.**

Review Completed	Indicator	Averaging Time	Level	Form
1971	Total Suspended Particles (TSP)	24-hour	260 µg/m ³ (primary) 150 µg/m ³ (secondary)	Not to be exceeded more than once per year
		Annual	75 µg/m ³ (primary) 60 µg/m ³ (secondary)	Annual geometric mean
1987	PM ₁₀	24-hour	150 µg/m ³	Not to be exceeded more than once per year on average over a 3-year period
		Annual	50 µg/m ³	Annual arithmetic mean, averaged over 3 years
1997	PM _{2.5}	24-hour	65 µg/m ³	98 th percentile, averaged over 3 years
		Annual	15.0 µg/m ³	Annual arithmetic mean, averaged over 3 years ^a
	PM ₁₀	24-hour	150 µg/m ³	99 th percentile, averaged over 3 years ^b
		Annual	50 µg/m ³	Annual arithmetic mean, averaged over 3 years
2006	PM _{2.5}	24-hour	35 µg/m ³	98 th percentile, averaged over 3 years
		Annual	15.0 µg/m ³	Annual arithmetic mean, averaged over 3 years ^c
	PM ₁₀	24-hour ^d	150 µg/m ³	Not to be exceed more than once per year on average over a 3-year period
2012	PM _{2.5}	24-hour	35 µg/m ³	98 th percentile, averaged over 3 years
		Annual	12.0 µg/m ³ (primary) 15.0 µg/m ³ (secondary)	Annual mean, averaged over 3 years ^e
	PM ₁₀	24-hour	150 µg/m ³	Not to be exceeded more than once per year on average over 3 years

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

^a The level of the 1997 annual PM_{2.5} standard was to be compared to measurements made at the community-oriented monitoring site recording the highest concentration or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged (i.e., “spatial averaging”) (62 FR 38652, July 18, 1997).

^b When the 1997 standards were vacated (see below), the form of the 1987 standards remained in place (i.e., not to be exceeded more than once per year on average over a 3-year period).

^c The EPA tightened the constraints on the spatial averaging criteria by further limiting the conditions under which some areas may average measurements from multiple community-oriented monitors to determine compliance (71 FR 61144, October 17, 2006).

^d The EPA revoked the annual PM₁₀ NAAQS in 2006 (71 FR 61144, October 17, 2006).

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

1 **1.3.1 Reviews Completed in 1971 and 1987**

2 The EPA first established NAAQS for PM in 1971 (36 FR 8186, April 30, 1971), based
3 on the original Air Quality Criteria Document (AQCD) (DHEW, 1969).⁹ The federal reference
4 method (FRM) specified for determining attainment of the original standards was the high-
5 volume sampler, which collects PM up to a nominal size of 25 to 45 micrometers (μm) (referred
6 to as total suspended particulates or TSP). The primary standards were set at $260 \mu\text{g}/\text{m}^3$, 24-hour
7 average, not to be exceeded more than once per year, and $75 \mu\text{g}/\text{m}^3$, annual geometric mean. The
8 secondary standards were set at $150 \mu\text{g}/\text{m}^3$, 24-hour average, not to be exceeded more than once
9 per year, and $60 \mu\text{g}/\text{m}^3$, annual geometric mean.

10 In October 1979 (44 FR 56730, October 2, 1979), the EPA announced the first periodic
11 review of the air quality criteria and NAAQS for PM. Revised primary and secondary standards
12 were promulgated in 1987 (52 FR 24634, July 1, 1987). In the 1987 decision, the EPA changed
13 the indicator for particles from TSP to PM_{10} , in order to focus on the subset of inhalable particles
14 small enough to penetrate to the thoracic region of the respiratory tract (including the
15 tracheobronchial and alveolar regions), referred to as thoracic particles.¹⁰ The level of the 24-
16 hour standards (primary and secondary) was set at $150 \mu\text{g}/\text{m}^3$, and the form was one expected
17 exceedance per year, on average over three years. The level of the annual standards (primary and
18 secondary) was set at $50 \mu\text{g}/\text{m}^3$, and the form was annual arithmetic mean, averaged over three
19 years.

20 **1.3.2 Review Completed in 1997**

21 In April 1994, the EPA announced its plans for the second periodic review of the air
22 quality criteria and NAAQS for PM, and in 1997 the EPA promulgated revisions to the NAAQS
23 (62 FR 38652, July 18, 1997). In the 1997 decision, the EPA determined that the fine and coarse
24 fractions of PM_{10} should be considered separately. This determination was based on evidence
25 that serious health effects were associated with short- and long-term exposures to fine particles in
26 areas that met the existing PM_{10} standards. The EPA added new standards, using $\text{PM}_{2.5}$ as the
27 indicator for fine particles (with $\text{PM}_{2.5}$ referring to particles with a nominal mean aerodynamic
28 diameter less than or equal to $2.5 \mu\text{m}$). The new primary standards were as follows: (1) an annual
29 standard with a level of $15.0 \mu\text{g}/\text{m}^3$, based on the 3-year average of annual arithmetic mean

⁹ Prior to the review initiated in 2007 (see below), the AQCD provided the scientific foundation (i.e., the air quality criteria) for the NAAQS. Beginning in that review, the ISA has replaced the AQCD.

¹⁰ PM_{10} refers to particles with a nominal mean aerodynamic diameter less than or equal to $10 \mu\text{m}$. More specifically, $10 \mu\text{m}$ is the aerodynamic diameter for which the efficiency of particle collection is 50 percent.

1 PM_{2.5} concentrations from single or multiple community-oriented monitors;¹¹ and (2) a 24-hour
2 standard with a level of 65 µg/m³, based on the 3-year average of the 98th percentile of 24-hour
3 PM_{2.5} concentrations at each monitor within an area. Also, the EPA established a new reference
4 method for the measurement of PM_{2.5} in the ambient air and adopted rules for determining
5 attainment of the new standards. To continue to address the health effects of the coarse fraction
6 of PM₁₀ (referred to as thoracic coarse particles or PM_{10-2.5}; generally including particles with a
7 nominal mean aerodynamic diameter greater than 2.5 µm and less than or equal to 10 µm), the
8 EPA retained the annual primary PM₁₀ standard and revised the form of the 24-hour primary
9 PM₁₀ standard to be based on the 99th percentile of 24-hour PM₁₀ concentrations at each monitor
10 in an area. The EPA revised the secondary standards by setting them equal in all respects to the
11 newly established primary standards.

12 Following promulgation of the 1997 PM NAAQS, petitions for review were filed by
13 several parties, addressing a broad range of issues. In May 1999, the U.S. Court of Appeals for
14 the District of Columbia Circuit (D.C. Circuit) upheld the EPA’s decision to establish fine
15 particle standards, holding that "the growing empirical evidence demonstrating a relationship
16 between fine particle pollution and adverse health effects amply justifies establishment of new
17 fine particle standards." *American Trucking Associations v. EPA*, 175 F. 3d at 1027, 1055-56
18 (D.C. Cir. 1999). The D.C. Circuit also found "ample support" for the EPA's decision to regulate
19 coarse particle pollution, but vacated the 1997 PM₁₀ standards, concluding that the EPA had not
20 provided a reasonable explanation justifying use of PM₁₀ as an indicator for coarse particles.
21 *American Trucking Associations v. EPA*, 175 F. 3d at 1054-55. Pursuant to the D.C. Circuit’s
22 decision, the EPA removed the vacated 1997 PM₁₀ standards, and the pre-existing 1987 PM₁₀
23 standards remained in place (65 FR 80776, December 22, 2000). The D.C. Circuit also upheld
24 the EPA’s determination not to establish more stringent secondary standards for fine particles to
25 address effects on visibility. *American Trucking Associations v. EPA*, 175 F. 3d at 1027.

26 The D.C. Circuit also addressed more general issues related to the NAAQS, including
27 issues related to the consideration of costs in setting NAAQS and the EPA’s approach to
28 establishing the levels of NAAQS. Regarding the cost issue, the court reaffirmed prior rulings
29 holding that in setting NAAQS the EPA is “not permitted to consider the cost of implementing
30 those standards.” *American Trucking Associations v. EPA*, 175 F. 3d at 1040-41. Regarding the
31 levels of NAAQS, the court held that the EPA’s approach to establishing the level of the

¹¹ The 1997 annual PM_{2.5} standard was to be compared with measurements made at the community-oriented monitoring site recording the highest concentration or, if specific constraints were met, measurements from multiple community-oriented monitoring sites could be averaged (i.e., “spatial averaging”). In the 2012 review, the EPA replaced the term “community-oriented” monitor with the term “area-wide” monitor. Area-wide monitors are those sited at the neighborhood scale or larger, as well as those monitors sited at micro- or middle-scales that are representative of many such locations in the same CBSA (78 FR 3236, January 15, 2013).

1 standards in 1997 (i.e., both for PM and for the ozone NAAQS promulgated on the same day)
2 effected “an unconstitutional delegation of legislative authority.” *American Trucking*
3 *Associations v. EPA*, 175 F. 3d at 1034-40. Although the court stated that “the factors EPA uses
4 in determining the degree of public health concern associated with different levels of ozone and
5 PM are reasonable,” it remanded the rule to the EPA, stating that when the EPA considers these
6 factors for potential non-threshold pollutants “what EPA lacks is any determinate criterion for
7 drawing lines” to determine where the standards should be set.

8 The D.C. Circuit’s holding on the cost and constitutional issues were appealed to the
9 United States Supreme Court. In February 2001, the Supreme Court issued a unanimous decision
10 upholding the EPA’s position on both the cost and constitutional issues. *Whitman v. American*
11 *Trucking Associations*, 531 U.S. 457, 464, 475-76. On the constitutional issue, the Court held
12 that the statutory requirement that NAAQS be “requisite” to protect public health with an
13 adequate margin of safety sufficiently guided the EPA’s discretion, affirming the EPA’s
14 approach of setting standards that are neither more nor less stringent than necessary.

15 The Supreme Court remanded the case to the Court of Appeals for resolution of any
16 remaining issues that had not been addressed in that court’s earlier rulings. *Id.* at 475-76. In a
17 March 2002 decision, the Court of Appeals rejected all remaining challenges to the standards,
18 holding that the EPA’s PM_{2.5} standards were reasonably supported by the administrative record
19 and were not “arbitrary and capricious” *American Trucking Associations v. EPA*, 283 F. 3d 355,
20 369-72 (D.C. Cir. 2002).

21 **1.3.3 Review Completed in 2006**

22 In October 1997, the EPA published its plans for the third periodic review of the air
23 quality criteria and NAAQS for PM (62 FR 55201, October 23, 1997). After the CASAC and
24 public review of several drafts, the EPA’s NCEA finalized the AQCD in October 2004 (U.S.
25 EPA, 2004a, U.S. EPA, 2004b). The EPA’s OAQPS finalized a Risk Assessment and Staff Paper
26 in December 2005 (Abt Associates, 2005, U.S. EPA, 2005).¹² On December 20, 2005, the EPA
27 announced its proposed decision to revise the NAAQS for PM and solicited public comment on a
28 broad range of options (71 FR 2620, January 17, 2006). On September 21, 2006, the EPA
29 announced its final decisions to revise the primary and secondary NAAQS for PM to provide
30 increased protection of public health and welfare, respectively (71 FR 61144, October 17, 2006).
31 With regard to the primary and secondary standards for fine particles, the EPA revised the level

¹² Prior to the review initiated in 2007, the Staff Paper presented the EPA staff’s considerations and conclusions regarding the adequacy of existing NAAQS and, when appropriate, the potential alternative standards that could be supported by the evidence and information. More recent reviews present this information in the Policy Assessment.

1 of the 24-hour PM_{2.5} standards to 35 µg/m³, retained the level of the annual PM_{2.5} standards at
2 15.0 µg/m³, and revised the form of the annual PM_{2.5} standards by narrowing the constraints on
3 the optional use of spatial averaging. With regard to the primary and secondary standards for
4 PM₁₀, the EPA retained the 24-hour standards, with levels at 150 µg/m³, and revoked the annual
5 standards.¹³ The Administrator judged that the available evidence generally did not suggest a link
6 between long-term exposure to existing ambient levels of coarse particles and health or welfare
7 effects. In addition, a new reference method was added for the measurement of PM_{10-2.5} in the
8 ambient air in order to provide a basis for approving federal equivalent methods (FEMs) and to
9 promote the gathering of scientific data to support future reviews of the PM NAAQS.

10 Several parties filed petitions for review following promulgation of the revised PM
11 NAAQS in 2006. These petitions addressed the following issues: (1) selecting the level of the
12 primary annual PM_{2.5} standard; (2) retaining PM₁₀ as the indicator of a standard for thoracic
13 coarse particles, retaining the level and form of the 24-hour PM₁₀ standard, and revoking the
14 PM₁₀ annual standard; and (3) setting the secondary PM_{2.5} standards identical to the primary
15 standards. On February 24, 2009, the U.S. Court of Appeals for the District of Columbia Circuit
16 issued its opinion in the case *American Farm Bureau Federation v. EPA*, 559 F. 3d 512 (D.C.
17 Cir. 2009). The court remanded the primary annual PM_{2.5} NAAQS to the EPA because the
18 Agency failed to adequately explain why the standards provided the requisite protection from
19 both short- and long-term exposures to fine particles, including protection for at-risk populations.
20 *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 520-27 (D.C. Cir. 2009). With regard
21 to the standards for PM₁₀, the court upheld the EPA’s decisions to retain the 24-hour PM₁₀
22 standard to provide protection from thoracic coarse particle exposures and to revoke the annual
23 PM₁₀ standard. *American Farm Bureau Federation*, 559 F. 2d at 533-38. With regard to the
24 secondary PM_{2.5} standards, the court remanded the standards to the EPA because the Agency
25 failed to adequately explain why setting the secondary PM standards identical to the primary
26 standards provided the required protection for public welfare, including protection from visibility
27 impairment. *American Farm Bureau Federation*, 559 F. 2d at 528-32. The EPA responded to the
28 court’s remands as part of the next review of the PM NAAQS, which was initiated in 2007
29 (discussed below).

¹³ In the 2006 proposal, the EPA proposed to revise the 24-hour PM₁₀ standard in part by establishing a new PM_{10-2.5} indicator for thoracic coarse particles (i.e., particles generally between 2.5 and 10 µm in diameter). The EPA proposed to include any ambient mix of PM_{10-2.5} that was dominated by resuspended dust from high density traffic on paved roads and by PM from industrial sources and construction sources. The EPA proposed to exclude any ambient mix of PM_{10-2.5} that was dominated by rural windblown dust and soils and by PM generated from agricultural and mining sources. In the final decision, the existing PM₁₀ standard was retained, in part due to an “inability...to effectively and precisely identify which ambient mixes are included in the [PM_{10-2.5}] indicator and which are not” (71 FR 61197, October 17, 2006).

1 **1.3.4 Review Completed in 2012**

2 In June 2007, the EPA initiated the fourth periodic review of the air quality criteria and
3 the PM NAAQS by issuing a call for information in the *Federal Register* (72 FR 35462, June 28,
4 2007). Based on the NAAQS review process, as revised in 2008 and again in 2009,¹⁴ the EPA
5 held science/policy issue workshops on the primary and secondary PM NAAQS (72 FR 34003,
6 June 20, 2007; 72 FR 34005, June 20, 2007), and prepared and released the planning and
7 assessment documents that comprise the review process (i.e., IRP (U.S. EPA, 2008), ISA (U.S.
8 EPA, 2009a), REA planning documents for health and welfare (U.S. EPA, 2009b, U.S. EPA,
9 2009c), a quantitative health risk assessment (U.S. EPA, 2010a) and an urban-focused visibility
10 assessment (U.S. EPA, 2010b), and PA (U.S. EPA, 2011)). In June 2012, the EPA announced its
11 proposed decision to revise the NAAQS for PM (77 FR 38890, June 29, 2012).

12 In December 2012, the EPA announced its final decisions to revise the primary NAAQS
13 for PM to provide increased protection of public health (78 FR 3086, January 15, 2013). With
14 regard to primary standards for PM_{2.5}, the EPA revised the level of the annual PM_{2.5} standard¹⁵ to
15 12.0 µg/m³ and retained the 24-hour PM_{2.5} standard, with its level of 35 µg/m³. For the primary
16 PM₁₀ standard, the EPA retained the 24-hour standard to continue to provide protection against
17 effects associated with short-term exposure to thoracic coarse particles (i.e., PM_{10-2.5}). With
18 regard to the secondary PM standards, the EPA generally retained the 24-hour and annual PM_{2.5}
19 standards¹⁶ and the 24-hour PM₁₀ standard to address visibility and non-visibility welfare effects.

20 As with previous reviews, petitioners challenged the EPA's final rule. Petitioners argued
21 that the EPA acted unreasonably in revising the level and form of the annual standard and in
22 amending the monitoring network provisions. On judicial review, the revised standards and
23 monitoring requirements were upheld in all respects. *NAM v EPA*, 750 F.3d 921 (D.C. Cir.
24 2014).

25 **1.3.5 Review Completed in 2020**

26 In December 2014, the EPA announced the initiation of the periodic review of the air
27 quality criteria for PM and of the PM_{2.5} and PM₁₀ NAAQS and issued a call for information in
28 the *Federal Register* (79 FR 71764, December 3, 2014). On February 9 to 11, 2015, the EPA's
29 NCEA and OAQPS held a public workshop to inform the planning for the current review of the
30 PM NAAQS (announced in 79 FR 71764, December 3, 2014). Workshop participants, including

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

¹⁵ The EPA also eliminated the option for spatial averaging.

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

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

9 The input received at the workshop guided the EPA staff in developing a draft IRP,
10 which was reviewed by the CASAC Particulate Matter Panel and discussed on public
11 teleconferences held in May 2016 (81 FR 13362, March 14, 2016) and August 2016 (81 FR
12 39043, June 15, 2016). Advice from the CASAC, supplemented by the Particulate Matter Panel,
13 and input from the public were considered in developing the final IRP for this review (U.S. EPA,
14 2016). The final IRP discusses the approaches to be taken in developing key scientific, technical,
15 and policy documents in this review and the key policy-relevant issues that will frame the EPA's
16 consideration of whether the current primary and/or secondary NAAQS for PM should be
17 retained or revised.

18 In May 2018, the Administrator issued a memorandum describing a “back-to-basics”
19 process for reviewing the NAAQS (Pruitt, 2018). This memo announced the Agency's intention
20 to conduct the current review of the PM NAAQS in such a manner as to ensure that any
21 necessary revisions were finalized by December 2020. Following this memo, on October 10,
22 2018 the Administrator additionally announced that the role of reviewing the key science
23 assessments developed as part of the ongoing review of the PM NAAQS (i.e., drafts of the ISA
24 and PA) would be performed only by the seven-member chartered CASAC (i.e., without the
25 support of the CASAC Particulate Matter Panel that reviewed the draft IRP).¹⁷

26 The EPA released the draft ISA in October 2018 (83 FR 53471, October 23, 2018). The
27 draft ISA was reviewed by the chartered CASAC at a public meeting held in Arlington, VA in
28 December 2018 (83 FR 55529, November 6, 2018) and was discussed on a public teleconference
29 in March 2019 (84 FR 8523, March 8, 2019). The CASAC provided its advice on the draft ISA
30 in a letter to the EPA Administrator dated April 11, 2019 (Cox, 2019a). The EPA took steps to
31 address these comments in the final ISA, which was released in December 2019 (U.S. EPA,
32 2019).

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

¹⁷ Announcement available at: <https://www.regulations.gov/document/EPA-HQ-OAR-2015-0072-0223>

1 meeting held in Cary, NC. Public comments were received via a separate public teleconference
2 (84 FR 51555, September 30, 2019). A public meeting to discuss the chartered CASAC letter
3 and response to charge questions on the draft PA was held in Cary, NC in December 2019 (84
4 FR 58713, November 1, 2019), and the CASAC provided its advice on the draft PA, including its
5 advice on the current primary and secondary PM standards, in a letter to the EPA Administrator
6 dated December 16, 2019 (Cox, 2019b). With regard to the primary standards, the CASAC
7 recommended retaining the current 24-hour PM_{2.5} and PM₁₀ standards but did not reach
8 consensus on the adequacy of the current annual PM_{2.5} standard. With regard to the secondary
9 standards, the CASAC recommended retaining the current standards. In response to the
10 CASAC’s comments, the 2020 final PA incorporated a number of changes (U.S. EPA, 2020), as
11 described in detail in section I.C.5 of the 2020 proposal (85 FR 24100, April 30, 2020).

12 On April 14, 2020, the EPA proposed to retain all of the primary and secondary PM
13 standards, without revision. These proposed decisions were published in the Federal Register on
14 April 30, 2020 (85 FR 24094, April 30, 2020). The EPA’s final decision on the PM NAAQS was
15 published in the Federal Register on December 18, 2020 (85 FR 82684, December 18, 2020). In
16 the 2020 rulemaking, the EPA retained the primary and secondary PM_{2.5} and PM₁₀ standards,
17 without revision. The EPA received three petitions for judicial review (described in more detail
18 in section 1.4.3 below), as well as three petitions for reconsideration of the 2020 final action.

19 **1.4 RECONSIDERATION OF THE 2020 PM NAAQS FINAL ACTION**

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

26 **1.4.1 Decision to Initiate a Reconsideration**

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

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

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

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

1 available scientific evidence and technical information indicate that the current standards may
2 not be adequate to protect public health and welfare, as required by the Clean Air Act. We note
3 that the 2020 PA concluded that the scientific evidence and information supported revising the
4 level of the primary annual PM_{2.5} standard to below the current level of 12 µg/m³ while retaining
5 the primary 24-hour PM_{2.5} standard (U.S. EPA, 2020). The EPA also notes that the 2020 PA
6 concluded that the available scientific evidence and information supported retaining the primary
7 PM₁₀ standard and secondary PM standards without revision (U.S. EPA, 2020).

8 **1.4.2 Process for Reconsideration of the 2020 PM NAAQS Decision**

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

16 On March 31, 2021, the Administrator announced his decision to reestablish the
17 membership of the CASAC to “ensure the agency received the best possible scientific insight to
18 support our work to protect human health and the environment.”²¹ Consistent with this
19 memorandum, a call for nominations of candidates to the EPA’s chartered CASAC was
20 published in the Federal Register (86 FR 17146, April 1, 2021). On June 17, 2021, the
21 Administrator announced his selection of the seven members to serve on the chartered CASAC.²²
22 ²³ Additionally, a call for nominations of candidates to a PM-specific panel was published in the
23 Federal Register (86 FR 33703, June 25, 2021). The members of the PM CASAC panel were
24 announced on August 30, 2021.²⁴

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

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

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

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

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

1 reconsideration of the 2020 PM NAAQS final decision. The draft ISA Supplement focuses on a
2 thorough evaluation of some studies that became available after the literature cutoff date of the
3 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address
4 key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. In
5 selecting the health effects to evaluate within the draft ISA Supplement, the EPA focused on the
6 strongest causality determinations for health effects categories presented in the 2019 ISA, and
7 the subsequent use of the health effects evidence in the 2020 PA with respect to which were most
8 useful in informing staff conclusions (U.S. EPA, 2021).²⁵ Specifically, within the draft ISA
9 Supplement, the focus is only on the health effects evidence where the 2019 ISA concluded a
10 “causal relationship” (U.S. EPA, 2021, section 1.2.1). Consistent with the rationale for the health
11 effects, the selection of the welfare effects to evaluate within the draft ISA Supplement were
12 based on the causality determinations reported in the 2019 ISA and the subsequent use of
13 scientific evidence in the 2020 PA.²⁶ Specifically, for welfare effects, the focus within the draft
14 ISA Supplement is on visibility effects. The draft ISA Supplement also considers recent health
15 effects evidence that addresses key scientific topics where the literature has evolved since the
16 2020 review was completed, specifically since the literature cutoff date for the 2019 ISA.²⁷

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

- 20 • Health effects:
 - 21 – Health effect categories where the 2019 PM ISA concluded a “*causal*
22 *relationship*” (i.e., short- and long-term PM_{2.5} exposure and cardiovascular effects

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

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

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

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

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

7 • Welfare Effects:

- 8 – U.S. and Canadian studies that provide new information on public preferences for
9 visibility impairment and/or developed methodologies or conducted quantitative
10 analyses of light extinction

11 • Key Scientific Topics

- 12 – Experimental studies (i.e., controlled human exposure and animal toxicological)
13 conducted at near-ambient PM_{2.5} concentrations
- 14 – At-Risk Populations
 - 15 ○ U.S. and Canadian-based epidemiologic or exposure studies examining
16 potential disparities in either PM_{2.5} exposures or the risk of health effects
17 by race/ethnicity or socioeconomic status (SES)
- 18 – U.S. and Canadian-based epidemiologic studies that examined the relationship
19 between PM_{2.5} exposures and COVID-19 infection and/or death

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

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

1 **1.4.3 Ongoing Litigation**

2 Following publication of the 2020 final action, several parties filed petitions for review of
3 the EPA’s final decision in the D.C. Circuit and the Court consolidated the cases. In order to
4 consider whether reconsideration of the 2020 final action was warranted, the EPA moved for two
5 90-day abeyances in these consolidated cases, which the Court granted. After the EPA
6 announced that is reconsidering the 2020 final decision, the EPA filed a motion with the Court to
7 hold the consolidated cases in abeyance until March 1, 2023. The court has not yet acted on the
8 EPA’s motion, which the court granted on October 1, 2021.

1 REFERENCES

- 2 Abt Associates, Inc. (2005). Particulate matter health risk assessment for selected urban areas:
3 Draft report. EPA Contract No. 68-D-03-002. U.S. Environmental Protection Agency.
4 Research Triangle Park, NC. Available at:
5 <http://www3.epa.gov/ttn/naaqs/standards/pm/data/PMrisk20051220.pdf>.
- 6 Cox, LA. (2019a). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory
7 Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's
8 *Integrated Science Assessment for Particulate Matter (External Review Draft - October*
9 *2018)*. April 11, 2019. EPA-CASAC-19-002. Office of the Administrator, Science
10 Advisory Board U.S. EPA HQ, Washington DC. Available at:
11 [https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/932](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/932D1DF8C2A9043F852581000048170D?OpenDocument&TableRow=2.3#2)
12 [D1DF8C2A9043F852581000048170D?OpenDocument&TableRow=2.3#2](https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/932D1DF8C2A9043F852581000048170D?OpenDocument&TableRow=2.3#2).
- 13 Cox, LA. (2019b). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory
14 Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's
15 *Policy Assessment for the Review of the National Ambient Air Quality Standards for*
16 *Particulate Matter (External Review Draft - September 2019)*. December 16, 2019. EPA-
17 CASAC-20-001. Office of the Administrator, Science Advisory Board U.S. EPA HQ,
18 Washington DC. Available at:
19 [https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F](https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf)
20 [6C71737201612852584D20069DFB1/\\$File/EPA-CASAC-20-001.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf).
- 21 DHEW (1969). Air Quality Criteria for Particulate Matter. National Air Pollution Control
22 Administration. Washington, D.C. U.S. Department of Health. January 1969.
- 23 Pruitt, E. (2018). Memorandum from E. Scott Pruitt, Administrator, U.S. EPA to Assistant
24 Administrators. Back-to-Basics Process for Reviewing National Ambient Air Quality
25 Standards. May 9, 2018. Office of the Administrator U.S. EPA HQ, Washington DC.
26 Available at: [https://www.epa.gov/criteria-air-pollutants/back-basics-process-reviewing-](https://www.epa.gov/criteria-air-pollutants/back-basics-process-reviewing-national-ambient-air-quality-standards)
27 [national-ambient-air-quality-standards](https://www.epa.gov/criteria-air-pollutants/back-basics-process-reviewing-national-ambient-air-quality-standards).
- 28 U.S. EPA (2004a). Air Quality Criteria for Particulate Matter. (Vol I of II). Office of Research
29 and Development. Research Triangle Park, NC. U.S. EPA. EPA-600/P-99-002aF.
30 October 2004. Available at:
31 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100LFIQ.txt>.
- 32 U.S. EPA (2004b). Air Quality Criteria for Particulate Matter. (Vol II of II). Office of Research
33 and Development. Research Triangle Park, NC. U.S. EPA. EPA-600/P-99-002bF.
34 October 2004. Available at:
35 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100LG7Q.txt>.
- 36 U.S. EPA (2005). Review of the National Ambient Air Quality Standards for Particulate Matter:
37 Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. Office
38 of Air Quality Planning and Standards. Research Triangle Park, NC. U.S. EPA. EPA-

- 1 452/R-05-005a. December 2005. Available at:
2 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1009MZM.txt>.
- 3 U.S. EPA (2008). Integrated Review Plan for the National Ambient Air Quality Standards for
4 Particulate Matter. Office of Research and Development, National Center for
5 Environmental Assessment; Office of Air Quality Planning and Standards, Health and
6 Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA 452/R-08-
7 004. March 2008. Available at:
8 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1001FB9.txt>.
- 9 U.S. EPA (2009a). Integrated Science Assessment for Particulate Matter (Final Report). Office
10 of Research and Development, National Center for Environmental Assessment. Research
11 Triangle Park, NC. U.S. EPA. EPA-600/R-08-139F. December 2009. Available at:
12 <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546>.
- 13 U.S. EPA (2009b). Particulate Matter National Ambient Air Quality Standards: Scope and
14 Methods Plan for Health Risk and Exposure Assessment. Office of Air Quality Planning
15 and Standards, Health and Environmental Impacts Division. Research Triangle Park, NC.
16 U.S. EPA. EPA-452/P-09-002. February 2009. Available at:
17 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FLWP.txt>.
- 18 U.S. EPA (2009c). Particulate Matter National Ambient Air Quality Standards: Scope and
19 Methods Plan for Urban Visibility Impact Assessment. Office of Air Quality Planning
20 and Standards, Health and Environmental Impacts Division. Research Triangle Park, NC.
21 U.S. EPA. EPA-452/P-09-001. February 2009. Available at:
22 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FLUX.txt>.
- 23 U.S. EPA (2010a). Quantitative Health Risk Assessment for Particulate Matter (Final Report).
24 Office of Air Quality Planning and Standards, Health and Environmental Impacts
25 Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-10-005. June 2010.
26 Available at: <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1007RFC.txt>.
- 27 U.S. EPA (2010b). Particulate Matter Urban-Focused Visibility Assessment (Final Document).
28 Office of Air Quality Planning and Standards, Health and Environmental Impacts
29 Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-10-004. July 2010.
30 Available at: <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FO5D.txt>.
- 31 U.S. EPA (2011). Policy Assessment for the Review of the Particulate Matter National Ambient
32 Air Quality Standards. Office of Air Quality Planning and Standards, Health and
33 Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-
34 11-003. April 2011. Available at:
35 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100AUMY.txt>.
- 36 U.S. EPA (2016). Integrated review plan for the national ambient air quality standards for
37 particulate matter. Office of Air Quality Planning and Standards. Research Triangle Park,
38 NC. U.S. EPA. EPA-452/R-16-005. December 2016. Available at:
39 [https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-
40 plan.pdf](https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf).

1 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
2 U.S. Environmental Protection Agency, Office of Research and Development, National
3 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
4 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
5 [standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).

6 U.S. EPA (2020). Policy Assessment for the Review of the National Ambient Air Quality
7 Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health
8 and Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-
9 452/R-20-002. January 2020. Available at: [https://www.epa.gov/sites/default/files/2020-](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf)
10 [01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf).

11 U.S. EPA (2021). Supplement to the 2019 Integrated Science Assessment for Particulate Matter
12 (External Review Draft). U.S. Environmental Protection Agency, Office of Research and
13 Development, Center for Public Health and Environmental Assessment. Washington,
14 DC. U.S. EPA. EPA/600/R-21/198. December 2019. Available at:
15 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
16 [assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).

17
18

2 PM AIR QUALITY

This chapter provides an overview of recent ambient air quality with respect to PM. It summarizes information on the distribution of particle size in ambient air, including discussions about size fractions and components (section 2.1), ambient monitoring of PM in the U.S. (section 2.2), ambient concentrations of PM in the U.S. (section 2.3), and background PM (section 2.4).

2.1 DISTRIBUTION OF PARTICLE SIZE IN AMBIENT AIR

In ambient air, PM is a mixture of substances suspended as small liquid and/or solid particles. Particle size is an important consideration for PM, as distinct health and welfare effects have been linked with exposures to particles of different sizes. Particles in the atmosphere range in size from less than 0.01 to more than 10 micrometers (μm) in diameter (U.S. EPA, 2019b, section 2.2). When describing PM, subscripts are used to denote the aerodynamic diameter¹ of the particle size range in micrometers (μm) of 50% cut points of sampling devices. The EPA defines $\text{PM}_{2.5}$, also referred to as fine particles, as particles with aerodynamic diameters generally less than or equal to 2.5 μm . The size range for $\text{PM}_{10-2.5}$, also called coarse or thoracic coarse particles, includes those particles with aerodynamic diameters generally greater than 2.5 μm and less than or equal to 10 μm . PM_{10} , which is comprised of both fine and coarse fractions, includes those particles with aerodynamic diameters generally less than or equal to 10 μm . Figure 2-1 provides perspective on these particle size fractions. In addition, ultrafine particles (UFP) are often defined as particles with a diameter of less than 0.1 μm based on physical size, thermal diffusivity or electrical mobility (U.S. EPA, 2019b, section 2.2).

¹ Aerodynamic diameter is the size of a sphere of unit density (i.e., 1 g/cm^3) that has the same terminal settling velocity as the particle of interest (U.S. EPA, 2018, U.S. EPA, 2019b, section 4.1.1).

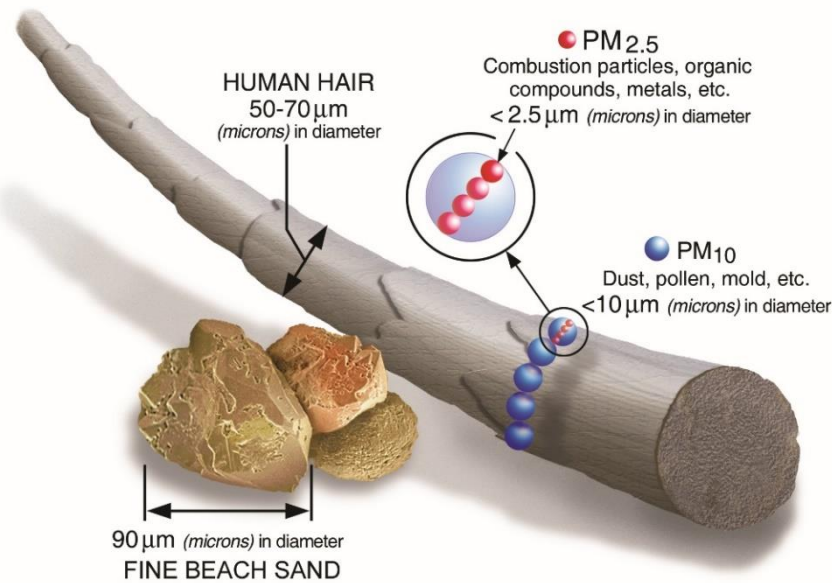


Figure 2-1. Comparisons of PM_{2.5} and PM₁₀ diameters to human hair and beach sand.

(Adapted from: <https://www.epa.gov/pm-pollution/particulate-matter-pm-basics>)

Atmospheric distributions of particle size generally exhibit distinct modes that roughly align with the PM size fractions defined above. The nucleation mode is made up of freshly generated particles, formed either during combustion or by atmospheric reactions of precursor gases. The nucleation mode is especially prominent near sources like heavy traffic, industrial emissions, biomass burning, or cooking (Vu et al., 2015). While nucleation mode particles are only a minor contributor to overall ambient PM mass and surface area, they are the main contributors to ambient particle number (U.S. EPA, 2019b, section 2.2). By number, most nucleation mode particles fall into the UFP size range, though some fraction of the nucleation mode number distribution can extend above 0.1 μm in diameter. Nucleation mode particles can grow rapidly through coagulation or uptake of gases by particle surfaces, giving rise to the accumulation mode. The accumulation mode is typically the predominant contributor to PM_{2.5} mass and surface area, though only a minor contributor to particle number (U.S. EPA, 2019b, section 2.2). PM_{2.5} sampling methods measure most of the accumulation mode mass, although a small fraction of particles that make up the accumulation mode are greater than 2.5 μm in diameter. Coarse mode particles are formed by mechanical generation, and through processes like dust resuspension and sea spray formation (Whitby et al., 1972). Most coarse mode mass is captured by PM_{10-2.5} sampling, but small fractions of coarse mode mass can be smaller than 2.5 μm or greater than 10 μm in diameter (U.S. EPA, 2019b, section 2.2).

Most particles are found in the lower troposphere, where they can have residence times ranging from a few hours to weeks. Particles are removed from the atmosphere by wet

1 deposition, such as when they are carried by rain or snow, or by dry deposition, when particles
2 settle out of suspension due to gravity. Atmospheric lifetimes are generally longest for $PM_{2.5}$,
3 which often remains in the atmosphere for days to weeks (U.S. EPA, 2019b, Table 2-1) before
4 being removed by wet or dry deposition. In contrast, atmospheric lifetimes for UFP and $PM_{10-2.5}$
5 are shorter. Within hours, UFP can undergo coagulation and condensation that lead to formation
6 of larger particles in the accumulation mode, or can be removed from the atmosphere by
7 evaporation, deposition, or reactions with other atmospheric components. $PM_{10-2.5}$ are also
8 generally removed from the atmosphere within hours, through wet or dry deposition (U.S. EPA,
9 2019b, Table 2-1).

10 **2.1.1 Sources of PM Emissions**

11 PM is composed of both primary (directly emitted particles) and secondary chemical
12 components. Primary PM is derived from direct particle emissions from specific PM sources
13 while secondary PM originates from gas-phase chemical compounds present in the atmosphere
14 that have participated in new particle formation or condensed onto existing particles (U.S. EPA,
15 2019b, section 2.3). Primary particles, and gas-phase compounds contributing to secondary
16 formation PM, are emitted from both anthropogenic and natural sources.

17 Anthropogenic sources of PM include both stationary and mobile sources. Stationary
18 sources include fuel combustion for electricity production and other purposes, industrial
19 processes, agricultural activities, and road and building construction and demolition. Mobile
20 sources of PM include diesel- and gasoline-powered highway vehicles and other engine-driven
21 sources (e.g., ships, aircraft, and construction and agricultural equipment). Both stationary and
22 mobile sources directly emit primary PM to ambient air, along with secondary PM precursors
23 (e.g., SO_2) that contribute to the secondary formation of PM in the atmosphere (U.S. EPA,
24 2019b, section 2.3, Table 2-2).

25 Natural sources of PM include dust from the wind erosion of natural surfaces, sea salt,
26 wildland fires, primary biological aerosol particles (PBAP) such as bacteria and pollen, oxidation
27 of biogenic hydrocarbons such as isoprene and terpenes to produce secondary organic aerosol
28 (SOA), and geogenic sources such as sulfate formed from volcanic production of SO_2 (U.S.
29 EPA, 2009, section 3.3, Table 3-2). While most of the above sources release or contribute
30 predominantly to fine aerosol, some sources including windblown dust, and sea salt also produce
31 particles in the coarse size range (U.S. EPA, 2019b, section 2.3.3).

32 Generally, the sources of PM for different size fractions vary. While $PM_{2.5}$ in ambient air
33 is largely emitted directly by sources such as those described above or through secondary PM
34 formation in the atmosphere, $PM_{10-2.5}$ is almost entirely from primary sources (i.e., directly
35 emitted) and is produced by surface abrasion or by suspension of sea spray or biological

1 materials such as microorganisms, pollen, and plant and insect debris (U.S. EPA, 2019b, section
2 2.3.2.1).

3 In sections 2.1.1.1 and 2.1.1.2 below, we describe the most recently available information
4 on sources contributing to PM_{2.5} and PM_{10-2.5} emissions into ambient air, respectively, based on
5 the 2017 National Emissions Inventory (NEI).² In section 2.1.1.3, we describe information on
6 sources contributing to emissions of PM components and precursor gases, with a focus on the
7 2017 NEI. Section 2.3.1 discusses emission trends and identifies the sectors that have
8 experienced the most change in direct PM and precursor emissions from 1990 to 2017. It should
9 be noted that major decreases have been observed in NO_x and SO₂ emissions over this time, with
10 continued reductions observed from the 2014 NEI to the 2017 NEI. For a more detailed review
11 of the changes in PM and PM precursor emissions from the 2014 NEI to the 2017 NEI, please
12 refer to the 2017 NEI Technical Support Document (U.S. EPA, 2021).

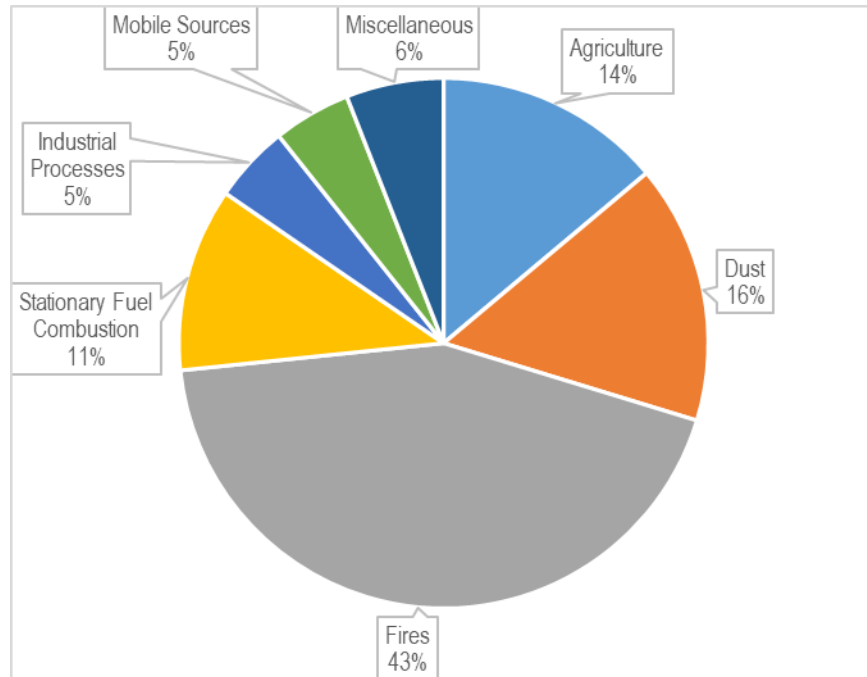
13 **2.1.1.1 Sources Contributing to Primary PM_{2.5} Emissions**

14 The National Emissions Inventory (NEI) is a comprehensive and detailed estimate of air
15 emissions of criteria pollutants, criteria pollutant precursors, and hazardous air pollutants from a
16 comprehensive set of air emissions sources, including point sources (e.g., electric generating
17 units, boilers, etc.), nonpoint (or area) sources (e.g., oil & gas, residential wood combustion, and
18 many other dispersed sources), mobiles sources, and events (large fires). There are over 3,000
19 sources for which the NEI is developed. The NEI is released every three years based primarily
20 upon data provided by state, local, and tribal air agencies for sources in their jurisdictions and
21 supplemented by data developed by the EPA. The NEI is built using the Emissions Inventory
22 System (EIS) first to collect the data from state, local, and tribal air agencies and then to blend
23 that data with other data sources.

24 Based on the 2017 NEI, approximately 5.7 million tons/year of PM_{2.5} were estimated to
25 be directly emitted to the atmosphere from a number of source sectors in the U.S. This total
26 excludes sources that are not a part of the NEI (e.g., windblown dust, geogenic sources). As
27 shown in Figure 2-2, nearly half of the total primary PM_{2.5} emissions nationally are contributed
28 by the dust and fire sectors together. Dust includes agricultural, construction, and road dust. Of
29 these, agricultural dust and road dust in sum make the greatest contributions to PM_{2.5} emissions
30 nationally. Fires include wildfires, prescribed fires, and agricultural fires, with wildfires and
31 prescribed fires accounting for most of the fire-related primary PM_{2.5} emissions nationally (U.S.

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

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



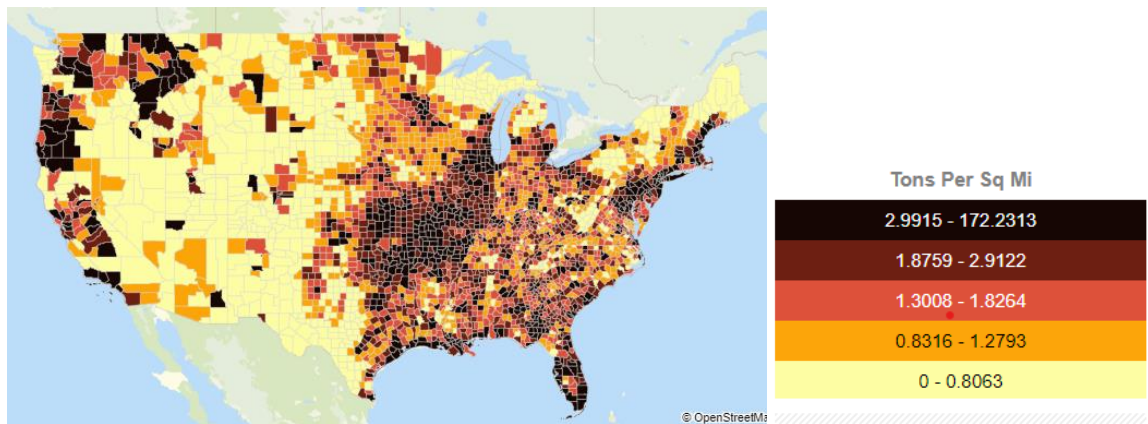
3
4 **Figure 2-2. Percent contribution of PM_{2.5} national emissions by source sectors.** (Source:
5 2017 NEI)

6 The relative contributions of specific sources to annual emissions of primary PM_{2.5} can
7 vary from location to location, with a notable difference in contributions of sources of PM_{2.5}
8 emissions in urban areas compared to national emissions. For example, the 2019 ISA illustrates
9 this variation of primary PM_{2.5} emissions with data from five urban counties in the U.S. (U.S.
10 EPA, 2019b, Figure 2-3).³ Across the majority of these urban areas, the largest PM_{2.5}-emitting
11 sectors are mobile sources and fuel combustion. This is in contrast to fires, which account for the
12 largest fraction of primary emissions nationally but make much smaller contributions in many
13 urban counties (U.S. EPA, 2019b, section 2.3.1.2, Figure 2-3). While primary PM_{2.5} from mobile
14 sources are a dominant contributor in some urban areas, accounting for an estimated 13 to 30%
15 of the total primary PM_{2.5} emissions, mobile sources contribute only about 5% to total primary
16 PM_{2.5} emissions nationally as shown in Figure 2-2.

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

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

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



4
5 **Figure 2-3. 2017 NEI PM_{2.5} Emissions Density Map, tons per square mile**

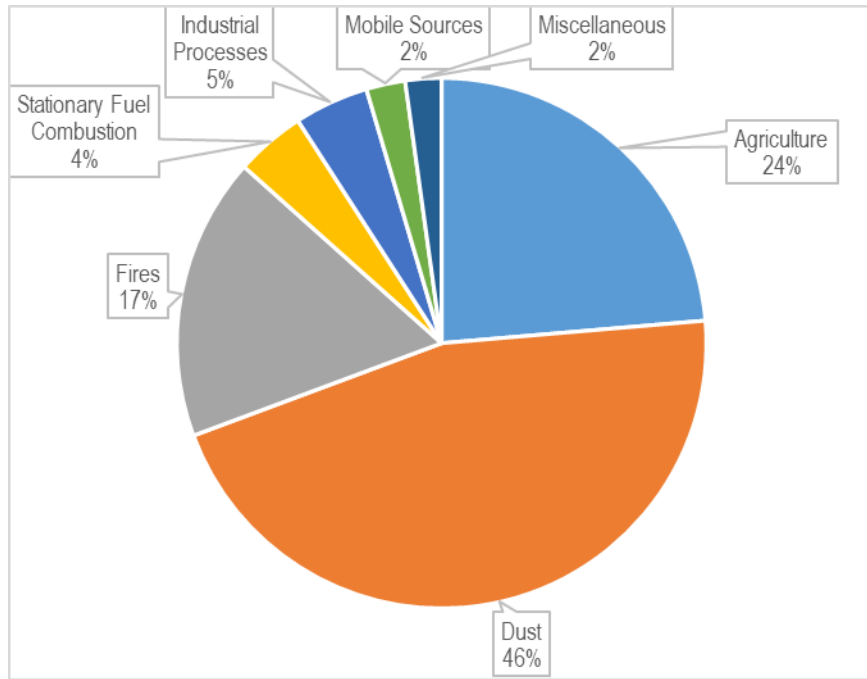
6 **2.1.1.2 Sources Contributing to Primary PM₁₀ Emissions**

7 Although the NEI does not estimate emissions of PM_{10-2.5} (coarse PM) specifically,
8 estimates of PM₁₀ emissions can provide insight into sources of coarse particles. Thus, the
9 discussion below focuses on PM₁₀ emissions. The relative contributions of key sources to
10 national PM₁₀ emissions, based on the 2017 NEI, are shown in Figure 2-4. Total PM₁₀ emissions
11 are estimated to be about 17 million tons. National emissions of PM₁₀ are dominated by dust and
12 agriculture, contributing a combined 70% of the total emissions. Current NEI estimates of dust
13 emissions across the U.S. are based on limited emissions profile and activity information. For a
14 number of reasons, quantification of dust emissions is highly uncertain. Much like wildfires, dust
15 emissions are common but intermittent emissions sources. Additionally, the suspension and
16 resuspension of dust is difficult to quantify. Moreover, some dust particles in the PM_{10-2.5} size
17 range are also transported internationally and are considered as a part of the background
18 component of PM as opposed to a primary emission of coarse PM (U.S. EPA, 2019b, section
19 2.3.3).

20 As with PM_{2.5}, the relative contributions of sources to total PM₁₀ emissions varies from
21 location to location (e.g., depending on local climate, geography, degree of urbanization, etc.).
22 However, unlike PM_{2.5}, the sectors included in Figure 2-4 are expected to be among the most
23 important contributors to coarse PM emissions at both the national and more regional levels,
24 particularly given the sources of the particles in these source categories (e.g., mineral dust,
25 primary biological aerosols (including pollen), sea spray). As noted previously, the NEI does not
26 include sources such as pollen, sea spray, windblown dust, or geogenic sources, though those
27 sources also likely contribute to PM₁₀ emissions. Figure 2-4 shows the national contributions to

1 PM₁₀ emissions from particular source sectors and Figure 2-5 exhibits the corresponding
2 emissions density map for PM₁₀.

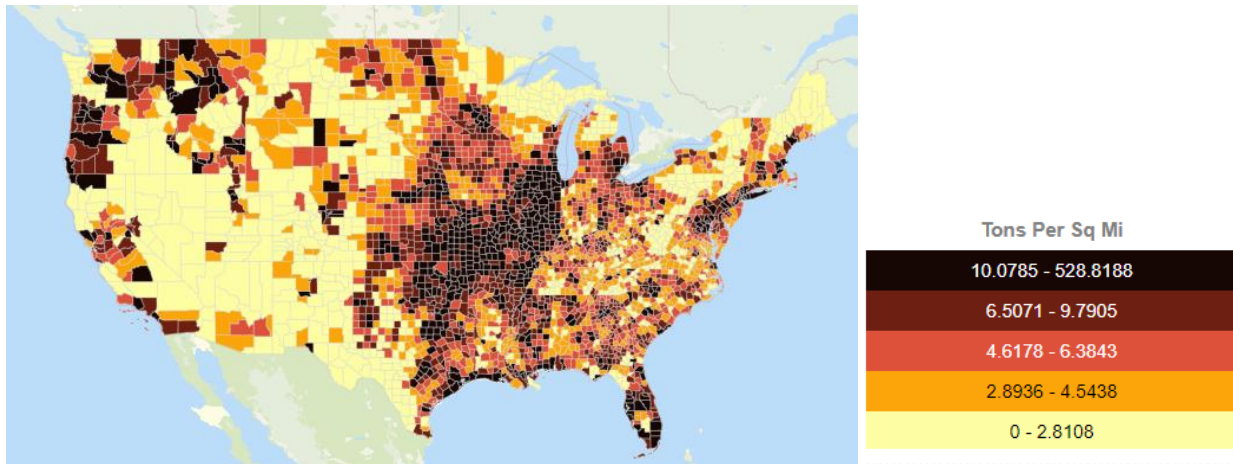
3



4

5 **Figure 2-4. Percent contribution of PM₁₀ emissions by national source sectors.** (Source:
6 2017 NEI)

7



8

9 **Figure 2-5. 2017 NEI PM₁₀ Emissions Density Map, tons per square mile**

10 2.1.1.3 Sources Contributing to Emissions of PM Components and Precursor Gases

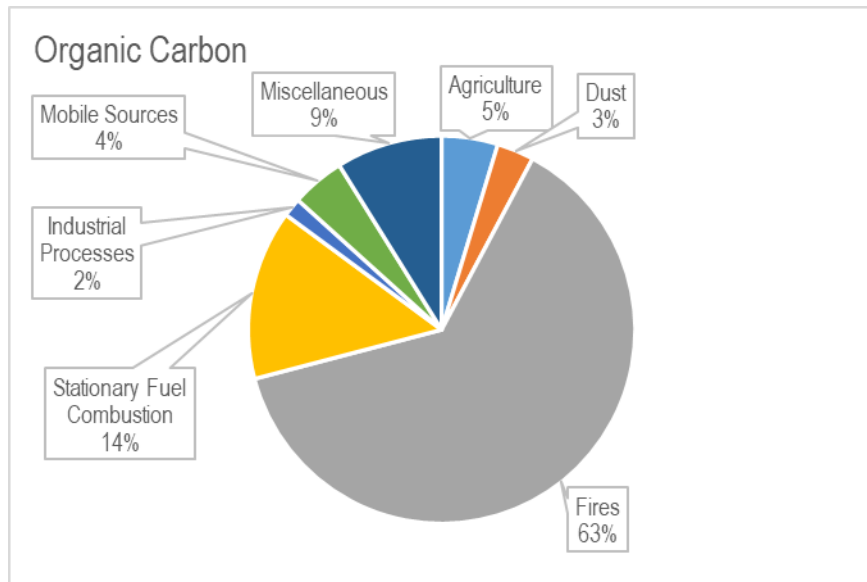
11 Understanding the components of PM is particularly important for providing insight into
12 which sources contribute to PM mass, as well as to better understand the health and welfare
13 effects of particles. Major components of PM_{2.5} mass include sulfate (SO₄²⁻), nitrate (NO₃⁻),

1 elemental or black carbon (EC or BC), organic carbon (OC), and crustal materials. Some of these
2 PM components are emitted directly to the air (e.g., EC/BC) while others are formed secondarily
3 through reactions by gaseous precursors (e.g., sulfate, nitrate). The following sections
4 specifically discuss the sources that contribute to the specific PM_{2.5} components, including
5 particulate carbon (section 2.1.1.3.1) and precursor gases (section 2.1.1.3.2).

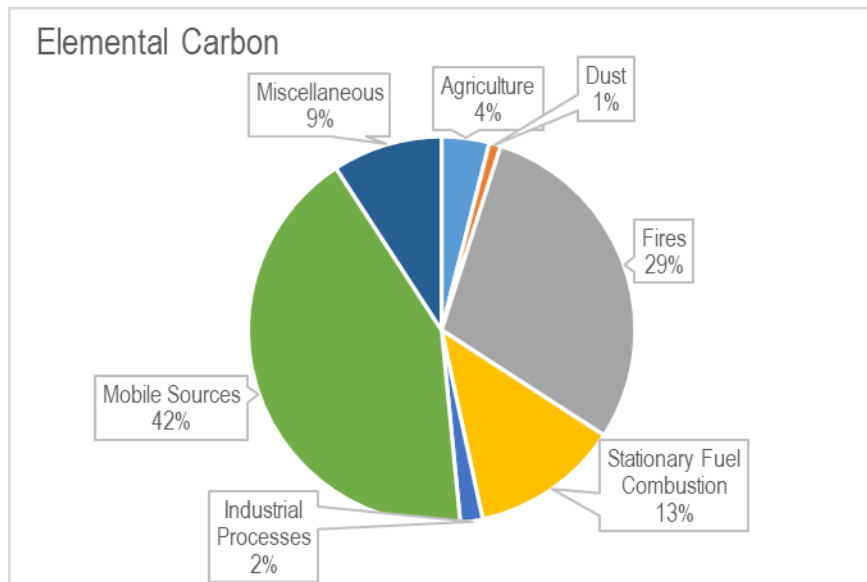
6 **2.1.1.3.1 Sources Contributing to Emissions of Particulate Carbon**

7 Of the directly emitted components of PM_{2.5}, emissions of elemental (or black) carbon
8 and organic carbon often make up the largest percentage of directly emitted PM_{2.5} mass. Figure
9 2-6 illustrates the sources that contribute to national emissions of elemental and organic carbon
10 based on the 2017 NEI. The top panel of Figure 2-6 shows that fires account for most (i.e., 63%)
11 of the 1.8 million tons of particulate OC emissions estimated in the 2017 NEI, while the bottom
12 panel of Figure 2-6 shows that fires and mobile sources (mostly diesel sources) contribute 71%
13 of the estimated ~ 284,000 tons of particulate EC in the 2017 NEI. It should be noted that the
14 fraction of EC to PM_{2.5} was lower in the 2017 NEI compared to the 2014 NEI, owing to a
15 significantly lower contribution of EC from fires in the 2017 NEI compared to previous NEIs.
16 This change in the EC fraction resulted from an in-house research program to investigate the
17 PM_{2.5} chemical composition of the emissions from fires burning different fuels and in different
18 combustion phases. It should be noted that the OC contributions on a percentage basis increased
19 in accordance with the EC decreases. While these results have not yet been directly published,
20 this information has been acknowledged and used in other EPA analyses (Kelly et al., 2019b,
21 Figure 13).

22



1



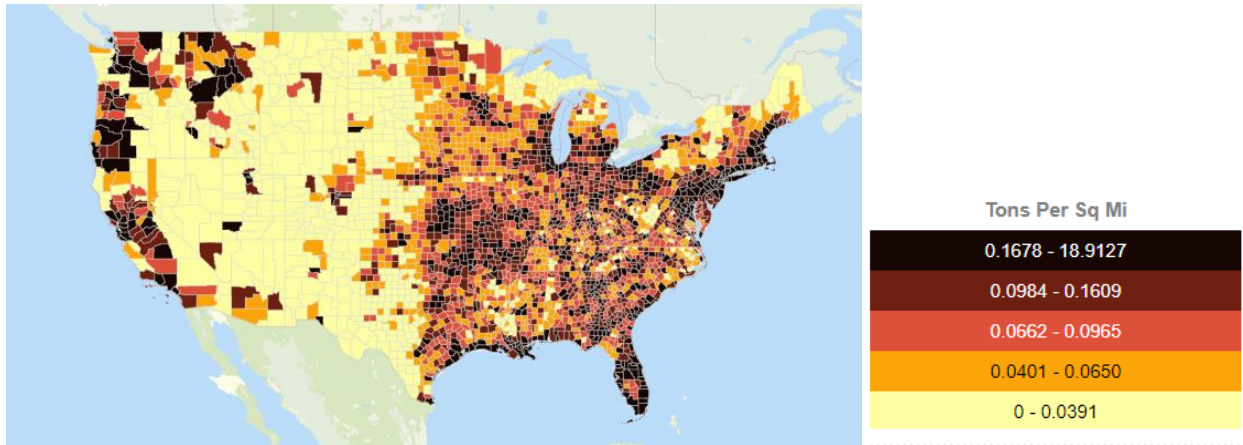
2

3

4

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

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



8
 9 **Figure 2-7. 2017 NEI Elemental Carbon Emissions Density Map, tons per square mile.**

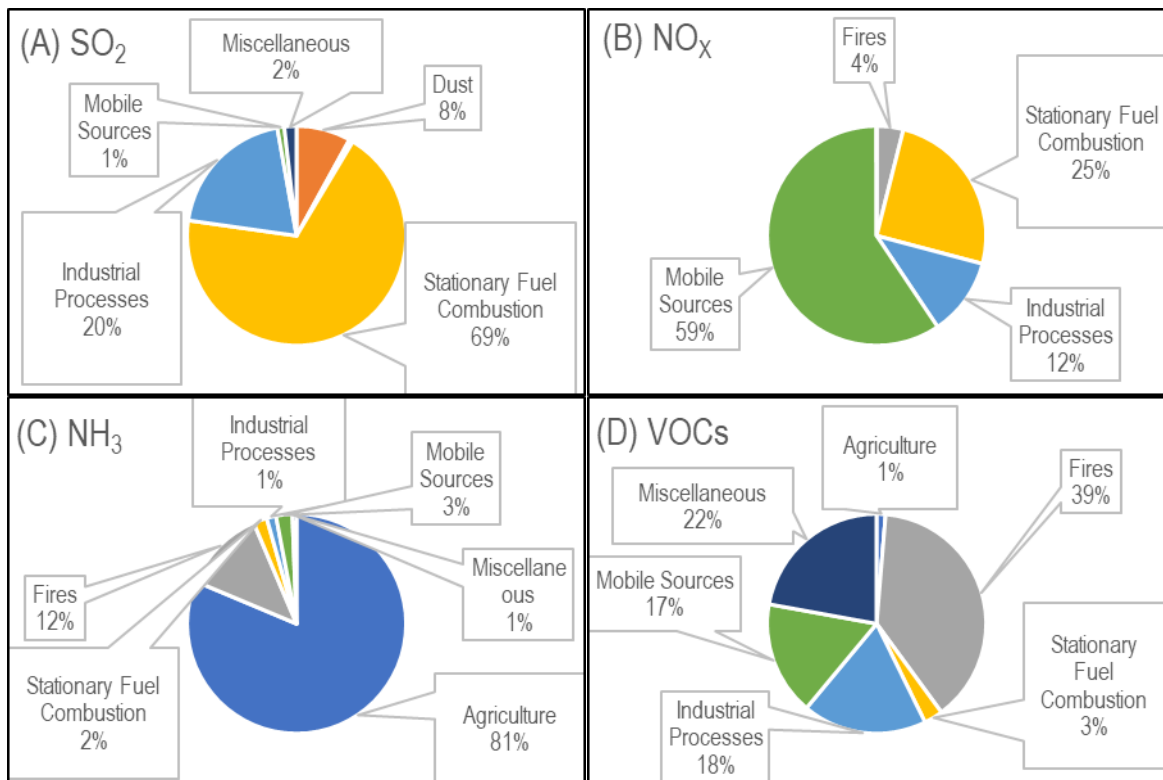
10 **2.1.1.3.2 Sources Contributing to Emissions of Precursor Gases**

11 As discussed further in the 2019 ISA (U.S. EPA, 2019b, section 2.3.2.1), secondary PM
 12 is formed in the atmosphere by photochemical oxidation reactions of both inorganic and organic
 13 gas-phase precursors. Precursor gases include SO₂, NO_x, and volatile organic compound (VOC)
 14 gases of anthropogenic or natural origin (U.S. EPA, 2019b, section 2.3.2.1). Anthropogenic SO₂
 15 and NO_x are the predominant precursor gases in the formation of secondary PM_{2.5}, and ammonia
 16 also plays an important role in the formation of nitrate PM by neutralizing sulfuric acid and nitric
 17 acid. In addition, atmospheric oxidation of VOCs, both anthropogenic and biogenic, is an
 18 important source of organic aerosols, particularly in summer. The semi-volatile and non-volatile
 19 products of VOC oxidation reactions can condense onto existing particles or can form new
 20 particles (U.S. EPA, 2009, section 3.3.2; U.S. EPA, 2019b, section 2.3.2).

21 Emissions of each of the precursor gases noted above are estimated in the NEI and have
 22 unique source signatures at the national level. Figure 2-8 illustrates the source contributions at
 23 the national level for these PM_{2.5} precursor gases. As shown in Panel A in Figure 2-8, stationary
 24 fuel combustion sources contribute nearly 70% of the estimated total of 2.8 million tons of
 25 national SO₂ national emissions. Within this source category, nearly all of the SO₂ emitted to the
 26 atmosphere comes from electricity generating units, or EGUs. Anthropogenic NO_x emissions,
 27 shown in panel B, are emitted by a range of combustion sources, including mobile sources (59%)

1 and stationary fuel combustion sources (25%). In the 2017 NEI, there is an estimated total of
 2 10.3 million tons of NO_x emitted. Of the total estimated 4.3 million tons of anthropogenic
 3 ammonia (NH₃) emissions shown in panel C of Figure 2-8, NH₃ emissions are dominated by the
 4 agriculture source categories. In these categories, NH₃ is predominantly emitted by livestock
 5 waste from animal husbandry operations (56%) and fertilizer application (25%). In urban areas,
 6 on-road mobile sources may also contribute significantly to NH₃ emissions (U.S. EPA, 2019b,
 7 Figure 2-3; Sun et al., 2014; U.S. EPA, 2020). Of the estimated 17.2 million tons of VOC
 8 emissions from anthropogenic sources, fires (39%) and “miscellaneous” (22%)⁴ are the highest
 9 contributors, followed by mobile sources (17%) and industrial processes (18%), as shown in
 10 Figure 2-8 panel D. It should be noted that as these traditional combustion sources of VOCs are
 11 reduced by regulations and controls, new non-combustion sources, such as volatile chemical
 12 products (solvents) are emerging as key contributors to anthropogenic VOC totals in some parts
 13 of the country, and particularly in urban corridors. In addition, biogenic sources (not shown in
 14 Figure 2-8) are significant contributors to both VOC and NO_x emissions.

15



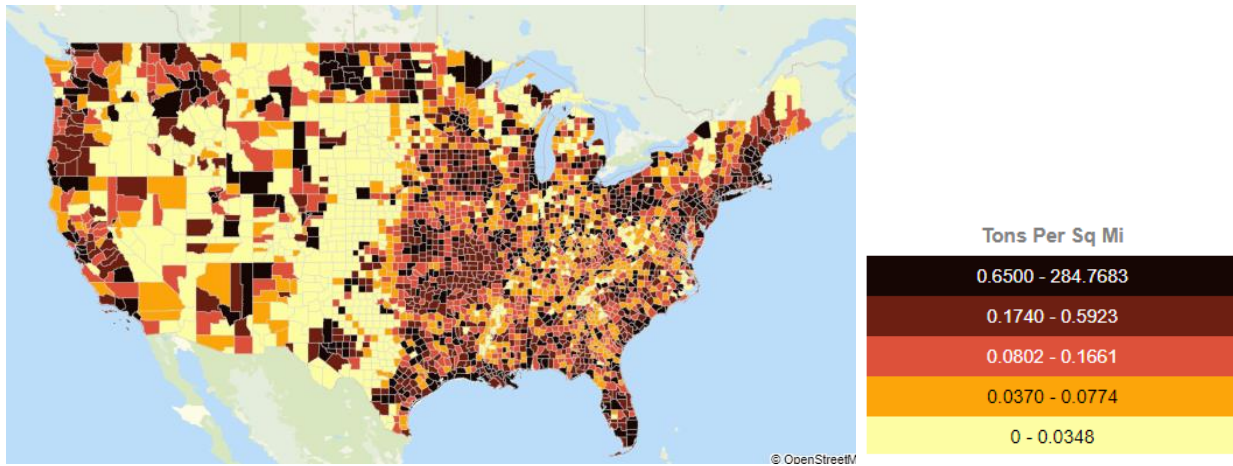
16

17
 18 **Figure 2-8. Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B),**
 19 **ammonia (panel C), and volatile organic compounds (panel D) national emissions by**
 20 **source sectors.** (Source: 2017 NEI). All graphics only show anthropogenic contributions.

⁴ The “miscellaneous” category includes such things as solvents, commercial cooking and waste disposal.

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

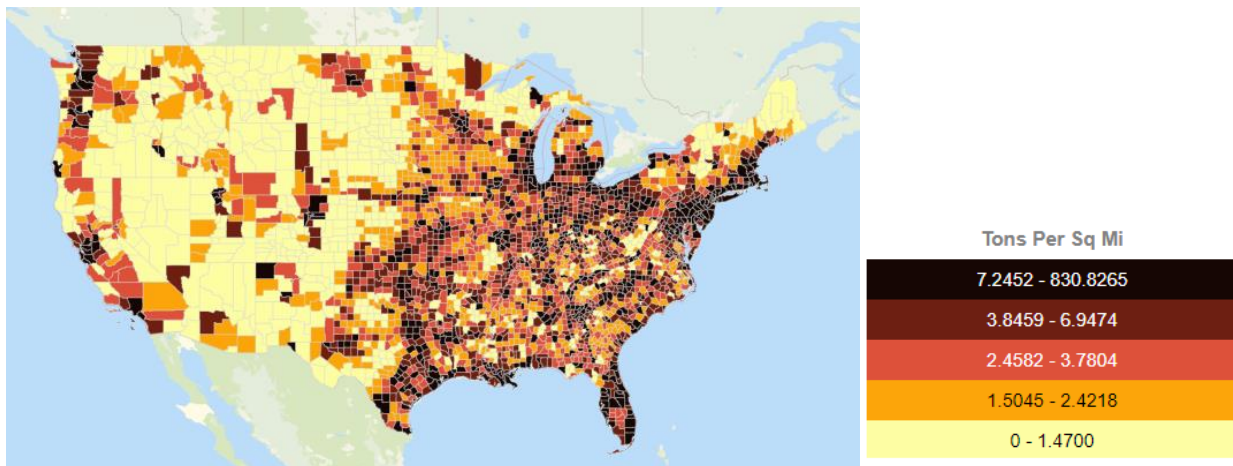
3



4

5 **Figure 2-9. SO₂ Emissions Density Map, tons per square mile.**

6



7

8 **Figure 2-10. NO_x Emissions Density Map, tons per square mile.**

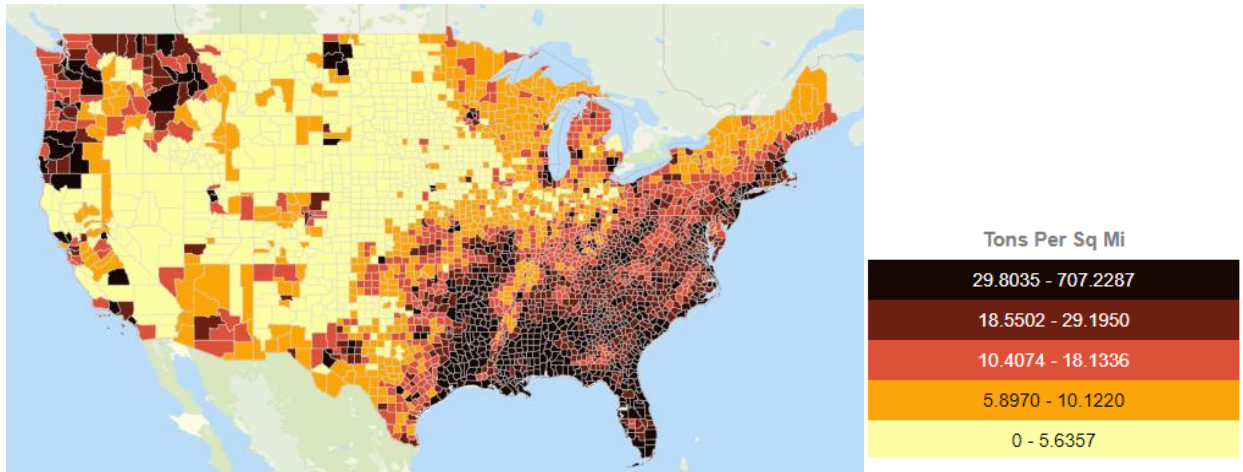
9



10

11 **Figure 2-11. NH₃ Emissions Density Map, tons per square mile.**

12



1
2 **Figure 2-12. Anthropogenic (including wildfires) VOC Emissions Density Map, tons per**
3 **square mile.**

4 **2.1.1.3.3 Uncertainty in Emission Estimates**

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

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

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

2.2 AMBIENT PM MONITORING METHODS AND NETWORKS

To promote uniform enforcement of the air quality standards set forth under the CAA and to achieve the degree of public health and welfare protection intended for the NAAQS, the EPA established PM Federal Reference Methods (FRMs)⁵ for both PM₁₀ and PM_{2.5} (40 CFR Appendix J and L to Part 50) and performance requirements for approval of Federal Equivalent Methods (FEMs) (40 CFR Part 53). Amended following the 2006 and 2012 PM NAAQS reviews, the current PM monitoring network relies on FRMs and automated continuous FEMs, in part to support changes necessary for implementation of the revised PM standards. The requirements for measuring ambient air quality and reporting ambient air quality data and related information are the basis for 40 CFR Appendices A through E to Part 58.

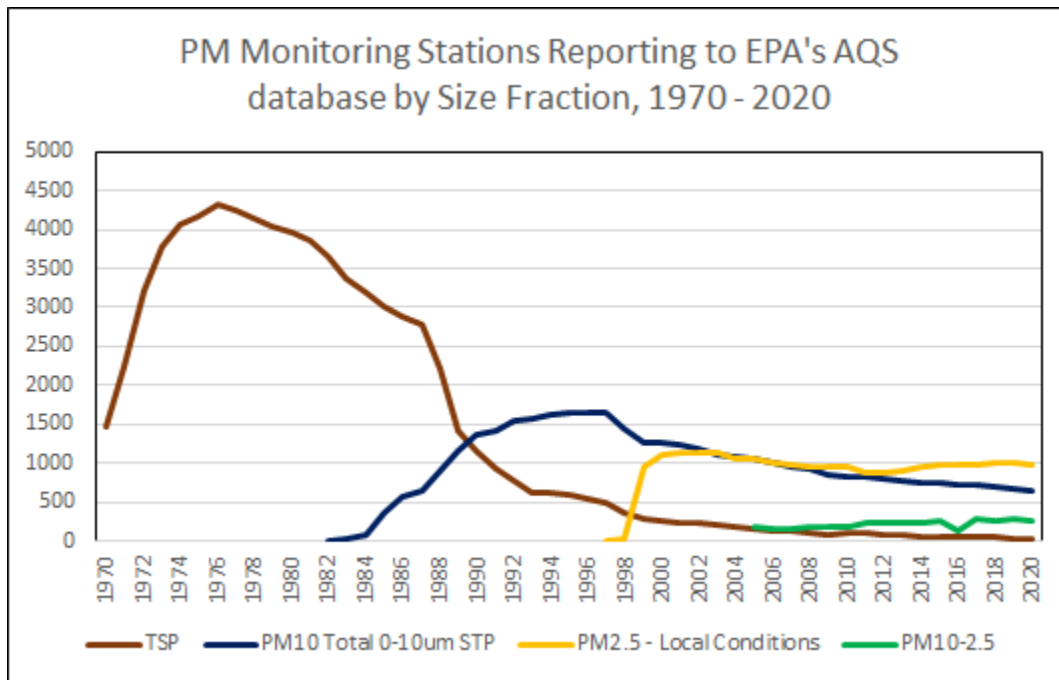
The EPA and its partners at state, local, and tribal monitoring agencies manage and operate the nation's ambient air monitoring networks. The EPA provides minimum monitoring requirements for criteria pollutants and related monitoring (e.g., the Chemical Speciation Network (CSN)), including identification of an FRM for criteria pollutants and guidance documents to support implementation and operation of the networks. Monitoring agencies carry out and perform ambient air monitoring in accordance with the EPA's requirements and guidance as well as often meeting their own state monitoring needs that may go beyond the minimum federal requirements. Data from the ambient air monitoring networks are available from two national databases: 1) the Air Quality System (AQS) database, which is the EPA's long-term repository of ambient air monitoring data and 2) the AirNow database, which provides near real-time data used in public reporting and forecasting of the Air Quality Index (AQI).⁶

The EPA and monitoring agencies manage and operate robust national networks for both PM₁₀ and PM_{2.5}, as these are the two measurement programs directly supporting the PM NAAQS. PM₁₀ measurements are based on gravimetric mass, while PM_{2.5} measurements include gravimetric mass and chemical speciation. A smaller network of stations is operating and reporting data for PM_{10-2.5} gravimetric mass and a few monitors are operated to support special projects, including pilot studies, for continuous speciation and particle count data. Monitoring networks and additional monitoring efforts for each of the various PM size fractions and for PM

⁵ FRMs provide the methodological basis for comparison to the NAAQS and also serve as the "gold standard" for the comparison of other methods being reviewed for potential approval as equivalent methods. The EPA keeps a complete list of designated reference and equivalent methods available on its Ambient Monitoring Technology Information Center (AMTIC) website (<https://www.epa.gov/amtic/air-monitoring-methods-criteria-pollutants>).

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

1 composition are discussed below.⁷ Section 2.2.1 provides information on monitoring for total
 2 suspended particulates (TSP), section 2.2.2 provides information on monitoring for PM₁₀, section
 3 2.2.3 provides information on monitoring PM_{2.5}, section 2.2.4 provides information on
 4 monitoring for PM_{10-2.5}, and section 2.2.5 provides information on additional PM metrics. All
 5 sampler and monitor counts provided in these sections are based on data submitted to the EPA
 6 for calendar year 2020, unless otherwise noted. Figure 2-13 below illustrates the changes in PM
 7 monitoring stations reporting to the EPA’s AQS database by size fraction since 1970.
 8



9
 10 **Figure 2-13. PM Monitoring stations reporting to EPA’s AQS database by PM size**
 11 **fraction, 1970-2020.**

12 **2.2.1 Total Suspended Particulates (TSP) Sampling**

13 The EPA first established NAAQS for PM in 1971, based on the original air quality
 14 criteria document (DHEW, 1969). The reference method specified for determining attainment of
 15 the original standards was the high-volume sampler, which collects PM up to a nominal size of
 16 25 to 45 µm (referred to as total suspended particles or TSP). TSP was replaced by PM₁₀ as the
 17 indicator for the PM NAAQS in the 1987 final rule (52 FR 24854, July 1, 1987). TSP sampling
 18 remains in operation at a limited number of locations primarily to provide aerosol collection for
 19 TSP lead (Pb) analysis as well as for instances where a state may continue to have state standards
 20 for TSP. The size of the TSP network peaked in the mid-1970s when over 4,300 TSP samplers

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

1 were in operation. As of 2020, there were 104 TSP samplers still in operation as part of the Pb
2 monitoring program; of these, 25 also report TSP mass.

3 **2.2.2 PM₁₀ Monitoring**

4 To support the 1987 PM₁₀ NAAQS, the EPA and its state and local partners implemented
5 the first size-selective PM monitoring network in 1990 with the establishment of a PM₁₀ network
6 consisting of mainly high-volume samplers. The network design criteria emphasize monitoring at
7 middle⁸ and neighborhood⁹ scales to effectively characterize the emissions from both mobile and
8 stationary sources, although not ruling out microscale¹⁰ monitoring in some instances (40 CFR
9 Part 58 Appendix D, 4.6 (b)). The PM₁₀ monitoring network peaked in size in 1995 with 1,665
10 stations reporting data.

11 In 2020, there were 680 PM₁₀ stations in operation to support comparison of the PM₁₀
12 data to the NAAQS, trends, and reporting and forecasting of the AQI. Though the PM₁₀ network

⁸ For PM₁₀, middle-scale is defined as follows: Much of the short-term public exposure to PM₁₀ is on this scale and on the neighborhood scale. People moving through downtown areas or living near major roadways or stationary sources, may encounter particulate pollution that would be adequately characterized by measurements of this spatial scale. Middle scale PM₁₀ measurements can be appropriate for the evaluation of possible short-term exposure public health effects. In many situations, monitoring sites that are representative of micro-scale or middle-scale impacts are not unique and are representative of many similar situations. This can occur along traffic corridors or other locations in a residential district. In this case, one location is representative of a neighborhood of small-scale sites and is appropriate for evaluation of long-term or chronic effects. This scale also includes the characteristic concentrations for other areas with dimensions of a few hundred meters such as the parking lot and feeder streets associated with shopping centers, stadia, and office buildings. In the case of PM₁₀, unpaved or seldomly swept parking lots associated with these sources could be an important source in addition to the vehicular emissions themselves.

⁹ For PM₁₀, neighborhood scale is defined as follows: Measurements in this category represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. In some cases, a location carefully chosen to provide neighborhood scale data would represent not only the immediate neighborhood but also neighborhoods of the same type in other parts of the city. Neighborhood scale PM₁₀ sites provide information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for extended periods. Neighborhood scale data could provide valuable information for developing, testing, and revising models that describe the larger-scale concentration patterns, especially those models relying on spatially smoothed emission fields for inputs. The neighborhood scale measurements could also be used for neighborhood comparisons within or between cities.

¹⁰ For PM₁₀, microscale is defined as follows: This scale would typify areas such as downtown street canyons, traffic corridors, and fence line stationary source monitoring locations where the general public could be exposed to maximum PM₁₀ concentrations. Microscale particulate matter sites should be located near inhabited buildings or locations where the general public can be expected to be exposed to the concentration measured. Emissions from stationary sources such as primary and secondary smelters, power plants, and other large industrial processes may, under certain plume conditions, likewise result in high ground level concentrations at the microscale. In the latter case, the microscale would represent an area impacted by the plume with dimensions extending up to approximately 100 meters. Data collected at microscale sites provide information for evaluating and developing hot spot control measures.

1 is relatively stable, monitoring agencies may continue divesting of some of the PM₁₀ monitoring
2 stations where concentration levels are low relative to the NAAQS.

3 While the PM₁₀ network is national in scope, there are areas of the west, such as
4 California and Arizona, with substantially higher PM₁₀ station density than the rest of the
5 country. In the PM₁₀ mass network, 385 of the stations operate automated continuous mass
6 monitors approved as FEMs and 295 operate FRMs. About 30 of the PM₁₀ stations have
7 collocation with both continuous FEMs and FRMs. More than half of the PM₁₀ stations with
8 FRMs operate on a sample frequency of one in every sixth day, with about 55 stations operating
9 every third day and another 55 stations operating every day.

10 **2.2.3 PM_{2.5} Monitoring**

11 To support the 1997 PM_{2.5} NAAQS, the first PM standard with PM_{2.5} as an indicator, the
12 EPA and states implemented a PM_{2.5} network consisting of ambient air monitoring sites with
13 mass and/or chemical speciation measurements. Network operation began in 1999 with nearly
14 1,000 monitoring stations operating FRMs to measure fine particle mass. The PM_{2.5} monitoring
15 program remains one of the major ambient air monitoring programs operated across the country.

16 For most urban locations, PM_{2.5} monitors are sited at the neighborhood scale,¹¹ where
17 PM_{2.5} concentrations are reasonably homogeneous throughout an entire urban sub-region. In each
18 CBSA with a monitoring requirement, at least one PM_{2.5} monitoring station representing area-
19 wide air quality is to be sited in an area of expected maximum concentration. Sites that represent
20 relatively unique microscale, localized hot-spot, or unique middle scale impact sites are only
21 eligible for comparison to the 24-hour PM_{2.5} NAAQS.

22 There are three main components of the current PM_{2.5} monitoring program: FRMs, PM_{2.5}
23 continuous mass monitors, and CSN samplers. The FRMs are primarily used for comparison to
24 the NAAQS, but also serve other important purposes such as developing trends and evaluating
25 the performance of PM_{2.5} continuous mass monitors. PM_{2.5} continuous mass monitors are
26 automated methods primarily used to support forecasting and reporting of the AQI, but are also
27 used for comparison to the NAAQS where approved as FEMs. The CSN and related Interagency
28 Monitoring of Protected Visual Environments (IMPROVE) network are used to provide

¹¹ For PM_{2.5}, neighborhood scale is defined as follows: Measurements in this category would represent conditions throughout some reasonably homogeneous urban sub-region with dimensions of a few kilometers and of generally more regular shape than the middle scale. Homogeneity refers to the particulate matter concentrations, as well as the land use and land surface characteristics. Much of the PM_{2.5} exposures are expected to be associated with this scale of measurement. In some cases, a location carefully chosen to provide neighborhood scale data would represent the immediate neighborhood as well as neighborhoods of the same type in other parts of the city. PM_{2.5} sites of this kind provide good information about trends and compliance with standards because they often represent conditions in areas where people commonly live and work for periods comparable to those specified in the NAAQS. In general, most PM_{2.5} monitoring in urban areas should have this scale.

1 chemical composition of the aerosol which serve a variety of objectives. This section provides an
2 overview of each of these components of the PM_{2.5} monitoring program and of recent changes to
3 PM_{2.5} monitoring requirements.

4 **2.2.3.1 Federal Reference Method and Continuous Monitors**

5 As noted above, the PM_{2.5} monitoring network began operation in 1999 with nearly 1,000
6 monitoring stations operating FRMs. The PM_{2.5} FRM network peaked in operation in 2001 with
7 over 1,150 monitoring stations. In the PM_{2.5} network for 2020 there were 527 FRM filter-based
8 samplers that provide 24-hour PM_{2.5} mass concentration data. Of these operating FRMs, 68 are
9 providing daily PM_{2.5} data, 340 every third day, and 119 every sixth day.

10 As of 2020, there are 950 continuous PM_{2.5} mass monitors that provide hourly data on a
11 near real-time basis reporting across the country. A total of 660 of the PM_{2.5} continuous monitors
12 are FEMs and therefore used both for comparison with the NAAQS and to report the AQI.
13 Another 290 monitors not approved as FEMs are operated primarily to report the AQI. These
14 legacy PM_{2.5} continuous monitors were largely purchased prior to the availability of PM_{2.5}
15 continuous FEMs.

16 The first method approved as a continuous PM_{2.5} FEM was the Met One BAM 1020. This
17 method, approved in 2008, accounts for just over a third of the operating PM_{2.5} continuous FEMs
18 in the country. The EPA has approved a total of 11 PM_{2.5} continuous methods as FEMs. Other
19 methods approved as continuous PM_{2.5} FEMs include beta attenuation from multiple instrument
20 manufacturers; optical methods such as the GRIMM and Teledyne T640; and methods
21 employing the Tapered Element Oscillating Microbalance (TEOM) with a Filter Dynamic
22 Measurement System (FDMS) manufactured by Thermo Fisher Scientific.

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

1 Automated PM_{2.5} FEMs include a wide variety of approved methods which can have
2 different measurement principles. Data aggregated across all automated FEMs for years 2018-
3 2020 result in a collocated precision of 12.8%. Bias can be calculated from the reference method
4 audit program and by comparing continuous FEMs to collocated FRMs run by the monitoring
5 agency. The 2018-2020 reference method audit program had a bias of -1.7% with a sample size
6 of 573 audits across all continuous FEMs. Continuous FEMs compared to collocated monitoring
7 agency FRMs were biased higher by 11.5% with a large sample size of 85,539 collocated pairs
8 for 2018-2020 (all cases where both the FRM and continuous FEM are at or above 3.0 µg/m³).
9 When evaluating automated FEMs as individual methods, only two of the seven methods with
10 available collocated precision data met the measurement uncertainty goal and six of the eleven
11 methods met the bias goal. However, for collocated precision data and when considering a
12 requirement for approval of candidate FEMs: “Statistical analyses based on the DQO model
13 show that the precision of a candidate method is not, statistically, very important to annual
14 concentration averages used for NAAQS attainment decisions, but would be important for a
15 daily standard” (71 FR 2620, January 17, 2006) In summary, PM_{2.5} automated FEMs tend to
16 have higher collocated precision than FRMs and tend to have a positive bias relative to state and
17 local operated FRMs.

18 **2.2.3.2 Chemical Speciation and IMPROVE Networks**

19 Due to the complex nature of fine particles, the EPA and states implemented the CSN to
20 better understand the components of fine particle mass at selected locations across the country.
21 The CSN was first piloted at 13 sites in 2000, and after the pilot phase, the program continued
22 with deployment of the Speciation Trends Network (STN) later that year. The CSN ultimately
23 grew to 54 trends sites and peaked in operation in 2005 with 252 stations: the 54 trends stations
24 and nearly 200 supplemental stations. The original CSN program had multiple sampler
25 configurations including the Thermo Andersen RAAS, Met One SASS/SuperSASS, and URG
26 MASS. During the 2000s, the EPA and states worked to align the network to one common
27 sampler for elements and ions, which was the Met One SASS/SuperSASS. In 2005, the CASAC
28 provided recommendations to the EPA for making changes to the CSN. These changes were
29 intended to improve data comparability with the rural IMPROVE carbon concentration data. To
30 accomplish this, the EPA replaced the existing carbon channel sampling and analysis methods
31 with a new modified IMPROVE version III module C sampler, the URG 3000N. Implementation
32 of the new carbon sampler and analysis was broken into three phases starting in May 2007
33 through October 2009.

1 In the 2020 PM_{2.5} CSN, long-term measurements are made at about 75 largely urban
2 locations comprised of either the STN or the National Core (NCore) network.¹² NCore is a
3 multipollutant network measuring particles, gases, and basic meteorology that has been in formal
4 operation since January 1, 2011. Particle measurements made at NCore include PM_{2.5} filter-based
5 mass, which is largely the FRM, except in some rural locations that utilize the IMPROVE
6 program PM_{2.5} mass filter-based measurement; PM_{2.5} speciation using either the CSN program or
7 IMPROVE program; and PM_{10-2.5} mass utilizing an FRM, FEM or IMPROVE for some of the
8 rural locations. As of 2020, the NCore network includes a total of 78 stations of which 63 are in
9 urban or suburban stations designed to provide representative population exposure and another
10 15 rural stations designed to provide background and transport information. The NCore network
11 is deployed in all 50 States, DC, and Puerto Rico with at least one station in each state and two or
12 more stations in larger population states (California, Florida, Illinois, Michigan, New York,
13 North Carolina, Ohio, Pennsylvania, and Texas).

14 Both the STN and NCore networks are intended to remain in operation indefinitely. The
15 CSN measurements at NCore and STN stations operate every third day. Six of these stations
16 have collocated sets of CSN samplers where the collocated samplers operate every sixth day to
17 provide precision calculations of each chemical species measured. Another approximately 70
18 CSN stations, known as supplemental sites, are intended to be potentially less permanent
19 locations used to support State Implementation Plan (SIP) development and other monitoring
20 objectives.¹³ Supplemental CSN stations typically operate every sixth day. In January 2015, 38
21 supplemental CSN stations that are largely located in the eastern half of the country stopped
22 operations to ensure a sustainable CSN network moving forward.¹⁴

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

¹² In most cases where a city has an STN station, it is located at the same site as the NCore station. In a few cases, a city may have an STN station located at a different location than the NCore station.

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

¹⁴ Based on assessments of the CSN network and IMPROVE protocol sites, monitoring resources were redistributed to focus on new or high priorities. More information on the CSN and IMPROVE protocol assessments is available at <https://www.epa.gov/amtic/csn-and-improve-protocol-network-assessment>.

¹⁵ Recognizing the importance of visual air quality, Congress included legislation in the 1977 Clean Air Act to prevent future and remedy existing visibility impairment in Class I areas. To aid the implementation of this legislation, the IMPROVE program was initiated in 1985 and substantially expanded in 2000-2003. This program implemented an extensive long-term monitoring program to establish the current visibility conditions, track changes in visibility and determine causal mechanism for the visibility impairment in the National Parks and Wilderness Areas. For more information, see <https://vista.cira.colostate.edu/Improve/>.

1 visibility in Class I areas¹⁶ as well as many other rural and some urban areas. As of 2018, the
2 IMPROVE network includes 110 monitoring locations that are part of the base network
3 supporting regional haze and another 38 locations operated as IMPROVE protocol sites where a
4 monitoring agency has requested participation in the program. These IMPROVE protocol sites
5 operate the same way as the IMPROVE program, but they may serve several monitoring
6 objectives (i.e., the same objectives as the CSN) and are not explicitly tied to the Regional Haze
7 Program. Samplers at IMPROVE stations operate every third day. In January 2016, eight
8 IMPROVE protocol stations stopped operating to ensure a sustainable IMPROVE program
9 moving forward. Details on the process and outcomes of the CSN supplemental and IMPROVE
10 protocol assessments used to identify sites that would no longer be funded are available on a
11 website.¹⁷ Together, the CSN and IMPROVE data provide chemical species information for fine
12 particles that are critical for use in health and epidemiologic studies to help inform reviews of the
13 primary PM NAAQS. CSN and IMPROVE data can also be used to better understand visibility
14 through calculation of light extinction using the IMPROVE algorithm¹⁸ to support reviews of the
15 secondary PM NAAQS.

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

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

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

¹⁸ The IMPROVE algorithm is an equation to estimate light extinction based on the measured concentration of several PM components and is used to track visibility progress in the Regional Haze Rule. More information about the IMPROVE algorithm is at available at: <http://vista.cira.colostate.edu/Improve/the-improve-algorithm>.

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

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

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

3 **2.2.3.3 Recent Changes to PM_{2.5} Monitoring Requirements**

4 Key changes made to the EPA's monitoring requirements as a result of the 2012 PM
5 NAAQS review included the addition of PM_{2.5} monitoring at near-road locations in core-based
6 statistical areas (CBSAs) over 1 million in population; the clarification of terms used in siting of
7 PM_{2.5} monitors and their applicability to the NAAQS; and the provision of flexibility on data
8 uses to monitoring agencies where their PM_{2.5} continuous monitors are not providing data that
9 meets the performance criteria used to approve the continuous method as an FEM. The addition
10 of PM_{2.5} monitoring at near-road locations was phased in from 2015 to 2017. On January 1,
11 2015, 22 CBSAs with a population of 2.5 million or more were required to have a PM_{2.5} FRM or
12 FEM operating at a near-road monitoring station. On January 1, 2017, 30 CBSAs with a
13 population between 1 million and 2.5 million were required to have a PM_{2.5} FRM or FEM
14 operating at a near-road monitoring station.

15 The terms clarified as a part of the 2012 rulemaking ensure consistency with all other
16 NAAQS and long-standing definitions used by the EPA (78 FR 3234, January 15, 2013). The
17 flexibility provided to monitoring agencies ensures that the incentives of utilizing PM_{2.5}
18 continuous monitors (e.g., efficiencies in operation and availability of hourly data in near-real
19 time) are realized without having potentially poor performing data being used in situations where
20 the data is not applicable to the NAAQS (78 FR 3241, January 15, 2013).

21 **2.2.4 PM_{10-2.5} Monitoring**

22 In the 2006 PM NAAQS review, the EPA promulgated a new FRM for the measurement
23 of PM_{10-2.5} mass in ambient air. Although the standard for coarse particles uses a PM₁₀ indicator,
24 a new FRM for PM_{10-2.5} mass was developed to provide a basis for approving FEMs and to
25 promote the gathering of scientific data to support future reviews of the PM NAAQS. The PM<sub>10-
26 2.5</sub> FRM (or approved FEMs, where available) was implemented at required NCore stations by
27 January 1, 2011. In addition to NCore, there are other collocated PM₁₀ and PM_{2.5} low-volume
28 FRMs operating across the country that are essentially providing the PM_{10-2.5} FRM measurement
29 by the difference method.

30 PM_{10-2.5} measurements are currently performed across the country at NCore stations,
31 IMPROVE monitoring stations, and at a few additional locations where state or local agencies
32 choose to operate a PM_{10-2.5} method. For urban NCore stations and other State and Local Air

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

1 Monitoring Stations (SLAMS) the method employed is either a PM_{10-2.5} FRM, which is
2 performed using a low-volume PM₁₀ FRM collocated with a low volume PM_{2.5} FRM of the same
3 make and model, or FEMs for PM_{10-2.5}, including filter-based dichotomous methods and
4 continuous methods of which several makes and models are approved. Filter-based PM_{10-2.5}
5 measurements at NCore (i.e., the FRM or dichotomous filter-based FEM) operate every third
6 day, while continuous methods have data available every hour of every day. PM_{10-2.5} filter-based
7 methods at other SLAMS typically operate every third or sixth day. For IMPROVE, which is
8 largely a rural network, PM_{10-2.5} measurements are made with two sample channels; one each for
9 PM₁₀ and PM_{2.5}. All IMPROVE program samplers operate every third day. All together there
10 were 287 stations in 2020 where PM_{10-2.5} data were being reported to the AQS database.

11 There is no operating chemical speciation network for characterizing the specific
12 components of coarse particles. In 2015, Washington University at St. Louis, under contract to
13 the EPA, reported on a coarse particle speciation pilot study with several objectives aimed at
14 addressing this issue, such as evaluating a coarse particle species analyte list and evaluating
15 sampling and analytical methods (U.S. EPA, 2015). The coarse particle speciation pilot study
16 provides useful information for any organization wishing to pursue coarse particle speciation.

17 **2.2.5 Additional PM Measurements and Metrics**

18 There are additional PM measurements and metrics made at a much smaller number of
19 stations. These measurements may be associated with special projects or are complementary
20 measurements to other networks where the monitoring agency has prioritized having the
21 measurements. None of these measurements are required by regulation. They include PM
22 measurements such as particle counts, continuous carbon, and continuous sulfate.

23 The EPA and state and local agencies have also been working together to pilot additional
24 PM methods at near-road monitoring stations that may be of interest to data users. These
25 methods include such techniques as particle counters, particle size distribution, and black carbon
26 by aethalometer. These methods and their rationale for use at near-road monitoring stations are
27 described in a Technical Assistance Document (TAD) on NO₂ near-road monitoring (U.S. EPA,
28 2012, section 16).

29 Aethalometer measurements of the concentration of optically absorbing particles have
30 been submitted to AQS for many years. Data uses include characterizing black carbon and wood
31 smoke. Ambient air monitoring stations that may have aethalometers include some of the near-
32 road monitoring stations and National Air Toxics Trends Stations (NATTS). In 2020, data from
33 72 monitoring sites across the county were reported from aethalometers and other related
34 commercially available continuous carbon analyzers. While aethalometer and related continuous

1 carbon data are available at high time resolutions (e.g., 5-minute data), they are typically
2 reported to the AQS database in 1-hour periods.

3 Continuous elemental and organic carbon data were monitored at select locations
4 participating in a pilot of the Sunset EC/OC analyzer as well as a few additional sites that were
5 already operating before the EPA initiated the pilot study.²² The Sunset EC/OC analyzer
6 provides high-time-resolution carbon data, typically every hour, but in some remote locations the
7 instrument is programmed to run every two hours to ensure collection of enough aerosol. The
8 data from the Sunset EC/OC analyzer was compared to filter-based carbon methods from the
9 carbon channel of the CSN program. The Sunset EC/OC analyzer was operated at each of the
10 study sites for at least three years. Results from this pilot study are available in an EPA report
11 (U.S. EPA, 2019a). A key finding from the study suggests that when the Sunset instrument was
12 working well, OC and optical EC were comparable to CSN OC and EC; however, the time and
13 resources needed to keep a Sunset analyzer operational did not merit replacement of CSN OC
14 and EC measurements.

15 As of 2020, continuous sulfate is measured at two remaining monitoring sites, one each
16 in Maine and North Carolina. Several other stations have historical data but are no longer
17 monitoring continuous sulfate. Discontinued monitoring efforts for continuous sulfate is likely an
18 outcome of the significantly lower sulfate concentrations throughout the east where these
19 methods were operated. The continuous sulfate analyzer provides hourly data and these data can
20 be readily compared to 24-hour sulfate data which are collected from the ion channel in both the
21 CSN and IMPROVE programs.

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

²² The six sites that participated in the study were Washington, DC; Chicago, IL; St. Louis, MO; Houston, TX; Las Vegas, NV; and Los Angeles, CA.

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

2.3 AMBIENT AIR CONCENTRATIONS

This section summarizes available information on recent ambient PM concentrations. Section 2.3.1 presents trends in emissions of PM and precursor gases, while section 2.3.2 presents trends in monitored ambient concentrations of PM in the U.S. Section 2.3.3 discusses approaches for predicting ambient PM_{2.5} by hybrid modeling approaches.

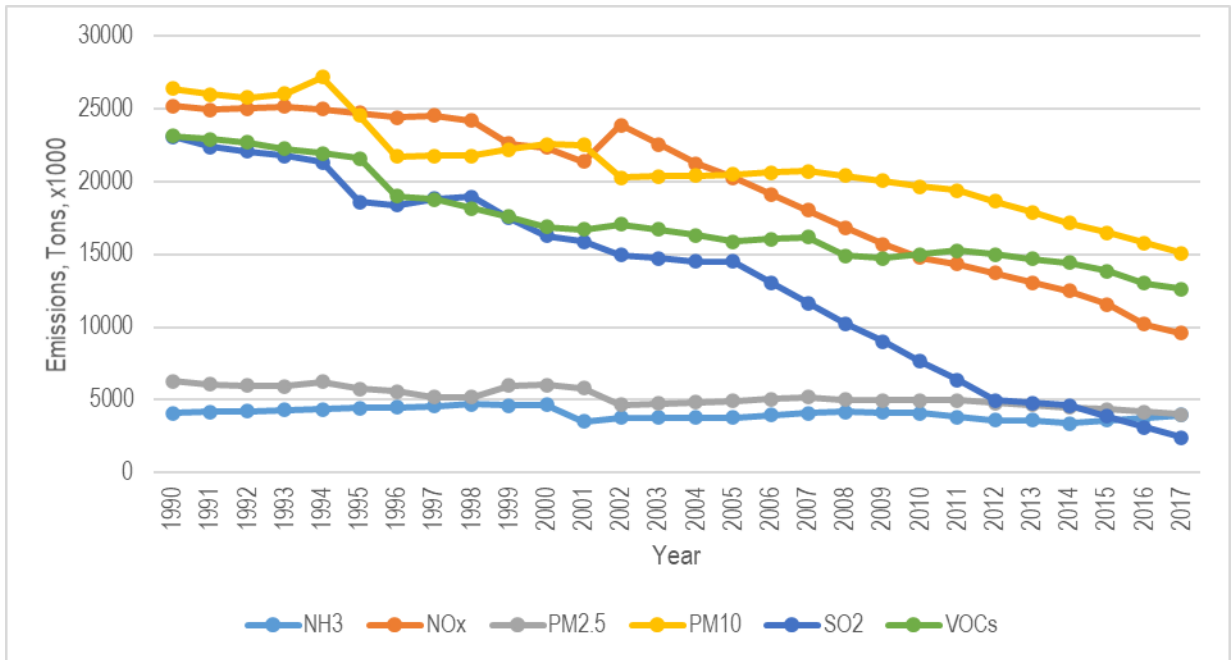
2.3.1 Trends in Emissions of PM and Precursor Gases

Direct emissions of PM have remained relatively unchanged in recent years, while emissions of some precursor gases have declined substantially.²⁴ As illustrated in Figure 2-14,²⁵ from 1990 to 2017, SO₂ emissions have undergone the largest declines while NH₃ emissions have undergone the smallest change. Declining SO₂ emissions during this time period are primarily a result of reductions at stationary sources such as EGUs, with substantial reductions also from mobile sources (U.S. EPA, 2019b, section 2.3.2.1). In more recent years (i.e., 2002 to 2017), emissions of SO₂ and NO_x have undergone the largest declines, while direct PM_{2.5} and NH₃ emissions have undergone the smallest changes, as shown in Table 2-1. Regional trends in emissions can differ from the national trends illustrated in Figure 2-14 and Table 2-1.²⁶ For example, Hand et al. (2012) studied reductions in EGU-related annual SO₂ emissions during the 2001-2010 period and found that while SO₂ emissions decreased throughout the U.S. by an average of 6.2% per year, the amount of change varied across the U.S. with the largest percent reductions in the western U.S. at 20.1% per year.

It should be noted that the reductions shown in PM_{2.5} and PM₁₀ emissions in Figure 2-14, a Table 2-1, and any subsequent discussions of emission trends are most likely due to changes in the methods used by the EPA to estimate emissions for source sectors over time. In all likelihood, emissions from dust and fires have increased over this time, which has been noted earlier in this document and mentioned broadly in the literature as well (Pu and Ginoux, 2017; Li et al., 2021; Liu et al., 2014; Schoennagel et al., 2017). It should also be noted that these data (in Figure 2-14 and Table 2-1) do not include emissions from wildfires, and these emissions can fluctuate greatly from year to year.

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

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



1
2 **Figure 2-14. National emission trends of PM_{2.5}, PM₁₀, and precursor gases from 1990 to**
3 **2017.**

4
5 **Table 2-1. Percent Changes in PM and PM precursor emissions in the NEI for the time**
6 **periods 1990-2017 and 2002-2017.**

Pollutant	Percent Change in Emissions: 1990 to 2017	Percent Change in Emissions: 2002 to 2017	Major Sources that contribute to changes over time
NH ₃	-3.1%	+5.6%	Agricultural Sources (Fertilizer and Livestock Waste), Fires
NO _x	-62%	-60%	EGUs, Mobile Sources
SO ₂	-90%	-84%	EGUs, other Stationary Sources
VOCs	-45%	-26%	Solvents, Fires, Mobile Sources
PM _{2.5}	-36%	-14%	Dust, Fires
PM ₁₀	-43%	-25%	Dust, Fires

7
8 **2.3.2 Trends in Monitored Ambient Concentrations**

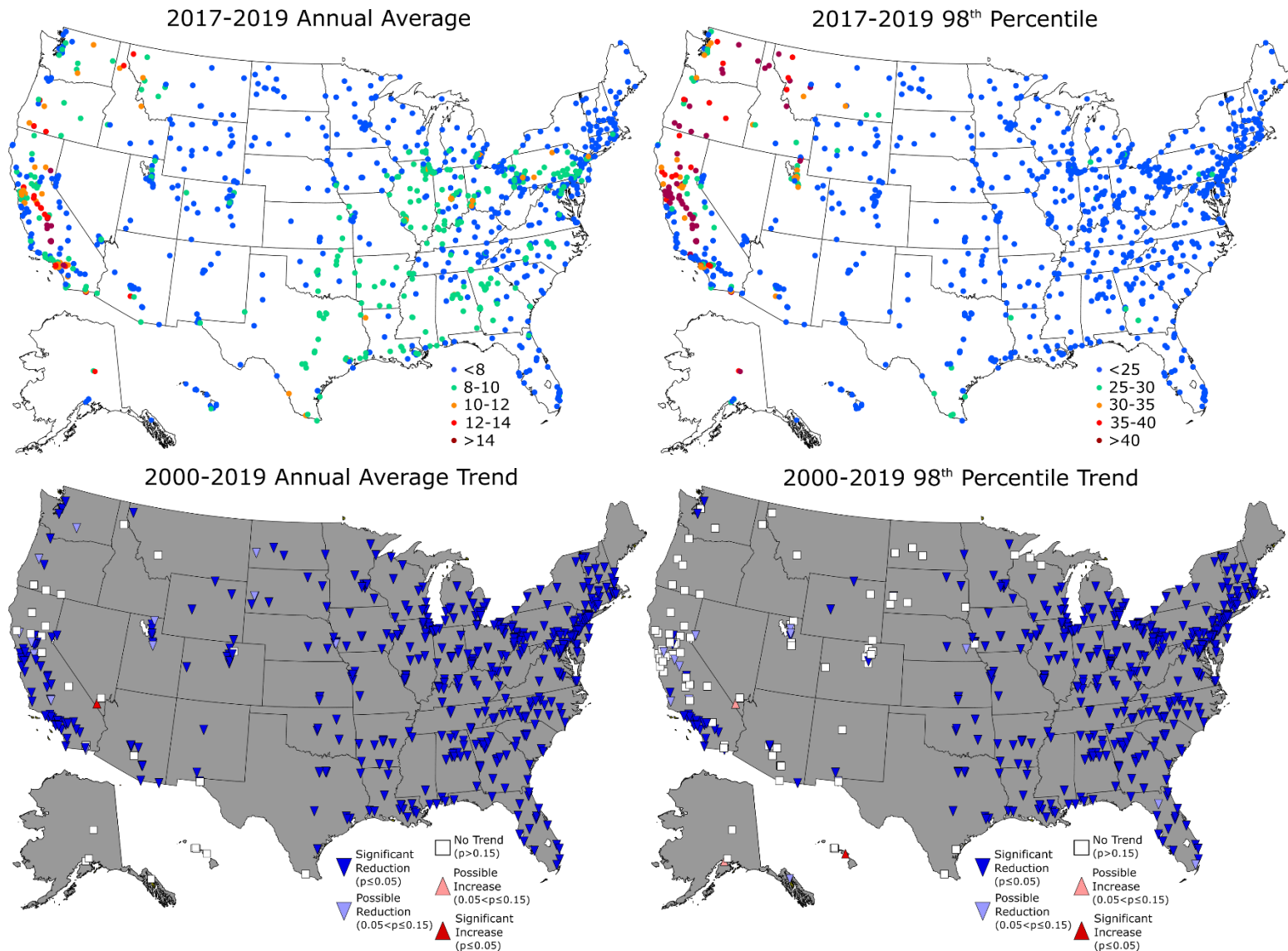
9 **2.3.2.1 National Characterization of PM_{2.5} Mass**

10 At long-term monitoring sites in the U.S., annual PM_{2.5} concentrations from 2017 to 2019
11 averaged 8.0 µg/m³ (with the 10th and 90th percentiles at 5.9 and 10.0 µg/m³, respectively) and
12 the 98th percentiles of 24-hour concentrations averaged 21.3 µg/m³ (with the 10th and 90th
13 percentiles at 14.0 and 29.7 µg/m³, respectively). Figure 2-15 (top panels) shows that the highest

1 ambient PM_{2.5} concentrations occur in the west, particularly in California and the Pacific
2 northwest. Much of the eastern U.S. has lower ambient concentrations, with annual average
3 concentrations generally well below 12.0 µg/m³ and 98th percentiles of 24-hour concentrations
4 generally at or below 30 µg/m³.

5 These concentrations are distinct from design values in part because they include days
6 with episodic events like wildfires and dust storms which can have very high PM_{2.5} and/or PM₁₀
7 concentrations. The EPA’s Exceptional Events Rule (81 FR 68216, October 3, 2016), most
8 recently updated in 2016, describes the process by which these events can be excluded from the
9 design values used for comparison to the NAAQS. For the remainder of Chapter 2, episodic
10 events are included in the calculations of PM concentrations. When design values are discussed
11 in Chapter 2, regionally-concurred exceptional events (as of June 2021) have been excluded from
12 the analysis.²⁷

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



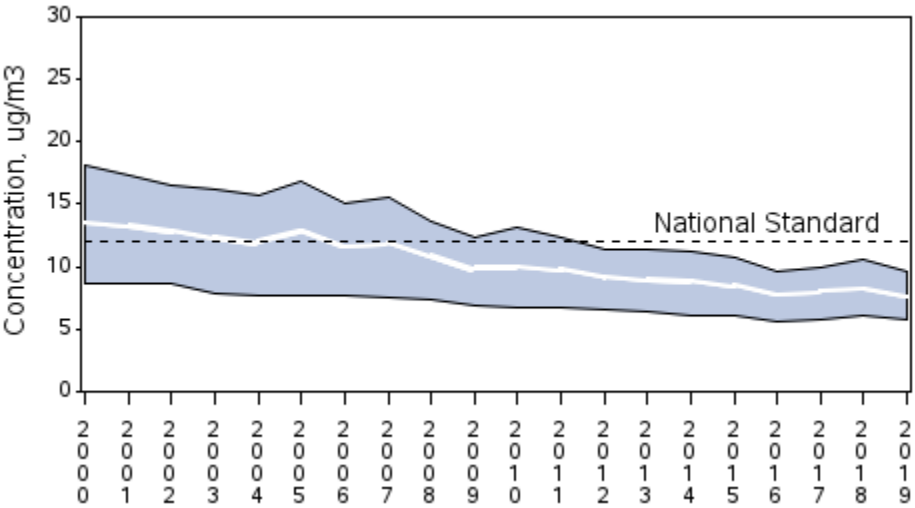
1

2

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

1 Analysis of monthly data indicate distinct peaks in national ambient PM_{2.5} concentrations
 2 during the summer and the winter (U.S. EPA, 2019b, Figure 2-22). Through 2008, the summer
 3 peaks reflected the highest national average PM_{2.5} concentrations. These summer peaks in
 4 ambient PM_{2.5} concentrations were largely a consequence of summertime peaks in SO₂
 5 emissions from power plants in the eastern U.S., and subsequent sulfate formation. However,
 6 substantial reductions in SO₂ emissions (see above and U.S. EPA, 2019b, sections 2.5.1.1.1 and
 7 2.5.2.2.1) have changed this pattern. Starting in 2009, winter peaks in national average PM_{2.5}
 8 concentrations have been higher than those in the summer (U.S. EPA, 2019b, section 2.5.2.2.1).
 9 This pattern is illustrated by data from 2013 to 2015, when average winter PM_{2.5} concentrations
 10 were about 11 µg/m³, average summer concentrations were about 9 µg/m³, and average spring
 11 and fall concentrations were about 7 µg/m³ (Chan et al., 2018).

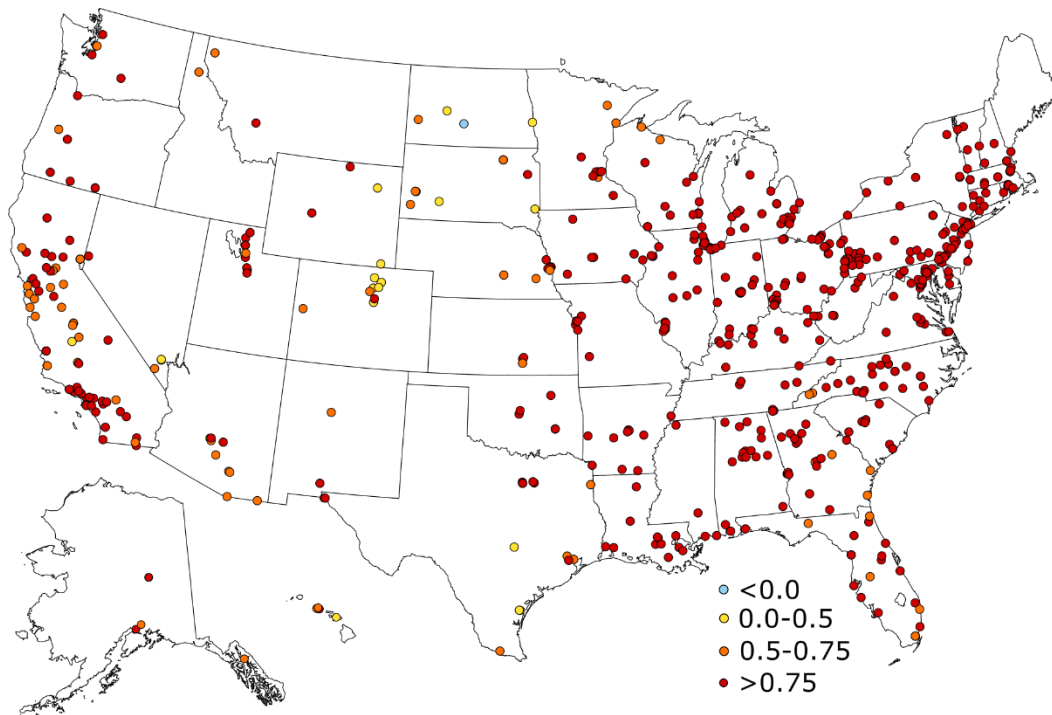
12 The ambient PM_{2.5} concentrations in Figure 2-15 reflect the substantial reductions that
 13 have occurred across much of the U.S. over recent years (Figure 2-15, bottom panels and Figure
 14 2-16). From 2000 to 2019, national annual average PM_{2.5} concentrations have declined from 13.5
 15 µg/m³ to 7.6 µg/m³, a 43% decrease (Figure 2-16).²⁸ These declines have occurred at both urban
 16 and rural monitoring sites, although urban PM_{2.5} concentrations remain consistently higher than
 17 those in rural areas (Chan et al., 2018) due to the so-called “urban increment” of PM_{2.5} from
 18 local sources in an urban area that is additive to the regional and natural background PM_{2.5}
 19 concentrations.



20
 21 **Figure 2-16. Seasonally-weighted annual average PM_{2.5} concentrations in the U.S. from**
 22 **2000 to 2019 (406 sites).** (Note: The white line indicates the mean concentration while the
 23 gray shading denotes the 10th and 90th percentile concentrations.)

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

1 Analyses at individual monitoring sites indicate that declines in ambient PM_{2.5}
2 concentrations have been most consistent across the eastern U.S. and in parts of coastal
3 California, where both annual average and 98th percentiles of 24-hour concentrations have
4 declined significantly (Figure 2-15, bottom panels). In contrast, trends in ambient PM_{2.5}
5 concentrations have been less consistent over much of the western U.S., with no significant
6 changes since 2000 observed at some sites in the Pacific northwest, the northern Rockies and
7 plains, and the southwest, particularly for 98th percentiles of 24-hour concentrations (Figure 2-
8 15, bottom panels). Trends in annual average PM_{2.5} concentrations have been highly correlated
9 with trends in 98th percentiles of 24-hour concentrations at individual sites (Figure 2-17). Such
10 correlations are highest across the eastern U.S. and in coastal California, and are somewhat
11 lower, though still generally positive, at sites in the Central and Western U.S. (i.e., outside of
12 coastal California).



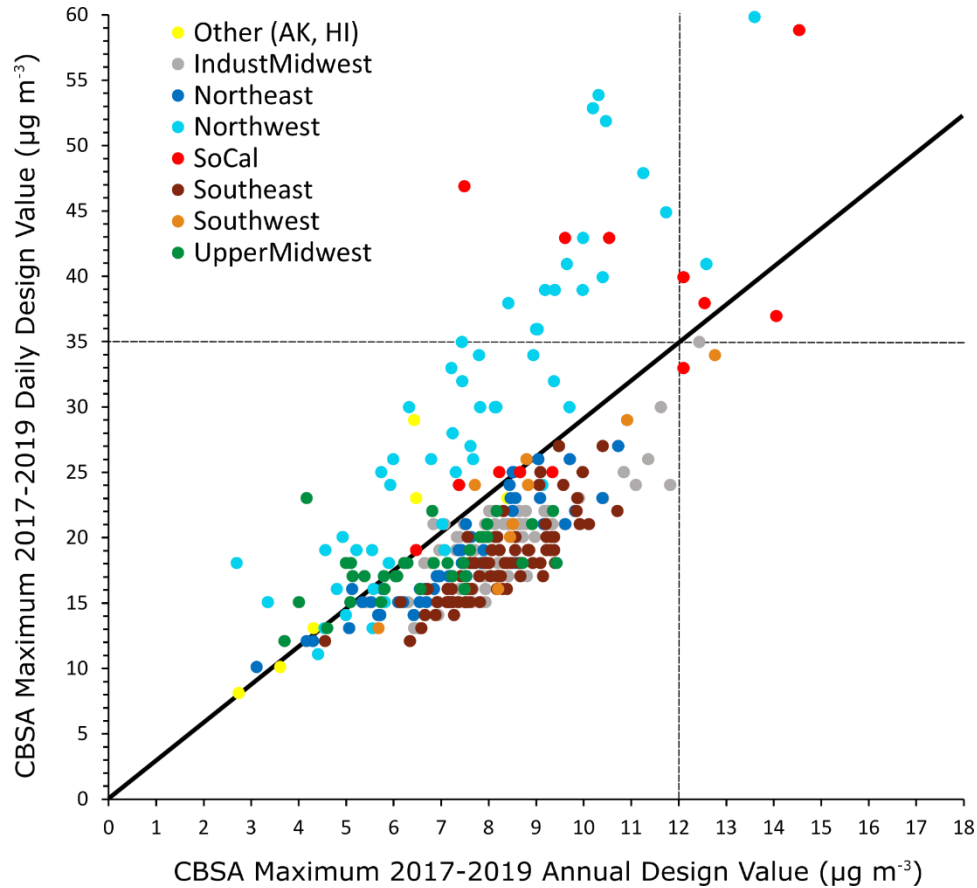
14 **Figure 2-17. Pearson's correlation coefficient between annual average and 98th percentile**
15 **of 24-hour PM_{2.5} concentrations from 2000-2019.**

16 **2.3.2.2 Characterization of PM_{2.5} Mass at Finer Spatial and Temporal Scales**

17 **2.3.2.2.1 CBSA Maximum Annual Versus Daily Design Values**

18 Analysis of recent air quality indicates that maximum annual and daily PM_{2.5} design
19 values within a CBSA are positively correlated with some noticeable regional variability (Figure
20 2-18). In the Southeast, Northeast, and Industrial Midwest regions, the annual design values are
21 high relative to the daily design values due in part to the infrequent impacts of episodic events

1 like wildfire or dust storms. On the other hand, the Northwest region has very high daily design
 2 values relative to the annual design values. This is due to episodically high PM_{2.5} concentrations
 3 that affect the region, both from wintertime stagnation events and summer/fall wildfire smoke
 4 events.²⁹ The relatively small population and low emissions in the region result in much lower
 5 PM_{2.5} concentrations during the other parts of the year not affected by these episodes.
 6



7
 8 **Figure 2-18. Scatterplot of CBSA maximum annual versus daily design values (2017-2019)**
 9 **with the solid black line representing the ratio of daily and annual NAAQS values.**

10 **2.3.2.2.2 PM_{2.5} Near Major Roadways**

11 Because of its longer atmospheric lifetime (U.S. EPA, 2019b, section 2.2), PM_{2.5} is
 12 expected to exhibit less spatial variability on an urban scale than UFP or PM_{10-2.5} (U.S. EPA,
 13 2019b, section 2.5.1.2.1). Analyses in the 2009 ISA for PM indicated that correlations between

²⁹ Due to the recent time period shown in Figure 2-18, it is likely that some of the annual and daily design values are affected by potential exceptional events associated with wildfire smoke that have yet to be regionally-concurred and removed from the design value calculations. The EPA defines exceptional events as unusual or natural-occurring events that that affect air quality but are not reasonably controllable using techniques that tribal, state, or local air agencies may implement. This is especially likely for the daily design values in the Northwest region, which experienced frequent wildfire smoke events during the 2017-2019 period.

1 PM_{2.5} monitoring sites up to a distance of 100 km from each other were greater than 0.75 in most
2 urban areas. However, more substantial spatial variation has been reported for some urban areas,
3 due in part to proximity between monitors and emissions sources (U.S. EPA, 2019b, section
4 2.5.1.2.1). The recent deployment of PM_{2.5} monitors near major roads in large urban areas
5 provides some insight into this spatial variation.

6 As discussed above, in the 2012 review of the PM NAAQS, the EPA required monitoring
7 of PM_{2.5}, along with NO₂ and CO, near major roads in CBSAs with populations greater than 1
8 million. PM_{2.5} monitoring was required to start for the largest CBSAs at the beginning of 2015,
9 and several years of data are now available for analysis at these sites. DeWinter et al. (2018)
10 analyzed these data and found that the average near-road increment (difference between near-
11 road PM_{2.5} concentrations and the concentrations at other sites in the same CBSA) was 1.2 µg/m³
12 for 2014-2015. Gantt et al. (2021) found that this near-road increment has a diurnal cycle, with a
13 peak during the morning rush hour. This near-road increment likely is additive to the urban
14 increment of PM_{2.5} from local sources in the CBSA including mobile sources on the numerous
15 non-highway roads that are not monitored by the near-road network. For 2016-2018, Gantt et al.
16 (2021) also reported that 52% and 24% of the time the near-road sites reported the highest annual
17 and 24-hour PM_{2.5} design value in the CBSA, respectively. Of the CBSAs with the highest
18 annual design values at near-road sites reported by Gantt et al. (2021), those design values were,
19 on average, 0.8 µg/m³ higher than at the highest measuring non-near-road sites (range is 0.1 to
20 2.1 µg/m³ higher at near-road sites).

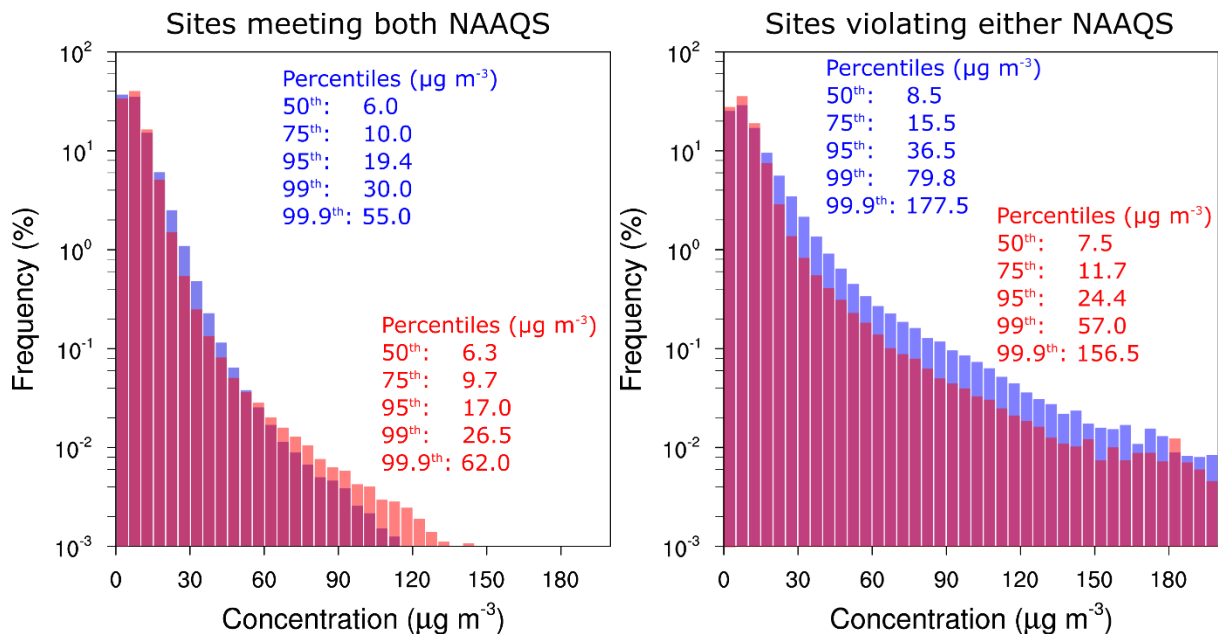
21 Although most near-road monitoring sites do not have sufficient data to evaluate long-
22 term trends in near-road PM_{2.5} concentrations, Gantt et al. (2021) analyzed data at one long-term
23 near-road-like site in Elizabeth, NJ,³⁰ and found that the annual average increment has generally
24 decreased between 2001 and 2018 from about 2.0 µg/m³ to about 1.3 µg/m³. The trend in the
25 near-road increment of elemental carbon at the Elizabeth, NJ site has shown a similar reduction,
26 with values of ~1.0 µg/m³ in 2001 decreasing to ~0.5 µg/m³ in 2018. These data are consistent
27 with the timing of EPA emission standards for motor vehicles.³¹ Although long-term data are not
28 available at other near-road sites, the national scope of the diesel vehicle controls suggests the
29 near-road environment across the U.S. may have experienced similar decreasing trends in near-
30 road PM_{2.5} increments.

31 **2.3.2.2.3 Sub-Daily Concentrations of PM_{2.5}**

³⁰ The Elizabeth Lab site in Elizabeth, NJ is situated approximately 30 meters from travel lanes of the Interchange 13 toll plaza of the New Jersey Turnpike and within 200 meters of travel lanes for Interstate 278 and the New Jersey Turnpike.

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

1 Ambient PM_{2.5} concentrations can exhibit a diurnal cycle that varies due to impacts from
 2 intermittent emission sources, meteorology, and atmospheric chemistry. The PM_{2.5} monitoring
 3 network in the U.S. has an increasing number of continuous FEM monitors reporting hourly
 4 PM_{2.5} mass concentrations that reflect this diurnal variation. The 2019 ISA describes a two-
 5 peaked diurnal pattern in urban areas, with morning peaks attributed to rush-hour traffic and
 6 afternoon peaks attributed to a combination of rush hour traffic, decreasing atmospheric dilution,
 7 and nucleation (U.S. EPA, 2019b, section 2.5.2.3, Figure 2-32). Because a focus on annual
 8 average and 24-hour average PM_{2.5} concentrations could mask sub-daily patterns, and because
 9 some health studies examine PM exposure durations shorter than 24-hours, it is useful to
 10 understand the broader distribution of sub-daily PM_{2.5} concentrations across the U.S. Figure 2-19
 11 below presents the frequency distribution of 2-hour average PM_{2.5} mass concentrations from all
 12 FEM PM_{2.5} monitors in the U.S. for 2017-2019.³² At sites meeting the current primary PM_{2.5}
 13 standards, these 2-hour concentrations generally remain below 10 µg/m³, and virtually never
 14 exceed 30 µg/m³. Two-hour concentrations are higher at sites violating the current standards,
 15 generally remaining below 16 µg/m³ and virtually never exceeding 80 µg/m³.



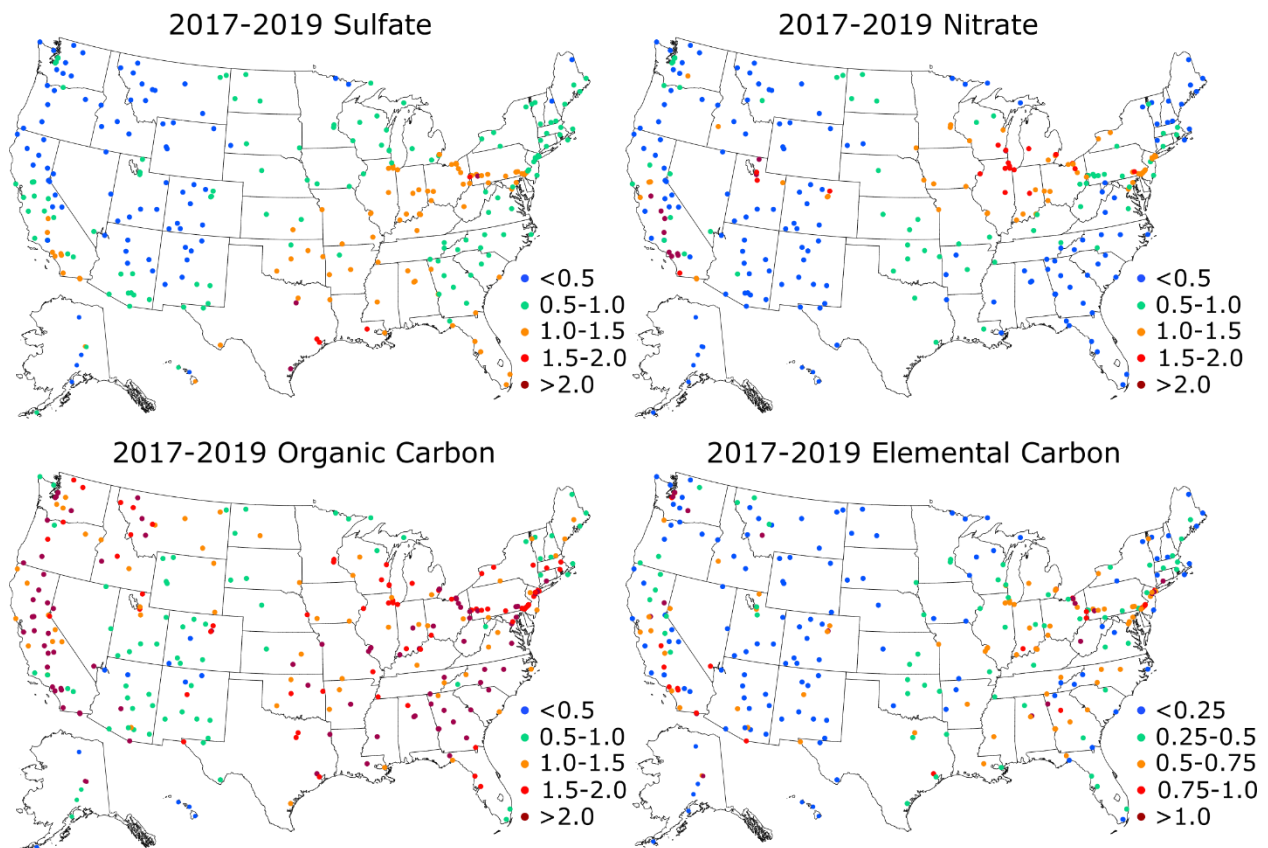
16
 17 **Figure 2-19. Frequency distribution of 2017-2019 2-hour averages for sites meeting both or**
 18 **violating either PM_{2.5} NAAQS for October to March (blue) and April to September**
 19 **(red).**

³² As discussed further in section 3.2, PM_{2.5} controlled human exposure studies often examine 2-hour exposures. Thus, when evaluating those studies in the context of the current primary PM_{2.5} standards, it is useful to consider the distribution of 2-hour PM_{2.5} concentrations. Similar analyses of 4-hour and 5-hour PM_{2.5} concentrations are presented in Appendix A, Figure A-2 and Figure A-3, respectively.

1 The extreme upper end of the distribution of 2-hour $PM_{2.5}$ concentrations is shifted higher
2 during the warmer months (red in Figure 2-19), generally corresponding to the period of peak
3 wildfire frequency (April to September) in the U.S. At sites meeting the current primary
4 standards, the highest 2-hour concentrations measured virtually never occur outside of the period
5 of peak wildfire frequency. Most of the sites measuring these very high concentrations are in the
6 northwestern U.S. and California, where wildfires have been relatively common in recent years
7 (see Appendix A, Figure A-1). When the period of peak wildfire frequency is excluded from the
8 analysis (blue in Figure 2-19), the extreme upper end of the distribution is reduced.

9 2.3.2.3 Chemical Composition of $PM_{2.5}$

10 Based on recent air quality data, the major chemical components of $PM_{2.5}$ have distinct
11 spatial distributions. Sulfate concentrations tend to be highest in the eastern U.S., while in the
12 Ohio Valley, Salt Lake Valley, and California nitrate concentrations are highest and relatively
13 high concentrations of organic carbon are widespread across most of the Continental U.S., as
14 shown in Figure 2-20. Elemental carbon, crustal material, and sea-salt are found to have the
15 highest concentrations in the northeast U.S., southwest U.S., and coastal areas, respectively.



16
17 **Figure 2-20. Annual average $PM_{2.5}$ sulfate, nitrate, organic carbon, and elemental carbon**
18 **concentrations (in $\mu g/m^3$) from 2017-2019.**

1 An examination of PM_{2.5} composition trends can provide insight into the factors
2 contributing to overall reductions in ambient PM_{2.5} concentrations. The biggest change in PM_{2.5}
3 composition that has occurred in recent years is the reduction in sulfate concentrations due to
4 reductions in SO₂ emissions. Between 2000 and 2015, the nationwide annual average sulfate
5 concentration decreased by 17% at urban sites and 20% at rural sites. This change in sulfate
6 concentrations is most evident in the eastern U.S. and has resulted in organic matter or nitrate
7 now being the greatest contributor to PM_{2.5} mass in many locations (U.S. EPA, 2019b, Figure 2-
8 19). The overall reduction in sulfate concentrations has contributed substantially to the decrease
9 in national average PM_{2.5} concentrations as well as the decline in the fraction of PM₁₀ mass
10 accounted for by PM_{2.5} (U.S. EPA, 2019b, section 2.5.1.1.6; section 2.3.1 above).

11 **2.3.2.4 National Characterization of PM₁₀ Mass**

12 At long-term monitoring sites in the U.S., the 2017-2019 average of 2nd highest 24-hour
13 PM₁₀ concentration was 68 µg/m³ (with the 10th and 90th percentiles at 28 and 124 µg/m³,
14 respectively) (Figure 2-21, top panels).³³ The highest PM₁₀ concentrations tend to occur in the
15 western U.S. Seasonal analyses indicate that ambient PM₁₀ concentrations are generally higher in
16 the summer months than at other times of year, though the most extreme high concentration
17 events are more likely in the spring (U.S. EPA, 2019b, Table 2-5). This is due to fact that the
18 major PM₁₀ emission sources, dust and agriculture, are more active during the warmer and drier
19 periods of the year.

³³ The form of the current 24-hour PM₁₀ standard is one-expected-exceedance, averaged over three years.

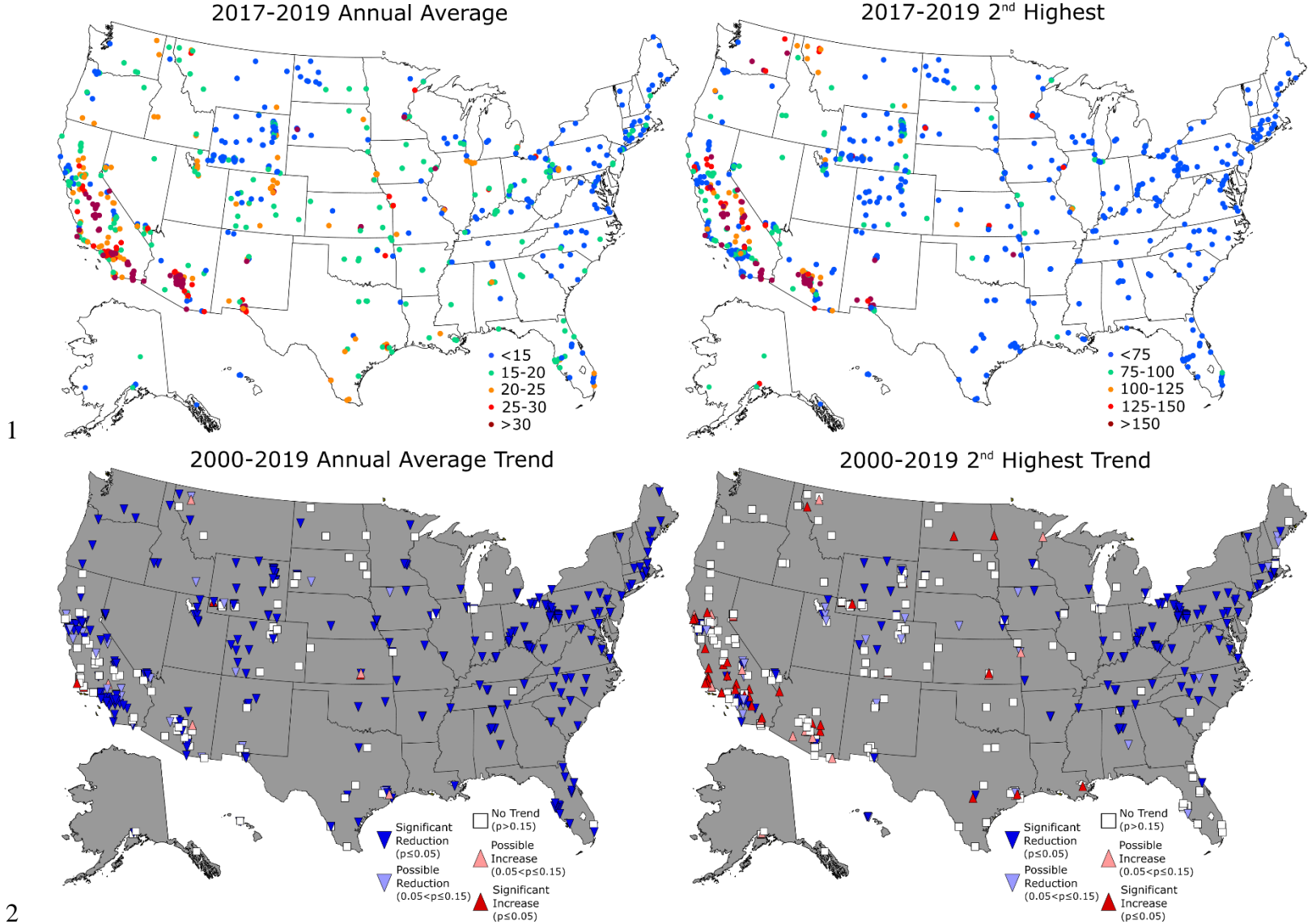
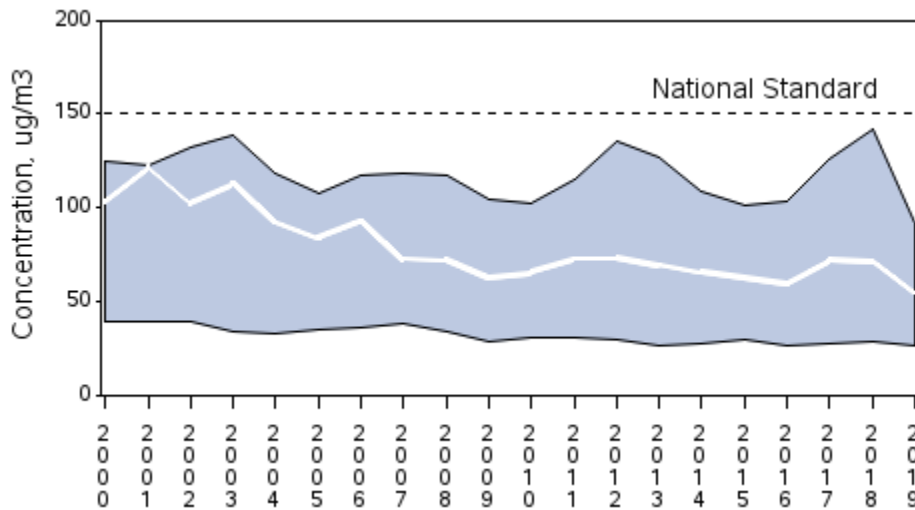


Figure 2-21. Annual average and 2nd highest PM₁₀ concentrations (in $\mu\text{g}/\text{m}^3$) from 2017-2019 (top) and linear trends and their associated significance in PM₁₀ concentrations from 2000-2019 (bottom).

1 Recent ambient PM₁₀ concentrations reflect reductions that have occurred across much of the
 2 U.S. (Figure 2-21, bottom panels). From 2000 to 2019, 2nd highest 24-hour PM₁₀ concentrations
 3 have declined by about 46% (Figure 2-22).³⁴ Analyses at individual monitoring sites indicate that
 4 annual average PM₁₀ concentrations have declined at most sites across the U.S., with much of the
 5 decrease in the eastern U.S. associated with reductions in PM_{2.5} concentrations. Annual second
 6 highest 24-hour PM₁₀ concentrations have generally declined in the eastern U.S., while
 7 concentrations in the much of the midwest and western U.S. have remained unchanged or
 8 increased since 2000 (Figure 2-21, bottom panels).



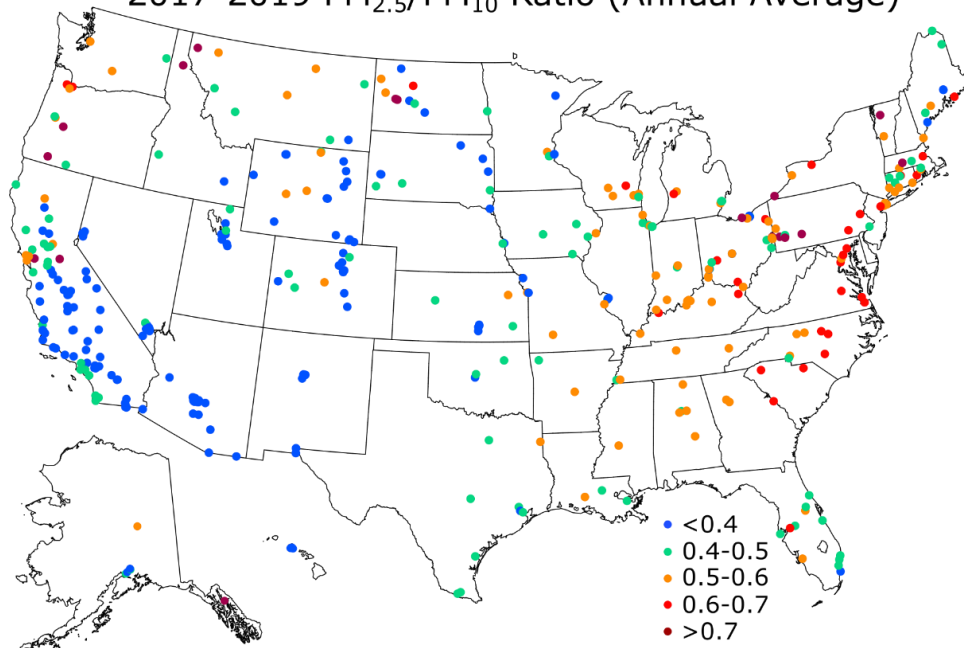
9
 10 **Figure 2-22. National trends in Annual 2nd Highest 24-Hour PM₁₀ concentrations from**
 11 **2000 to 2019 (262 sites).** (Note: The white line indicates the mean concentration while the
 12 gray shading denotes the 10th and 90th percentile concentrations.)

13 Compared to previous reviews, data available from the NCore monitoring network in the
 14 current reconsideration allows a more comprehensive analysis of the relative contributions of
 15 PM_{2.5} and PM_{10-2.5} to PM₁₀ mass. PM_{2.5} generally contributes more to annual average PM₁₀ mass
 16 in the eastern U.S. than the western U.S. (Figure 2-23). At most sites in the eastern U.S., the
 17 majority of PM₁₀ mass is comprised of PM_{2.5}. As ambient PM_{2.5} concentrations have declined in
 18 the eastern U.S. (section 2.3.2.2, above), the ratios of PM_{2.5} to PM₁₀ have also declined.

19

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

2017-2019 PM_{2.5}/PM₁₀ Ratio (Annual Average)

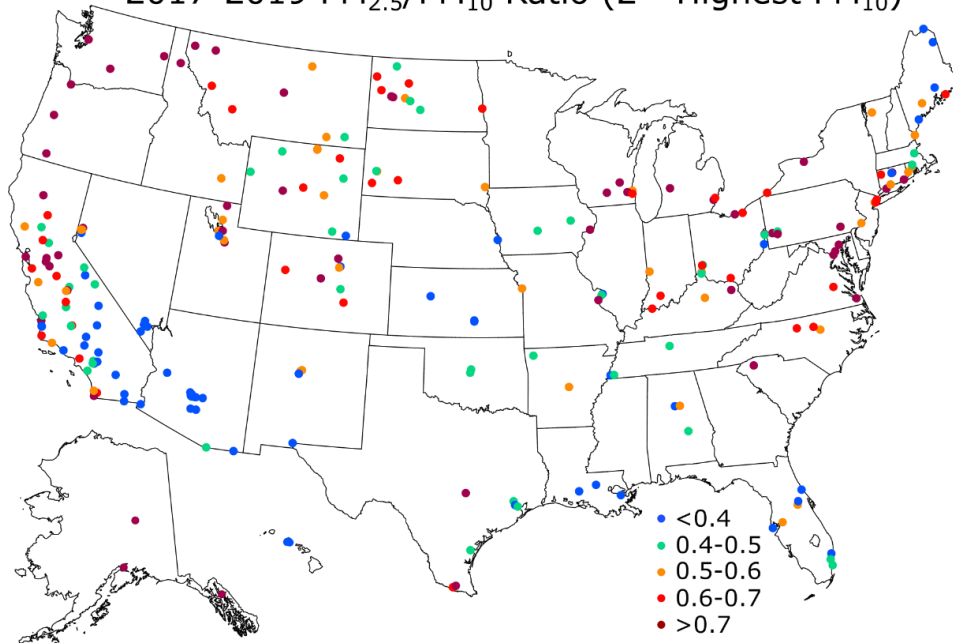


1
2 **Figure 2-23. Annual average PM_{2.5}/PM₁₀ ratio for 2017-2019.**

3 For days with very high PM₁₀ concentrations (Figure 2-24), the PM_{2.5}/PM₁₀ ratios are
4 typically higher than the annual average ratios. This is particularly true in the northwestern U.S.
5 where the high PM₁₀ concentrations can occur during wildfires with high PM_{2.5}.

6

2017-2019 PM_{2.5}/PM₁₀ Ratio (2nd Highest PM₁₀)

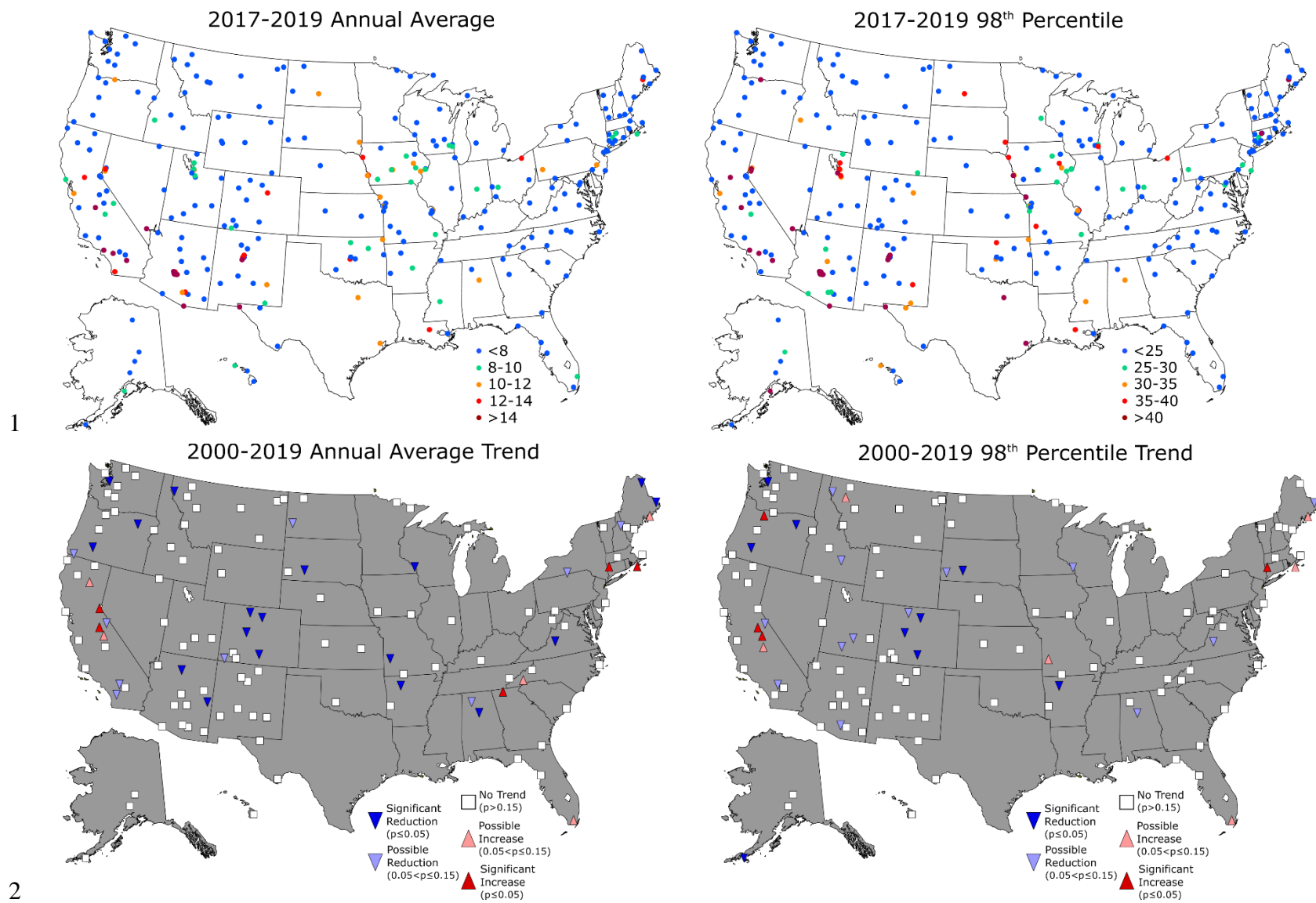


7
8 **Figure 2-24. PM_{2.5}/PM₁₀ ratio on the date of the second highest PM₁₀ concentrations for**
9 **2017-2019.**

1 **2.3.2.5 National Characterization of PM_{10-2.5} Mass**

2 Since the 2012 review, the availability of PM_{10-2.5} ambient concentration data has greatly
3 increased. As illustrated in Figure 2-25³⁵ (top panels), annual average and 98th percentile PM_{10-2.5}
4 concentrations exhibit less distinct differences between the eastern and western U.S. than for
5 either PM_{2.5} or PM₁₀. Additionally, compared to PM_{2.5} and PM₁₀, changes in PM_{10-2.5}
6 concentrations have been small in magnitude and inconsistent in direction (Figure 2-25, lower
7 panels).

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



1

2

3

4

Figure 2-25. Annual average and 98th percentile PM_{10-2.5} concentrations ($\mu\text{g}/\text{m}^3$) from 2017-2019 (top) and linear trends and their associated significance in PM_{10-2.5} concentrations from 2000-2019 (bottom).

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

Compared to PM_{2.5} mass, there is relatively little data on U.S. particle number concentrations, which are dominated by UFP. In the published literature, annual average particle number concentrations reaching about 20,000 to 30,000 cm⁻³ have been reported in U.S. cities (U.S. EPA, 2019b). In addition, based on UFP measurements in two urban areas (New York City, Buffalo) and at a background site (Steuben County) in New York, there is a pronounced difference in particle number concentration between different types of locations (Figure 2-26; U.S. EPA, 2019b, Figure 2-18). Urban particle number counts were several times higher than at the background site, and the highest particle number counts in an urban area with multiple sites (Buffalo) were observed at a near-road location. Hourly data indicate that particle numbers remain fairly constant throughout the day at the background site, that they peak around 8:00 a.m. in Buffalo and New York City (NYC), and that they remain high into the evening hours with distinct rush hour and early afternoon peaks.

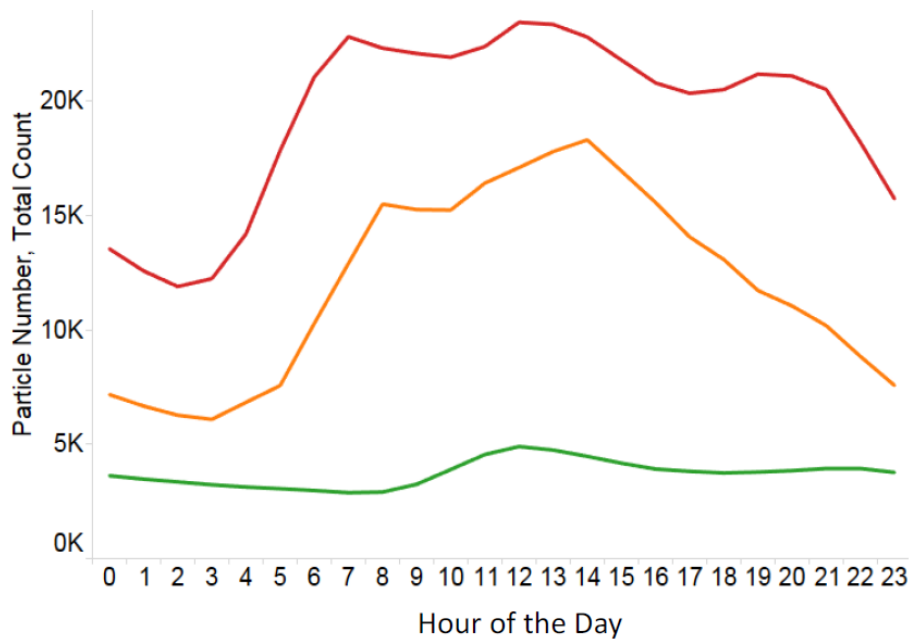
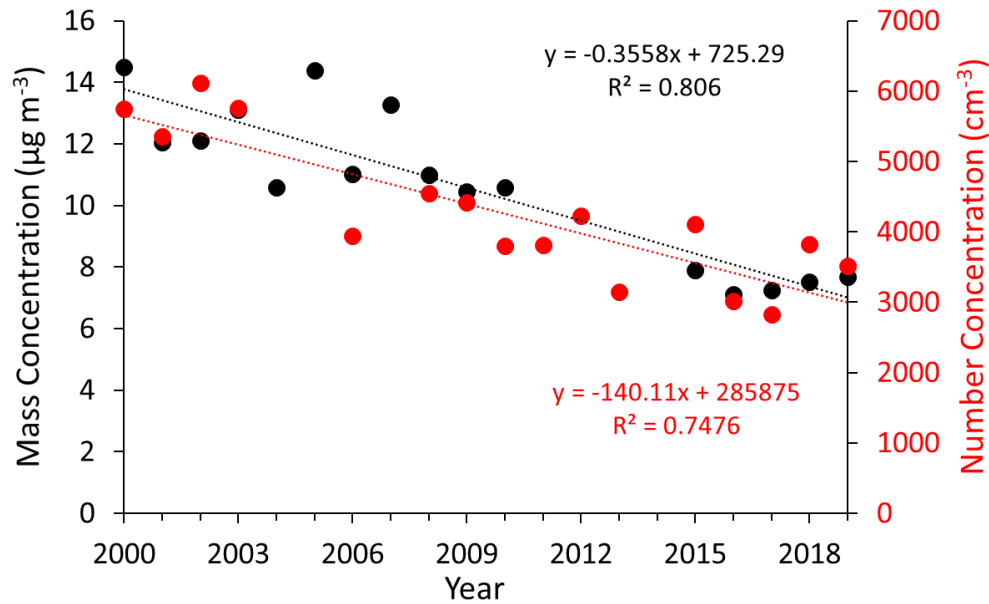


Figure 2-26. Average hourly particle number concentrations from three locations in the State of New York for 2014 to 2015 (green is Steuben County, orange is Buffalo, red is New York City). (Source: Figure 2-18 in U.S. EPA, 2019b).

Long-term trends in UFP are generally not available at U.S. monitoring sites. However, data on number size distribution have been reported for an 8-year period from 2002 to 2009 in Rochester, NY. Number concentrations averaged 4,730 cm⁻³ for 0.01 to 0.05 μm particles and 1,838 cm⁻³ for 0.05 to 0.1 μm particles (Wang et al., 2011). On average over the 8 years that UFP data were collected in Rochester, total particle number concentrations declined from the earlier period evaluated (i.e., 2001 to 2005) to the later period (2006 to 2009). This decline was

1 most evident for particles between 0.01 and 0.1 μm and was attributed to changes in local
2 sources resulting from the 2007 Heavy Duty Highway Rule (66 FR 5002, January 18, 2001), a
3 reduction in local industrial activity, and the closure of a nearby coal-fired power plant (Wang et
4 al., 2011; U.S. EPA, 2019b, section 2.5.2.1.4).

5 In addition, at a site in Illinois the annual average particle number concentration declined
6 between 2000 and 2019, closely matching the reductions in annual $\text{PM}_{2.5}$ mass over that same
7 period (Figure 2-27, below). Particle number concentrations at this site are closer to those of the
8 background site in Figure 2-27 than the urban sites. A recent study found that particle number
9 concentrations in an urban area (Pittsburgh, PA) decreased between 2001-2002 and 2016-2017
10 along with decreases in $\text{PM}_{2.5}$ associated with SO_2 emission reductions (Saha et al., 2018).
11 However, the relationship between changes in ambient $\text{PM}_{2.5}$ and UFPs cannot be
12 comprehensively characterized due to the high variability and limited monitoring of UFPs.
13



14
15 **Figure 2-27. Time series of annual average mass and number concentrations (left) and**
16 **scatterplot of mass vs. number concentration (right) between 2000-2019 in Bondville, IL.**

17 2.3.3 Characterizing Ambient $\text{PM}_{2.5}$ Concentrations for Exposure

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

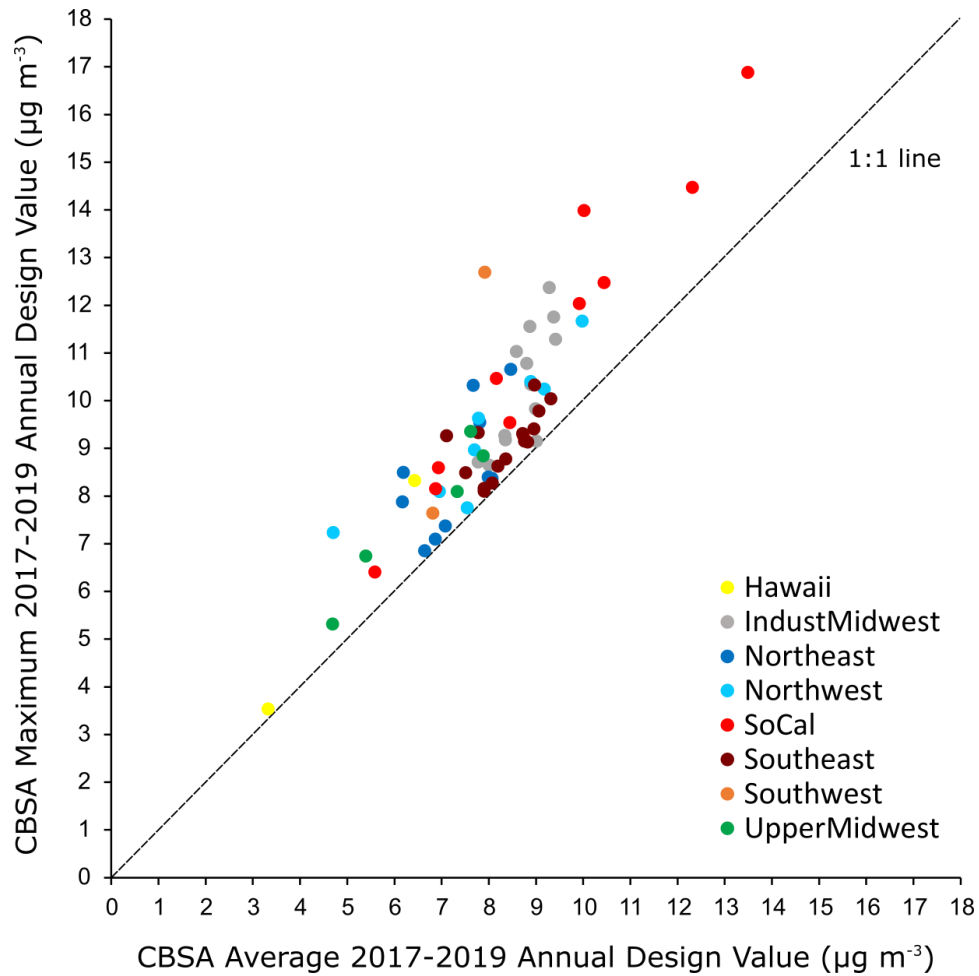
1 hybrid modeling approaches tend to broaden the areas captured in the exposure assessment, and
2 in doing so, the studies that utilize these methods tend to report lower mean PM_{2.5} concentrations
3 than monitor-based approaches. Further, other aspects of the method used to calculate PM_{2.5}
4 concentrations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

6 **2.3.3.1 Predicted Ambient PM_{2.5} and Exposure Based on Monitored Data**

7 Ambient concentrations of PM_{2.5} are often characterized using measurements from
8 national monitoring networks due to the accuracy and precision of the measurements and the
9 public availability of data. For applications requiring PM_{2.5} characterizations across urban areas,
10 data averaging techniques such as area-wide and population-weighted averaging of monitors are
11 sometimes used to provide complete coverage from the site measurements (U.S. EPA, 2019b,
12 chapter 3).

13 For an area to meet the NAAQS, all valid design values in that area, including the highest
14 annual and 24-hour values, must be at or below the levels of the standards. Because monitors are
15 often required in locations with high PM_{2.5} concentrations (section 2.2.3), areas meeting an
16 annual PM_{2.5} standard with a particular level would be expected to have long-term average PM_{2.5}
17 concentrations (i.e., averaged across space and over time in the area) somewhat below that
18 standard level. Figure 2-28 and Figure 2-29 indicate that, based on recent air quality in U.S.
19 CBSAs, maximum annual PM_{2.5} design values are often 10% to 20% higher than annual average
20 concentrations (i.e., averaged across multiple monitors in the same CBSA). The difference
21 between the maximum annual design value and average concentration in an area can be smaller
22 or larger than this range, likely depending on factors such as the number of monitors, monitor
23 siting characteristics, and the distribution of ambient PM_{2.5} concentrations. Given that higher
24 PM_{2.5} concentrations have been reported at some near-road monitoring sites, relative to the
25 surrounding area (section 2.3.2.2), recent requirements for PM_{2.5} monitoring at near-road
26 locations in large urban areas (section 2.2.3.3) may increase the ratios of maximum annual
27 design values to averaged concentrations in some areas. Such ratios may also depend on how the
28 average concentrations are calculated (i.e., averaged across monitors versus across modeled grid
29 cells). Compared to annual design values, Figure 2-29 indicates a more variable relationship
30 between maximum 24-hour PM_{2.5} design values and annual average concentrations.

31



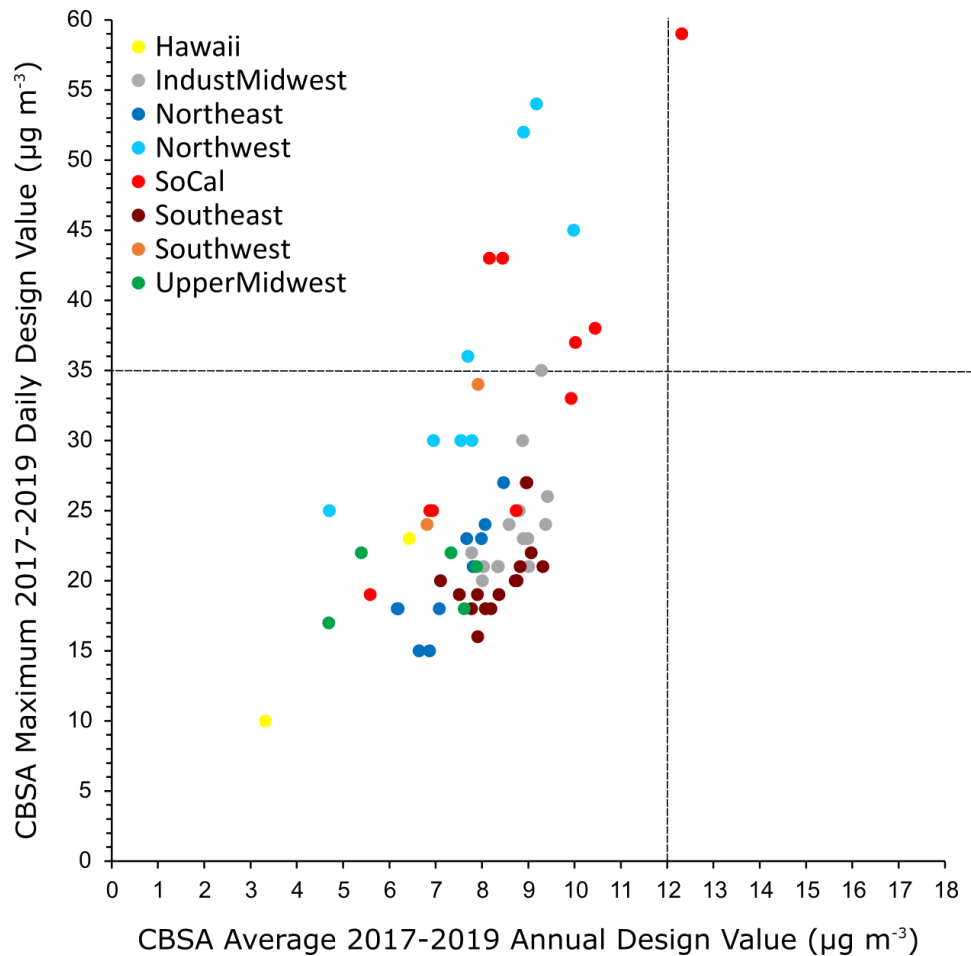
1
 2 **Figure 2-28. Comparison of CBSA average annual design values and CBSA maximum**
 3 **annual design values for 2017-2019.** (Note: Includes all CBSAs with at least 3 valid annual
 4 DVs.)

5

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

Years of Monitoring Data	Number of Monitors per CBSA	Number of CBSAs	Ratio of Maximum Annual DV to CBSA Average	Ratio of Maximum 24-hour DV to CBSA Average
2009-2011	3 or more	67	1.12	1.13
	4 or more	33	1.14	1.16
	5 or more	18	1.17	1.19
2012-2014	3 or more	60	1.15	1.15
	4 or more	38	1.17	1.18
	5 or more	23	1.19	1.21
2015-2017	3 or more	65	1.16	1.19
	4 or more	38	1.19	1.21
	5 or more	30	1.20	1.24
2017-2019	3 or more	67	1.16	1.22
	4 or more	47	1.19	1.25
	5 or more	32	1.21	1.26

3



2

3 **Figure 2-29. Comparison of CBSA average annual design values and CBSA maximum**
 4 **daily design values for 2017-2019.** (Note: Dashed lines indicate the level of the current 24-
 5 hour PM_{2.5} standard (35 µg/m³) and the current annual PM standard (12 µg/m³). Includes all
 6 CBSAs with at least 3 valid daily and 3 valid annual DVs.)

7

2.3.3.2 Predicted Ambient PM_{2.5} Based on Hybrid Modeling Approaches

8

9

10

11

12

13

14

15

16

17

Ambient concentrations of PM_{2.5} are often characterized using measurements from national monitoring networks due to the accuracy and precision of the measurements and the public availability of data. For applications requiring PM_{2.5} characterizations across urban areas, data averaging techniques such as area-wide and population-weighted averaging of monitors are sometimes used to provide complete coverage from the site measurements (U.S. EPA, 2019b, chapter 3). Yet data averaging methods may not adequately represent the spatial heterogeneity of PM_{2.5} within an area and are not practical for large unmonitored areas or time periods. As a result, additional methods have been developed to improve PM_{2.5} characterizations in areas where monitoring is relatively sparse or unavailable. Methods include interpolation of monitored data, land-use regression models, chemical-transport models (CTMs), models based on satellite-

1 derived aerosol optical depth (AOD), and hybrid spatiotemporal models that combine
2 information from the individual approaches (U.S. EPA, 2019b, chapter 3). A number of recent
3 studies have employed such methods to estimate PM_{2.5} air quality concentrations across the U.S.
4 and Canada, and to estimate population exposures for use in epidemiologic analyses (U.S. EPA,
5 2019b, sections 3.3 and 3.4). Given the increasing availability and application of these methods,
6 in this section we provide an overview of recently developed hybrid modeling methods, their
7 predictions and performance, and how predictions from various methods compare to each other.

8 **2.3.3.2.1 Overview of Hybrid Methods**

9 Hybrid methods are broadly classified into four categories: (1) methods based primarily
10 on interpolation of monitor data, (2) Bayesian statistical downscalers, (3) methods based
11 primarily on satellite-derived AOD, and (4) methods based on machine-learning algorithms.
12 Each method is discussed briefly below.

13 Interpolation-based methods are the simplest approach for developing spatial fields of
14 PM_{2.5} concentrations and rely on the moderate degree of spatial autocorrelation in PM_{2.5} in many
15 areas of the U.S. Interpolation methods often use inverse-distance or inverse-distance-squared
16 weighted averaging of monitoring data to predict PM_{2.5} concentrations at unmonitored receptor
17 points. Examples include the Voronoi neighbor averaging (VNA) approach and the enhanced
18 VNA approach (eVNA). The VNA approach applies weighted averaging to the concentrations
19 monitored in the Voronoi cells neighboring the cell containing the prediction point (Abt
20 Associates, 2014). In the eVNA approach, monitored data are further weighted by the ratio of
21 CTM predictions in the grid-cell containing the prediction point to the grid-cell containing the
22 monitor (Abt Associates, 2014).

23 Bayesian statistical modeling has been used to calibrate CTM PM_{2.5} predictions or
24 satellite-derived AOD estimates to surface measurements (Berrocal et al., 2012; Wang et al.,
25 2018b, Berrocal et al., 2020). This approach, commonly referred to as a Bayesian downscaler
26 because it “downscales” grid-cell average values to points, first regresses the PM_{2.5} predictions
27 or AOD estimates on monitoring data. The resulting relationships are then used to develop a
28 gridded PM_{2.5} field from the CTM or AOD input field. Bayesian downscalers have been applied
29 to develop gridded daily PM_{2.5} fields at 12-km resolution for the conterminous U.S. (Wang et al.,
30 2018b; U.S. EPA, 2017). An ensemble technique that optimally combines predictions of CTM
31 and AOD downscalers has also been developed to predict PM_{2.5} at high resolution over Colorado
32 during the fire season (Geng et al., 2018).

33 Surface PM_{2.5} concentrations can also be predicted based on satellite retrievals of AOD
34 and the relationship between surface PM_{2.5} and AOD from CTM simulations (van Donkelaar et
35 al., 2010). For example, in van Donkelaar et al. (2015a), satellite-based approaches (van

1 Donkelaar et al., 2010; van Donkelaar et al., 2013) were used to estimate a gridded field of
2 global mean PM_{2.5} concentration for the 2001-2010 period that was combined with information
3 from radiometrically stable satellite instruments (Boys et al., 2014) to develop global PM_{2.5}
4 fields over the 1998-2012 period (van Donkelaar et al., 2015a). Motivated by the limited use of
5 surface measurements in this approach, van Donkelaar et al. (2015b) developed an updated
6 method that incorporates additional information from PM_{2.5} monitoring networks to improve
7 performance. Specifically, geographically weighted regression (GWR) of residual PM_{2.5} (i.e., the
8 difference between monitored PM_{2.5} and predictions based on satellite-derived AOD) with land-
9 use and other variables is performed to improve PM_{2.5} concentration estimates in areas such as
10 North America where monitoring is relatively dense (van Donkelaar et al., 2019; van Donkelaar
11 et al., 2015b). This approach has been used to create long-term PM_{2.5} fields globally and for
12 North America at about 1-km resolution. However, the developers caution that PM_{2.5} gradients
13 may not be fully resolved at 1-km resolution due to the influence of coarser-scale data used in
14 the model³⁶ and report that mean error variance decreases when averaging the 1-km fields to
15 coarser resolution (van Donkelaar et al., 2019).

16 Daily PM_{2.5} fields based on non-parametric (i.e., machine learning) methods have also
17 been developed to characterize PM_{2.5} over the U.S. Non-parametric methods facilitate the use of
18 large numbers of predictor variables that may have complex nonlinear relationships with PM_{2.5}
19 concentrations that would be challenging to specify with a parametric method. For example, a
20 neural network algorithm was used to predict daily PM_{2.5} fields at 1-km resolution over the
21 conterminous U.S. during 2000-2012 using more than 50 predictor variables including satellite-
22 derived AOD, CTM predictions, satellite-derived absorbing aerosol index, meteorological data,
23 and land-use variables (Di et al., 2016). A random forest algorithm was also applied to develop
24 daily PM_{2.5} fields at 12-km resolution over the conterminous U.S. in 2011 and provide variable
25 importance information for about 40 predictor variables including CTM results and satellite-
26 derived AOD (Hu et al., 2017). Satellite-derived AOD and the convolution layer for nearby
27 PM_{2.5} measurements are ranked among the top five most important predictor variables for the
28 importance metrics considered. An ensemble model based on random forest, neural network, and
29 gradient boosting methods has also been recently applied to develop daily 1-km PM_{2.5}
30 concentration fields over the U.S. for the 2000-2015 period (Di et al., 2019). A wide range of
31 parametric and non-parametric hybrid PM_{2.5} models have recently been reviewed in Chapter 3 of
32 the 2019 ISA (U.S. EPA, 2019b).

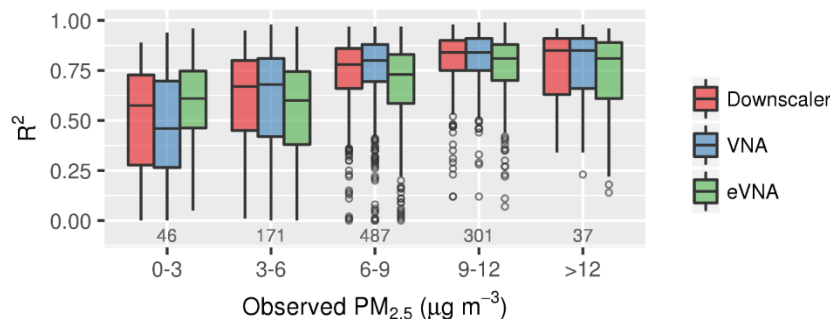
33 **2.3.3.2.2 Performance of the Methods**

³⁶ See <https://sites.wustl.edu/acag/datasets/surface-pm2-5/>

1 The performance of hybrid modeling methods is often evaluated against surface
2 measurements using n-fold cross validation (i.e., 1/n of the data are reserved for validation with
3 the rest used for model training, and the process is repeated n times). Although model evaluation
4 methods are not consistent across studies, ten-fold cross-validation statistics are often reported
5 and support use of the hybrid methods just described. For example, the neural network achieved
6 total R^2 of 0.84 and root-mean-square error (RMSE) of $2.94 \mu\text{g m}^{-3}$ for daily $\text{PM}_{2.5}$ predictions at
7 sites in the conterminous U.S. during 2000-2012 (Di et al., 2016). The random forest achieved
8 total R^2 of 0.80 and RMSE of $2.83 \mu\text{g m}^{-3}$ for daily $\text{PM}_{2.5}$ predictions at U.S. sites in 2011 (Hu et
9 al., 2017). The satellite-derived AOD approach with GWR yielded an R^2 of 0.79 and RMSE of
10 $1.7 \mu\text{g m}^{-3}$ in cross validation for longer-term $\text{PM}_{2.5}$ predictions at sites in North America (van
11 Donkelaar et al., 2015b). The Bayesian downscalers had weaker performance in cross validation
12 (e.g., national R^2 : 0.66-0.70; Wang et al., 2018b; Kelly et al., 2019a) than the other methods,
13 possibly due to the relatively small number of predictor variables. However, the downscalers
14 have advantages of simplicity, computational efficiency, and lower potential for overfitting
15 compared with the machine learning methods.

16 Although model validation analyses often report favorable performance in terms of
17 aggregate cross-validation statistics, studies have reported heterogeneity in performance by
18 season, region, and concentration range. For example, several methods had relatively high cross-
19 validation R^2 in summer compared with other seasons (Kelly et al., 2019a ; Hu et al., 2017; Di et
20 al., 2016; van Donkelaar et al., 2015b). Also, studies have noted relatively weak performance in
21 parts of the western U.S., possibly due to the sharp concentration gradients, complex terrain, low
22 concentrations (and therefore signal-to-noise ratio), less dense monitoring, prevalence of
23 wildfire, and challenges in satellite retrievals and CTM modeling (Di et al., 2016; Wang et al.,
24 2018b; Hu et al., 2017; Kelly et al., 2019a). Predictive capability in terms of cross-validation R^2
25 has also been reported to weaken with decreasing $\text{PM}_{2.5}$ concentration in several studies (e.g.,
26 Kelly et al., 2019a; Di et al., 2016; van Donkelaar et al., 2019). This trend could be due in part to
27 increases in the fraction of the $\text{PM}_{2.5}$ distribution that is explained by less predictable stochastic
28 variation as $\text{PM}_{2.5}$ concentrations decrease (Just et al., 2020). Trends in model performance
29 associated with $\text{PM}_{2.5}$ concentration (e.g., Figure 2-30) could also be due to the relatively sparse
30 monitoring in remote areas, where $\text{PM}_{2.5}$ concentrations tend to be low. Consistent with this
31 hypothesis, studies have reported degradation of model performance metrics with increasing
32 distance to the nearest in-sample monitor, suggesting that predictions are most reliable in densely
33 monitored urban areas (Jin et al., 2019; Huang et al., 2018; Kelly et al., 2019a; Berrocal et al.,
34 2020).

35



1 **Figure 2-30. R² for ten-fold cross-validation of daily PM_{2.5} predictions in 2015 from three**
 2 **methods for individual sites as a function of observed concentration.** Text indicates the
 3 number of monitors in the PM_{2.5} concentration range. Downscaler: Bayesian downscaler of
 4 CMAQ predictions; VNA: Voronoi Neighbor Averaging; eVNA: enhanced-VNA. From
 5 Kelly et al., 2019a.
 6

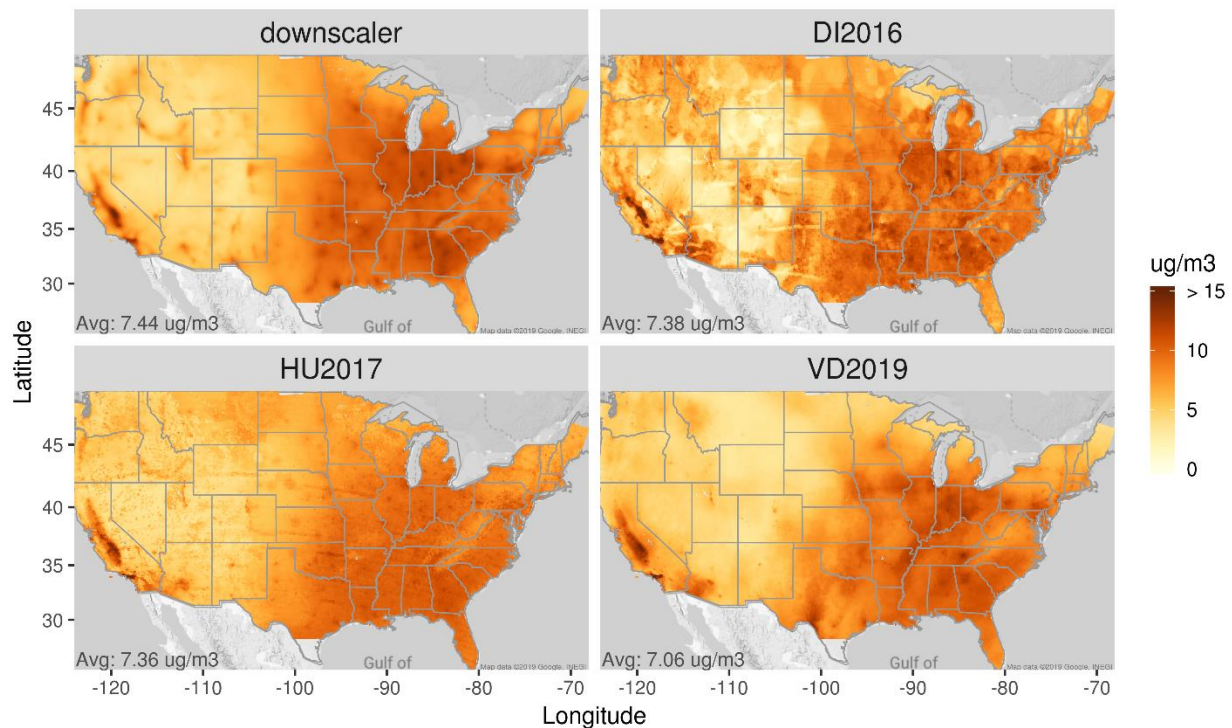
7 A limited number of studies have intercompared concentration predictions based on
 8 different PM_{2.5} characterization methods. Huang et al. (2018) compared PM_{2.5} concentrations
 9 from the method of Di et al. (2016) with concentrations from the CTM-based data fusion method
 10 of Friberg et al. (2016) and the satellite-derived AOD approach of Hu et al. (2014) for North
 11 Carolina. They reported general agreement in concentrations among methods, with some
 12 differences along the coast and in forested regions where monitoring is less dense. Yu et al.
 13 (2018) compared PM_{2.5} concentrations from fourteen approaches of varying complexity for
 14 developing PM_{2.5} spatial fields over the Atlanta, Georgia region. They reported that predictions
 15 of the methods can differ considerably, and the hybrid approaches that incorporate CTM
 16 predictions generally outperformed the simpler techniques (e.g., monitor interpolation). Also,
 17 model predictions appeared to be more reliable in the urban center based on relatively low cross
 18 validation R² for sites away from the urban core. Jin et al. (2019) reported increasing uncertainty
 19 in hybrid model predictions with distance to the nearest AQS monitor. Keller and Peng (2019)
 20 reported that a prediction model incorporating CTM output outperformed a monitor averaging
 21 approach and error reduction could be achieved by restricting the study to areas near monitors.
 22 Diao et al. (2019) reviewed publicly available PM_{2.5} products and identified inconsistencies in
 23 PM_{2.5} predictions from several methods. Kelly et al. (2021) reported broad agreement among
 24 model predictions at the national scale but differences in the intra-urban variations in PM_{2.5}
 25 concentrations.

26 **2.3.3.2.3 Comparison of PM_{2.5} Fields Across Approaches**

27 To illustrate features of the spatial fields reported in the literature, the annual mean PM_{2.5}
 28 concentrations for 2011 from four methods is shown in Figure 2-31, where predictions from the
 29 methods were averaged to a common 12-km grid. The fields were developed using a Bayesian
 30 downscaler (downscaler, Berrocal et al., 2012), neural network (DI2016, Di et al., 2016), random

1 forest (HU2017, Hu et al., 2017), and GWR of residuals from satellite-based PM_{2.5} estimates
 2 (VD2019; van Donkelaar et al., 2019). Annual mean concentrations were developed from daily
 3 PM_{2.5} predictions in the downscaler, DI2016, and HU2017 cases and from monthly PM_{2.5}
 4 predictions in the VD2019 case. General features of the 2011 fields are in reasonable agreement
 5 across methods, with elevated concentrations across broad areas of the eastern U.S. and in the
 6 San Joaquin Valley and South Coast Air Basin of California. The national mean PM_{2.5}
 7 concentration for the VD2019 case (7.06 μg m⁻³) is slightly lower than those of the other cases
 8 (7.36-7.44 μg m⁻³), possibly because the VD2019 fields were developed using monthly (rather
 9 than daily) PM_{2.5} measurements. Use of monthly averages provides greater influence on the
 10 annual mean of sites with less frequent monitoring that tend to be in rural areas with relatively
 11 low concentrations. Mean PM_{2.5} concentrations predicted by the four methods in nine U.S.
 12 climate regions (Karl and Koss, 1984) are provided in Table 2-3.

13



14

15 **Figure 2-31. Comparison of 2011 annual average PM_{2.5} concentrations from four methods.**
 16 (Note: These four methods include: downscaler (Berrocal et al., 2012), DI2016 (Di et al.,
 17 2016), HU2017 (Hu et al., 2017), and VD2019 (van Donkelaar et al., 2019). Predictions have
 18 been averaged to a common 12-km grid for this comparison.)

19

20

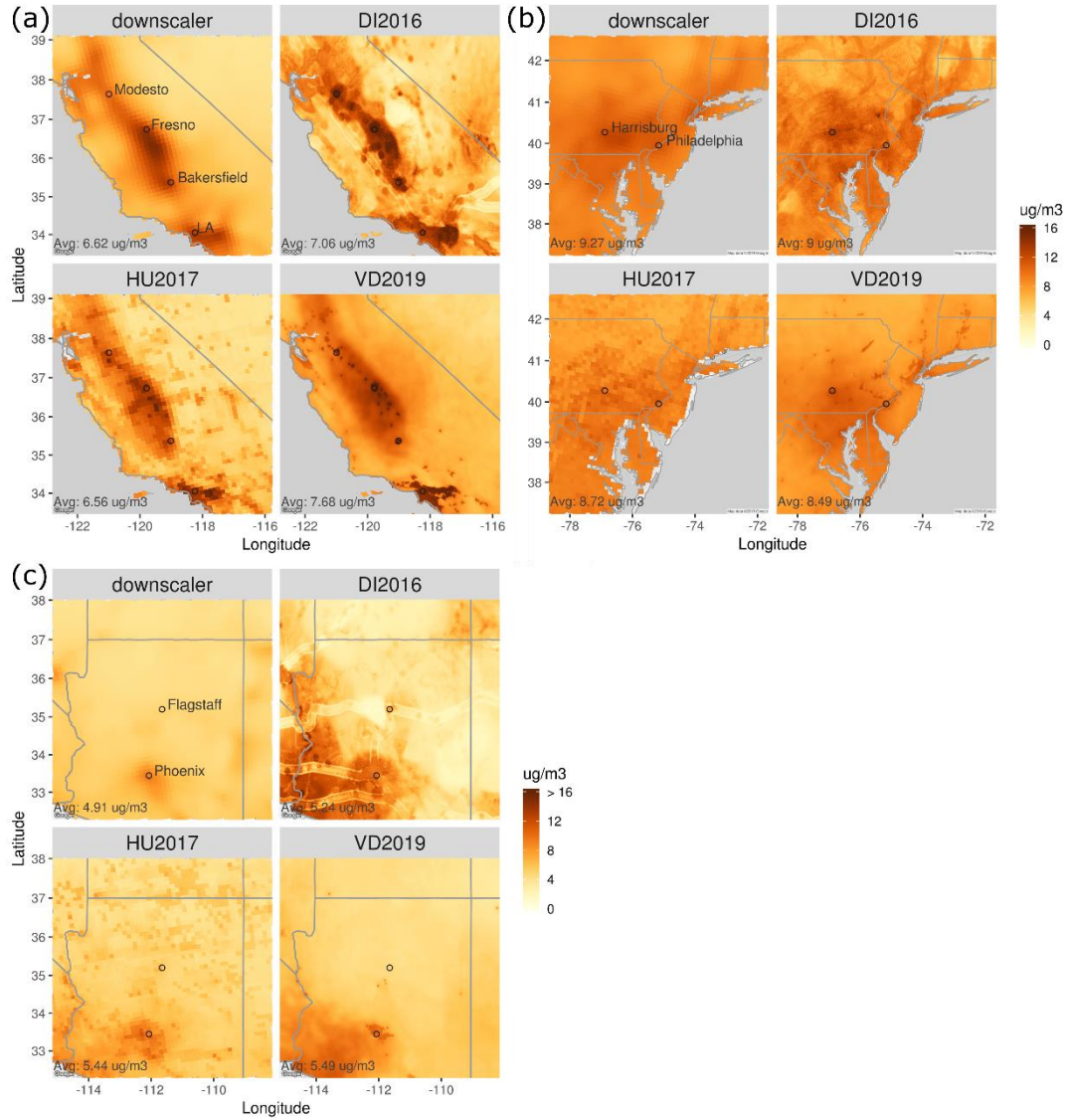
1 **Table 2-3. Mean 2011 PM_{2.5} concentration by region for predictions in Figure 2-29**

Region ¹	downscaler	HU2017	DI2016	VD2019
Northeast	8.5	8.0	8.2	7.5
Southeast	9.9	10.0	9.4	9.8
Ohio Valley	10.7	9.6	9.8	10.0
Upper Midwest	8.8	7.9	7.9	7.1
South	8.8	8.9	9.0	8.7
Southwest	5.0	5.3	5.2	5.1
N. Rockies & Plains	5.6	5.9	5.6	4.5
Northwest	5.0	5.3	6.1	4.9
West	5.5	5.7	6.0	6.5

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

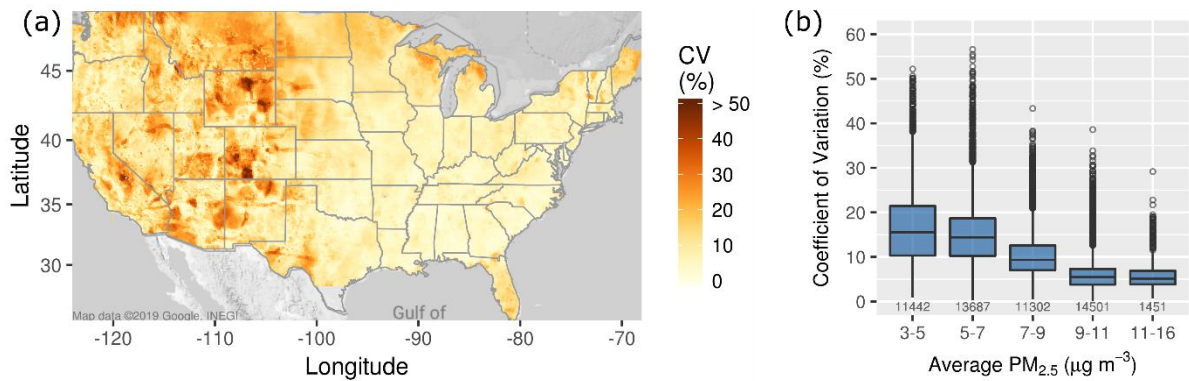
2

3 In Figure 2-32, PM_{2.5} concentrations predicted by the four methods are shown at their
 4 native resolution for regions centered on California, New Jersey, and Arizona. Predictions have
 5 sharper spatial gradients and span a wider range of concentrations for the western regions
 6 centered on California and Arizona (Figure 2-32, panels a and c) than the eastern region centered
 7 on New Jersey (Figure 2-32, panel b). Despite general agreement among predictions for the
 8 California and the eastern U.S. areas, the spatial texture of the concentration fields differs among
 9 methods. For instance, the 12-km Bayesian downscaler produces the smoothest PM_{2.5}
 10 concentration field, and the 1-km neural network (DI2016) produces the field with the greatest
 11 variance. Some of the largest differences in PM_{2.5} concentration among methods occurred over
 12 southwest Arizona. The DI2016 and VD2019 methods predict higher concentrations in this area
 13 than the downscaler and HU2017 methods, and the DI2016 approach predicts distinct spatial
 14 features associated with Interstate 40, 10, and 8 that are not apparent in the other fields (Figure 2-
 15 32, panel c).

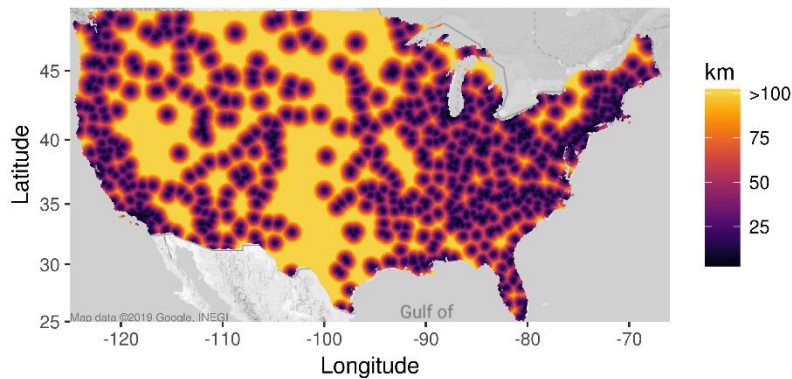


1
 2 **Figure 2-32. Comparison of 2011 annual average PM_{2.5} concentrations from four methods**
 3 **for regions centered on the (a) California (b) New Jersey, and (c) Arizona.** Predictions
 4 are shown at their native resolution (i.e., about 1-km for DI2016 and VD2019 and 12-km for
 5 downscaler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

6
 7 In Figure 2-33, the coefficient of variation (CV; i.e., the standard deviation divided by the
 8 mean) among methods is shown in percentage units based on predictions that were averaged to a
 9 common 12-km grid. The largest values occur in the western U.S. (Figure 2-33, panel a), where
 10 spatial gradients are high, terrain is complex, wildfire is prevalent, monitoring is relatively
 11 sparse, and PM_{2.5} concentrations are low on average. The distance from the grid-cell center to the
 12 nearest monitor is greater than 100 km for broad areas of the west (Figure 2-34).

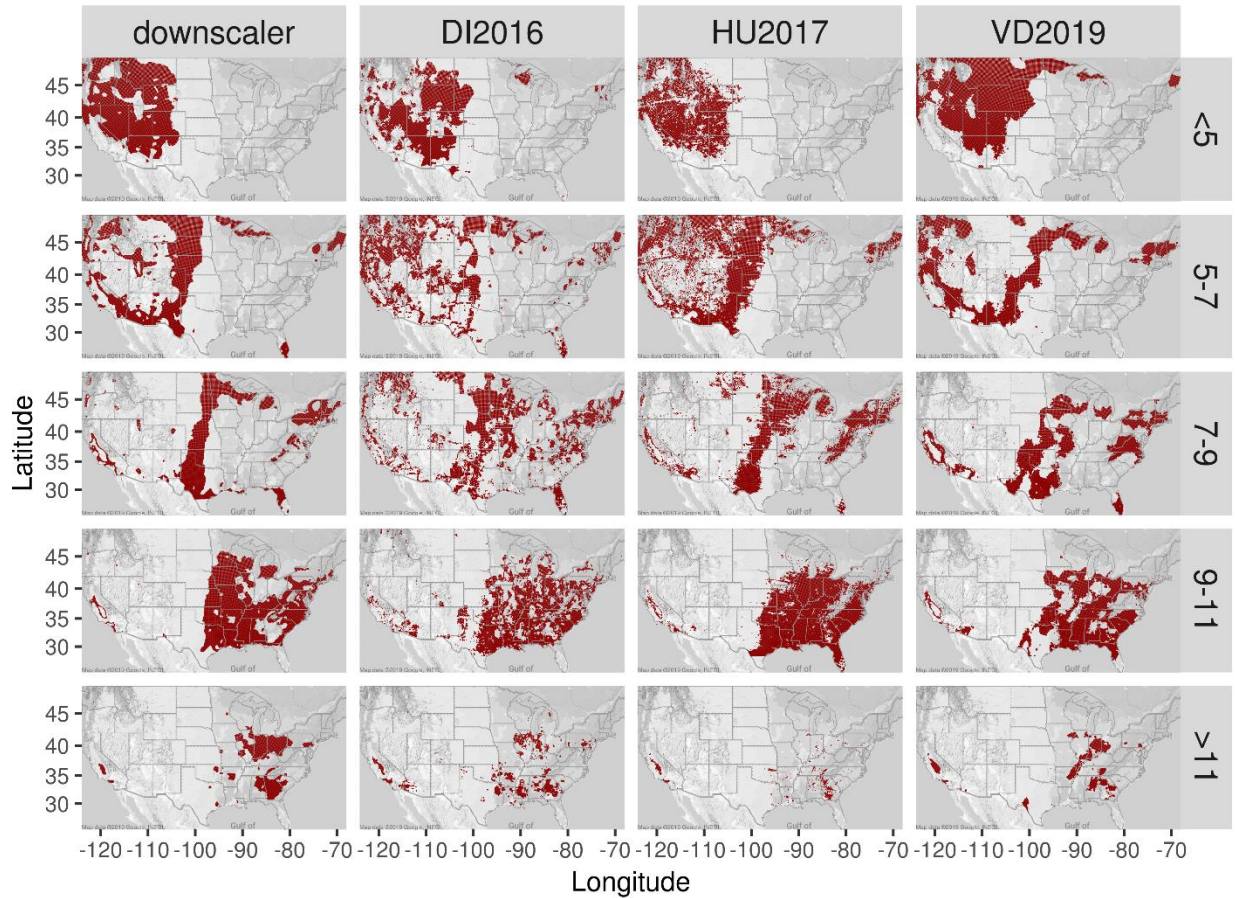


1
2 **Figure 2-33. (a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in**
3 **percentage units for the four models in Figure 2-29. (b) Boxplot distributions of CV for**
4 **grid cells binned by the average PM_{2.5} concentration for the four models. (Note: The box**
5 **brackets the interquartile range (IQR), the horizontal line within the box represents the**
6 **median, the whiskers represent 1.5 times the IQR from either end of the box, and circles**
7 **represent individual values less than and greater than the range of the whiskers.)**



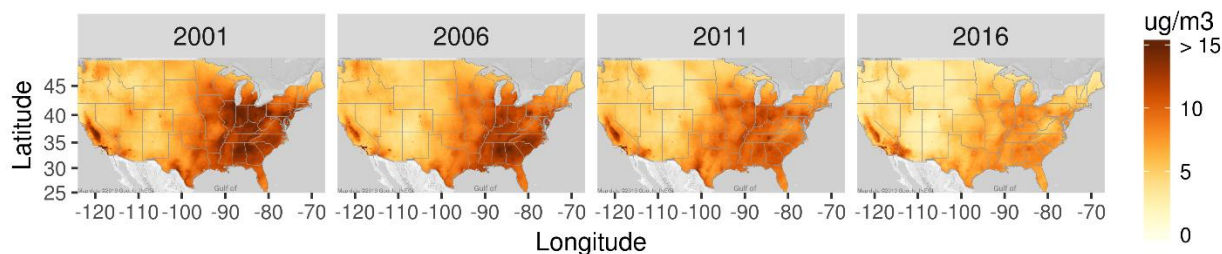
9
10 **Figure 2-34. Distance from the center of the 12-km grid cells to the nearest PM_{2.5}**
11 **monitoring site for PM_{2.5} measurements from the AQS database and IMPROVE**
12 **network.**

13
14 Concentrations less than 5 $\mu\text{g}/\text{m}^3$ occur exclusively in the western U.S. for the downscaler
15 and HU2017 methods, and the western U.S. plus a few areas along the northern U.S. border in
16 the eastern U.S. for the DI2016 and VD2019 methods (Figure 2-35, top row). Concentrations
17 between 5 and 7 $\mu\text{g}/\text{m}^3$ are predicted in the western U.S. and parts of New England for all
18 methods and over Florida by the downscaler and DI2016 approaches (Figure 2-35, second row).
19 The CV among methods increases with decreasing concentration (Figure 2-33 above, panel b),
20 and the median CV is about 15% for grid cells with mean concentrations less than 7 $\mu\text{g}/\text{m}^3$. As
21 illustrated by Figure 2-33 and Figure 2-35, the low-concentration areas with relatively large CVs
22 are in the western U.S. and along the northern and southern border of the eastern U.S.



1
 2 **Figure 2-35. Location of PM_{2.5} predictions by range in annual average concentration for**
 3 **the four prediction methods at their native resolution.** (Note: Concentration ranges: < 5
 4 µg/m³, 5-7 µg/m³, 7-9 µg/m³, 9-11 µg/m³, and >11 µg/m³.)

5
 6 The comparison of PM_{2.5} concentrations across approaches was based on the 2011 period
 7 due to the availability of predictions from multiple methods for that year. As discussed earlier in
 8 this chapter, PM_{2.5} concentrations have declined over the U.S. in the last several decades. Annual
 9 mean PM_{2.5} concentrations predicted by the VD2019 method for 2011 are compared with
 10 predictions for 2001, 2006, and 2016 in Figure 2-36. The VD2019 fields capture the trend of
 11 decreasing PM_{2.5} over the U.S. during this period, and the areas with annual mean PM_{2.5}
 12 concentration greater than 11 µg/m³ in 2016 are limited to California and southwest Arizona.
 13



1
2 **Figure 2-36. Annual mean PM_{2.5} from the VD2019 method (van Donkelaar et al., 2019) for**
3 **2001, 2006, 2011, and 2016.**

4 **2.3.3.2.4 Comparison of PM_{2.5} Fields in Estimating Exposure and Relative to**
5 **Design Values**

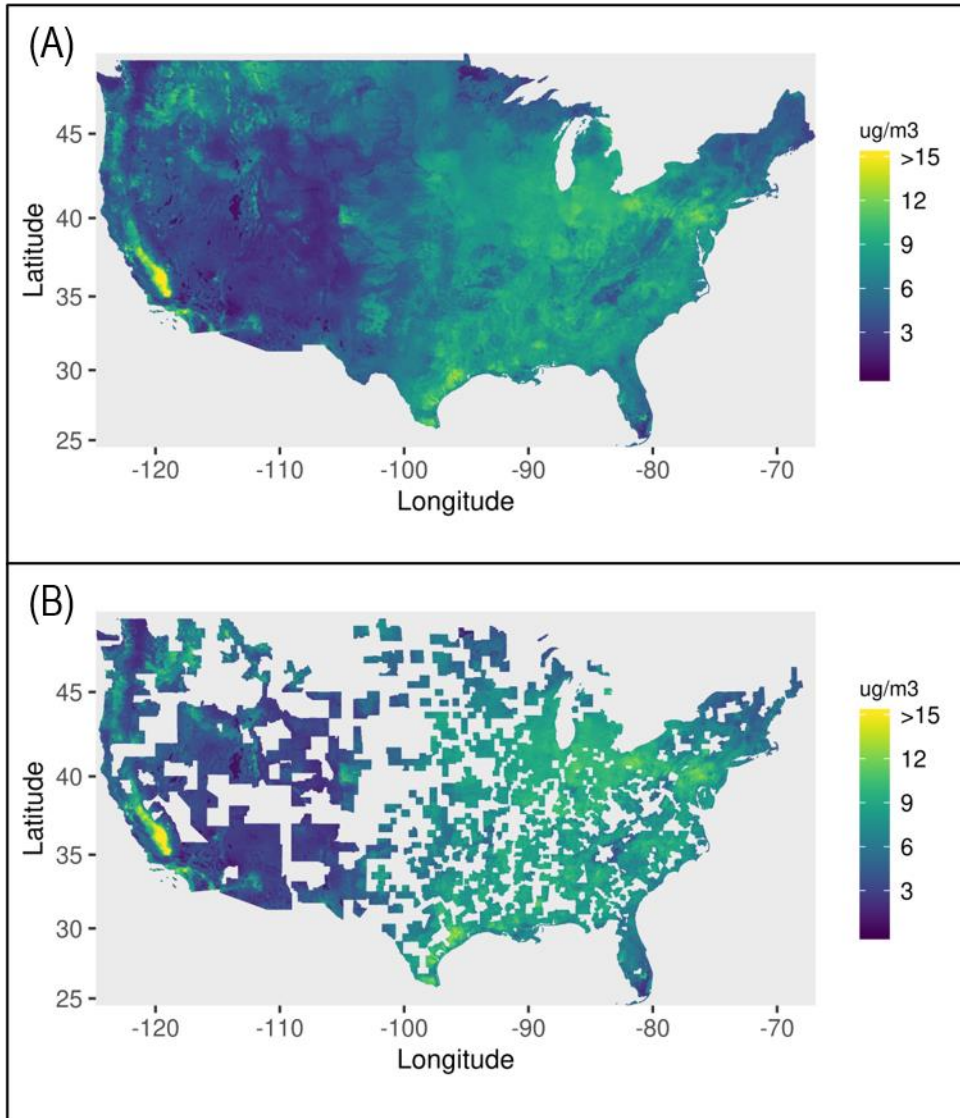
6 Two types of hybrid approaches that have been utilized in several key PM_{2.5}
7 epidemiologic studies in the 2019 ISA and draft ISA Supplement include neural network
8 approaches and use of GWR of residual PM_{2.5} with land-use and other variables to improve
9 estimates of PM_{2.5} concentration in the US. As such, we further compare these two types of
10 approaches across various scales and taking into account population weighting approaches
11 utilized in epidemiologic studies when estimating PM_{2.5} exposure. Additionally, we assess how
12 average PM_{2.5} concentrations computed using these hybrid surfaces compare to the maximum
13 design values measured at ground-based monitors. For this assessment, we evaluate the DI2019³⁷
14 and HA2020³⁸ surfaces. This analysis may help to inform how the magnitude of the overall study
15 reported mean PM_{2.5} concentrations in epidemiologic studies may be influenced by the approach
16 used to compute that mean and how that value might compare to monitor reported
17 concentrations.

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

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

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

1 more urban or densely populated. Further, the areas that are not included in the CBSA-only
2 analysis tend to have lower PM_{2.5} concentrations. These areas tend to be more rural or less
3 densely populated areas, and likely correspond to those locations where monitoring data
4 availability is limited or nonexistent.
5



6
7 **Figure 2-37. Spatial distribution of the annual average PM_{2.5} concentrations for 2015 using**
8 **the DI2019 surface nationwide (panel A) and for CBSAs only (panel B).**

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

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

5 **Table 2-4. Average Annual PM_{2.5} Concentration (µg/m³) by Year.**

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

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

6
7

1 **Table 2-5. Three-Year Average of the Average Annual PM_{2.5} Concentrations (µg/m³).**

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

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

2
3 At the national scale, the average annual PM_{2.5} concentrations are slightly higher when
4 using the DI2019 surface compared to the HA2020 surface but are generally similar. The
5 average annual PM_{2.5} concentrations are also slightly lower using the HA2020 surface compared
6 to the DI2019 surface when the analyses are conducted for CBSAs. However, regardless of
7 which surface is used, the average annual PM_{2.5} concentrations for the CBSA-only analyses are
8 somewhat higher than for the nationwide analyses (4-8% higher), likely reflecting the more
9 urban or densely populated areas in the CBSA-only analyses that typically have higher PM_{2.5} in
10 ambient air compared to more rural or less densely populated areas captured in the nationwide
11 analyses.

12 Similarly, as shown in Table 2-5, for both the DI2019 and HA2020 surfaces, the
13 nationwide average annual PM_{2.5} concentrations, averaged over three years, are lower than the
14 CBSA only average annual PM_{2.5} concentrations, averaged over three years. For the national
15 scale, 3-year averages of the average annual PM_{2.5} concentrations generally range from about 5.3
16 µg/m³ to 8.1 µg/m³, compared to the CBSA scale, which ranges from 5.7 µg/m³ to 8.7 µg/m³.

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

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

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

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

Years of Monitoring Data	No. of CBSAs ^a	Average Annual PM _{2.5} Concentration (µg/m ³) ^b	Population Weighted Average Annual PM _{2.5} Concentration (µg/m ³) ^b	Average Maximum Annual DVs (µg/m ³) ^b	Ratio of Average Maximum Annual DVs to Average Annual PM _{2.5} Concentrations	Ratio of Average Maximum Annual DVs to Population Weighted Average Annual PM _{2.5} Concentrations
DI2019 Surface from Di et al. (2019)						
2008-2010	67	8.61	10.17	11.67	1.48	1.15
2011-2013	64	8.10	9.37	10.91	1.47	1.17
2014-2016	61	7.22	8.26	9.57	1.41	1.17
HA2020 Surface from Hammer et al. (2020) and van Donkelaar et al. (2019)						
2008-2010	67	8.25	9.93	11.67	1.50	1.18
2011-2013	64	7.92	9.34	10.91	1.43	1.17
2014-2016	61	6.98	8.19	9.57	1.43	1.18
^a The number of CBSAs with 3 or more valid design values for the 3-year period						
^b Averaged across CBSAs						

14
 15 As shown in Table 2-6, the results using the DI2019 and HA2020 surfaces are similar for
 16 the average annual PM_{2.5} concentrations, by each 3-year period. When population weighting is
 17 not applied, the average annual PM_{2.5} concentrations generally range from 7.0 to 8.6 µg/m³.
 18 When population weighting is applied, the average annual PM_{2.5} concentrations are slightly
 19 higher, ranging from 8.2 to 10.2 µg/m³. As with CBSAs versus the national comparison above,

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

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

3 For the CBSAs included in the population weighted analyses, the average maximum
4 annual design values generally range from 9.5 to 11.7 µg/m³. As shown in Table 2-6, these
5 analyses show that the results are similar for both the DI2019 and HA2020 surfaces and the
6 maximum annual PM_{2.5} design values are often 40% to 50% higher than average annual PM_{2.5}
7 concentrations when population weighting is not applied. However, when population weighting
8 is applied, the ratio of the maximum annual PM_{2.5} design values to the average annual PM_{2.5}
9 concentrations are lower than when not population weighted, and generally range from 15% to
10 18%.

11 **2.3.3.2.5 Summary**

12 Hybrid PM_{2.5} modeling methods have improved the ability to estimate PM_{2.5} exposure for
13 populations throughout the conterminous U.S. compared with the earlier approaches based on
14 monitoring data alone. Excellent performance in cross-validation tests suggests that hybrid
15 methods are reliable for estimating PM_{2.5} exposure in many applications. As discussed in
16 Chapter 3 of this draft PA, good agreement in health study results between monitor- and model-
17 based methods for urban areas (McGuinn et al., 2017) and general consistency in results for the
18 conterminous U.S. (Jerrett et al., 2017; Di et al., 2016) also suggests that the fields are reliable
19 for use in health studies. However, there are also important limitations associated with the
20 modeled fields. First, performance evaluations for the methods are weighted toward densely
21 monitored urban areas at the scales of representation of the monitoring networks. Predictions at
22 different scales or in sparsely monitored areas are relatively untested. Second, studies have
23 reported heterogeneity in performance with relatively weak performance in parts of the western
24 U.S., at low concentrations, at greater distance to monitors, and under conditions where the
25 reliability and availability of key input datasets (e.g., satellite retrievals and air quality modeling)
26 are limited. Differences in predictions among different hybrid methods have also been reported
27 and tend to be most important under conditions with the performance issues just noted.
28 Differences in predictions could also be related to the different approaches used to create long-
29 term PM_{2.5} fields (e.g., averaging daily PM_{2.5} fields vs. developing long-term average fields),
30 which is important due to variable monitoring schedules. More work is warranted on identifying
31 the most appropriate model performance metrics and comprehensively characterizing model
32 performance to further inform our understanding of the implications of using these fields to
33 estimate PM_{2.5} exposures in health studies.

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

1 concentrations at the national scale and on average across all CBSAs in the U.S. The spatial scale
2 can affect the magnitude of the average annual PM_{2.5} concentration with somewhat higher
3 concentrations (4-8% higher) resulting from averaging across all CBSAs in the U.S. versus
4 averaging across the entire U.S. Additionally, when average annual PM_{2.5} concentrations from
5 the hybrid modeled surfaces are compared to the average maximum annual design value
6 measured at ground-based monitors in a subset of CBSAs, the average of the maximum annual
7 design values tends to be a 40-50% higher than the average annual PM_{2.5} concentration estimated
8 from the hybrid modeling surfaces. When population weighting is introduced, the average of the
9 maximum annual design values tends to only be 15-18% higher than the average annual PM_{2.5}
10 concentration estimated from the hybrid modeling surfaces. This analysis may help better
11 explain why reported study means from different epidemiologic studies can vary and why these
12 mean values tend to be lower than concentrations reported at ground-based monitors. However,
13 it is important to recognize that these results only reflect two surfaces and two types of
14 approaches and that the use of different hybrid methods to estimate mean PM_{2.5} concentrations
15 may influence the comparability across studies.

16 **2.4 BACKGROUND PM**

17 For the purposes of this assessment, we define background PM as all particles that are
18 formed by sources or processes that cannot be influenced by actions within the jurisdiction of
19 concern. For this document, U.S. background PM is defined as any PM formed from emissions
20 other than U.S. anthropogenic (i.e., manmade) emissions. Potential sources of U.S. background
21 PM include both natural sources (i.e., PM that would exist in the absence of any anthropogenic
22 emissions of PM or PM precursors) and transboundary sources originating outside U.S. borders.

23 Ambient monitoring networks provide long-term records of speciated PM concentrations
24 across the U.S., which can inform estimates of individual source contributions to background PM
25 levels in different parts of the country. However, even the most remote monitors within the U.S.
26 can be periodically affected by U.S. anthropogenic emissions. Monitor data are also limited in
27 more remote areas due to a sparser monitoring network where PM concentrations are more likely
28 influenced by background sources. Chemical transport models (CTMs) offer complementary
29 information to ambient monitor networks by providing more spatially and temporally
30 comprehensive estimates of atmospheric composition. CTMs can also be applied to isolate
31 contributions from specific emission sources to PM concentrations in different areas via source
32 apportionment or “zero-out” modeling (i.e., estimating what the residual concentrations would be
33 were emissions from the emission source of interest to be entirely removed).

34 At annual and national scales, estimated background PM concentrations in the U.S. are
35 small compared to contributions from domestic anthropogenic emissions. For example, based on

1 zero-out modeling in the 2012 review of the PM NAAQS, annual background PM_{2.5}
2 concentrations were estimated to range from 0.5 - 3 µg/m³ across the sites examined. The
3 magnitude and sources of background PM can vary widely by region and time of year. Coastal
4 sites may experience a consistent contribution of PM from sea spray aerosol, while other areas
5 covered with dense vegetation may be impacted by biogenic aerosol production during the
6 summertime. Sources of background PM also operate across a range of time scales. While some
7 sources like biogenic aerosol vary at monthly to seasonal scales, many sources of background
8 PM are episodic in nature. These episodic sources (e.g., large wildfires) can be characterized by
9 infrequent contributions to high-concentration events occurring over shorter periods of time (e.g.,
10 hours to several days). Such episodic events are sporadic and do not necessarily occur in all
11 years. While these exceptional episodes can lead to violations of the daily PM_{2.5} standard (35
12 µg/m³) in some cases (Schweizer et al., 2017), such events are routinely screened for and usually
13 identifiable in the monitoring data. As described further below, contributions to background PM
14 in the U.S. result mainly from sources within North America. Contributions from
15 intercontinental events have also been documented (e.g., transport from dust storms occurring in
16 deserts in North Africa and Asia), but these events are less common and represent a relatively
17 small fraction of background PM in most places.

18 While the potential sources of background PM discussed above include sources of both
19 fine (PM_{2.5}) and coarse (PM_{10-2.5}) particles, background contributions to ambient UFP are less
20 well characterized and are not discussed here due to lack of information. Section 2.4.1 below
21 further discusses background PM from natural sources inside the U.S. Section 2.4.2 characterizes
22 the role of international transport of PM from sources outside U.S. borders.

23 **2.4.1 Natural Sources**

24 As noted in section 2.1.1, sources that contribute to natural background PM include dust
25 from the wind erosion of natural surfaces, sea salt, wildland fires, primary biological aerosol
26 particles (PBAP) such as bacteria and pollen, oxidation of biogenic hydrocarbons such as
27 isoprene and terpenes to produce SOA, and geogenic sources such as sulfate formed from
28 volcanic production of SO₂ and oceanic production of dimethyl-sulfide (DMS). While most of
29 the above sources release or contribute predominantly to fine aerosol, some sources including
30 windblown dust, and sea salt also produce particles in the coarse size range (U.S. EPA, 2019b,
31 section 2.3.3).

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

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

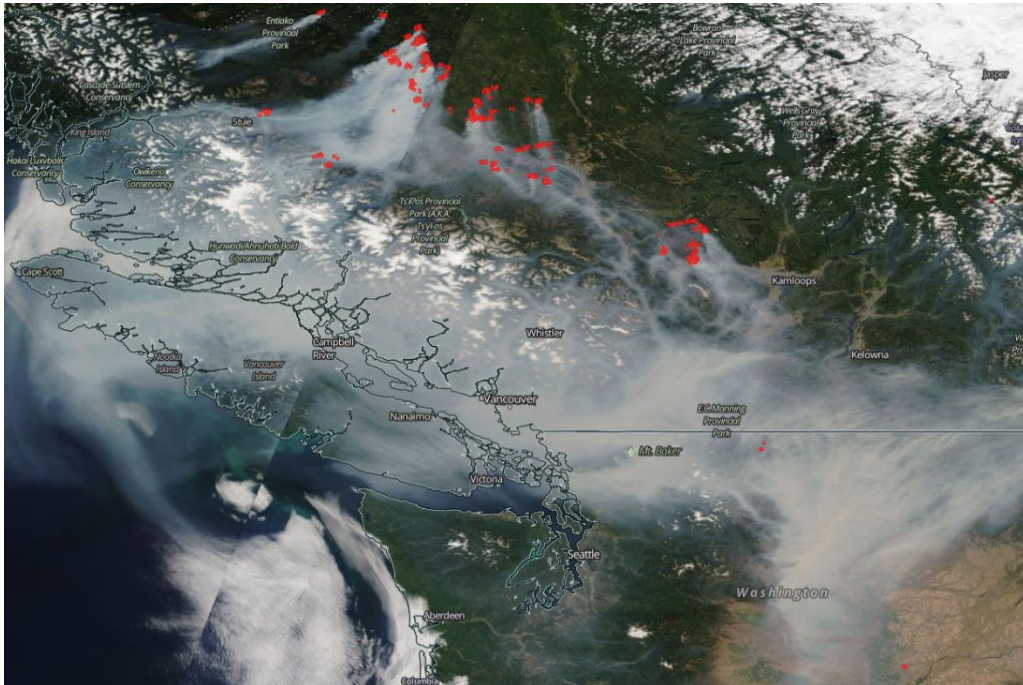
10 Soil dust and sea salt have been estimated to account for less than 10% of urban PM_{2.5} on
11 average in the U.S. (Karagulian et al., 2015), although episodic contributions from these sources
12 can be much higher in some locations. For example, during a dust storm affecting Phoenix in
13 July of 2011, peak hourly average PM₁₀ concentrations were greater than 5,000 µg/m³, with area-
14 wide average hourly concentrations ranging from a few hundred to a few thousand µg/m³
15 (Vukovic et al., 2014). Dust can also account for much of the PM that originates from outside the
16 U.S., which we discuss further below (U.S. EPA, 2019b, section 2.5.4.2). In addition to sea salt
17 aerosol, biological production of the sulfate precursor DMS can also occur in some marine
18 environments, although the impact of DMS emissions on annual mean sulfate concentrations is
19 likely very small in the U.S. (<0.2 µg/m³) and confined to coastal areas (Sarwar et al., 2018).

20 Wildfires release large amounts of particles and gaseous PM precursors. Invasive species,
21 historical fire management practices, frequency of drought, and extreme heat have resulted in
22 longer fire seasons (Jolly et al., 2015) and more large fires (Dennison et al., 2014) over time. In
23 addition to emissions from fires in the U.S., emissions from fires in other countries can be
24 transported to the U.S. Transport of smoke from fires in Canada, Mexico, Central America, and
25 Siberia have been documented in multiple studies (U.S. EPA, 2009). According to the NEI,
26 wildfire smoke contributes between 10 and 20% of primary PM emissions in the U.S. per year
27 (U.S. EPA, 2019b, section 2.3.1), with much higher localized contributions near fire-affected
28 areas.

29 To illustrate how episodic impacts from a large natural source can affect PM
30 concentrations in the U.S., Figure 2-38 and Figure 2-39 show an example from a recent wildfire
31 event. In summer 2017, smoke from wildfires in British Columbia, Canada led to severe air
32 quality degradation in parts of the Pacific Northwest. A NASA Worldview⁴⁰ image from August
33 4, 2017 (Figure 2-38) shows smoke from multiple fire detections across southern British
34 Columbia crossing into northern Washington state. Smoke from these fires was also captured at

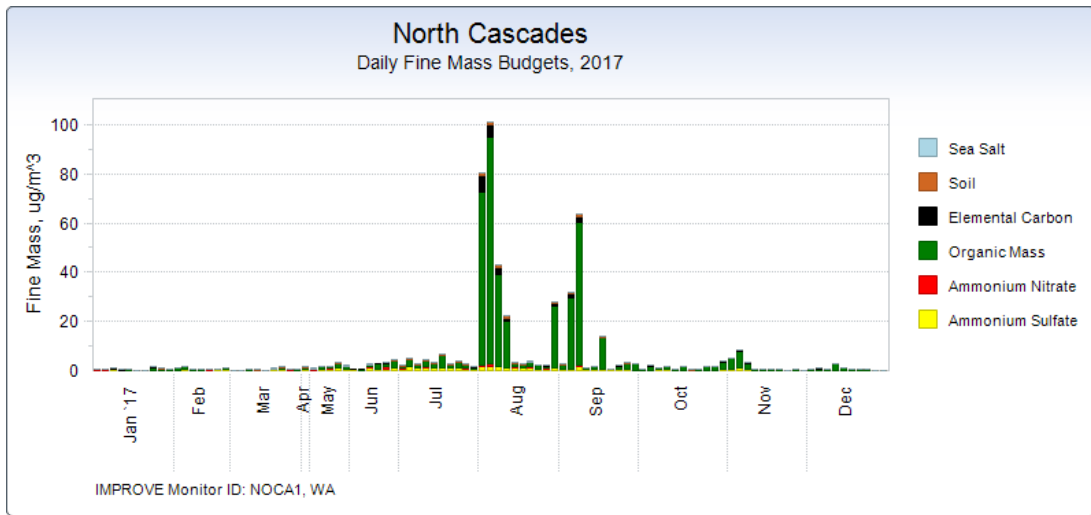
⁴⁰ Available from <https://worldview.earthdata.nasa.gov>.

1 the North Cascades IMPROVE monitor (Figure 2-39), where daily fine PM concentrations were
2 increased from a typical baseline of less than $10 \mu\text{g}/\text{m}^3$ to $\sim 100 \mu\text{g}/\text{m}^3$ during this time.



3
4 **Figure 2-38. Smoke and fire detections observed by the MODIS instrument onboard the**
5 **Aqua satellite on August 4th, 2017 accessed through NASA Worldview.**

6



7
8 **Figure 2-39. Fine PM mass time series during 2017 from the North Cascades IMPROVE**
9 **site in north central Washington state.**⁴¹

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

1 Later in August and September 2017, many other wildfires occurred in Washington state
2 and Oregon, making this fire season one of the worst for the Pacific Northwest in recent history.
3 The severe fires in British Columbia, Washington and Oregon during 2017 have been linked to
4 the combination of usually hot temperatures in August/September in the region following a very
5 wet preceding winter season. While many of the most severe wildfire events in the U.S. occur in
6 the western part of the country during the late summer, most of the contiguous U.S. is affected
7 by wildfire smoke during some part of the year (Kaulfus et al., 2017).

8 **2.4.2 International Transport**

9 Background PM contributions from international sources include PM that is both natural
10 and anthropogenic in origin crossing into U.S. borders from Canada and Mexico or from longer
11 range intercontinental transport. While in general the biggest contributions to U.S. background
12 PM from international sources come from nearby Canada and Mexico, large episodic events
13 from intercontinental sources can sometimes occur (e.g., windblown dust from Asia or Africa).
14 This section discusses transboundary PM transport within North America (section 2.4.2.1) as
15 well as long range intercontinental transport from anthropogenic (section 2.4.2.2) and natural
16 (section 2.4.2.3) sources.

17 **2.4.2.1 Transboundary Transport in North America**

18 As discussed above, some of the largest potential international sources of U.S.
19 background PM originate elsewhere in North America. PM produced from fires in both Canada
20 and Mexico can affect air quality in the U.S., particularly in border states (Park et al., 2007;
21 Miller et al., 2011; Wang et al., 2018a). Anthropogenic emissions from Canada and Mexico can
22 also influence U.S. PM air quality. An inverse modeling study by Henze et al. (2009) estimated
23 that in 2001 anthropogenic SO_x emissions from Canada and Mexico accounted for 6% and 4%
24 respectively of total daily inorganic PM_{2.5} in the U.S. These authors also estimated that SO_x
25 emissions related to international shipping accounted for approximately 2% of total inorganic
26 PM in the U.S.

27 **2.4.2.2 Long Range Transport from Anthropogenic Sources**

28 Due to the relatively short atmospheric lifetime of particles (~days to weeks), long range
29 transport of aerosols does not contribute significant PM mass to the U.S. Heald et al. (2006)
30 estimated that transport from Asia accounted for less than 0.2 μg/m³ of sulfate PM_{2.5} in the
31 Northwestern U.S. in spring, and Leibensperger et al. (2011) estimated intercontinental
32 contributions from Asian anthropogenic SO₂ and NO_x emissions of 0.1 - 0.25 μg/m³ annually in
33 the western U.S. Leibensperger et al. (2011) also concluded that much of the intercontinental
34 influence captured by the GEOS-Chem model was in fact local PM production attributable to

1 domestic emissions in receptor countries arising from changes in global oxidant budgets, rather
2 than impacts from PM directly transported across geopolitical boundaries. The studies above are
3 also consistent with findings from other analyses. A report from the United Nations on global air
4 quality synthesizing results across many studies estimated an annual average contribution of
5 approximately $0.1 \mu\text{g}/\text{m}^3$ sulfate PM in North America due to transport from East Asia
6 (TFHTAP, 2006).

7 **2.4.2.3 Long Range Transport from Natural Sources**

8 Long range transport of dust from both Asia (Vancuren and Cahill, 2002; Yu et al., 2008)
9 and North Africa (Prospero, 1999b; Prospero, 1999a; Chiapello et al., 2005; McKendry et al.,
10 2007) has been shown to occasionally contribute to surface PM concentrations in some regions
11 of the U.S. The likelihood of such long-range dust transport events depends on large-scale
12 meteorological patterns, which can vary significantly across seasons and between years. Yu et al.
13 (2015) found that the transport of North African dust across the Atlantic Ocean is strongly
14 negatively correlated with precipitation in the Sahel during the preceding year. Dust from Africa
15 has also shown a decreasing trend of approximately 10% per decade from 1982 to 2008 based on
16 measurements of aerosol optical depth and surface concentrations in Barbados. This trend was
17 attributed to a corresponding decrease in surface winds over source regions (Ridley et al., 2014).
18 Variability in springtime Asian dust transport to the U.S. has been linked to north-south shifts in
19 trans-Pacific flow modulated by the El Nino-Southern Oscillation (Achakulwisut et al., 2017), as
20 well as to variations in regional precipitation affecting both dust emissions in Asia and
21 atmospheric residence times during transport (Fischer et al., 2009).

22 On average, intercontinental dust transport is estimated to contribute about $1\text{-}2 \mu\text{g}/\text{m}^3$ to
23 annual $\text{PM}_{2.5}$ at some U.S. sites (Jaffe et al., 2005; TFHTAP, 2006; Creamean et al., 2014).
24 However, daily concentrations can be substantially larger for individual events, especially for
25 coarser particles. For example, Jaffe et al. (2003) found evidence of Asian dust events in 1998
26 and 2001 contributing $30\text{-}40 \mu\text{g}/\text{m}^3$ to daily PM_{10} at sites throughout the U.S., although the
27 authors also note that large events of this scale are rare and only occurred twice during their 15-
28 year study period. Similar magnitudes have also been reported for individual North African
29 events; analysis of a multidecadal record of African dust reaching Miami indicated
30 concentrations of PM ranging from ~ 10 to $120 \mu\text{g}/\text{m}^3$ (Prospero, 1999a; Prospero, 1999b).⁴² In
31 June 2020 a large dust transport episode originating in North Africa may have contributed up to
32 $50 \mu\text{g}/\text{m}^3$ for several days at multiple sites in the southeastern U.S. (Pu and Jin, 2021).

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

1 2.4.3 Estimating Background PM with Recent Data

2 As discussed above, the 2009 PM ISA estimated background PM concentrations at
3 several remote IMPROVE sites in different regions of the U.S. for 2004 using a combination of
4 monitor data and zero-out air quality modeling. Revisiting the speciated IMPROVE PM data at
5 the monitors included in the 2009 ISA assessment provides some insights into how contributions
6 from different PM sources may have changed, and what those changes (or lack thereof) mean for
7 our current understanding of background PM in the U.S.

8 Figure 2-40 shows observed annual average PM_{2.5} in 2004 and 2016 at the same remote
9 monitors examined in the 2009 ISA. The comparisons show decreases in both total PM_{2.5} and
10 ammonium sulfate across all sites examined, consistent with decreases in anthropogenic SO₂ and
11 other PM precursors observed over this time period. It is likely that most of the remaining
12 ammonium sulfate observed at these sites is also a result of domestic anthropogenic emissions
13 and therefore not relevant for assessments of background PM.

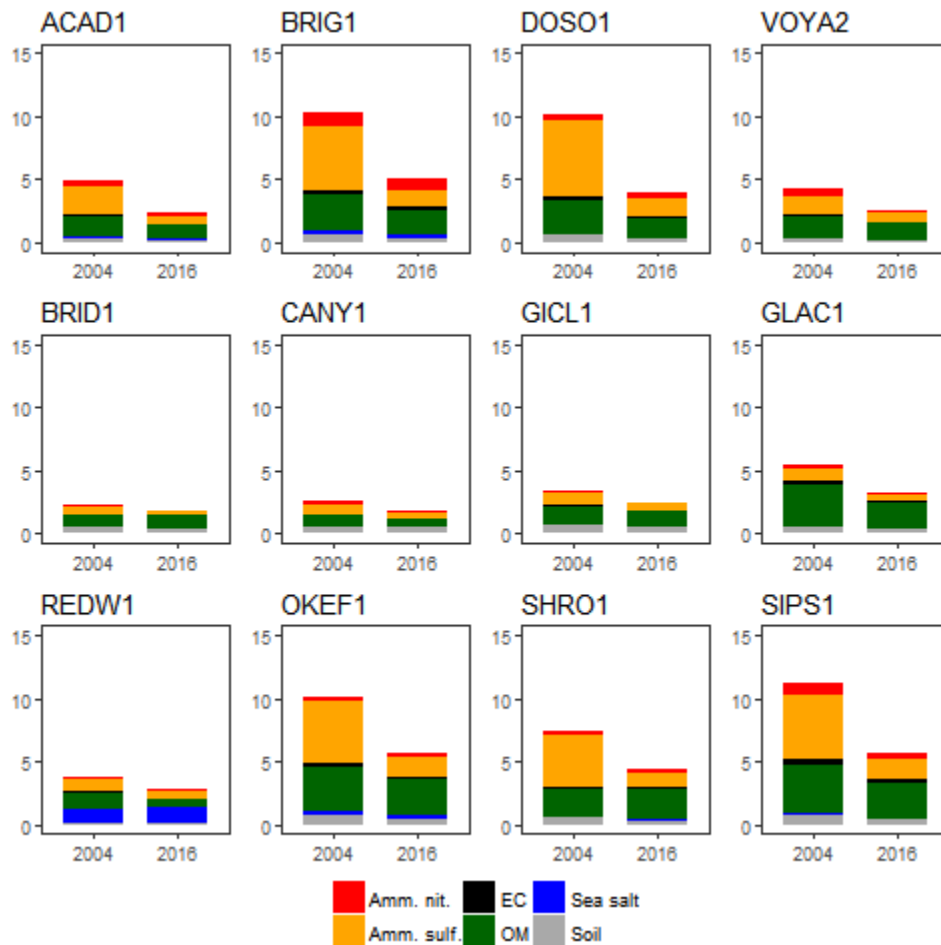
14 Sea salt and dust aerosol are likely natural in origin at these remote sites. With the
15 exception of REDW1, a coastal site in California, soil and sea salt aerosol together account for
16 less than about 0.5 μg/m³ of the annual average PM_{2.5} at all monitors examined here, which is
17 below the values cited from the literature for long range dust contributions discussed above.
18 Contributions from ammonium nitrate and elemental carbon could be from either anthropogenic
19 or natural sources, but together represent less than about 0.5 μg/m³ at most of the sites in 2016.
20 The largest contribution from nitrate occurs at the BRIG1 monitor in New Jersey and is likely
21 anthropogenic given the high density of NO_x from vehicle emissions in that region.

22 After ammonium sulfate, the next largest contributing species for most of the sites is
23 organic matter, which for many of the monitors in Figure 2-40 represents 50% or more of total
24 PM in both 2004 and 2016. In addition to the IMPROVE sites from the 2019 ISA, Figure 2-40
25 also shows comparisons for three sites in the Southeast U.S. As a region, the Southeast has the
26 highest levels of biogenic aerosol production in the country, so the organic matter contribution at
27 these three sites likely represents an upper bound for the country of what natural biogenic
28 organic aerosol production could be under present atmospheric conditions. The organic aerosol
29 components shown in Figure 2-37 will also include the influence of fires for some monitors. The
30 highest organic matter contribution for any of the sites shown in Figure 2-40, including the three
31 Southeast monitors, is approximately 2 μg/m³. While contributions from ammonium sulfate have
32 decreased substantially at some of the monitors, particularly the eastern sites, contributions from
33 organic aerosol are roughly consistent between 2004 and 2016, as are the contributions from the
34 other species assumed to be mostly natural in origin (soil and sea salt). Therefore, while no new
35 zero-out modeling was done for the reconsideration, revisiting these monitors with more recent

1 data suggests that estimates of background concentrations at these monitors are still around 1-3
 2 $\mu\text{g}/\text{m}^3$ and have not changed significantly since the 2012 PM NAAQS review.

3 While estimates of total annual background concentrations have generally not changed
 4 significantly since the 2012 review, our scientific understanding of organic aerosol formation has
 5 evolved. Organic aerosol can be produced from a variety of natural and anthropogenic processes,
 6 which presents a challenge for source attribution techniques. Additionally, new research over the
 7 past decade has identified a host of new sources and chemical pathways for SOA formation that
 8 have only recently begun to be implemented into CTMs. Further research implementing these
 9 new sources and pathways into CTMs is needed to understand 1) the behavior of these different
 10 algorithms under a range of possible atmospheric conditions, and 2) what the implications are for
 11 understanding SOA formation in the U.S.

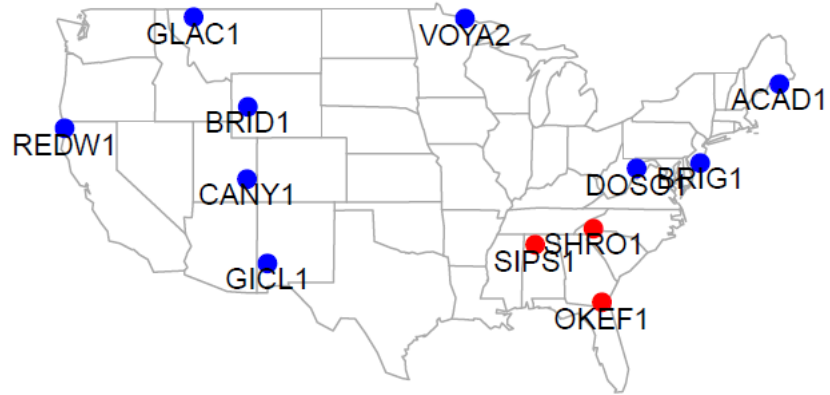
12



13

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

16



1
 2 **Figure 2-41. Site locations for the IMPROVE monitors in Figure 2-40.** (Note: Monitors also
 3 assessed in the 2009 ISA are shown in blue. Monitors only examined in this assessment are
 4 shown in red.)

1 **REFERENCES**

- 2 Abt Associates, Inc. (2014). User's guide: modeled attainment test software. U.S. Environmental
3 Protection Agency. Research Triangle Park, NC.
- 4 Achakulwisut, P, Shen, L and Mickley, LJ (2017). What controls springtime fine dust variability
5 in the western United States? Investigating the 2002-2015 increase in fine dust in the US
6 southwest. *Journal of Geophysical Research: Atmospheres* 122(22): 12449-12467.
- 7 Berrocal, VJ, Gelfand, AE and Holland, DM (2012). Space-time data fusion under error in
8 computer model output: An application to modeling air quality. *Biometrics* 68(3): 837-
9 848.
- 10 Berrocal, VJ, Guan, Y, Muyskens, A, Wang, H, Reich, BJ, Mulholland, JA and Chang, HH
11 (2020). A comparison of statistical and machine learning methods for creating national
12 daily maps of ambient PM_{2.5} concentration. *Atmospheric Environment* 222: 117130.
- 13 Boys, BL, Martin, RV, van Donkelaar, A, MacDonell, RJ, Hsu, NC, Cooper, MJ, Yantosca, RM,
14 Lu, Z, Streets, DG, Zhang, Q and Wang, SW (2014). Fifteen-year global time series of
15 satellite-derived fine particulate matter. *Environmental Science and Technology* 48(19):
16 11109-11118.
- 17 Chan, EAW, Gantt, B and McDow, S (2018). The reduction of summer sulfate and switch from
18 summertime to wintertime PM_{2.5} concentration maxima in the United States.
19 *Atmospheric Environment* 175: 25-32.
- 20 Chiapello, I, Moulin, C and Prospero, JM (2005). Understanding the long-term variability of
21 African dust transport across the Atlantic as recorded in both Barbados surface
22 concentrations and large-scale Total Ozone Mapping Spectrometer (TOMS) optical
23 thickness. *Journal of Geophysical Research* 110(D18): D18S10.
- 24 Creamean, JM, Spackman, JR, Davis, SM and White, AB (2014). Climatology of long-range
25 transported Asian dust along the West Coast of the United States. *Journal of Geophysical*
26 *Research: Atmospheres* 119(21): 12171-12185.
- 27 Day, M, Pouliot, G, Hunt, S, Baker, KR, Beardsley, M, Frost, G, Mobley, D, Simon, H,
28 Henderson, B and Yelverton, T (2019). Reflecting on progress since the 2005 NARSTO
29 emissions inventory report. *Journal of the Air Waste Management Association* 69(9):
30 1023-1048.
- 31 Dennison, PE, Brewer, SC, Arnold, JD and Moritz, MA (2014). Large wildfire trends in the
32 western United States, 1984-2011. *Geophysical Research Letters* 41(8): 2928-2933.
- 33 DeWinter, J, Brown, S, Seagram, A, Landsberg, K and Eisinger, D (2018). A national-scale
34 review of air pollutant concentrations measured in the U.S. near-road monitoring network
35 during 2014 and 2015. *Atmospheric Environment* 183.

- 1 DHEW (1969). Air Quality Criteria for Particulate Matter. National Air Pollution Control
2 Administration. Washington, D.C. U.S. Department of Health. January 1969.
- 3 Di, Q, Amini, H, Shi, L, Kloog, I, Silvern, R, Kelly, J, Sabath, MB, Choirat, C, Koutrakis, P and
4 Lyapustin, A (2019). An ensemble-based model of PM_{2.5} concentration across the
5 contiguous United States with high spatiotemporal resolution. *Environment International*
6 130: 104909.
- 7 Di, Q, Kloog, I, Koutrakis, P, Lyapustin, A, Wang, Y and Schwartz, J (2016). Assessing PM_{2.5}
8 exposures with high spatiotemporal resolution across the Continental United States.
9 *Environmental Science and Technology* 50(9): 4712-4721.
- 10 Diao, M, Holloway, T, Choi, S, O'Neill, SM, Al-Hamdan, MZ, Van Donkelaar, A, Martin, RV,
11 Jin, X, Fiore, AM and Henze, DK (2019). Methods, availability, and applications of
12 PM_{2.5} exposure estimates derived from ground measurements, satellite, and atmospheric
13 models. *Journal of the Air & Waste Management Association* 69(12): 1391-1414.
- 14 Fischer, EV, Hsu, NC, Jaffe, DA, Jeong, MJ and Gong, SL (2009). A decade of dust: Asian dust
15 and springtime aerosol load in the US Pacific Northwest. *Geophysical Research Letters*
16 36.
- 17 Friberg, MD, Zhai, X, Holmes, HA, Chang, HH, Strickland, MJ, Sarnat, SE, Tolbert, PE,
18 Russell, AG and Mulholland, JA (2016). Method for fusing observational data and
19 chemical transport model simulations to estimate spatiotemporally resolved ambient air
20 pollution. *Environmental Science and Technology* 50(7): 3695-3705.
- 21 Gantt, B, Owen, RC and Watkins, N (2021). Characterizing Nitrogen Oxides and Fine
22 Particulate Matter near Major Highways in the United States Using the National Near-
23 Road Monitoring Network. *Environmental science & technology* 55(5): 2831-2838.
- 24 Geng, G, Murray, NL, Tong, D, Fu, JS, Hu, X, Lee, P, Meng, X, Chang, HH and Liu, Y (2018).
25 Satellite-based daily PM_{2.5} estimates during fire seasons in Colorado. *Journal of*
26 *Geophysical Research: Atmospheres* 123(15): 8159-8171.
- 27 Hammer, MS, van Donkelaar, A, Li, C, Lyapustin, A, Sayer, AM, Hsu, NC, Levy, RC, Garay,
28 MJ, Kalashnikova, OV and Kahn, RA (2020). Global estimates and long-term trends of
29 fine particulate matter concentrations (1998–2018). *Environmental Science &*
30 *Technology* 54(13): 7879-7890.
- 31 Hand, JL, Schichtel, BA, Malm, WC and Pitchford, ML (2012). Particulate sulfate ion
32 concentration and SO₂ emission trends in the United States from the early 1990s through
33 2010. *Atmospheric Chemistry and Physics* 12(21): 10353-10365.
- 34 Heald, CL, Jacob, DJ, Park, RJ, Alexander, B, Fairlie, TD, Yantosca, RM and Chu, DA (2006).
35 Transpacific transport of Asian anthropogenic aerosols and its impact on surface air
36 quality in the United States. *Journal of Geophysical Research: Atmospheres* 111(D14).

- 1 Henze, DK, Seinfeld, JH and Shindell, DT (2009). Inverse modeling and maing US air quality
2 influences of inorganic PM(2.5) precursor emissions using the adjoint of GEOS-Chem.
3 Atmospheric Chemistry and Physics 9(16): 5877-5903.
- 4 Hu, X, Belle, JH, Meng, X, Wildani, A, Waller, LA, Strickland, MJ and Liu, Y (2017).
5 Estimating PM_{2.5} Concentrations in the Conterminous United States Using the Random
6 Forest Approach. Environmental Science and Technology 51(12): 6936-6944.
- 7 Hu, X, Waller, LA, Lyapustin, A, Wang, Y and Liu, Y (2014). 10-year spatial and temporal
8 trends of PM_{2.5} concentrations in the southeastern US estimated using high-resolution
9 satellite data. Atmospheric Chemistry and Physics 14(12): 6301-6314.
- 10 Huang, Ra, Zhai, X, Ivey, CE, Friberg, MD, Hu, X, Liu, Y, Di, Q, Schwartz, J, Mulholland, JA
11 and Russell, AG (2018). Air pollutant exposure field modeling using air quality model-
12 data fusion methods and comparison with satellite AOD-derived fields: application over
13 North Carolina, USA. Air Quality, Atmosphere and Health 11(1): 11-22.
- 14 Jaffe, D, Snow, J and Cooper, O (2003). The 2001 Asian Dust Events: Transport and impact on
15 surface aerosol concentrations in the U.S. Eos 84(46): 501-506.
- 16 Jaffe, D, Tamura, S and Harris, J (2005). Seasonal cycle and composition of background fine
17 particles along the west coast of the US. Atmospheric Environment 39(2): 297-306.
- 18 Jerrett, M, Turner, MC, Beckerman, BS, Pope, CA, van Donkelaar, A, Martin, RV, Serre, M,
19 Crouse, D, Gapstur, SM, Krewski, D, Diver, WR, Coogan, PF, Thurston, GD and
20 Burnett, RT (2017). Comparing the health effects of ambient particulate matter estimated
21 using ground-based versus remote sensing exposure estimates. Environmental health
22 perspectives 125(4): 552-559.
- 23 Jin, X, Fiore, AM, Civerolo, K, Bi, J, Liu, Y, van Donkelaar, A, Martin, RV, Al-Hamdan, M,
24 Zhang, Y, Insaf, TZ, Kioumourtzoglou, M-A, He, MZ and Kinney, PL (2019).
25 Comparison of multiple PM_{2.5} exposure products for estimating health benefits of
26 emission controls over New York State, USA. Environmental Research Letters 14(8):
27 084023.
- 28 Jolly, WM, Cochrane, MA, Freeborn, PH, Holden, ZA, Brown, TJ, Williamson, GJ and
29 Bowman, DMJS (2015). Climate-induced variations in global wildfire danger from 1979
30 to 2013. Nature Communications 6.
- 31 Just, AC, Arfer, KB, Rush, J, Dorman, M, Shtein, A, Lyapustin, A and Kloog, I (2020).
32 Advancing methodologies for applying machine learning and evaluating spatiotemporal
33 models of fine particulate matter (PM_{2.5}) using satellite data over large regions.
34 Atmospheric Environment 239: 117649.
- 35 Karagulian, F, Belis, CA, Dora, CFC, Pruess-Ustuen, AM, Bonjour, S, Adair-Rohani, H and
36 Amann, M (2015). Contributions to cities' ambient particulate matter (PM): A systematic
37 review of local source contributions at global level. Atmospheric Environment 120: 475-
38 483.

- 1 Karl, T and Koss, WJ (1984). *Regional and national monthly, seasonal, and annual temperature*
2 *weighted by area, 1895-1983*. National Climatic Data Center. Asheville, NC.
- 3 Kaulfus, AS, Nair, U, Jaffe, D, Christopher, SA and Goodrick, S (2017). Biomass burning smoke
4 climatology of the United States: implications for particulate matter air quality.
5 *Environmental Science and Technology* 51(20): 11731-11741.
- 6 Keller, JP and Peng, RD (2019). Error in estimating area-level air pollution exposures for
7 epidemiology. *Environmetrics* 0(0): e2573.
- 8 Kelly, JT, Jang, C, Timin, B, Di, Q, Schwartz, J, Liu, Y, van Donkelaar, A, Martin, RV,
9 Berrocal, V and Bell, ML (2021). Examining PM_{2.5} concentrations and exposure using
10 multiple models. *Environmental Research* 196: 110432.
- 11 Kelly, JT, Jang, CJ, Timin, B, Gantt, B, Reff, A, Zhu, Y, Long, S and Hanna, A (2019a). A
12 system for developing and projecting PM_{2.5} spatial fields to correspond to just meeting
13 National Ambient Air Quality Standards. *Atmospheric Environment: X* 2: 100019.
- 14 Kelly, JT, Koplitz, SN, Baker, KR, Holder, AL, Pye, HOT, Murphy, BN, Bash, JO, Henderson,
15 BH, Possiel, NC, Simon, H, Eyth, AM, Jang, CJ, Phillips, S and Timin, B (2019b).
16 Assessing PM_{2.5} model performance for the conterminous U.S. with comparison to model
17 performance statistics from 2007-2015. *Atmospheric Environment* 214: 116872.
- 18 Leibensperger, EM, Mickley, LJ, Jacob, DJ and Barrett, SRH (2011). Intercontinental influence
19 of NO(x) and CO emissions on particulate matter air quality. *Atmospheric Environment*
20 45(19): 3318-3324.
- 21 Li, Y, Mickley, LJ and Kaplan, JO (2021). Response of dust emissions in southwestern North
22 America to 21st century trends in climate, CO₂ fertilization, and land use: implications
23 for air quality. *Atmos. Chem. Phys.* 21(1): 57-68.
- 24 Liu, Y, Goodrick, S and Heilman, W (2014). Wildland fire emissions, carbon, and climate:
25 Wildfire–climate interactions. *Forest Ecology and Management* 317: 80-96.
- 26 McGuinn, LA, Ward-Caviness, C, Neas, LM, Schneider, A, Di, Q, Chudnovsky, A, Schwartz, J,
27 Koutrakis, P, Russell, AG, Garcia, V, Kraus, WE, Hauser, ER, Cascio, W, Diaz-Sanchez,
28 D and Devlin, RB (2017). Fine particulate matter and cardiovascular disease:
29 Comparison of assessment methods for long-term exposure. *Environmental Research*
30 159: 16-23.
- 31 McKendry, IG, Strawbridge, KB, O'Neill, NT, MacDonald, AM, Liu, PSK, Leitch, WR,
32 Anlauf, KG, Jaegle, L, Fairlie, TD and Westphal, DL (2007). Trans-Pacific transport of
33 Saharan dust to western North America: A case study. *Journal of Geophysical Research*
34 112(D1): D01103.
- 35 Miller, DJ, Sun, K, Zondlo, MA, Kanter, D, Dubovik, O, Welton, EJ, Winker, DM and Ginoux,
36 P (2011). Assessing boreal forest fire smoke aerosol impacts on U.S. air quality: A case
37 study using multiple data sets. *Journal of Geophysical Research* 116(D22): D22209.

- 1 Park, RJ, Jacob, DJ and Logan, JA (2007). Fire and biofuel contributions to annual mean aerosol
2 mass concentrations in the United States. *Atmospheric Environment* 41(35): 7389-7400.
- 3 Prospero, JM (1999a). Long-term measurements of the transport of African mineral dust to the
4 southeastern United States: implications for regional air quality. *Journal of Geophysical*
5 *Research* 104(D13): 15917-15927.
- 6 Prospero, JM (1999b). Long-range transport of mineral dust in the global atmosphere: Impact of
7 African dust on the environment of the southeastern United States. *Proceedings of the*
8 *National Academy of Sciences* 96(7): 3396-3403.
- 9 Pu, B and Ginoux, P (2017). Projection of American dustiness in the late 21st century due to
10 climate change. *Scientific Reports* 7(1): 5553.
- 11 Pu, B and Jin, Q (2021). A Record-Breaking Trans-Atlantic African Dust Plume Associated with
12 Atmospheric Circulation Extremes in June 2020 %J *Bulletin of the American*
13 *Meteorological Society*. 102(7): E1340-E1356.
- 14 Ridley, DA, Heald, CL and Prospero, JM (2014). What controls the recent changes in African
15 mineral dust aerosol across the Atlantic? *Atmospheric Chemistry and Physics* 14(11):
16 5735-5747.
- 17 Saha, PK, Robinson, ES, Shah, RU, Zimmerman, N, Apte, JS, Robinson, AL and Presto, AA
18 (2018). Reduced ultrafine particle concentration in urban air: changes in nucleation and
19 anthropogenic emissions. *Environmental Science and Technology* 52(12): 6798-6806.
- 20 Sarwar, G, Xing, Ji, Fahey, K, Foley, K, Wong, D, Mathur, R, Gan, CM, Gantt, B and Simon, H
21 (2018). Dimethylsulfide Chemistry: Annual, Seasonal, and Spatial Impacts on Sulfate.
22 *International Technical Meeting on Air Pollution Modelling and its Application*: 347-
23 352.
- 24 Schichtel, BA, Malm, WC, Bench, G, Fallon, S, McDade, CE, Chow, JC and Watson, JG (2008).
25 Fossil and contemporary fine particulate carbon fractions at 12 rural and urban sites in the
26 United States. *Journal of Geophysical Research: Atmospheres* 113(D2).
- 27 Schoennagel, T, Balch, JK, Brenkert-Smith, H, Dennison, PE, Harvey, BJ, Krawchuk, MA,
28 Mietkiewicz, N, Morgan, P, Moritz, MA, Rasker, R, Turner, MG and Whitlock, C
29 (2017). Adapt to more wildfire in western North American forests as climate changes.
30 *Proceedings of the National Academy of Sciences* 114(18): 4582.
- 31 Schweizer, D, Cisneros, R, Traina, S, Ghezzehei, TA and Shaw, G (2017). Using National
32 Ambient Air Quality Standards for fine particulate matter to assess regional wildland fire
33 smoke and air quality management. *Journal of Environmental Management* 201: 345-
34 356.
- 35 Sun, K, Tao, L, Miller, DJ, Khan, MA and Zondlo, MA (2014). On-Road Ammonia Emissions
36 Characterized by Mobile, Open-Path Measurements. *Environmental Science &*
37 *Technology* 48(7): 3943-3950.

- 1 TFHTAP (2006). Hemispheric transport of air pollution 2010. Part A: Ozone and particulate
2 matter. Air Pollution Studies No. 17. United Nations Publications. Geneva, Switzerland.
- 3 U.S. EPA (2009). Integrated Science Assessment for Particulate Matter (Final Report). Office of
4 Research and Development, National Center for Environmental Assessment. Research
5 Triangle Park, NC. U.S. EPA. EPA-600/R-08-139F. December 2009. Available at:
6 <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546>.
- 7 U.S. EPA (2012). Near-road NO₂ monitoring technical assistance document. U.S.
8 Environmental Protection Agency, Office of Air Quality Planning and Standards.
9 Research Triangle Park, NC. U.S. EPA. EPA-454/B-12-002. June 2012. Available at:
10 <http://nepis.epa.gov/exe/ZyPURL.cgi?Dockey=P100EPRZ.txt>.
- 11 U.S. EPA (2015). Pilot study on coarse PM monitoring. EPA-454/R-15-001. U.S. EPA Office of
12 Air Quality Planning and Standards. Research Triangle Park, NC.
- 13 U.S. EPA (2017). Bayesian space-time downscaling fusion model (downscaler) -Derived
14 Estimates of Air Quality for 2013. Office of Air Quality Planning and Standards, Office
15 of Air and Radiation. Research Triangle Park, NC. U.S. EPA. EPA-450/R-17-001.
16 Available: <https://www.epa.gov/hesc/rsig-related-downloadable-data-files>.
- 17 U.S. EPA (2018). Integrated Science Assessment (ISA) for Particulate Matter (External Review
18 Draft). U.S. Environmental Protection Agency, Office of Research and Development,
19 National Center for Environmental Assessment. Washington, DC. U.S. EPA.
20 EPA/600/R-18/179. October 2018. Available at:
21 <https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=341593>.
- 22 U.S. EPA (2019a). Review of Sunset Organic and Elemental Carbon (OC and EC)
23 Measurements During EPA's Sunset Carbon Evaluation Project. U.S. Environmental
24 Protection Agency, Office of Air Quality Planning and Standards. Research Triangle
25 Park, NC. U.S. EPA. EPA-454/R-19-005. May 2019. Available at:
26 [https://www.epa.gov/sites/production/files/2019-05/documents/sunset_epa-454r-19-
27 005_1.pdf](https://www.epa.gov/sites/production/files/2019-05/documents/sunset_epa-454r-19-005_1.pdf).
- 28 U.S. EPA (2019b). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
29 U.S. Environmental Protection Agency, Office of Research and Development, National
30 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
31 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-
32 standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 33 U.S. EPA (2020). Addendum SPECIATE Verison 5.1 Database Development Documentation.
34 U.S. Environmental Protection Agency. Washington, DC. U.S. EPA. EPA-600/R-20/189.
35 June 2020. Available at: [https://www.epa.gov/sites/default/files/2020-
36 07/documents/speciate_5.1.pdf](https://www.epa.gov/sites/default/files/2020-07/documents/speciate_5.1.pdf).
- 37 U.S. EPA (2021). 2017 National Emissions Inventory: January 2021 Updated Release, Technical
38 Support Document. U.S. Environmental Protection Agency, Office of Air Quality
39 Planning and Standards. Research Triangle Park, NC. U.S. EPA. February 2021.

- 1 Available at: [https://www.epa.gov/sites/production/files/2021-](https://www.epa.gov/sites/production/files/2021-02/documents/nei2017_tsd_full_jan2021.pdf)
2 [02/documents/nei2017_tsd_full_jan2021.pdf](https://www.epa.gov/sites/production/files/2021-02/documents/nei2017_tsd_full_jan2021.pdf).
- 3 van Donkelaar, A, Martin, RV, Brauer, M and Boys, BL (2015a). Use of satellite observations
4 for long-term exposure assessment of global concentrations of fine particulate matter.
5 *Environmental Health Perspectives* 123(2): 135-143.
- 6 van Donkelaar, A, Martin, RV, Brauer, M, Kahn, R, Levy, R, Verduzco, C and Villeneuve, PJ
7 (2010). Global estimates of ambient fine particulate matter concentrations from satellite-
8 based aerosol optical depth: development and application. *Environmental Health*
9 *Perspectives* 118(6): 847-855.
- 10 van Donkelaar, A, Martin, RV, Li, C and Burnett, RT (2019). Regional estimates of chemical
11 composition of fine particulate matter using a combined geoscience-statistical method
12 with information from satellites, models, and monitors. *Environmental Science &*
13 *Technology* 53(5).
- 14 van Donkelaar, A, Martin, RV, Spurr, RJ and Burnett, RT (2015b). High-resolution satellite-
15 derived PM_{2.5} from optimal estimation and geographically weighted regression over
16 North America. *Environmental Science and Technology* 49(17): 10482-10491.
- 17 van Donkelaar, A, Martin, RV, Spurr, RJD, Drury, E, Remer, LA, Levy, RC and Wang, J (2013).
18 Optimal estimation for global ground-level fine particulate matter concentrations. *Journal*
19 *of Geophysical Research: Atmospheres* 118(11): 5621-5636.
- 20 Vancuren, RA and Cahill, TA (2002). Asian aerosols in North America: Frequency and
21 concentration of fine dust. *Journal of Geophysical Research* 107(D24): 4804.
- 22 Vu, TV, Delgado-Saborit, JM and Harrison, RM (2015). Review: Particle number size
23 distributions from seven major sources and implications for source apportionment
24 studies. *Atmospheric Environment* 122: 114-132.
- 25 Vukovic, A, Vujadinovic, M, Pejanovic, G, Andric, J, Kumjian, MR, Djurdjevic, V, Dacic, M,
26 Prasad, AK, El-Askary, HM, Paris, BC, Petkovic, S, Nickovic, S and Sprigg, WA (2014).
27 Numerical simulation of "an American haboob". *Atmospheric Chemistry and Physics*
28 14(7): 3211-3230.
- 29 Wang, S-C, Wang, Y, Estes, M, Lei, R, Talbot, R, Zhu, L and Hou, P (2018a). Transport of
30 Central American fire emissions to the U.S. Gulf Coast: climatological pathways and
31 impacts on ozone and PM_{2.5}. *Journal of Geophysical Research: Atmospheres* 123(15):
32 8344-8361.
- 33 Wang, Y, Hopke, PK, Chalupa, DC and Utell, MJ (2011). Long-term study of urban ultrafine
34 particles and other pollutants. *Atmospheric Environment* 45(40): 7672-7680.
- 35 Wang, Y, Hu, X, Chang, H, Waller, L, Belle, J and Liu, Y (2018b). A Bayesian downscaler
36 model to estimate daily PM_{2.5} levels in the conterminous U.S. *International Journal of*
37 *Environmental Research and Public Health* 15(9): 1999.

- 1 Whitby, KT, Husar, RB and Liu, BYH (1972). The aerosol size distribution of Los Angeles
2 smog. *Journal of Colloid and Interface Science* 39: 177-204.
- 3 Xu, L, Guo, H, Boyd, CM, Klein, M, Bougiatioti, A, Cerully, KM, Hite, JR, Isaacman-Vanwertz,
4 G, Kreisberg, NM, Knote, C, Olson, K, Koss, A, Goldstein, AH, Hering, SV, de Gouw, J,
5 Baumann, K, Lee, SH, Nenes, A, Weber, RJ and Ng, NL (2015). Effects of
6 anthropogenic emissions on aerosol formation from isoprene and monoterpenes in the
7 southeastern United States. *Proceedings of the National Academy of Sciences* 112(1): 37-
8 42.
- 9 Yu, H, Chin, M, Bian, H, Yuan, T, Prospero, JM, Omar, AH, Remer, LA, Winker, DM, Yang, Y,
10 Zhang, Ya and Zhang, Z (2015). Quantification of trans-Atlantic dust transport from
11 seven-year (2007-2013) record of CALIPSO lidar measurements. *Remote Sensing of*
12 *Environment* 159: 232-249.
- 13 Yu, H, Remer, LA, Chin, M, Bian, H, Kleidman, RG and Diehl, T (2008). A satellite-based
14 assessment of transpacific transport of pollution aerosol. *Journal of Geophysical*
15 *Research* 113(D14): 1-15.
- 16 Yu, H, Russell, A, Mulholland, J, Odman, T, Hu, Y, Chang, HH and Kumar, N (2018). Cross-
17 comparison and evaluation of air pollution field estimation methods. *Atmospheric*
18 *Environment* 179: 49-60.
- 19
20

1	2	PM AIR QUALITY	2-1
2	2.1	Distribution of Particle Size in Ambient Air.....	2-1
3	2.1.1	Sources of PM Emissions	2-3
4	2.2	Ambient PM Monitoring Methods and Networks.....	2-14
5	2.2.1	Total Suspended Particulates (TSP) Sampling	2-15
6	2.2.2	PM ₁₀ Monitoring.....	2-16
7	2.2.3	PM _{2.5} Monitoring	2-17
8	2.2.4	PM _{10-2.5} Monitoring.....	2-22
9	2.2.5	Additional PM Measurements and Metrics	2-23
10	2.3	Ambient Air Concentrations	2-25
11	2.3.1	Trends in Emissions of PM and Precursor Gases	2-25
12	2.3.2	Trends in Monitored Ambient Concentrations	2-26
13	2.3.3	Predicted Ambient PM _{2.5} Based on Hybrid Modeling Approaches.....	2-43
14	2.4	Background PM.....	2-53
15	2.4.1	Natural Sources	2-55
16	2.4.2	International Transport.....	2-57
17	2.4.3	Estimating Background PM with Recent Data	2-59
18	References	2-62	
19			
20			

1 Table 2-1. Percent Changes in PM and PM precursor emissions in the NEI for the time
2 periods 1990-2017 and 2002-2017. 2-26
3 Table 2-2. Nationwide averages of ratios of maximum annual PM_{2.5} design values to average
4 composite monitor PM_{2.5} concentrations across CBSAs. 2-45
5 Table 2-3. Mean 2011 PM_{2.5} concentration by region for predictions in Figure 2-29..... 2-52
6 Table 2-4. Average Annual PM_{2.5} Concentration (µg/m³) by Year..... 2-58
7 Table 2-5. Three-Year Average of the Average Annual PM_{2.5} Concentrations (µg/m³)..... 2-59
8 Table 2-6. Average Annual PM_{2.5} Concentrations and Ratios to Regulatory Design Values.
9 2-60
10
11

1	Figure 2-1. Comparisons of PM _{2.5} and PM ₁₀ diameters to human hair and beach sand.	2-2
2	Figure 2-2. Percent contribution of PM _{2.5} national emissions by source sectors.	2-5
3	Figure 2-3. 2017 NEI PM _{2.5} Emissions Density Map, tons per square mile	2-6
4	Figure 2-4. Percent contribution of PM ₁₀ emissions by national source sectors.	2-7
5	Figure 2-5. 2017 NEI PM ₁₀ Emissions Density Map, tons per square mile	2-7
6	Figure 2-6. Percent contribution to organic carbon (top panel) and elemental carbon (bottom	
7	panel) national emissions by source sectors.	2-9
8	Figure 2-7. 2017 NEI Elemental Carbon Emissions Density Map, tons per square mile. ..	2-10
9	Figure 2-8. Percent contribution to sulfur dioxide (panel A), oxides of nitrogen (panel B),	
10	ammonia (panel C), and volatile organic compounds (panel D) national emissions by source	
11	sectors. (Source: 2017 NEI). All graphics only show anthropogenic contributions.....	2-11
12	Figure 2-9. SO ₂ Emissions Density Map, tons per square mile.....	2-12
13	Figure 2-10. NO _x Emissions Density Map, tons per square mile.	2-12
14	Figure 2-11. NH ₃ Emissions Density Map, tons per square mile.	2-12
15	Figure 2-12. Anthropogenic (including wildfires) VOC Emissions Density Map, tons per	
16	square mile.	2-13
17	Figure 2-13. PM Monitoring stations reporting to EPA’s AQS database by PM size fraction,	
18	1970-2020.	2-15
19	Figure 2-14. National emission trends of PM _{2.5} , PM ₁₀ , and precursor gases from 1990 to	
20	2017.	
21	2-26
22	Figure 2-15. Annual average and 98 th percentile of 24-hour PM _{2.5} concentrations (in µg/m ³)	
23	from 2017-2019 (top) and linear trends and their associated significance (based on p-values)	
24	in PM _{2.5} concentrations from 2000-2019 (bottom).....	2-28
25	Figure 2-16. Seasonally-weighted annual average PM _{2.5} concentrations in the U.S. from 2000	
26	to 2019 (406 sites).....	
27	2-29	
28	Figure 2-17. Pearson’s correlation coefficient between annual average and 98 th percentile of	
29	24-hour PM _{2.5} concentrations from 2000-2019.	2-30
30	Figure 2-18. Scatterplot of CBSA maximum annual versus daily design values (2017-2019)	
31	with the solid black line representing the ratio of daily and annual NAAQS values.	2-31
32	Figure 2-19. Frequency distribution of 2017-2019 2-hour averages for sites meeting both or	
33	violating either PM _{2.5} NAAQS for October to March (blue) and April to September (red). ..	2-
34	33	
35	Figure 2-20. Annual average PM _{2.5} sulfate, nitrate, organic carbon, and elemental carbon	
36	concentrations (in µg/m ³) from 2017-2019.	2-34
37	Figure 2-21. Annual average and 2 nd highest PM ₁₀ concentrations (in µg/m ³) from 2017-2019	
38	(top) and linear trends and their associated significance in PM ₁₀ concentrations from 2000-	

1 2019 (bottom). 2-36

2 2-36

3 Figure 2-22. National trends in Annual 2nd Highest 24-Hour PM₁₀ concentrations from 2000

4 to 2019 (262 sites)..... 2-37

5 2-37

6 Figure 2-23. Annual average PM_{2.5}/PM₁₀ ratio for 2017-2019. 2-38

7 Figure 2-24. PM_{2.5}/PM₁₀ ratio on the date of the second highest PM₁₀ concentrations for

8 2017-2019. 2-38

9 Figure 2-25. Annual average and 98th percentile PM_{10-2.5} concentrations (µg/m³) from 2017-

10 2019 (top) and linear trends and their associated significance in PM_{10-2.5} concentrations from

11 2000-2019 (bottom). 2-40

12 2-40

13 Figure 2-26. Average hourly particle number concentrations from three locations in the State

14 of New York for 2014 to 2015..... 2-41

15 Figure 2-27. Time series of annual average mass and number concentrations (left) and

16 scatterplot of mass vs. number concentration (right) between 2000-2019 in Bondville, IL. ... 2-

17 42

18 Figure 2-28. Comparison of CBSA average annual design values and CBSA maximum

19 annual design values for 2017-2019. 2-44

20 Figure 2-29. Comparison of CBSA average annual design values and CBSA maximum daily

21 design values for 2017-2019..... 2-46

22 Figure 2-30. R² for ten-fold cross-validation of daily PM_{2.5} predictions in 2015 from three

23 methods for individual sites as a function of observed concentration. 2-50

24 Figure 2-31. Comparison of 2011 annual average PM_{2.5} concentrations from four methods. 2-

25 51

26 Figure 2-32. Comparison of 2011 annual average PM_{2.5} concentrations from four methods for

27 regions centered on the (a) California (b) New Jersey, and (c) Arizona 2-53

28 Figure 2-33. (a) Spatial distribution of the CV (i.e., standard deviation divided by mean) in

29 percentage units for the four models in Figure 2-29. (b) Boxplot distributions of CV for grid

30 cells binned by the average PM_{2.5} concentration for the four models. 2-54

31 Figure 2-34. Distance from the center of the 12-km grid cells to the nearest PM_{2.5} monitoring

32 site for PM_{2.5} measurements from the AQS database and IMPROVE network. 2-54

33 Figure 2-35. Location of PM_{2.5} predictions by range in annual average concentration for the

34 four prediction methods at their native resolution. 2-55

35 Figure 2-36. Annual mean PM_{2.5} from the VD2019 method (van Donkelaar et al., 2019) for

36 2001, 2006, 2011, and 2016..... 2-56

37 Figure 2-37. Spatial distribution of the annual average PM_{2.5} concentrations for 2015 using

38 the DI2019 surface nationwide (panel A) and for CBSAs only (panel B). 2-57

1	Figure 2-38. Smoke and fire detections observed by the MODIS instrument onboard the	
2	Aqua satellite on August 4 th , 2017 accessed through NASA Worldview.	2-65
3	Figure 2-39. Fine PM mass time series during 2017 from the North Cascades IMPROVE site	
4	in north central Washington state.	2-65
5	Figure 2-40. Speciated annual average IMPROVE PM _{2.5} in µg/m ³ at select remote monitors	
6	during 2004 and 2016.	2-69
7	Figure 2-41. Site locations for the IMPROVE monitors in Figure 2-40.	2-70
8		

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27

3 RECONSIDERATION OF THE PRIMARY STANDARDS FOR PM_{2.5}

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

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

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

3.1 BACKGROUND ON THE CURRENT STANDARDS

The current primary PM_{2.5} standards were retained in 2020 based on the Administrator’s judgments regarding the available scientific evidence, the available risk information regarding the risk that may be allowed by such standards, and the appropriate degree of public health protection provided by the existing standards (85 FR 82718, December 18, 2020). With the 2020 final decision, the EPA retained the primary 24-hour PM_{2.5}, with its level of 35 µg/m³, and the primary annual PM_{2.5} standard, with its level of 12.0 µg/m³. This decision drew upon the scientific evidence assessed in the 2019 ISA, the evidence and quantitative risk information in the 2020 PA, the advice and recommendations of the CASAC, and public comments on the proposed decision (85 FR 24094, April 30, 2020).

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

Epidemiologic studies reported PM_{2.5} health effect associations with mortality and/or morbidity across multiple U.S. cities and in diverse populations, including in studies examining populations and lifestages that may be at comparatively higher risk of experiencing a PM_{2.5}-related health effect (e.g., older adults, children). The 2019 ISA cited extensive evidence indicating that “both the general population as well as specific populations and lifestages are at

1 risk for PM_{2.5}-related health effects” (U.S. EPA, 2019, p. 12-1). In support of the causal and
2 likely to be causal determinations, the 2019 ISA cites substantial evidence for:

- 3 • PM-related mortality and cardiovascular effects in older adults (U.S. EPA, 2019, sections
4 11.1, 11.2, 6.1, and 6.2);
- 5 • PM-related cardiovascular effects in people with pre-existing cardiovascular disease (U.S.
6 EPA, 2019, section 6.1);
- 7 • PM-related respiratory effects in people with pre-existing respiratory disease, particularly
8 asthma (U.S. EPA, 2019, section 5.1);
- 9 • PM-related impairments in lung function growth and asthma development in children (U.S.
10 EPA, 2019, sections 5.1, 5.2, and 12.5.1.1).

11 The 2019 ISA also noted that stratified analyses (i.e., analyses that allow for comparison of PM-
12 related health effects in subgroups to health effects for full populations) provided strong
13 evidence for racial and ethnic differences in PM_{2.5} exposures and PM_{2.5}-related health risk. Such
14 analyses indicated that certain racial and ethnic groups such as Hispanic and non-Hispanic Black
15 populations have higher PM_{2.5} exposures than non-Hispanic White populations, thus contributing
16 to risk of adverse health effects in non-white populations (U.S. EPA, 2019, section 12.5.4).
17 Stratified analyses focused on other groups also suggested that populations with pre-existing
18 cardiovascular or respiratory disease, populations that are overweight or obese, populations that
19 have particular genetic variants, and populations that are of low socioeconomic status could be at
20 increased risk for PM_{2.5}-related adverse health effects (U.S. EPA, 2019, chapter 12).

21 The risk information available in the 2020 review included risk estimates for air quality
22 conditions just meeting the existing primary PM_{2.5} standards, and also for air quality conditions
23 just meeting potential alternative standards. The general approach to estimating PM_{2.5}-associated
24 health risks combined concentration-response functions from epidemiologic studies with model-
25 based PM_{2.5} air quality surfaces, baseline health incidence data, and population demographics for
26 47 urban areas (U.S. EPA, 2020, section 3.3, Figure 3-10, Appendix C). The risk assessment
27 estimated that the existing primary PM_{2.5} standards could allow a substantial number of PM_{2.5}-
28 associated deaths in the U.S. Uncertainty in risk estimates (e.g., in the size of risk estimates) can
29 result from a number of factors, including assumptions about the shape of the concentration-
30 response relationship with mortality at low ambient PM concentrations, the potential for
31 confounding and/or exposure measurement error, and the methods used to adjust PM_{2.5} air
32 quality. In light of the limitations and uncertainties, these risk estimates were given little weight
33 by the Administrator in his decision on the standards (85 FR 82717, December 18, 2020).

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

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

3 **3.1.1 Considerations Regarding the Adequacy of the Existing Standards in the 2020**
4 **Review**

5 With the 2020 final decision, the EPA retained the primary 24-hour PM_{2.5} standard, with
6 its level of 35 µg/m³, and the primary annual PM_{2.5} standard, with its level of 12.0 µg/m³. The
7 Administrator’s conclusions regarding the adequacy of the primary PM_{2.5} standards at the time of
8 the 2020 review was based on consideration of the evidence, analyses and conclusions contained
9 in the 2019 ISA; the quantitative risk assessment in the 2020 PA; advice from the CASAC; and
10 public comments. Key considerations informing the Administrator’s decisions that the 2012
11 standards should be retained are summarized below.

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

17 With regard to indicator, the Administrator recognized that, consistent with the evidence
18 available in prior reviews, the scientific evidence in the 2020 review continued to provide strong
19 support for health effects following short- and long-term PM_{2.5} exposures. He noted the 2020 PA
20 conclusions that the information continued to support the PM_{2.5} mass-based indicator and
21 remained too limited to support a distinct standard for any specific PM_{2.5} component or group of
22 components, and too limited to support a distinct standard for the ultrafine fraction. Thus, the
23 Administrator concluded that it was appropriate to retain PM_{2.5} as the indicator for the primary
24 standards for fine particulates (85 FR 82715, December 18, 2020).

25 With respect to averaging time and form, the Administrator noted that the scientific
26 evidence continued to provide strong support for health effects associations with both long-term
27 (e.g., annual or multi-year) and short-term (e.g., mostly 24-hour) exposures to PM_{2.5}, consistent
28 with the conclusions in the 2020 PA. In the 2019 ISA, epidemiologic and controlled human
29 exposure studies examined a variety of PM_{2.5} exposure durations. Epidemiologic studies
30 continued to provide strong support for health effects associated with short-term PM_{2.5} exposures
31 based on 24-hour PM_{2.5} averaging periods, and the EPA noted that associations with sub-daily
32 estimates are less consistent and, in some cases, smaller in magnitude (U.S. EPA, 2019, section
33 1.5.2.1; U.S. EPA, 2020, section 3.5.2.2). In addition, controlled human exposure and panel-
34 based studies of sub-daily exposures typically examined subclinical effects, rather than the more
35 serious population-level effects that have been reported to be associated with 24-hour exposures

1 (e.g., mortality, hospitalizations). Taken together, the 2019 ISA concludes that epidemiologic
2 studies did not indicate that sub-daily averaging periods were more closely associated with
3 health effects than the 24-hour average exposure metric (U.S. EPA, 2019, section 1.5.2.1).
4 Additionally, while controlled human exposure studies provided consistent evidence for
5 cardiovascular effects following PM_{2.5} exposures for less than 24 hours (i.e., < 30 minutes to 5
6 hours), exposure concentrations in the studies were well-above the ambient concentrations
7 typically measured in locations meeting the existing standards (U.S. EPA, 2020, section 3.2.3.1).
8 Thus, these studies also did not suggest the need for additional protection against sub-daily PM_{2.5}
9 exposures (U.S. EPA, 2020, section 3.5.2.2). Therefore, the Administrator judged that the 24-
10 hour averaging time remained appropriate (85 FR 82715, December 18, 2020).

11 With regard to the form of the 24-hour standard (98th percentile, averaged over three
12 years), the Administrator noted that epidemiologic studies continued to provide strong support
13 for health effect associations with short-term (e.g., mostly 24-hour) PM_{2.5} exposures (U.S. EPA,
14 2020, section 3.5.2.3) and that controlled human exposure studies provided evidence for health
15 effects following single short-term “peak” PM_{2.5} exposures. Thus, the evidence supported
16 retaining a standard focused on providing supplemental protection against short-term peak
17 exposures and supported a 98th percentile form for a 24-hour standard. The Administrator further
18 noted that this form also provided an appropriate balance between limiting the occurrence of
19 peak 24-hour PM_{2.5} concentrations and identifying a stable target for risk management programs
20 (U.S. EPA, 2020, section 3.5.2.3). As such, the Administrator concluded to retain the form and
21 averaging time of the current 24-hour standard (98th percentile, averaged over three years) and
22 annual standard (annual average, averaged over three years) (85 FR 82715, December 18, 2020).

23 With regard to the level of the standards, in reaching his final decision, the Administrator
24 considered the large body of evidence presented and assessed in the 2019 ISA (U.S. EPA, 2019),
25 the policy-relevant and risk-based conclusions and rationales as presented in the 2020 PA (U.S.
26 EPA, 2020), advice from the CASAC, and public comments. In particular, in considering the
27 2019 ISA and 2020 PA, he considered key epidemiologic studies that evaluated associations
28 between PM_{2.5} air quality distributions and mortality and morbidity, including key accountability
29 studies; the availability of experimental studies to support biological plausibility; controlled
30 human exposure studies examining effects following short-term PM_{2.5} exposures; air quality
31 analyses; and the important uncertainties and limitations associated with the information (85 FR
32 82715, December 18, 2020).

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

1 mean of the distribution), but also provided some control over short-term peak PM_{2.5}
2 concentrations. On the other hand, the 24-hour standard, with its 98th percentile form, was most
3 effective at limiting peak 24-hour PM_{2.5} concentrations, but in doing so also had an effect on
4 annual average PM_{2.5} concentrations. Thus, while either standard could be viewed as providing
5 some measure of protection against both average exposures and peak exposures, the 24-hour and
6 annual standards were not expected to be equally effective at limiting both types of exposures.
7 Thus, consistent with previous reviews, the Administrator’s consideration of the public health
8 protection provided by the existing primary PM_{2.5} standards was based on his consideration of
9 the combination of the annual and 24-hour standards. Specifically, he recognized that the annual
10 standard was more likely to appropriately limit the “typical” daily and annual exposures that are
11 most strongly associated with the health effects observed in epidemiologic studies. The
12 Administrator concluded that an annual standard (as the arithmetic mean, averaged over three
13 years) remained appropriate for targeting protection against the annual and daily PM_{2.5} exposures
14 around the middle portion of the PM_{2.5} air quality distribution. Further, recognizing that the 24-
15 hour standard (with its 98th percentile form) was more directly tied to short-term peak PM_{2.5}
16 concentrations, and more likely to appropriately limit exposures to such concentrations, the
17 Administrator concluded that the current 24-hour standard (with its 98th percentile form,
18 averaged over three years) remained appropriate to provide a balance between limiting the
19 occurrence of peak 24-hour PM_{2.5} concentrations and identifying a stable target for risk
20 management programs. However, the Administrator recognized that changes in PM_{2.5} air quality
21 to meet an annual standard would likely result not only in lower short- and long-term PM_{2.5}
22 concentrations near the middle of the air quality distribution, but also in fewer and lower short-
23 term peak PM_{2.5} concentrations. The Administrator further recognized that changes in air quality
24 to meet a 24-hour standard, with a 98th percentile form, would result not only in fewer and lower
25 peak 24-hour PM_{2.5} concentrations, but also in lower annual average PM_{2.5} concentrations (85
26 FR 82715-82716, December 18, 2020).

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

1 meeting the standards (U.S. EPA, 2020, section 3.2.3.1). In particular, controlled human
2 exposure studies often reported statistically significant effects on one or more indicators of
3 cardiovascular function following 2-hour exposures to PM_{2.5} concentrations at and above 120
4 µg/m³ (at and above 149 µg/m³ for vascular impairment, the effect shown to be most consistent
5 across studies). To provide insight into what these studies may indicate regarding the primary
6 PM_{2.5} standards, the 2020 PA (U.S. EPA, 2020, p. 3-49) noted that 2-hour ambient
7 concentrations of PM_{2.5} at monitoring sites meeting the current standards almost never exceeded
8 32 µg/m³. In fact, even the extreme upper end of the distribution of 2-hour PM_{2.5} concentrations
9 at sites meeting the primary PM_{2.5} standards remained well-below the PM_{2.5} exposure
10 concentrations consistently shown in controlled human exposure studies to elicit effects (i.e.,
11 99.9th percentile of 2-hour concentrations at these sites is 68 µg/m³ during the warm season).
12 Thus, the available experimental evidence did not indicate the need for additional protection
13 against exposures to peak PM_{2.5} concentrations, beyond the protection provided by the
14 combination of the 24-hour and the annual standards (U.S. EPA, 2020, section 3.2.3.1; 85 FR
15 82716, December 18, 2020).

16 With respect to the epidemiologic evidence, the Administrator noted that the studies did
17 not indicate that associations in those studies were strongly influenced by exposures to peak
18 concentrations in the air quality distribution and thus did not indicate the need for additional
19 protection against short-term exposures to peak PM_{2.5} concentrations (U.S. EPA, 2020, section
20 3.5.1). The Administrator noted that this was consistent with CASAC consensus support for
21 retaining the current 24-hour standard. Thus, the Administrator concluded that the 24-hour
22 standard with its level of 35 µg/m³ was adequate to provide supplemental protection (i.e., beyond
23 that provided by the annual standard alone) against short-term exposures to peak PM_{2.5}
24 concentrations (85 FR 82716, December 18, 2020).

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

1 consideration of the uncertainties in the reported associations between daily or annual average
2 PM_{2.5} exposures and mortality or morbidity in the epidemiologic studies. Such an approach is
3 consistent with setting standards that are neither more nor less stringent than necessary,
4 recognizing that a zero-risk standard is not required by the CAA (85 FR 82716, December 18,
5 2020).

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

18 Taking into consideration the views expressed by the CASAC members who supported
19 retaining the annual standard, the Administrator recognized that epidemiologic studies examined
20 associations between distributions of PM_{2.5} air quality and health outcomes, and they did not
21 identify particular PM_{2.5} exposures that cause effects (U.S. EPA, 2020, section 3.1.2). While the
22 Administrator remained concerned about placing too much weight on epidemiologic studies to
23 inform conclusions on the adequacy of the primary standards, he noted the approach to
24 considering such studies in the 2012 review. In the 2012 review, it was noted that the evidence of
25 an association in any epidemiologic study was “strongest at and around the long-term average
26 where the data in the study are most concentrated” (78 FR 3140, January 15, 2013). In
27 considering the characterization of epidemiologic studies, the Administrator viewed that when
28 assessing the mean concentrations of the key short-term and long-term epidemiologic studies in
29 the U.S. that use ground-based monitoring (i.e., those studies where the mean is most directly
30 comparable to the current annual standard), the majority of studies had mean concentrations at or
31 above the level of the existing annual standard, with the mean of the study-reported means or
32 medians equal to 13.5 µg/m³, a concentration level above the existing level of the primary annual
33 standard of 12 µg/m³. The Administrator further noted his caution in directly comparing the
34 reported study mean values to the standard level given that study-reported mean concentrations,
35 by design, are generally lower than the design value of the highest monitor in an area, which
36 determines compliance. In the 2020 PA, analyses of recent air quality in U.S. CBSAs indicated

1 that maximum annual PM_{2.5} design values for a given three-year period were often 10% to 20%
2 higher than average monitored concentrations (i.e., averaged across multiple monitors in the
3 same CBSA) (U.S. EPA, 2020, Appendix B, section B.7). He further noted his concern in
4 placing too much weight on any one epidemiologic study but instead judged that it was more
5 appropriate to focus on the body of studies together and therefore noted the calculation of the
6 mean of study-reported means (or medians). Thus, while the Administrator was cautious in
7 placing too much weight on the epidemiologic evidence alone, he noted that: (1) the reported
8 mean concentration in the majority of the key U.S. epidemiologic studies using ground-based
9 monitoring data were above the level of the existing annual standard; (2) the mean of the
10 reported study means (or medians) (i.e., 13.5 µg/m³) was above the level of the current standard;²
11 (3) air quality analyses showed the study means to be lower than their corresponding design
12 values by 10-20%; and (4) these analyses must be considered in light of uncertainties inherent in
13 the epidemiologic evidence. When taken together, the Administrator judged that, even if it were
14 appropriate to place more weight on the epidemiologic evidence, this information did not call
15 into question the adequacy of the current standards (85 FR 82716-82717, December 18, 2020).

16 In addition to the evidence, the Administrator also considered the potential implications
17 of the risk assessment. He noted that all risk assessments have limitations and that he remained
18 concerned about the uncertainties in the underlying epidemiologic data used in the risk
19 assessment. The Administrator also noted that in previous reviews, these uncertainties and
20 limitations have often resulted in less weight being placed on quantitative estimates of risk than
21 on the underlying scientific evidence itself (e.g., 78 FR 3086, 3098-99, January 15, 2013). These
22 uncertainties and limitations included uncertainty in the shapes of concentration-response
23 functions, particularly at low concentrations; uncertainties in the methods used to adjust air
24 quality; and uncertainty in estimating risks for populations, locations and air quality distributions
25 different from those examined in the underlying epidemiologic study (U.S. EPA, 2020, section
26 3.3.2.4). Additionally, the Administrator noted similar concern expressed by some members of
27 the CASAC who support retaining the existing standards; they highlighted similar uncertainties
28 and limitations in the risk assessment (Cox, 2019). In light of all of this, the Administrator
29 judged it appropriate to place little weight on quantitative estimates of PM_{2.5}-associated mortality
30 risk in reaching conclusions about the level of the primary PM_{2.5} standards (85 FR 82717,
31 December 18, 2020).

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

² The median of the study-reported mean (or median) PM_{2.5} concentrations is 13.3 µg/m³, which was also above the level of the existing standard.

1 those reductions resulted in public health improvements. While the Administrator agreed with
2 public commenters that well-designed and conducted accountability studies can be informative,
3 he viewed that interpreting such studies in the context of the primary PM_{2.5} standards was
4 complicated by the fact that some of the available studies had not evaluated PM_{2.5} specifically
5 (e.g., as opposed to PM₁₀ or total suspended particulates), did not show changes in PM_{2.5} air
6 quality, or had not been able to disentangle health impacts of the interventions from background
7 trends in health (U.S. EPA, 2020, section 3.5.1). He further recognized that the small number of
8 available studies that did report public health improvements following past declines in ambient
9 PM_{2.5} had not examined air quality meeting the existing standards (U.S. EPA, 2020, Table 3-3).
10 This included U.S. studies that reported increased life expectancy, decreased mortality, and
11 decreased respiratory effects following past declines in ambient PM_{2.5} concentrations. Such
12 studies examined “starting” annual average PM_{2.5} concentrations (i.e., prior to the reductions
13 being evaluated) ranging from about 13.2 to > 20 µg/m³ (i.e., U.S. EPA, 2020, Table 3-3). Given
14 the lack of available accountability studies reporting public health improvements attributable to
15 reductions in ambient PM_{2.5} in locations meeting the existing standards, together with his broader
16 concerns regarding the lack of experimental studies examining PM_{2.5} exposures typical of areas
17 meeting the existing standards, the Administrator judged that there was considerable uncertainty
18 in the potential for increased public health protection from further reductions in ambient PM_{2.5}
19 concentrations beyond those achieved under the existing primary PM_{2.5} standards (85 FR 82717,
20 December 18, 2020).

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

35 In reaching this conclusion, the Administrator judged that the existing standards provided
36 an adequate margin of safety. With respect to the annual standard, the level of 12 µg/m³ was

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

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

20 In reaching his final decision, the Administrator concluded that the scientific evidence
21 and technical information continued to support the existing annual and 24-hour $\text{PM}_{2.5}$ standards.
22 This conclusion reflected the Administrator’s view that there were important limitations and
23 uncertainties that remained in the evidence. The Administrator concluded that these limitations
24 contributed to considerable uncertainty regarding the potential public health implications of
25 revising the existing primary $\text{PM}_{2.5}$ standards. Given this uncertainty, and noting the advice from
26 some CASAC members, he concluded that the primary $\text{PM}_{2.5}$ standards, including the indicators
27 ($\text{PM}_{2.5}$), averaging times (annual and 24-hour), forms (arithmetic mean and 98th percentile,
28 averaged over three years) and levels (12.0 $\mu\text{g}/\text{m}^3$, 35 $\mu\text{g}/\text{m}^3$), when taken together, remained
29 requisite to protect the public health. Therefore, in the 2020 review, the Administrator reached
30 the conclusion that the primary 24-hour and annual $\text{PM}_{2.5}$ standards, together, were requisite to
31 protect public health from fine particles with an adequate margin of safety, including the health
32 of at-risk populations, and retained the standards, without revision (85 FR 82718, December 18,
33 2020).

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

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

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

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

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

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

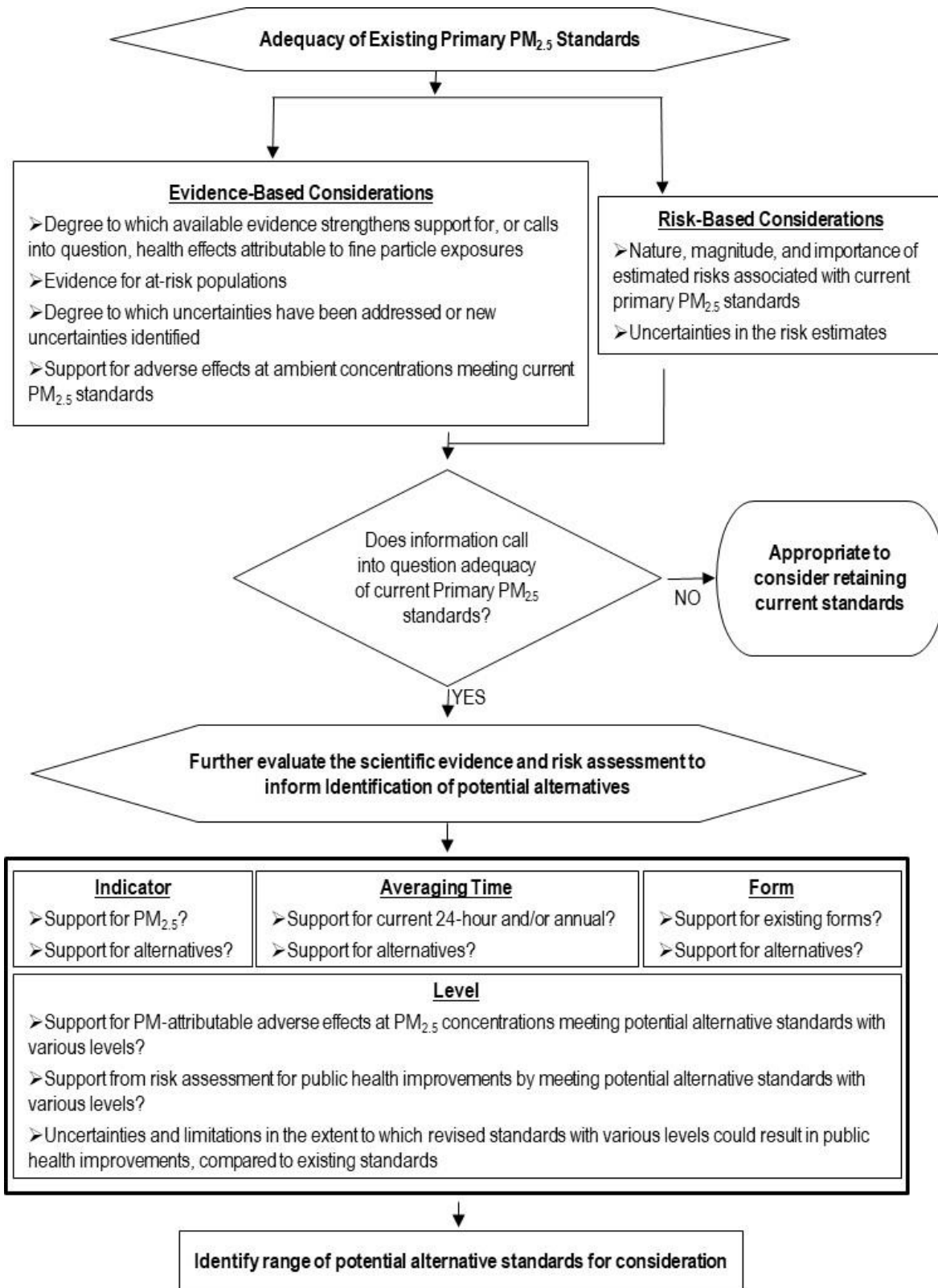
1 the 2020 review regarding health effects related to exposure to PM_{2.5} in ambient air. Information
2 available in this reconsideration that may be informative to public health judgments regarding
3 significance or adversity of key effects will also be considered. Additionally, the available risk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

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

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

25 The approach to reaching preliminary conclusions on the current primary PM_{2.5} standards
26 and, as appropriate, on potential alternative standards is summarized in general terms in Figure
27 3-1.

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



1
2
3

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

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

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

23 **3.3 HEALTH EFFECTS EVIDENCE**

24 In this section, we draw from the EPA’s synthesis and assessment of the scientific
25 evidence presented in the 2019 ISA (U.S. EPA, 2019) and the draft ISA Supplement (U.S. EPA,
26 2021a) to consider the following policy-relevant question:

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

- 1 • **To what extent does the currently available scientific evidence, as assessed in the**
2 **2019 ISA and the draft ISA Supplement, support or call into question the public**
3 **health protection afforded by the current suite of PM_{2.5} standards?**

4 The 2019 ISA uses a weight-of-evidence framework for characterizing the strength of the
5 available scientific evidence for health effects attributable to PM exposures (U.S. EPA, 2015b,
6 Preamble, section 5). This framework provides the basis for robust, consistent, and transparent
7 evaluation of the scientific evidence, including its uncertainties, and for drawing conclusions on
8 PM-related health effects. As in previous reviews, the 2019 ISA adopts a five-level hierarchy to
9 classify the overall weight of evidence into one of the following categories: causal relationship;
10 likely to be a causal relationship; suggestive of, but not sufficient to infer, a causal relationship;
11 inadequate to infer a causal relationship; and not likely to be a causal relationship (U.S. EPA,
12 2015b, Preamble Table II). In using the weight-of-evidence approach to inform judgments about
13 the causal nature of relationships between PM exposure and health effects, evidence is evaluated
14 for major outcome categories or groups of related outcomes (e.g., respiratory effects), integrating
15 evidence from across disciplines, including epidemiologic, controlled human exposure, and
16 animal toxicological studies and evaluating the coherence of evidence across a spectrum of
17 related endpoints (U.S. EPA, 2015b, Preamble, section 5.c.). In this draft PA, we consider the
18 full body of health evidence, including evidence from the 2019 ISA and draft ISA Supplement,
19 placing the greatest emphasis on the health effects for which the evidence has been judged in the
20 2019 ISA to demonstrate a “causal” or a “likely to be causal” relationship with PM exposures.
21 The 2019 ISA defines these causality determinations as follows (U.S. EPA, 2019, p. p-20; U.S.
22 EPA, 2015b):

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

30 While the 2019 ISA provides the broad scientific foundation for this reconsideration, we
31 recognize that additional literature has become available since the literature cutoff date of the
32 2019 ISA that expands the body of evidence that can inform the Administrator’s judgments on
33 the adequacy of the current primary PM_{2.5} standards. As such, the draft ISA Supplement builds
34 on the information in the 2019 ISA with a targeted identification and evaluation of new scientific
35 information (U.S. EPA, 2021a, section 1.2). The draft ISA Supplement focuses on PM_{2.5} health
36 effects evidence where the 2019 ISA concludes a “causal relationship,” because such health
37 effects are given the most weight in an Administrator’s decisions in a NAAQS review. The draft

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

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

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

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

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

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

5 **3.3.1 Nature of Effects**

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

- 9 • **To what extent does the currently available scientific evidence strengthen, or
10 otherwise alter, our preliminary conclusions regarding health effects attributable to
11 long- or short-term fine particle exposures? Have previously identified uncertainties
12 been reduced? What important uncertainties remain and have new uncertainties
13 been identified?**

14 In answering these questions, as noted above, we consider the full body of evidence assessed in
15 the 2019 ISA, along with the targeted evaluation of recent evidence in the draft ISA Supplement.
16 In so doing, we place particular emphasis on health outcomes for which the evidence in the 2019
17 ISA supports either a “causal” or a “likely to be causal” relationship. While the strongest
18 evidence focuses on PM_{2.5}, the 2019 ISA also assesses the evidence for the ultrafine fraction of
19 PM_{2.5} (ultrafine particles or UFP), generally considered as particulates with a diameter less than
20 or equal to 0.1 μm⁷ (typically based on physical size, thermal diffusivity or electrical mobility)
21 (U.S. EPA, 2019, Preface, p. 11). Table 3-1 lists causality determinations for all of the health
22 effect categories and exposure durations for both PM_{2.5} and UFP, which we consider within this
23 chapter (adapted from U.S. EPA, 2019, Table 1-4).
24

⁷ Definitions of UFP vary across the scientific literature and, as discussed in sections 3.3.1.5 and 3.3.1.6, UFP exposures in animal toxicological and controlled human exposure studies typically use a particle concentrator, which can result in exposures to particles > 0.1 μm in diameter in some studies of UFP-related health effects.

1 **Table 3-1. Key causality determinations for PM_{2.5} and UFP exposures.**

Health Outcome	Size Fraction	Exposure Duration	2009 ISA	2019 ISA
Mortality	PM _{2.5}	Long-term	Causal	Causal
		Short-term		
Cardiovascular effects	PM _{2.5}	Long-term	Causal	Causal
		Short-term		
	UFP	Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Respiratory effects	PM _{2.5}	Long-term	Likely to be causal	Likely to be causal
		Short-term		
	UFP	Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Cancer	PM _{2.5}	Long-term	Suggestive of, but not sufficient to infer	Likely to be causal
Nervous System effects	PM _{2.5}	Long-term	---	Likely to be causal
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
	UFP	Long-term	---	Suggestive of, but not sufficient to infer
		Short-term	Inadequate	Suggestive of, but not sufficient to infer
Metabolic effects	PM _{2.5}	Long-term	---	Suggestive of, but not sufficient to infer
		Short-term	---	Suggestive of, but not sufficient to infer
Reproduction and Fertility	PM _{2.5}	Long-, Short-term	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Pregnancy and Birth Outcomes				

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

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

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

2

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

12 **3.3.1.1 Mortality**

13 Long-term PM_{2.5} exposures

14 The 2009 ISA reported that the evidence was “sufficient to conclude that the relationship
15 between long-term PM_{2.5} exposures and mortality is causal” (U.S. EPA, 2009, p. 7-96). The
16 strongest evidence supporting this conclusion was provided by epidemiologic studies,
17 particularly those examining two seminal cohorts, the American Cancer Society (ACS) and the
18 Harvard Six Cities cohorts. Analyses of the Harvard Six Cities cohort included demonstrations
19 that reductions in ambient PM_{2.5} concentrations are associated with reduced mortality risk
20 (Laden et al., 2006) and with increases in life expectancy (Pope et al., 2009). Further support was
21 provided by other cohort studies conducted in North America and Europe that also reported
22 positive associations between long-term PM_{2.5} exposures and risk of mortality (U.S. EPA, 2009).

23 Cohort studies, assessed in the 2019 ISA, continue to provide consistent evidence of
24 positive associations between long-term PM_{2.5} exposures and mortality. These studies add
25 support for associations with total and non-accidental mortality,⁸ as well as with specific causes
26 of death, including cardiovascular disease and respiratory disease (U.S. EPA, 2019, section
27 11.2.2). Many of these studies have extended the follow-up periods originally evaluated in the
28 ACS and Harvard Six Cities cohorts and continue to observe positive associations between long-
29 term PM_{2.5} exposures and mortality (U.S. EPA, 2019, section 11.2.2.1; Figures 11-18 and 11-
30 19). Adding to the evaluations of the ACS and Six Cities cohorts, studies conducted in other
31 cohorts also demonstrate consistent, positive associations between long-term PM_{2.5} exposure and
32 mortality across various demographic groups (e.g., age, sex, occupation), spatial and temporal
33 extents, exposure assessment metrics, and statistical techniques (U.S. EPA, 2019, sections

⁸ The majority of these studies examined non-accidental mortality outcomes, though some Medicare studies lack cause-specific death information and, therefore, examine total mortality.

1 11.2.2.1, 11.2.5; U.S. EPA, 2021a, Table 11-8). This includes some of the largest cohort studies
2 conducted to date, with analyses of the U.S. Medicare cohort that include nearly 61 million
3 enrollees (Di et al., 2017b) and studies that control for a range of individual and ecological
4 covariates, such as race, age, socioeconomic status, smoking status, body mass index, and annual
5 weather variables (e.g., temperature, humidity).

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

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

30 A series of epidemiologic studies evaluated in the 2019 ISA tested the hypothesis that
31 past reductions in ambient PM_{2.5} concentrations have been associated with increased life
32 expectancy or a decreased mortality rate (U.S. EPA, 2019, section 11.2.2.5). In their original
33 study, Pope et al. (2009) used air quality data in a cross-sectional analysis from 51 metropolitan
34 areas across the U.S., beginning in the 1970s through the early 2000s, to demonstrate that a
35 10 µg/m³ decrease in long-term PM_{2.5} concentration was associated with a 0.61-year increase in
36 life expectancy. In a subsequent analysis, these authors extended the period of analysis to include

1 2000 to 2007 (Correia et al., 2013), a time period with lower ambient PM_{2.5} concentrations. In
2 this follow-up study, a decrease in long-term PM_{2.5} concentration continued to be associated with
3 an increase in life expectancy, though the magnitude of the increase was smaller than during the
4 earlier time period (i.e., a 10 µg/m³ decrease in long-term PM_{2.5} concentration was associated
5 with a 0.35-year increase in life expectancy). Additional studies conducted in the U.S. or Europe
6 similarly report that reductions in ambient PM_{2.5} are associated with improvements in longevity
7 (U.S. EPA, 2019, section 11.2.2.5). Multiple epidemiologic studies that conducted accountability
8 analyses and were published after the literature cutoff date for the 2019 ISA were evaluated in
9 the draft ISA Supplement (U.S. EPA, 2021a, section 3.2.1.3). These studies are consistent with
10 and expand upon the body of evidence from the 2019 ISA. For example, Bennett et al. (2019)
11 reported that PM_{2.5} concentrations above the lowest observed concentration (2.8 µg/m³) were
12 associated with a 0.15 year decrease in national life expectancy for women and 0.13 year
13 decrease in national life expectancy for men (U.S. EPA, 2021a, section 3.2.2.2.4, Figure 3-25).
14 Another study compared participants living in areas with PM_{2.5} concentrations >12 µg/m³ to
15 participants living in areas with PM_{2.5} concentrations < 12 µg/m³ and reported that the number of
16 years of life lost due to living in areas with higher PM_{2.5} concentrations was 0.84 years over a 5-
17 year period (Ward-Caviness et al., 2020; U.S. EPA, 2021a, section 3.2.2.2.4).

18 Since the 2009 ISA there is an emerging group of studies that used causal modeling
19 statistical methods to further assess relationship between long-term PM_{2.5} exposure and mortality
20 (U.S. EPA, 2019, section 11.2.2.4). The goal of causal modeling methods is to “estimate the
21 difference (or ratio) in the expected value of [an] outcome in the population under the exposure
22 they received versus what it would have been had they received an alternative exposure”
23 (Schwartz et al., 2015). Multiple epidemiologic studies that implemented causal modeling
24 methods and were published since the literature cutoff date of the 2019 ISA were evaluated in
25 the draft ISA Supplement (U.S. EPA, 2021a, section 3.2.2.3). These studies use a variety of
26 statistical methods including generalized propensity score (GPS), inverse probability weighting
27 (IPW), and difference-in-difference (DID) to reduce uncertainties related to confounding bias in
28 the association between long-term PM_{2.5} exposure and mortality. Studies that employed these
29 causal modeling methods reported consistent positive associations that further inform the
30 relationship between long-term PM_{2.5} exposure and total mortality (U.S. EPA, 2021a, section
31 3.2.2.3). These studies provide further support of associations seen in cohort studies and
32 referenced just above.

33 The 2019 ISA and draft ISA Supplement also evaluate the degree to which recent studies
34 that examine the relationship between long-term PM_{2.5} exposure and mortality have addressed
35 key policy-relevant issues and/or previously identified data gaps in the scientific evidence,
36 including methods to estimate exposure, methods to control for confounding, like copollutant

1 confounding, and the shape of the concentration-response curve. For example, based on its
2 assessment of the evidence, the 2019 ISA concludes that positive associations between long-term
3 PM_{2.5} exposures and mortality are robust across recent analyses using various approaches to
4 estimate PM_{2.5} exposures (e.g., based on monitors, modeling, satellites, or hybrid methods that
5 combine information from multiple sources) (U.S. EPA, 2019, section 11.2.5.1). This includes a
6 study Hart et al. (2015) reporting that correction for bias due to exposure measurement error
7 increases the magnitude of the hazard ratios (confidence intervals widen but the association
8 remains statistically significant), suggesting that failure to correct for exposure measurement
9 error could result in attenuation or underestimation of risk estimates.

10 The 2019 ISA additionally concludes that positive associations between long-term PM_{2.5}
11 exposures and mortality are robust across statistical models that use different approaches to
12 control for confounders or different sets of confounders (U.S. EPA, 2019, sections 11.2.3 and
13 11.2.5), across diverse geographic regions and populations, and across a range of temporal
14 periods including the periods of declining PM concentrations (U.S. EPA, 2019, sections 11.2.2.5
15 and 11.2.5.3). Additional evidence further demonstrates that associations with mortality remain
16 robust in copollutants analyses (U.S. EPA, 2019, section 11.2.3), and that associations persist in
17 analyses restricted to long-term exposures below 12 µg/m³ (Di et al., 2017b) or 10 µg/m³ (Shi et
18 al., 2016) (i.e., indicating that risks are not disproportionately driven by the upper portions of the
19 air quality distribution). Recent studies further assess potential copollutant confounding as
20 reflected in the studies evaluated in the draft ISA Supplement that indicate while there is some
21 evidence of potential confounding of the PM_{2.5}-mortality association by copollutants in the some
22 of the studies (i.e., those studies of the MAPLE cohort), this result is inconsistent with other
23 recent studies evaluated in the 2019 ISA that were conducted in the U.S. and Canada that found
24 associations in both single and copollutant models (U.S. EPA, 2019; U.S. EPA, 2021a, section
25 3.2.2.4 and 3.1.2.2.8). Additionally, a few studies use statistical techniques to reduce
26 uncertainties related to potential confounding in order to further inform conclusions on causality
27 for long-term PM_{2.5} exposure and mortality. For example, studies by Greven et al. (2011), Pun et
28 al. (2017), and Eum et al. (2018) decompose ambient PM_{2.5} into “spatial” and “spatiotemporal”
29 components in order to evaluate the potential for bias due to unmeasured spatial confounding.
30 Eum et al. (2018) and Wu et al. (2020a) also attempted to address long-term trends and
31 meteorological variables as potential confounders and found that not adjusting for temporal
32 trends could overestimate the association, while effect estimates in analyses that excluded
33 meteorological variables remained unchanged compared to main analyses. The results of these

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

3 An additional important consideration in characterizing the public health impacts
4 associated with PM_{2.5} exposure is whether concentration-response relationships are linear across
5 the range of concentrations or if nonlinear relationships exist along any part of this range. Studies
6 evaluated in the 2019 ISA and draft ISA Supplement examine this issue, and continue to provide
7 evidence of linear, no-threshold relationships between long-term PM_{2.5} exposures and all-cause
8 and cause-specific mortality (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section
9 3.2.2.2.7, Table 3-6). Across the studies evaluated in the 2019 ISA and draft ISA Supplement, a
10 variety of statistical methods have been used to assess whether there is evidence of deviations in
11 linearity (U.S. EPA, 2019, Table 11-7; U.S. EPA, 2021a, section 2.2.3.2). Studies have also
12 conducted cut-point analyses that focus on examining risk at specific ambient PM_{2.5}
13 concentrations. These studies reported results that generally support a linear, no-threshold
14 relationships between long-term PM_{2.5} exposures and total (nonaccidental) mortality, especially
15 at lower ambient PM_{2.5} concentration, with confidence in the linear relationship as low as 5 to 8
16 µg/m³ in some studies (U.S. EPA, 2019, section 11.2.4; U.S. EPA, 2021a, section 2.2.3.2). There
17 was also some limited evidence indicating that the slope of the concentration-response (C-R)
18 function may be steeper (supralinear) at lower concentrations for cardiovascular mortality (U.S.
19 EPA, 2021a, section 2.2.3.2).

20 The biological plausibility of PM_{2.5}-attributable mortality is supported by the coherence
21 of effects across scientific disciplines (i.e., animal toxicological, controlled human exposure
22 studies, and epidemiologic) when evaluating respiratory and cardiovascular morbidity effects,
23 which are some of the largest contributors to total (nonaccidental) mortality. The 2019 ISA
24 outlines the available evidence for biologically plausible pathways by which inhalation exposure
25 to PM_{2.5} could progress from initial events (e.g., pulmonary inflammation, autonomic nervous
26 system activation) to endpoints relevant to population outcomes, particularly those related to
27 cardiovascular diseases such as coronary heart disease (CHD), stroke and atherosclerosis (U.S.
28 EPA, 2019, section 6.2.1, Table 11-8), and metabolic effects, including diabetes (U.S. EPA,
29 2019, section 7.3.1). The 2019 ISA notes “more limited evidence from respiratory morbidity”
30 (U.S. EPA, 2019, p. 11-101) such as development of chronic obstructive pulmonary disease

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

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

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

10 As such, these studies reduce key uncertainties identified in the previous review,
11 including those related to potential copollutant confounding, and provide additional information
12 on the shape of the concentration-response curve. As assessed in the 2019 ISA, experimental and
13 epidemiologic evidence for cardiovascular effects, and respiratory effects to a more limited
14 degree, supports the plausibility of mortality due to long-term PM_{2.5} exposures. The 2019 ISA
15 concludes that, “collectively, this body of evidence is sufficient to conclude that a causal
16 relationship exists between long-term PM_{2.5} exposure and total mortality” (U.S. EPA, 2019,
17 section 11.2.7; p. 11-102) which is supported and extended by recent evidence evaluated in the
18 draft Supplement (U.S. EPA, 2021a, section 3.2.2.4).

19 Short-term PM_{2.5} exposures

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

35 Multicity studies evaluated in the 2019 ISA and draft ISA Supplement provide evidence
36 of primarily positive associations between daily PM_{2.5} exposures and mortality, with percent

1 increases in total mortality ranging from 0.19% (Lippmann et al., 2013) to 2.80% (Kloog et al.,
2 2013)¹⁰ at lags of 0 to 1 days in single pollutant-models. Whereas most studies rely on assigning
3 exposures using data from ambient monitors, associations are also reported in studies that
4 employ hybrid modeling approaches using additional PM_{2.5} data (i.e., from satellites, land use
5 information, and air quality modeling, in addition to monitors), allowing for the inclusion of
6 more rural locations in analyses (Kloog et al., 2013, Shi et al., 2016). Consistent with the
7 evidence assessed in previous ISAs, recent studies report more variable results with wider
8 confidence intervals for respiratory mortality (Lavigne et al., 2018; Shin et al., 2021).

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

28 With regard to copollutants, studies evaluated in the 2019 ISA provide additional
29 evidence that associations between short-term PM_{2.5} exposures and mortality remain positive and
30 relatively unchanged in copollutant models with both gaseous pollutants and PM_{10-2.5} (U.S. EPA,
31 2019, Section 11.1.4). Additionally, the low ($r < 0.4$) to moderate correlations ($r = 0.4-0.7$)
32 between PM_{2.5} and gaseous pollutants and PM_{10-2.5} increase the confidence in PM_{2.5} having an
33 independent effect on mortality (U.S. EPA, 2019, section 11.1.4). Consistent with the studies

¹⁰ As detailed in the Preface to the ISA, risk estimates are for a 10 µg/m³ increase in 24-hour avg PM_{2.5} concentrations, unless otherwise noted (U.S. EPA, 2019).

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

6 The generally positive associations reported with mortality are supported by a small group
7 of studies employing causal modeling methods or quasi-experimental statistical approaches (U.S.
8 EPA, 2019, section 11.1.2.1). For example, two studies by Schwartz et al. (Schwartz et al., 2015;
9 Schwartz et al., 2017) report associations between PM_{2.5} instrumental variables and mortality
10 (U.S. EPA, 2019, Table 11-2), including in an analysis limited to days with 24-hour average
11 PM_{2.5} concentrations <30 µg/m³ (Schwartz et al., 2017). In addition to the main analyses, these
12 studies conducted Granger-like causality tests as sensitivity analyses to examine whether there
13 was evidence of an association between mortality and PM_{2.5} after the day of death, which would
14 support the possibility that unmeasured confounders were not accounted for in the statistical
15 model. Neither study reports evidence of an association with PM_{2.5} after death (i.e., they do not
16 indicate unmeasured confounding). A quasi-experimental study examines whether a specific
17 regulatory action in Tokyo, Japan (i.e., a diesel emission control ordinance) resulted in a
18 subsequent reduction in daily mortality (Yorifuji et al., 2016). The authors report a reduction in
19 mortality in Tokyo due to the ordinance, compared to Osaka, which did not have a similar diesel
20 emission control ordinance in place. In another study, Schwartz et al. (2018b) utilized three
21 causal methods including instrumental variable analysis, a negative exposure control, and
22 marginal structural models to estimate the association between PM_{2.5} and daily mortality
23 (Schwartz et al., 2018b). Results from this study continue to support a relationship between
24 short-term PM_{2.5} exposure and mortality. Additional epidemiologic studies evaluated in the draft
25 ISA Supplement that employed causal modeling methods to examine the association between
26 short-term PM_{2.5} exposure and mortality also report consistent positive associations in studies
27 that examine effects across multiple cities in the U.S. (U.S. EPA, 2021a).

28 The positive associations for total mortality reported across the majority of studies
29 evaluated are further supported by analyses reporting generally consistent, positive associations
30 with both cardiovascular and respiratory mortality (U.S. EPA, 2019, section 11.1.3). Recent
31 multicity studies evaluated in the draft ISA Supplement add to the body of evidence indicating a
32 relationship between short-term PM_{2.5} exposure and cause-specific mortality, with more
33 variability in the magnitude and precision of associations for respiratory mortality (U.S. EPA,
34 2021a; Figure 3-14). For both cardiovascular and respiratory mortality, there has been a limited
35 assessment of potential copollutant confounding, though initial evidence indicates that
36 associations remain positive and relatively unchanged in models with gaseous pollutants and

1 PM_{10-2.5}. This evidence further supports the copollutant analyses conducted for total mortality.
2 The strong evidence for ischemic events and heart failure, as detailed in the assessment of
3 cardiovascular morbidity (U.S. EPA, 2019, Chapter 6), provides biological plausibility for
4 PM_{2.5}-related cardiovascular mortality, which comprises the largest percentage of total mortality
5 (i.e., ~33%) (NHLBI, 2017). Although there is evidence for exacerbations of COPD and asthma,
6 the collective body of respiratory morbidity evidence provides limited biological plausibility for
7 PM_{2.5}-related respiratory mortality (U.S. EPA, 2019, Chapter 5).

8 In the 2009 ISA, one of the main uncertainties identified was the regional and city-to-city
9 heterogeneity in PM_{2.5}-mortality associations. Recent studies examine both city-specific as well
10 as regional characteristics to identify the underlying contextual factors that could contribute to
11 this heterogeneity (U.S. EPA, 2019, section 11.1.6.3). Analyses focusing on effect modification
12 of the PM_{2.5}-mortality relationship by PM_{2.5} components, regional patterns in PM_{2.5} components
13 and city-specific differences in composition and sources indicate some differences in the PM_{2.5}
14 composition and sources across cities and regions, but these differences do not fully explain the
15 observed heterogeneity. Additional studies find that factors related to potential exposure
16 differences, such housing stock and commuting, as well as city-specific factors (e.g., land-use,
17 port volume, and traffic information), may explain some of the observed heterogeneity (U.S.
18 EPA, 2019, section 11.1.6.3). Collectively, studies evaluated in the 2019 ISA and the draft ISA
19 Supplement indicate that the heterogeneity in PM_{2.5}-mortality risk estimates cannot be attributed
20 to one factor, but instead a combination of factors including, but not limited to, PM composition
21 and sources as well as community characteristics that could influence exposures (U.S. EPA,
22 2019, section 11.1.12; U.S. EPA, 2021a, section 3.2.1.2.1)).

23 A number of studies conducted systematic evaluations of the lag structure of associations
24 for the PM_{2.5}-mortality relationship by examining either a series of single-day or multiday lags
25 and these studies continue to support an immediate effect (i.e., lag 0 to 1 days) of short-term
26 PM_{2.5} exposures on mortality (U.S. EPA, 2019, section 11.1.8.1; U.S. EPA, 2021a, section
27 3.2.1.1). Recent studies also conducted analyses comparing the traditional 24-hour average
28 exposure metric with a sub-daily metric (i.e., 1-hour max). These initial studies provide evidence
29 of a similar pattern of associations for both the 24-hour average and 1-hour max metric, with the
30 association larger in magnitude for the 24-hour average metric.

31 Multicity studies indicate that positive and statistically significant associations with
32 mortality persist in analyses restricted to short-term PM_{2.5} exposures below 35 µg/m³ (Lee et al.,
33 2015),¹¹ below 30 µg/m³ (Shi et al., 2016), and below 25 µg/m³ (Di et al., 2017a), indicating that

¹¹ Lee et al. (2015) also report that positive and statistically significant associations between short-term PM_{2.5} exposures and mortality persist in analyses restricted to areas with long-term concentrations below 12 µg/m³.

1 risks associated with short-term PM_{2.5} exposures are not disproportionately driven by the peaks
2 of the air quality distribution. Additional studies examine the shape of the C-R relationship and
3 whether a threshold exists specifically for PM_{2.5} (U.S. EPA, 2019, section 11.1.10). These
4 studies have used various statistical approaches and consistently demonstrate a linear
5 relationship with no evidence of a threshold. Moreover, recent studies evaluated in the draft ISA
6 Supplement provide additional support for a linear, no-threshold C-R relationship between short-
7 term PM_{2.5} exposure and mortality, with confidence in the shape decreasing at concentrations
8 below 5 µg/m³ (Liu et al., 2019; Lavigne et al., 2018). Recent analyses provide initial evidence
9 indicating that PM_{2.5}-mortality associations persist and may be stronger (i.e., a steeper slope) at
10 lower concentrations (e.g., Di et al., 2017a; Figure 11-12 in U.S. EPA, 2019). However, given
11 the limited data available at the lower end of the distribution of ambient PM_{2.5} concentrations,
12 the shape of the C-R curve remains uncertain at these low concentrations. Although difficulties
13 remain in assessing the shape of the PM_{2.5}-mortality C-R relationship, to date, studies have not
14 conducted systematic evaluations of alternatives to linearity, and recent studies continue to
15 provide evidence of a no-threshold linear relationship, with less confidence at concentrations
16 lower than 5 µg/m³.

17 Overall, recent epidemiologic studies build upon and extend the conclusions of the 2009
18 ISA for the relationship between short-term PM_{2.5} exposures and total mortality. Supporting
19 evidence for PM_{2.5}-related cardiovascular morbidity, and more limited evidence from respiratory
20 morbidity, provides biological plausibility for mortality due to short-term PM_{2.5} exposures. The
21 primarily positive associations observed across studies conducted in diverse geographic locations
22 is further supported by the results from co-pollutant analyses indicating robust associations,
23 along with evidence from analyses of the concentration-response relationship. The 2019 ISA
24 states that, collectively, “this body of evidence is sufficient to conclude that a causal relationship
25 exists between short-term PM_{2.5} exposure and total mortality” (U.S. EPA, 2019, pp. 11-58).
26 Recent evidence evaluated in the draft ISA Supplement provides “additional support to the
27 evidence base that contributed to the conclusion of a causal relationship between short-term
28 PM_{2.5} exposure and mortality” (U.S. EPA, 2021a, section 3.2.1.4, pp 3-69).

29 **3.3.1.2 Cardiovascular Effects**

30 Long-term PM_{2.5} exposures

31 The scientific evidence reviewed in the 2009 ISA was “sufficient to infer a causal
32 relationship between long-term PM_{2.5} exposure and cardiovascular effects” (U.S. EPA, 2009).
33 The strongest line of evidence comprised findings from several large epidemiologic studies of
34 U.S. and Canadian cohorts that consistently showed positive associations between long-term
35 PM_{2.5} exposure and cardiovascular mortality (Krewski et al., 2009, Miller et al., 2007, et al.,).

1 Studies of long-term PM_{2.5} exposure and cardiovascular morbidity were limited in number.
2 Biological plausibility and coherence with the epidemiologic findings were provided by studies
3 using genetic mouse models of atherosclerosis demonstrating enhanced atherosclerotic plaque
4 development and inflammation, as well as changes in measures of impaired heart function,
5 following 4- to 6-month exposures to PM_{2.5} concentrated ambient particles (CAPs), and by a
6 limited number of studies reporting CAPs-induced effects on coagulation factors, vascular
7 reactivity, and worsening of experimentally induced hypertension in mice (U.S. EPA, 2009).

8 Consistent with the evidence assessed in the 2009 ISA, the 2019 ISA concludes that
9 recent studies, together with the evidence available in previous reviews, support a causal
10 relationship between long-term exposure to PM_{2.5} and cardiovascular effects. Additionally,
11 recent epidemiologic studies published since the completion of the 2019 ISA and evaluated in
12 the draft ISA Supplement expands the body of evidence and further supports such a conclusion
13 (U.S. EPA, 2021a). As discussed above (section 3.3.1.1), results from U.S. and Canadian cohort
14 studies evaluated in the 2019 ISA consistently report positive associations between long-term
15 PM_{2.5} exposure and cardiovascular mortality (U.S. EPA, 2019, Figure 6-19) in evaluations
16 conducted at varying spatial scales and employing a variety of exposure assessment and
17 statistical methods (U.S. EPA, 2019, section 6.2.10). Positive associations between long-term
18 PM_{2.5} exposures and cardiovascular mortality are generally robust in copollutant models adjusted
19 for ozone, NO₂, PM_{10-2.5}, or SO₂. In addition, most of the results from analyses examining the
20 shape of the concentration-response relationship for cardiovascular mortality support a linear
21 relationship with long-term PM_{2.5} exposures and do not identify a threshold below which effects
22 do not occur (U.S. EPA, 2019, section 6.2.16; Table 6-52).

23 The body of literature examining the relationship between long-term PM_{2.5} exposure and
24 cardiovascular morbidity has greatly expanded since the 2009 ISA, with positive associations
25 reported in several cohorts (U.S. EPA, 2019, section 6.2). Though results for cardiovascular
26 morbidity are less consistent than those for cardiovascular mortality (U.S. EPA, 2019, section
27 6.2), studies in the 2019 ISA and draft ISA Supplement provide some evidence for associations
28 between long-term PM_{2.5} exposures and the progression of cardiovascular disease. Positive
29 associations with cardiovascular morbidity (e.g., coronary heart disease, stroke, arrhythmias,
30 myocardial infarction (MI), and atherosclerosis progression) are observed in several
31 epidemiologic studies (U.S. EPA, 2019, sections 6.2.2. to 6.2.9; U.S. EPA, 2021a, section
32 3.1.1.4). Associations in such studies are supported by toxicological evidence for increased
33 plaque progression in mice following long-term exposure to PM_{2.5} collected from multiple
34 locations across the U.S. (U.S. EPA, 2019, section 6.2.4.2). A small number of epidemiologic
35 studies also report positive associations between long-term PM_{2.5} exposure and heart failure,
36 changes in blood pressure, and hypertension (U.S. EPA, 2019, sections 6.2.5 and 6.2.7).

1 Associations with heart failure are supported by animal toxicological studies demonstrating
2 decreased cardiac contractility and function, and increased coronary artery wall thickness
3 following long-term PM_{2.5} exposure (U.S. EPA, 2019, section 6.2.5.2). Similarly, a limited
4 number of animal toxicological studies demonstrating a relationship between long-term exposure
5 to PM_{2.5} and consistent increases in blood pressure in rats and mice are coherent with
6 epidemiologic studies reporting positive associations between long-term exposure to PM_{2.5} and
7 hypertension. Moreover, a number of studies assessed in the draft ISA Supplement focusing on
8 morbidity outcomes, including those that focused on incidence of MI, atrial fibrillation (AF),
9 stroke, and congestive heart failure (CHF), expand the evidence pertaining to the shape of the C-
10 R relationship between long-term PM_{2.5} exposure and cardiovascular effects. Additionally,
11 studies evaluated in the draft ISA Supplement report positive associations among those with pre-
12 existing conditions, among patients followed after a cardiac event procedure, and among those
13 with a first hospital admission for heart attacks among older adults enrolled in Medicare (U.S.
14 EPA, 2021a, sections 3.1.1 and 3.1.2). A number of these studies use statistical techniques that
15 allow for departures from linearity (U.S. EPA, 2021a, Table 3-3, and generally support the
16 evidence characterized in the 2019 ISA showing linear, no-threshold C-R relationship for most
17 CVD outcomes. However, there is some evidence for a sublinear or supralinear C-R relationship
18 for some outcomes (U.S. EPA, 2021a, section 3.1.2.2.9).¹² Moreover, several recent
19 epidemiologic studies evaluated in the draft ISA Supplement reported that the association
20 between long-term PM_{2.5} exposure with stroke persisted after adjustment for NO₂ but was
21 attenuated in the model with O₃ and oxidant gases represented by the redox weighted average of
22 NO₂ and O₃ (U.S. EPA, 2021a, section 3.1.2.2.8).

23 Longitudinal epidemiologic analyses also report positive associations with markers of
24 systemic inflammation (U.S. EPA, 2019, section 6.2.11), coagulation (U.S. EPA, 2019, section
25 6.2.12), and endothelial dysfunction (U.S. EPA, 2019, section 6.2.13). These results are coherent
26 with animal toxicological studies generally reporting increased markers of systemic
27 inflammation, oxidative stress, and endothelial dysfunction (U.S. EPA, 2019, section 6.2.12.2
28 and 6.2.14).

29 The 2019 ISA concludes that there is consistent evidence from multiple epidemiologic
30 studies illustrating that long-term exposure to PM_{2.5} is associated with mortality from
31 cardiovascular causes. Epidemiologic studies in the draft ISA Supplement support and extend the
32 findings characterized in the 2019 ISA, providing additional evidence of positive associations
33 between long-term PM_{2.5} exposure and cardiovascular morbidity (U.S. EPA, 2021a section

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

1 3.1.1.4). Associations with CHD, stroke and atherosclerosis progression were observed in several
2 additional epidemiologic studies, providing coherence with the mortality findings.

3 Results from copollutant models generally support the independence of the PM_{2.5}
4 associations (U.S. EPA, 2019, Table 3-2; U.S. EPA, 2021a). Additional evidence of the
5 independent effect of PM_{2.5} on the cardiovascular system is provided by experimental studies in
6 animals, which demonstrate biologically plausible pathways by which long-term inhalation
7 exposure to PM_{2.5} could potentially result in outcomes such as CHD, stroke, CHF and
8 cardiovascular mortality. The combination of epidemiologic and experimental evidence results in
9 the 2019 ISA conclusion that “a causal relationship exists between long-term exposure to PM_{2.5}
10 and cardiovascular effects” (U.S. EPA, 2019, section 6.2.18). Studies evaluated in the draft ISA
11 Supplement support and extend the evidence that contributed to the conclusion of a causal
12 relationship between long-term PM_{2.5} exposure and cardiovascular effects (U.S. EPA, 2021a,
13 section 3.1.2.4).

14 Short-term PM_{2.5} exposures

15 The 2009 ISA concluded that “a causal relationship exists between short-term exposure
16 to PM_{2.5} and cardiovascular effects” (U.S. EPA, 2009). The strongest evidence in the 2009 ISA
17 was from epidemiologic studies of emergency department (ED) visits and hospital admissions
18 for IHD and HF, with supporting evidence from epidemiologic studies of cardiovascular
19 mortality (U.S. EPA, 2009). Animal toxicological studies provided coherence and biological
20 plausibility for the positive associations reported with myocardial ischemia ED visit and hospital
21 admissions. These included studies reporting reduced myocardial blood flow during ischemia
22 and studies indicating altered vascular reactivity. In addition, effects of PM_{2.5} exposure on a
23 potential indicator of ischemia (i.e., ST segment depression on an electrocardiogram) were
24 reported in both animal toxicological and epidemiologic panel studies.¹³ Key uncertainties from
25 the 2009 ISA resulted from inconsistent results across disciplines with respect to the relationship
26 between short-term exposure to PM_{2.5} and changes in blood pressure, blood coagulation markers,
27 and markers of systemic inflammation. In addition, while the 2009 ISA identified a growing
28 body of evidence from controlled human exposure and animal toxicological studies, uncertainties
29 remained with respect to biological plausibility.

30 Recent evidence assessed in the 2019 ISA and the draft ISA Supplement supports and
31 extends the evidence from the 2009 ISA indicating that there is a causal relationship between
32 short-term PM_{2.5} exposure and cardiovascular effects. This includes generally positive
33 associations observed in multicity epidemiologic studies of emergency department visits and

¹³ Some animal studies included in the 2009 ISA examined exposures to mixtures, such as motor vehicle exhaust or woodsmoke. In these studies, it was unclear if the resulting cardiovascular effects could be attributed specifically to the particulate components of the mixture.

1 hospital admissions for IHD, heart failure (HF), and combined cardiovascular-related endpoints.
2 In particular, nationwide studies of older adults (65 years and older) using Medicare records
3 report positive associations between PM_{2.5} exposures and hospital admissions for HF (U.S. EPA,
4 2019, section 6.1.3.1). Moreover, recent multicity studies, published after the literature cutoff
5 date of the 2019 ISA, are coherent with studies evaluated in the 2019 ISA that report positive
6 association between short-term PM_{2.5} exposure and ED visits and hospital admission for IHD,
7 heart attacks, and HF (U.S. EPA, 2021a, section 3.1). Epidemiologic studies conducted in single
8 cities contribute some support, though associations reported in single-city studies are less
9 consistently positive than in multicity studies, and include a number of studies reporting null
10 associations (U.S. EPA, 2019, sections 6.1.2 and 6.1.3). When considered as a whole; however,
11 the recent body of IHD and HF epidemiologic evidence supports the evidence from previous
12 ISAs reporting mainly positive associations between short-term PM_{2.5} concentrations and
13 emergency department visits and hospital admissions.

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

20 A number of controlled human exposure, animal toxicological, and epidemiologic panel
21 studies provide evidence that PM_{2.5} exposure could plausibly result in IHD or HF through
22 pathways that include endothelial dysfunction, arterial thrombosis, and arrhythmia (U.S. EPA,
23 2019, section 6.1.1). The most consistent evidence from recent controlled human exposure
24 studies is for endothelial dysfunction, as measured by changes in brachial artery diameter or flow
25 mediated dilation. All but one of the available controlled human exposure studies examining the
26 potential for endothelial dysfunction report an effect of PM_{2.5} exposure on measures of blood
27 flow (U.S. EPA, 2019, section 6.1.13.2). These studies report variable results regarding the
28 timing of the effect and the mechanism by which reduced blood flow occurs (i.e., availability vs
29 sensitivity to nitric oxide). Some controlled human exposure studies using CAPs report evidence
30 for small increases in blood pressure (U.S. EPA, 2019, section 6.1.6.3). In addition, although not
31 entirely consistent, there is also some evidence across controlled human exposure studies for
32 conduction abnormalities/arrhythmia (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate
33 variability (HRV) (U.S. EPA, 2019, section 6.1.10.2), changes in hemostasis that could promote
34 clot formation (U.S. EPA, 2019, section 6.1.12.2), and increases in inflammatory cells and
35 markers (U.S. EPA, 2019, section 6.1.11.2). A recent study by Wyatt et al. (2020a) adds to the
36 limited evidence base of controlled human exposure studies conducted at near ambient PM_{2.5}

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

4 Thus, when taken as a whole, controlled human exposure studies are coherent with
5 epidemiologic studies in that they demonstrate short-term exposures to PM_{2.5} may result in the
6 types of cardiovascular endpoints that could lead to emergency department visits and hospital
7 admissions in some people.

8 Animal toxicological studies published since the 2009 ISA also support a relationship
9 between short-term PM_{2.5} exposure and cardiovascular effects. A study demonstrating decreased
10 cardiac contractility and left ventricular pressure in mice is coherent with the results of
11 epidemiologic studies reporting associations between short-term PM_{2.5} exposure and heart failure
12 (U.S. EPA, 2019, section 6.1.3.3). In addition, and as with controlled human exposure studies,
13 there is generally consistent evidence in animal toxicological studies for indicators of endothelial
14 dysfunction (U.S. EPA, 2019, section 6.1.13.3). Studies in animals also provide evidence for
15 changes in a number of other cardiovascular endpoints following short-term PM_{2.5} exposure.
16 Although not entirely consistent, these studies provide some evidence of conduction
17 abnormalities and arrhythmia (U.S. EPA, 2019, section 6.1.4.4), changes in HRV (U.S. EPA,
18 2019, section 6.1.10.3), changes in blood pressure (U.S. EPA, 2019, section 6.1.6.4), and
19 evidence for systemic inflammation and oxidative stress (U.S. EPA, 2019, section 6.1.11.3).

20 In summary, recent evidence evaluated in the 2019 ISA and the draft ISA Supplement
21 further supports and extends the conclusions of the evidence base reported in the 2009 ISA. In
22 support of epidemiologic studies reporting robust associations in copollutant models, direct
23 evidence for an independent effect of PM_{2.5} on cardiovascular effects can be found in a number
24 of controlled human exposure and animal toxicological studies. Coherent with these results are
25 epidemiologic panel studies reporting that PM_{2.5} exposure is associated with some of the same
26 cardiovascular endpoints reported in experimental studies. For these effects, there are
27 inconsistencies in results across some animal toxicological, controlled human exposure, and
28 epidemiologic panel studies, though this may be due to substantial differences in study design
29 and/or study populations. Overall, the results from epidemiologic panel, controlled human
30 exposure, and animal toxicological studies, in particular those related to endothelial dysfunction,
31 impaired cardiac function, ST segment depression, thrombosis, conduction abnormalities, and
32 changes in blood pressure provide coherence and biological plausibility for the consistent results
33 from epidemiologic studies observing positive associations between short-term PM_{2.5}
34 concentrations and IHD and HF, and ultimately cardiovascular mortality. The 2019 ISA
35 concludes that, overall, “there continues to be sufficient evidence to conclude that a causal
36 relationship exists between short-term PM_{2.5} exposure and cardiovascular effects” (U.S. EPA,

1 2019, p. 6-138), which is further supported by recent studies evaluated in the draft ISA
2 Supplement (U.S. EPA, 2021a section 3.1.1.4).

3 **3.3.1.3 Respiratory Effects**

4 Long-term PM_{2.5} exposures

5 The 2009 ISA concluded that “a causal relationship is likely to exist between long-term
6 PM_{2.5} exposure and respiratory effects” (U.S. EPA, 2009). This conclusion was based mainly on
7 epidemiologic evidence demonstrating associations between long-term PM_{2.5} exposure and
8 changes in lung function or lung function growth in children. Biological plausibility was
9 provided by a single animal toxicological study examining pre- and post-natal exposure to PM_{2.5}
10 CAPs, which found impaired lung development. Epidemiologic evidence for associations
11 between long-term PM_{2.5} exposure and other respiratory outcomes, such as the development of
12 asthma, allergic disease, and COPD; respiratory infection; and the severity of disease was
13 limited, both in the number of studies available and the consistency of the results. Experimental
14 evidence for other outcomes was also limited, with one animal toxicological study reporting that
15 long-term exposure to PM_{2.5} CAPs results in morphological changes in nasal airways of healthy
16 animals. Other animal studies examined exposure to mixtures, such as motor vehicle exhaust and
17 woodsmoke, and effects were not attributed specifically to the particulate components of the
18 mixture.

19 Cohort studies evaluated in the 2019 ISA provided additional support for the relationship
20 between long-term PM_{2.5} exposure and decrements in lung function growth (as a measure of lung
21 development), indicating a robust and consistent association across study locations, exposure
22 assessment methods, and time periods (U.S. EPA, 2019, section 5.2.13). This relationship was
23 further supported by a retrospective study that reports an association between declining PM_{2.5}
24 concentrations and improvements in lung function growth in children (U.S. EPA, 2019,
25 section 5.2.11). Epidemiologic studies also examine asthma development in children (U.S. EPA,
26 2019, section 5.2.3), with prospective cohort studies reporting generally positive associations,
27 though several are imprecise (i.e., they report wide confidence intervals). Supporting evidence is
28 provided by studies reporting associations with asthma prevalence in children, with childhood
29 wheeze, and with exhaled nitric oxide, a marker of pulmonary inflammation (U.S. EPA, 2019,
30 section 5.2.13). Additionally, animal toxicological study showing the development of an allergic
31 phenotype and an increase in a marker of airway responsiveness provides biological plausibility
32 for allergic asthma (U.S. EPA, 2019, section 5.2.13). Other epidemiologic studies report a
33 PM_{2.5}-related acceleration of lung function decline in adults, while improvement in lung function
34 was observed with declining PM_{2.5} concentrations (U.S. EPA, 2019, section 5.2.11). A
35 longitudinal study found declining PM_{2.5} concentrations are also associated with an improvement

1 in chronic bronchitis symptoms in children, strengthening evidence reported in the 2009 ISA for
2 a relationship between increased chronic bronchitis symptoms and long-term PM_{2.5} exposure
3 (U.S. EPA, 2019, section 5.2.11). A common uncertainty across the epidemiologic evidence is
4 the lack of examination of copollutants to assess the potential for confounding. While there is
5 some evidence that associations remain robust in models with gaseous pollutants, a number of
6 these studies examining copollutant confounding were conducted in Asia, and thus have limited
7 generalizability due to high annual pollutant concentrations.

8 When taken together, the 2019 ISA concludes that the “epidemiologic evidence strongly
9 supports a relationship with decrements in lung function growth in children” and “with asthma
10 development in children, with increased bronchitis symptoms in children with asthma, with an
11 acceleration of lung function decline in adults, and with respiratory mortality and cause-specific
12 respiratory mortality for COPD and respiratory infection” (U.S. EPA, 2019, p. 1-34). In support
13 of the biological plausibility of such associations reported in epidemiologic studies of respiratory
14 health effects, animal toxicological studies continue to provide direct evidence that long-term
15 exposure to PM_{2.5} results in a variety of respiratory effects. Animal studies in the 2019 ISA show
16 pulmonary oxidative stress, inflammation, and morphologic changes in the upper (nasal) and
17 lower airways. Other results show that changes are consistent with the development of allergy
18 and asthma, and with impaired lung development. Overall, the 2019 ISA concludes that “the
19 collective evidence is sufficient to conclude that a causal relationship is likely to exist between
20 long-term PM_{2.5} exposure and respiratory effects” (U.S. EPA, 2019, section 5.2.13).

21 Short-term PM_{2.5} exposures

22 The 2009 ISA (U.S. EPA, 2009) concluded that a “causal relationship is likely to exist”
23 between short-term PM_{2.5} exposure and respiratory effects. This conclusion was based mainly on
24 the epidemiologic evidence demonstrating positive associations with various respiratory effects.
25 Specifically, the 2009 ISA described epidemiologic evidence as consistently showing
26 PM_{2.5}-associated increases in hospital admissions and emergency department visits for chronic
27 obstructive pulmonary disease (COPD) and respiratory infection among adults or people of all
28 ages, as well as increases in respiratory mortality. These results were supported by studies
29 reporting associations with increased respiratory symptoms and decreases in lung function in
30 children with asthma, though the epidemiologic evidence was inconsistent for hospital
31 admissions or emergency department visits for asthma. Studies examining copollutants models
32 showed that PM_{2.5} associations with respiratory effects were robust to inclusion of CO or SO₂ in
33 the model, but often were attenuated (though still positive) with inclusion of O₃ or NO₂. In
34 addition to the copollutants models, evidence supporting an independent effect of PM_{2.5} exposure
35 on the respiratory system was provided by animal toxicological studies of PM_{2.5} CAPs
36 demonstrating changes in some pulmonary function parameters, as well as inflammation,

1 oxidative stress, injury, enhanced allergic responses, and reduced host defenses. Many of these
2 effects have been implicated in the pathophysiology for asthma exacerbation, COPD
3 exacerbation, or respiratory infection. In the few controlled human exposure studies conducted in
4 individuals with asthma or COPD, PM_{2.5} exposure mostly had no effect on respiratory
5 symptoms, lung function, or pulmonary inflammation. Available studies in healthy people also
6 did not clearly demonstrate respiratory effects following short-term PM_{2.5} exposures.

7 Epidemiologic studies evaluated in the 2019 ISA continue to provide strong evidence for
8 a relationship between short-term PM_{2.5} exposure and several respiratory-related endpoints,
9 including asthma exacerbation (U.S. EPA, 2019, section 5.1.2.1), COPD exacerbation (U.S.
10 EPA, 2019, section 5.1.4.1), and combined respiratory-related diseases (U.S. EPA, 2019, section
11 5.1.6), particularly from studies examining emergency department visits and hospital admissions.
12 The generally positive associations between short-term PM_{2.5} exposure and asthma and COPD
13 emergency department visits and hospital admissions are supported by epidemiologic studies
14 demonstrating associations with other respiratory-related effects such as symptoms and
15 medication use that are indicative of asthma and COPD exacerbations (U.S. EPA, 2019, sections
16 5.1.2.2 and 5.4.1.2). The collective body of epidemiologic evidence for asthma exacerbation is
17 more consistent in children than in adults. Additionally, epidemiologic studies examining the
18 relationship between short-term PM_{2.5} exposure and respiratory mortality provide evidence of
19 consistent positive associations, demonstrating a continuum of effects (U.S. EPA, 2019, section
20 5.1.9).

21 Building off the studies evaluated in the 2009 and 2019 ISA, epidemiologic studies
22 expand the assessment of potential copollutant confounding. There is some evidence that PM_{2.5}
23 associations with asthma exacerbation, combined respiratory-related diseases, and respiratory
24 mortality remain relatively unchanged in copollutant models with gaseous pollutants (i.e., O₃,
25 NO₂, SO₂, with more limited evidence for CO) and other particle sizes (i.e., PM_{10-2.5}) (U.S. EPA,
26 2019, section 5.1.10.1).

27 In the 2019 ISA, the uncertainty related to whether there is an independent effect of PM_{2.5}
28 on respiratory health is also partially addressed by findings from animal toxicological studies.
29 Specifically, short-term exposure to PM_{2.5} enhanced asthma-related responses in an animal
30 model of allergic airways disease and enhanced lung injury and inflammation in an animal model
31 of COPD (U.S. EPA, 2019, sections 5.1.2.4.4 and 5.1.4.4.3). The experimental evidence
32 provides biological plausibility for some respiratory-related endpoints, including limited
33 evidence of altered host defense and greater susceptibility to bacterial infection as well as
34 consistent evidence of respiratory irritant effects. Animal toxicological evidence for other
35 respiratory effects is inconsistent. A recent study by Wyatt et al. (2020a) was conducted at near
36 ambient PM_{2.5} concentrations and adds to the limited evidence base of controlled human

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

3 The 2019 ISA concludes that “[t]he strongest evidence of an effect of short-term PM_{2.5}
4 exposure on respiratory effects is provided by epidemiologic studies of asthma and COPD
5 exacerbation. While animal toxicological studies provide biological plausibility for these
6 findings, some uncertainty remains with respect to the independence of PM_{2.5} effects” (U.S.
7 EPA, 2019, p. 5-155). When taken together, the 2019 ISA concludes that this evidence “is
8 sufficient to conclude that a causal relationship is likely to exist between short-term PM_{2.5}
9 exposure and respiratory effects” (U.S. EPA, 2019, p. 5-155).

10 **3.3.1.4 Cancer – Long-term PM_{2.5} Exposures**

11 The 2009 ISA concluded that the overall body of evidence was “suggestive of a causal
12 relationship between relevant PM_{2.5} exposures and cancer” (U.S. EPA, 2009). This conclusion
13 was based primarily on positive associations observed in a limited number of epidemiologic
14 studies of lung cancer mortality. The few epidemiologic studies that had evaluated PM_{2.5}
15 exposure and lung cancer incidence or cancers of other organs and systems generally did not
16 show evidence of an association. Toxicological studies did not focus on exposures to specific
17 PM size fractions, but rather investigated the effects of exposures to total ambient PM, or other
18 source-based PM such as wood smoke. Collectively, results of in vitro studies were consistent
19 with the larger body of evidence demonstrating that ambient PM and PM from specific
20 combustion sources are mutagenic and genotoxic. However, animal inhalation studies found
21 little evidence of tumor formation in response to chronic exposures. A small number of studies
22 provided preliminary evidence that PM exposure can lead to changes in methylation of DNA,
23 which may contribute to biological events related to cancer.

24 Since the 2009 ISA, additional cohort studies provide evidence that long-term PM_{2.5}
25 exposure is positively associated with lung cancer mortality and with lung cancer incidence, and
26 provide initial evidence for an association with reduced cancer survival (U.S. EPA, 2019, section
27 10.2.5). Re-analyses of the ACS cohort using different years of PM_{2.5} data and follow-up, along
28 with various exposure assignment approaches, provide consistent evidence of positive
29 associations between long-term PM_{2.5} exposure and lung cancer mortality (U.S. EPA, 2019,
30 Figure 10-3). Additional support for positive associations with lung cancer mortality is provided
31 by epidemiologic studies using individual-level data to control for smoking status, by studies of
32 people who have never smoked (though such studies generally report wide confidence intervals
33 due to the small number of lung cancer mortality cases within this population), and in analyses of
34 cohorts that relied upon proxy measures to account for smoking status (U.S. EPA, 2019, section
35 10.2.5.1.1). Although studies that have evaluated lung cancer incidence, including studies of

1 people who have never smoked, are limited in number, studies in the 2019 ISA generally report
2 positive associations with long-term PM_{2.5} exposures (U.S. EPA, 2019, section 10.2.5.1.2). A
3 subset of the studies focusing on lung cancer incidence also examined histological subtype,
4 providing some evidence of positive associations for adenocarcinomas, the predominate subtype
5 of lung cancer observed in people who have never smoked (U.S. EPA, 2019, section 10.2.5.1.2).
6 Associations between long-term PM_{2.5} exposure and lung cancer incidence were found to remain
7 relatively unchanged, though in some cases confidence intervals widened, in analyses that
8 attempted to reduce exposure measurement error by accounting for length of time at residential
9 address or by examining different exposure assignment approaches (U.S. EPA, 2019, section
10 10.2.5.1.2).

11 The 2019 ISA evaluates the degree to which epidemiologic studies have addressed the
12 potential for confounding by copollutants and the shape of the concentration-response
13 relationship. To date, relatively few studies have evaluated the potential for copollutant
14 confounding of the relationship between long-term PM_{2.5} exposure and lung cancer mortality or
15 incidence. The small number of such studies have generally focused on O₃ and report that PM_{2.5}
16 associations remain relatively unchanged in copollutant models (U.S. EPA, 2019, section
17 10.2.5.1.3). However, available studies have not systematically evaluated the potential for
18 copollutant confounding by other gaseous pollutants or by other particle size fractions (U.S.
19 EPA, 2019, section 10.2.5.1.3). Compared to total (non-accidental) mortality (U.S. EPA, 2019,
20 section 10.2.4.1.4), fewer studies have examined the shape of the concentration-response curve
21 for cause-specific mortality outcomes, including lung cancer. Several studies of lung cancer
22 mortality and incidence have reported no evidence of deviations from linearity in the shape of
23 the concentration-response relationship (Lepeule et al., 2012; Raaschou-Nielsen et al., 2013;
24 Puett et al., 2014), though authors provided only limited discussions of results (U.S. EPA, 2019,
25 section 10.2.5.1.4).

26 In support of the biological plausibility of an independent effect of PM_{2.5} on lung cancer,
27 the 2019 ISA notes evidence from recent experimental and epidemiologic studies demonstrating
28 that PM_{2.5} exposure can lead to a range of effects indicative of mutagenicity, genotoxicity, and
29 carcinogenicity, as well as epigenetic effects (U.S. EPA, 2019, section 10.2.7). For example,
30 both in vitro and in vivo toxicological studies have shown that PM_{2.5} exposure can result in DNA
31 damage (U.S. EPA, 2019, section 10.2.2). Although such effects do not necessarily equate to
32 carcinogenicity, the evidence that PM exposure can damage DNA, and elicit mutations, provides
33 support for the plausibility of epidemiologic associations with lung cancer mortality and
34 incidence. Additional supporting studies indicate the occurrence of micronuclei formation and
35 chromosomal abnormalities (U.S. EPA, 2019, section 10.2.2.3), and differential expression of
36 genes that may be relevant to cancer pathogenesis, following PM exposures. Experimental and

1 epidemiologic studies that examine epigenetic effects indicate changes in DNA methylation,
2 providing some support for PM_{2.5} exposure contributing to genomic instability (U.S. EPA, 2019,
3 section 10.2.3). Overall, there is limited evidence that long-term PM_{2.5} exposure is associated
4 with cancers in other organ systems, but there is some evidence that PM_{2.5} exposure may reduce
5 survival in individuals with cancer (U.S. EPA, 2019 section 10.2.7; U.S. EPA, 2021a, section
6 2.1.1.4.1).

7 Epidemiologic evidence for associations between PM_{2.5} and lung cancer mortality and
8 incidence, together with evidence supporting the biological plausibility of such associations,
9 contributes to the 2019 ISA’s conclusion that the evidence “is sufficient to conclude that a causal
10 relationship is likely to exist between long-term PM_{2.5} exposure and cancer” (U.S. EPA, 2019,
11 section 10.2.7).

12 **3.3.1.5 Nervous System Effects**

13 Long-term PM_{2.5} exposures

14 Reflecting the very limited evidence available in the 2012 review, the 2009 ISA did not
15 make a causality determination for long-term PM_{2.5} exposures and nervous system effects (U.S.
16 EPA, 2009). Since the last review, this body of evidence has grown substantially (U.S. EPA,
17 2019, section 8.2). Animal toxicology studies assessed in the 2019 ISA report that long-term
18 PM_{2.5} exposures can lead to morphologic changes in the hippocampus and to impaired learning
19 and memory. This evidence is consistent with epidemiologic studies reporting that long-term
20 PM_{2.5} exposure is associated with reduced cognitive function (U.S. EPA, 2019, section 8.2.5).
21 Further, while the evidence is limited, the presence of early markers of Alzheimer’s disease
22 pathology has been demonstrated in rodents following long-term exposure to PM_{2.5} CAPs. These
23 findings support reported associations with neurodegenerative changes in the brain
24 (i.e., decreased brain volume), all-cause dementia, or hospitalization for Alzheimer’s disease in a
25 small number of epidemiologic studies (U.S. EPA, 2019, section 8.2.6). Additionally, loss of
26 dopaminergic neurons in the substantia nigra, a hallmark of Parkinson disease, has been reported
27 in mice (U.S. EPA, 2019, section 8.2.4), though epidemiologic studies provide only limited
28 support for associations with Parkinson’s disease (U.S. EPA, 2019, section 8.2.6). Overall, the
29 lack of consideration of copollutant confounding introduces some uncertainty in the
30 interpretation of epidemiologic studies of nervous system effects, but this uncertainty is partly
31 addressed by the evidence for an independent effect of PM_{2.5} exposures provided by
32 experimental animal studies.

33 In addition to the findings described above, which are most relevant to older adults,
34 several studies of neurodevelopmental effects in children have also been conducted. Positive
35 associations between long-term exposure to PM_{2.5} during the prenatal period and autism

1 spectrum disorder (ASD) are observed in multiple epidemiologic studies (U.S. EPA, 2019,
2 section 8.2.7.2), while studies of cognitive function provide little support for an association (U.S.
3 EPA, 2019, section 8.2.5.2). Interpretation of these epidemiologic studies is limited due to the
4 small number of studies, their lack of control for potential confounding by copollutants, and
5 uncertainty regarding the critical exposure windows. Biological plausibility is provided for the
6 ASD findings by a study in mice that found inflammatory and morphologic changes in the
7 corpus collosum and hippocampus, as well as ventriculomegaly (i.e., enlarged lateral ventricles)
8 in young mice following prenatal exposure to PM_{2.5} CAPs.

9 Taken together, the 2019 ISA concludes that studies indicate long-term PM_{2.5} exposures
10 can lead to effects on the brain associated with neurodegeneration (i.e., neuroinflammation and
11 reductions in brain volume), as well as cognitive effects in older adults (U.S. EPA, 2019, Table
12 1-2). Animal toxicology studies provide evidence for a range of nervous system effects in adult
13 animals, including neuroinflammation and oxidative stress, neurodegeneration, and cognitive
14 effects, and effects on neurodevelopment in young animals. The epidemiologic evidence is more
15 limited, but studies generally support associations between long-term PM_{2.5} exposure and
16 changes in brain morphology, cognitive decrements and dementia. There is also initial, and
17 limited, evidence for neurodevelopmental effects, particularly ASD. The consistency and
18 coherence of the evidence supports the 2019 ISA’s conclusion that “the collective evidence is
19 sufficient to conclude that a causal relationship is likely to exist between long-term PM_{2.5}
20 exposure and nervous system effects” (U.S. EPA, 2019, section 8.2.9).

21 **3.3.1.6 Other Effects**

22 Compared to the health outcomes discussed above, the 2019 ISA concludes that there is
23 greater uncertainty in the evidence linking PM_{2.5}, or UFP, exposures with other health outcomes,
24 reflected in conclusions that the evidence is “suggestive of, but not sufficient to infer, a causal
25 relationship.” The sections below summarize the 2019 ISA conclusions for these outcomes for
26 long-term (section 3.3.1.6.1) and short-term (section 3.3.1.6.2) PM_{2.5} and UFP exposures.
27 Section 3.3.1.6.3 summarizes information assessed in the draft ISA Supplement related to the
28 emerging area of COVID-19 infection and death.

29 **3.3.1.6.1 Long-term Exposures**

30 As indicated in Table 3-1 above, the 2019 ISA concludes that the evidence is “suggestive
31 of, but not sufficient to infer, a causal relationship” between long-term PM_{2.5} exposures and
32 metabolic effects and reproductive and developmental effects (reproduction and fertility;
33 pregnancy and birth outcomes). These conclusions reflect evidence that is “generally supportive
34 but not entirely consistent or is limited overall” where “[c]hance, confounding, and other biases

1 cannot be ruled out” (U.S. EPA, 2019, Preface, p. P-20). The basis for these causality
2 determinations is summarized briefly below.

3 *PM_{2.5} – Metabolic effects*

4 There were no causality determinations for long-term PM_{2.5} exposure and metabolic
5 effects in the 2009 ISA (U.S. EPA, 2009). However, the literature pertaining to the effect of
6 long-term exposure to PM_{2.5} and metabolic effects has expanded substantially since the 2009
7 ISA, and consists of both epidemiologic and experimental evidence (U.S. EPA, 2019, section
8 7.2). Epidemiologic studies report positive associations between long-term PM_{2.5} exposure and
9 diabetes-related mortality. In addition, although results were not consistent across cohorts, there
10 is some evidence from epidemiologic studies for positive associations with incident diabetes,
11 metabolic syndrome, and alterations in glucose and insulin homeostasis. Consideration of
12 copollutant confounding was limited. In animal toxicologic studies, there is some support for a
13 relationship between long-term PM_{2.5} exposure and metabolic effects from experimental studies
14 demonstrating increased blood glucose, insulin resistance, and inflammation and visceral
15 adiposity but the experimental evidence was not entirely consistent. Based on this evidence, the
16 2019 ISA concludes that, “[o]verall, the collective evidence is suggestive of, but is not sufficient
17 to infer, a causal relationship between long-term PM_{2.5} exposure and metabolic effects” (U.S.
18 EPA, 2019, p. 7-52).

19 *PM_{2.5} – Reproductive and developmental effects*

20 The 2009 ISA determined that the evidence was “suggestive of a causal relationship” for
21 the association between long-term PM_{2.5} exposure and reproductive and developmental
22 outcomes. The body of literature characterizing these relationships has grown since the 2009
23 ISA, with much of the evidence focusing on reproduction and fertility or pregnancy and birth
24 outcomes, though important uncertainties persist (U.S. EPA, 2019, sections 9.1.1, 9.1.2, 9.1.5).

25 Effects of PM_{2.5} exposure on sperm have been studied in both epidemiology and
26 toxicology studies and shows the strongest evidence in epidemiologic studies for impaired sperm
27 motility and in animal toxicological studies for impaired spermiation. Epidemiologic evidence on
28 sperm morphology have reported inconsistent results. Evidence for effects of PM_{2.5} exposure on
29 female reproduction also comes from both epidemiology and toxicology studies. In the
30 epidemiologic literature, results on human fertility and fecundity are limited, but the evidence on
31 in vitro fertilization indicates a modest association of PM_{2.5} exposures with decreased odds of
32 becoming pregnant. Studies in rodents have shown ovulation and estrus are affected by PM_{2.5}
33 exposure. Biological plausibility for outcomes related to male and female fertility and
34 reproduction comes from laboratory animal studies demonstrating genetic and epigenetic
35 changes in germ cells with PM_{2.5} exposure. The 2019 ISA concludes that, “[c]ollectively, the

1 evidence is suggestive of, but not sufficient to infer, a causal relationship between PM_{2.5}
2 exposure and male and female reproduction and fertility” (U.S. EPA, 2019, p. 9-43).

3 With regard to pregnancy and birth outcomes, while the collective evidence for many of
4 the outcomes examined is not consistent, there are some animal toxicology and epidemiologic
5 studies that indicate an association between PM_{2.5} exposures and reduced fetal growth, low birth
6 weight and preterm birth. Most of the epidemiologic studies do not control for co-pollutant
7 confounding and do not identify a specific sensitive window of exposure, but results from animal
8 toxicologic studies provide biological plausibility for these outcomes, as well as support for
9 multiple sensitive windows for PM_{2.5} exposure-associated outcomes. There is also epidemiologic
10 evidence for congenital heart defects of different types, as well as biological plausibility to
11 support this outcome from the animal toxicology literature. However, evidence for a relationship
12 between PM_{2.5} exposure and various pregnancy-related pathologies, including gestational
13 hypertension, pre-eclampsia and gestational diabetes is inconsistent. Biological plausibility for
14 effects of PM_{2.5} exposure and various pregnancy and birth outcomes is provided by studies
15 showing that PM_{2.5} exposure in laboratory rodents resulted in impaired implantation and vascular
16 endothelial dysfunction. Coherence with toxicological studies is provided by epidemiologic
17 studies in humans reporting associations with epigenetic changes to the placenta and impaired
18 fetal thyroid function. When taken together, the 2019 ISA concludes that the available evidence,
19 including uncertainties that evidence, is “suggestive of, but not sufficient to infer, a causal
20 relationship between exposure to PM_{2.5} and pregnancy and birth outcomes” (U.S. EPA, 2019, p.
21 9-44).

22 *UFP – Nervous System Effects*

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

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

8 **3.3.1.6.2 Short-term Exposures**

9 As indicated in Table 3-1 above, the 2019 ISA concludes that the evidence is “suggestive
10 of, but not sufficient to infer, a causal relationship” between short-term PM_{2.5} exposures and
11 metabolic effects and nervous system effects. Additionally, the 2019 ISA concludes that the
12 evidence is “suggestive” for short-term UFP exposures and cardiovascular effects, respiratory
13 effects, and nervous system effects. As for the outcomes related to long-term exposures,
14 discussed above, these conclusions reflect evidence that is “generally supportive but not entirely
15 consistent or is limited overall” where “[c]hance, confounding, and other biases cannot be ruled
16 out” (U.S. EPA, 2019, Preface, p.P-20). The basis for these causality determinations is
17 summarized briefly below.

18 *PM_{2.5} – Metabolic effects*

19 There were no studies of the effect of short-term PM_{2.5} exposure and metabolic effects
20 reviewed in the 2009 ISA (U.S. EPA, 2009). New evidence for a relationship between short-term
21 PM_{2.5} exposure and metabolic effects is based on a small number of epidemiologic and animal
22 toxicological studies reporting effects on glucose and insulin homeostasis and other indicators of
23 metabolic function such as inflammation in the visceral adipose tissue and liver (U.S. EPA,
24 2019, section 7.1). The 2019 ISA concludes that, overall, the collective evidence “is suggestive
25 of, but not sufficient to infer, a causal relationship between short-term PM_{2.5} exposure and
26 metabolic effects” (U.S. EPA, 2019, p. 7-11).

27 *PM_{2.5} – Nervous system effects*

28 The evidence reviewed in the 2009 ISA was characterized as "inadequate to infer" a
29 causal relationship between short-term PM_{2.5} exposure and nervous system effects (U.S. EPA,
30 2009), based on a small number of experimental animal studies. Studies assessed in the 2019
31 ISA provide additional evidence that short-term exposure to PM_{2.5} can affect the nervous system
32 (U.S. EPA, 2019, section 8.1). The strongest evidence is provided by experimental studies in
33 mice that show effects on the brain. These toxicological studies demonstrate changes in
34 neurotransmitters in the hypothalamus that are linked to sympathetic nervous system and
35 hypothalamic-pituitary-adrenal (HPA) stress axis activation, as well as upregulation of

1 inflammation-related genes, changes in cytokine levels, and other changes that are indicative of
2 brain inflammation. In addition, an association of short-term PM_{2.5} exposure with hospital
3 admissions for Parkinson’s disease was observed indicating the potential for exacerbation of
4 neurological diseases. The 2019 ISA concludes that, overall, the collective evidence “is
5 suggestive of, but not sufficient to infer, a causal relationship between short-term exposure to
6 PM_{2.5} and nervous system effects” (U.S. EPA, 2019, p. 8-15).

7 *UFP – Cardiovascular effects*

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

30 *UFP – Respiratory effects*

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

1 ambient UFP were associated with lung function decrements in adults with asthma. The few
2 available experimental studies provided limited coherence with epidemiologic findings for
3 asthma exacerbation. Studies assessed in the 2019 ISA add to this evidence base and support
4 epidemiologic evidence for asthma exacerbation and combined respiratory-related diseases but
5 do not rule out chance, confounding, and other biases (U.S. EPA, 2019, section 5.5). For
6 example, associations persist in one epidemiologic study with adjustment for NO₂, but not in
7 another. Additional supporting evidence, showing decrements in lung function and enhancement
8 of allergic inflammation and other allergic responses, is provided by a controlled human
9 exposure study in adults with asthma and by animal toxicological studies in an animal model of
10 allergic airway disease. For combined respiratory-related diseases, recent findings add
11 consistency for hospital admissions and emergency department visits and indicate lung function
12 changes among adults with asthma or COPD. Uncertainty remains regarding the characterization
13 of UFP exposures and the potential for copollutant confounding in epidemiologic studies, which
14 limits inference about an independent effect of UFP exposures (U.S. EPA, 2019, section 5.5).
15 The 2019 ISA concludes that, overall, the evidence is “suggestive of, but not sufficient to infer, a
16 causal relationship between short-term UFP exposure and respiratory effects” (U.S. EPA, 2019,
17 p. 5-303).

18 *UFP- Nervous system effects*

19 The 2009 ISA reported limited animal toxicological evidence of a relationship between
20 short-term exposure to UFP and nervous system effects, without supporting epidemiologic
21 studies. Several experimental studies evaluated in the 2019 ISA add to this evidence base. In the
22 2019 ISA, the strongest evidence for a relationship between short-term UFP exposure and
23 nervous system effects is provided by animal toxicological studies that show inflammation and
24 oxidative stress in multiple brain regions following exposure to UFP. There is a lack of evidence
25 from epidemiologic studies (U.S. EPA, 2019, section 8.5). The 2019 ISA concludes that, overall,
26 the collective evidence is “suggestive of, but not sufficient to infer, a causal relationship between
27 short-term UFP exposure and nervous system effects” (U.S. EPA, 2019, p. 8-86).

28 **3.3.1.6.3 COVID-19 Infection and Death**

29 With the advent of the global COVID-19 pandemic, a number of recent studies evaluated
30 in the draft ISA Supplement examined the role of ambient air pollution, specifically PM_{2.5}, on
31 COVID-19 infections and deaths, including a few studies within the U.S. and Canada (U.S. EPA,
32 2021a; section 3.3.2). While there is no exact corollary within the 2019 ISA for these types of
33 studies, the 2019 ISA presented evidence that evaluates the potential relationship between short-
34 and long-term PM_{2.5} exposure and respiratory infection (U.S. EPA, 2019, section 5.1.5 and
35 5.2.6). Studies assessed in the 2019 ISA report that some evidence of positive associations

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

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

25 3.3.1.7 Summary

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

- 29 • **To what extent does the scientific evidence strengthen, or otherwise alter, our**
30 **preliminary conclusions regarding health effects attributable to long- or short-term**
31 **fine particle exposures? Have previously identified uncertainties been reduced?**
32 **What important uncertainties remain and have new uncertainties been identified?**

33 We consider these questions in the context of the evidence for effects of long- and short-
34 term PM_{2.5} exposures. Studies reviewed in the 2019 ISA and the draft ISA Supplement expand
35 our understanding of the PM_{2.5}-related health effects from long- and short- term exposures, as
36 well as reduced important uncertainties identified in prior reviews. Epidemiologic studies

1 consistently report positive associations between PM_{2.5} exposures and a wide range of health
2 outcomes, including total and cause-specific mortality (e.g., cardiovascular and respiratory
3 mortality), cardiovascular and respiratory morbidity, lung cancer, and nervous system effects.
4 Such associations have been reported in analyses employing a variety of study designs,
5 approaches to estimating PM_{2.5} exposures, statistical models, and long-term exposure windows
6 (i.e., the exposure period that is associated with the health outcome). Recent U.S. and Canadian
7 epidemiologic studies evaluated in the draft ISA Supplement provide additional support for the
8 conclusions of the 2019 ISA. Overall, these studies support, and in some instances strengthen,
9 the evidence presented in the 2019 ISA of long-term PM_{2.5} exposures and health effects. Cohort
10 studies assessed in the draft ISA Supplement add to the large body of evidence exhibiting
11 consistent, positive associations between long-term PM_{2.5} exposure and mortality detailed in the
12 2019 ISA. While relatively fewer recent U.S. and Canadian epidemiologic studies examined
13 short-term PM_{2.5} exposure and mortality, these studies continue to provide evidence of positive
14 associations with all-cause and total (nonaccidental) mortality, in addition to cause-specific
15 mortality outcomes. Further, the 2019 ISA and draft ISA Supplement include retrospective
16 studies that demonstrate improvements in health outcomes, including increased life expectancy,
17 decreasing mortality, or decreasing respiratory effects, as a result of decreases in ambient PM_{2.5}
18 concentrations over time. Lastly, the biological plausibility of PM_{2.5}-attributable mortality is
19 supported by the coherence of effects across scientific disciplines (i.e., animal toxicological,
20 controlled human exposure studies, and epidemiologic) when evaluating respiratory and
21 cardiovascular morbidity effects, which are some of the largest contributors to total
22 (nonaccidental) mortality.

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

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

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

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

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

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

10 3.3.2 Public Health Implications and At-Risk Populations

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

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

18 The information available in this reconsideration has not altered our understanding of
19 human populations at risk of health effects from PM_{2.5} exposures. As recognized in the 2020
20 review, the 2019 ISA cites extensive evidence indicating that “both the general population as
21 well as specific populations and lifestages are at risk for PM_{2.5}-related health effects” (U.S. EPA,
22 2019, p. 12-1). Factors that may contribute to increased risk of PM_{2.5}-related health effects
23 include lifestage (children and older adults), pre-existing diseases (cardiovascular disease and
24 respiratory disease), race/ethnicity, and socioeconomic status.¹⁴

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

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

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

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

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

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

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

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

16 There is strong evidence for racial and ethnic disparities in PM_{2.5} exposures and PM_{2.5}-
17 related health risk, as assessed in the 2019 ISA and with even more evidence available since the
18 literature cutoff date for the 2019 ISA and evaluated in the draft ISA Supplement. There is strong
19 evidence demonstrating that Black and Hispanic populations, in particular, have higher PM_{2.5}
20 exposures than non-Hispanic White populations (U.S. EPA, 2019, Figure 12-2; U.S. EPA,
21 2021a, Figure 3-38). Black populations or individuals that live in predominantly Black
22 neighborhoods experience higher PM_{2.5} exposures, in comparison to non-Hispanic White
23 populations. There is also consistent evidence across multiple studies that demonstrate increased
24 risk of PM_{2.5}-related health effects, with the strongest evidence for health risk disparities for
25 mortality (U.S. EPA, 2019, section 12.5.4). There is also evidence of health risk disparities for
26 both Hispanic and non-Hispanic Black populations compared to non-Hispanic White populations
27 for cause-specific mortality and incident hypertension (U.S. EPA, 2021a, 3.3.3.2).

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

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

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

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

17 **Table 3-2. National demographic information, 2019.**

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

\$10,000 to \$14,999		4.0
\$15,000 to \$24,999		8.3
\$25,000 to \$34,999		8.4
\$35,000 to \$49,999		11.9
\$50,000 to \$74,999		17.4
\$75,000 to \$99,999		12.8
\$100,000 to \$149,999		15.7
\$150,000 to \$199,999		7.2
\$200,000 or more		8.5
Educational Attainment⁴		
Less than high school	25,618,541	11.4
High school graduate (or equivalent)	60,482,353	26.9
Some college, no degree	44,914,086	20
Associate's degree	19,381,937	8.6
Bachelor's degree	45,730,479	20.3
Graduate or professional degree	28,771,172	12.8

¹ Numbers within selected characteristics may not sum to total due to rounding

² NH = non-Hispanic

³ Household income in the past 12 months in 2019 inflation-adjusted dollars.

⁴ Educational attainment for population aged 25 years and older.

Adapted from the 2019 American Community Survey and Housing Survey. Available at:

Demographics: <https://data.census.gov/cedsci/table?q=United%20States&tid=ACSDP1Y2019.DP05>

Income: <https://data.census.gov/cedsci/table?q=United%20States&t=Income%20and%20Poverty&tid=ACSST1Y2019.S1901>

Education:

<https://data.census.gov/cedsci/table?q=United%20States&t=Education%3AEducational%20Attainment&tid=ACSST1Y2019.S1501>

1
2 As noted above, individuals with pre-existing cardiovascular disease and pre-existing
3 respiratory disease may also be at increased risk of PM_{2.5}-related health effects. Table 3-3 below
4 considers the currently available information that helps to characterize key features of
5 populations with cardiovascular or respiratory diseases or conditions. The National Center for
6 Health Statistics data for 2018 indicate that, for adult populations, older adults (e.g., those 65
7 years and older) have a higher prevalence of cardiovascular diseases compared to younger adults
8 (e.g., those 64 years and younger). For respiratory diseases, older adults also have a higher
9 prevalence of emphysema than younger adults, and adults 44 years or older have a higher
10 prevalence of chronic bronchitis. However, the prevalence for asthma is generally similar across
11 all adult age groups.

12 With respect to race, American Indians or Alaskan Natives have the highest prevalence of
13 all heart disease and coronary heart disease, while Blacks have the highest prevalence of
14 hypertension and stroke. Hypertension has the highest prevalence across all racial groups
15 compared to other cardiovascular diseases or conditions, ranging from approximately 22% to
16 32% of each racial group. Overall, the prevalence of cardiovascular diseases or conditions is
17 lowest for Asians compared to Whites, Blacks, and American Indians or Alaskan Natives.

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

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

	Adults (18+)	Age (%) ¹				Race (%) ²				Ethnicity (%) ³	
Chronic Disease or Condition	N (in thousands)	18-44	44-64	65-74	75+	White	Black	American Indian or Alaska Native	Asian	Hispanic	Non-Hispanic
All (N, in thousands)	249,456	115,008	83,038	30,809	20,601	193,454	30,813	2,810	15,960	40,749	208,706
Selected Cardiovascular Diseases/Conditions											
All heart disease	30,252	4.8	11.8	23.6	37.3	11.5	10.0	14.6	7.7	8.2	11.7
Coronary heart disease	15,780	1.0	6.0	15.5	23.9	5.7	5.4	8.6	4.4	5.1	5.7
Hypertension	67,856	8.8	34.4	54.4	61.1	23.9	32.2	27.2	21.9	23.7	25.1
Stroke	7,801	0.6	3.1	6.9	11.8	2.6	3.9	3.0	2.7	2.5	2.9
Selected Respiratory Diseases											
Asthma ⁴	19,233	7.2	8.3	8.6	6.7	7.5	9.1	9.5	3.7	6.0	8.1
COPD – chronic bronchitis	9,003	2.2	4.5	5.1	5.6	3.6	3.4	*	1.1	2.7	3.6
COPD – emphysema	3,780	0.2	1.6	4.1	4.5	1.4	1.1	0.4	0.7	1.0	1.4
¹ Percentage of individual adults within each age group with disease, based on N (at the top of each age column). ² Percentage of individual adults within each race group with disease, based on N (at the top of each race column). ³ Percentage of individual adults within each ethnic group with disease, based on N (at the top of each ethnic column). ⁴ Asthma prevalence is reported for “still has asthma.” * Estimate does not meet NCHS standards of reliability. Source: (Insert cites); National Center for Health Statistics, Summary Health Statistics, National Health Interview Survey, 2018; Tables A-1 and A-2.											

2

1 Taken together, this information indicates that the groups at increased risk of PM_{2.5}-
2 related health effects represent a substantial portion of the total U.S. population. In evaluating the
3 primary PM_{2.5} standards, an important consideration is the potential PM_{2.5}-related public health
4 impacts in these populations.

5 **3.3.3 PM_{2.5} Concentrations in Key Studies Reporting Health Effects**

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

- 11 • **What are the short- or long-term PM_{2.5} exposures that have been associated with**
12 **health effects and to what extent does the evidence support the occurrence of such**
13 **effects for air quality meeting the current primary PM_{2.5} standards?**

14 In addressing these questions, we emphasize health outcomes for which the 2019 ISA concludes
15 that the evidence supports a “causal” or a “likely to be causal” relationship with PM_{2.5} exposures.
16 As discussed above, this includes mortality, cardiovascular effects, and respiratory effects
17 associated with short- or long-term PM_{2.5} exposures and cancer and nervous system effects
18 associated with long-term PM_{2.5} exposures. While the causality determinations in the 2019 ISA
19 are informed by studies evaluating a wide range of PM_{2.5} concentrations, this section considers
20 the degree to which the evidence in the 2019 ISA and draft ISA Supplement supports the
21 occurrence of PM-related effects at concentrations relevant to informing conclusions on the
22 primary PM_{2.5} standards. Section 3.3.3.1 considers the exposure concentrations that have been
23 evaluated in experimental studies and section 3.3.3.2 considers the ambient concentrations in
24 locations evaluated by epidemiologic studies.

25 **3.3.3.1 PM Exposure Concentrations Evaluated in Experimental Studies**

26 As stated in the 2019 ISA, the evidence for a particular PM_{2.5}-related health outcome is
27 strengthened when results from experimental studies demonstrate biologically plausible
28 mechanisms through which adverse human health outcomes could occur (U.S. EPA, 2015b,
29 Preamble p. 20). Two types of experimental studies are of particular importance in understanding
30 the effects of PM exposures: controlled human exposure and animal toxicology studies. In such
31 studies, investigators expose human volunteers or laboratory animals to known concentrations of
32 air pollutants under carefully regulated environmental conditions and activity levels. Thus,
33 controlled human exposure and animal toxicology studies can provide information on the health
34 effects of experimentally administered pollutant exposures under highly controlled laboratory
35 conditions (U.S. EPA, 2015b, Preamble, p. 11).

1 In this section, we consider the PM_{2.5} exposure concentrations shown to result effects in
2 controlled human exposure studies and in animal toxicology studies. We particularly consider
3 the consistency of specific PM_{2.5}-related effects across studies, the potential adversity of such
4 effects, and the degree to which exposures shown to cause effects are likely to occur in areas
5 meeting the current primary standards. To address these issues, we consider the following
6 question:

- 7 • **To what extent does the evidence from controlled human exposure or animal
8 toxicology studies support the potential for adverse cardiovascular, respiratory, or
9 other effects following PM_{2.5} exposures likely to occur in areas meeting the current
10 or alternative primary standards?**

11 Controlled Human Exposure Studies

12 As discussed in detail in the 2019 ISA (U.S. EPA, 2019, section 6.1), controlled human
13 exposure studies have reported that PM_{2.5} exposures lasting from less than one hour up to five
14 hours can impact cardiovascular function.¹⁶ The most consistent evidence from these studies is
15 for impaired vascular function (U.S. EPA, 2019, section 6.1.13.2). In addition, although less
16 consistent, the 2019 ISA notes that studies examining PM_{2.5} exposures also provide evidence for
17 increased blood pressure (U.S. EPA, 2019, section 6.1.6.3), conduction abnormalities/arrhythmia
18 (U.S. EPA, 2019, section 6.1.4.3), changes in heart rate variability (U.S. EPA, 2019, section
19 6.1.10.2), changes in hemostasis that could promote clot formation (U.S. EPA, 2019, section
20 6.1.12.2), and increases in inflammatory cells and markers (U.S. EPA, 2019, section 6.1.11.2).
21 The 2019 ISA concludes that, when taken as a whole, controlled human exposure studies
22 demonstrate that short-term exposure to PM_{2.5} may impact cardiovascular function in ways that
23 could lead to more serious outcomes (U.S. EPA, 2019, section 6.1.16). Thus, such studies can
24 provide insight into the potential for specific PM_{2.5} exposures to result in physiological changes
25 that could increase the risk of more serious effects.

26 Table 3-4 below summarizes information from the 2019 ISA and draft ISA Supplement
27 on available controlled human exposure studies that evaluate effects on markers of
28 cardiovascular function following exposures to PM_{2.5}, either as concentrated ambient particles
29 (CAP) or in unfiltered versus filtered air.¹⁷

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

¹⁷ Table 3-4 identifies controlled human exposure studies included in the 2019 ISA and draft ISA Supplement that examine the potential for PM_{2.5} exposures to alter markers of cardiovascular function and is ordered by exposure concentration. Studies that focus on specific components of PM_{2.5} (e.g., endotoxin), or studies that evaluated PM_{2.5} exposures only in the presence of an intervention (e.g., dietary intervention) or other pollutant (e.g., ozone), are not included.

1 **Table 3-4. Summary of information from PM_{2.5} controlled human exposure studies.**

Study	Population	Exposure Details (average concentration; duration)	Results
Bräuner et al., 2008	Healthy adults	10.5 µg/m ³ PM _{2.5} (unfiltered) vs below detection (filtered); 24 h	No significant effect on markers of vascular function
Hemmingsen et al., 2015a, Hemmingsen et al., 2015b	Healthy, overweight older adults	24 µg/m ³ (unfiltered) vs 3.0 µg/m ³ (filtered) Copenhagen PM; 5 h	Impaired vascular function and altered heart rate variability; no significant changes in blood pressure or markers of inflammation or oxidative stress
Wyatt et al., 2020a *	Healthy young adults (18-35)	37.8 µg/m ³ CAP vs 2.1 µg/m ³ (filtered); 4h	Increased blood inflammatory markers; Inconsistent changes in HRV
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	64 µg/m ³ CAP (lower exposure); 2 h	No significant change in blood markers of inflammation or oxidative stress
Huang et al., 2012	Healthy adults	90 µg/m ³ CAP; 2 h	No significant changes in heart rate variability
Devlin et al., 2003	Healthy older adults	99 µg/m ³ CAP ¹ ; 2 h	Decreased heart rate variability
Hazucha et al., 2013	Adult current and former smokers	109 µg/m ³ CAP; 2 h	No significant changes in markers of inflammation or coagulation
Ghio et al., 2000	Healthy young adults	120 µg/m ³ CAP; 2 h	Increased fibrinogen (coagulation)
Ghio et al., 2003	Healthy young adults	120 µg/m ³ CAP; 2 h	Increased fibrinogen; no significant effect on markers of inflammation
Urch et al., 2010	Non-asthmatic and mild asthmatic adults	140 µg/m ³ CAP (higher exposure); 2 h	Increased blood inflammatory markers
Brook et al., 2009	Healthy adults	149 µg/m ³ CAP; 2 h	Impaired vascular function, increased blood pressure; no significant change in markers of inflammation (compared to filtered air)
Ramanathan et al., 2016	Healthy adults	149 µg/m ³ CAP; 2 h	Decreased anti-oxidant/anti-inflammatory capacity when baseline capacity was low
Sivagangabalan et al., 2011	Healthy adults	150 µg/m ³ CAP; 2 h	Increase in indicator of possible arrhythmia; no significant effect on heart rate

Kusha et al., 2012	Healthy adults	154 $\mu\text{g}/\text{m}^3$ CAP; 2 h	No significant effect on indicator of possible arrhythmia
Gong et al., 2003	Adults with and without asthma	174 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Increased heart rate; No significant effect on indicators of arrhythmia, inflammation, coagulation; inconsistent effects on blood pressure
Gong et al., 2004	Older adults with and without COPD	200 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Decreased heart rate variability, increase in markers of inflammation (without COPD only); inconsistent effect on arrhythmia; no significant effect on markers of blood coagulation
Liu et al., 2015	Healthy adults	238 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increase in urinary markers of oxidative stress and vascular dysfunction; no significant effect on blood markers of oxidative stress, vascular function, or inflammation
Bellavia et al., 2013	Healthy adults	~242 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increased blood pressure
Behbod et al., 2013	Healthy adults	~250 $\mu\text{g}/\text{m}^3$ CAP; 130 min	Increase in markers of inflammation
Tong et al., 2015	Healthy older adults	253 $\mu\text{g}/\text{m}^3$ CAP; 2 h	Impaired vascular function and increased blood pressure; no significant change in markers of inflammation or coagulation
Lucking et al., 2011	Healthy young men	320 $\mu\text{g}/\text{m}^3$ (unfiltered) vs 7.2 $\mu\text{g}/\text{m}^3$ (filtered); 1 h	Impaired vascular function and increased potential for coagulation; no significant effect on blood pressure, markers of inflammation, or arterial stiffness
Vieira et al., 2016a, Vieira et al., 2016b	Healthy adults; Heart failure patients	325 $\mu\text{g}/\text{m}^3$ (unfiltered) vs 25 $\mu\text{g}/\text{m}^3$ (filtered) diesel exhaust; 21-min	Increase in marker of potential impairment in heart function, impaired vascular function (heart failure patients); no significant effect on blood pressure, heart rate or heart rate variability, markers of inflammation, markers of coagulation, or arterial stiffness
* Study newly assessed in the draft ISA Supplement			
¹ The published study reports an average CAP concentration of 41 $\mu\text{g}/\text{m}^3$, but communication with the study authors revealed an error in that reported concentration (Jenkins, 2016).			

1
2 Most of the controlled human exposure studies in Table 3-4 exposed participants to
3 average $\text{PM}_{2.5}$ concentrations at or above about 100 $\mu\text{g}/\text{m}^3$, with exposure durations typically up
4 to about two hours. Statistically significant effects on one or more indicators of cardiovascular
5 function are often, though not always, reported following 2-hour exposures to average $\text{PM}_{2.5}$
6 concentrations at and above about 120 $\mu\text{g}/\text{m}^3$, with less consistent evidence for effects following
7 exposures to concentrations lower than 120 $\mu\text{g}/\text{m}^3$. Impaired vascular function, the effect

1 identified in the 2019 ISA as the most consistent across studies (U.S. EPA, 2019, section
2 6.1.13.2), is shown following 2-hour exposures to PM_{2.5} concentrations at and above 149 µg/m³.
3 Mixed results are reported in the three studies that evaluated longer exposure durations (i.e.,
4 longer than 2 hours) and lower (i.e., near-ambient) PM_{2.5} concentrations, with significant effects
5 for some outcomes reported following 5-hour exposures to 24 µg/m³ in Hemmingsen et al.
6 (2015b), but not for other outcomes following 5-hour exposures in Hemmingsen et al. (2015a)
7 and not following 24-hour exposures to 10.5 µg/m³ in Bräuner et al. (2008). Wyatt et al. (2020a)
8 adds to this limited evidence base of controlled human exposure studies conducted at near
9 ambient concentrations. This study was a randomized double-blind crossover study in healthy
10 young participants (18-35 years, n=21) who were subject to intermittent moderate exercise and
11 found significant effects for some cardiovascular and (e.g., systematic inflammation markers,
12 cardiac repolarization, and decreased pulmonary function) following 4-hour exposures to 37.8
13 µg/m³. The higher ventilation rate and longer exposure duration in this study compared to most
14 controlled human exposure studies is roughly equivalent to a 2-hour exposure of 75-100 µg/m³
15 of PM_{2.5}. Therefore, dosimetric consideration may explain the observed changes in lung function
16 and inflammation in young healthy individuals. While this study provides evidence of some
17 effects at lower PM_{2.5} concentrations, overall there is inconsistent evidence for changes in lung
18 function and inflammation in other controlled human exposure studies evaluated in the 2019 ISA
19 (U.S. EPA, 2019, sections 5.1.7., 5.1.2.3.3, and 6.1.11.2.1; U.S. EPA, 2021a, section 3.3.1).

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

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

11 Nonetheless, we note the findings in several of these controlled human exposure studies
12 conducted at near-ambient PM_{2.5} concentrations and the potential of these studies to provide
13 some insight into what these controlled human exposure studies may indicate regarding short-
14 term exposure to peak PM_{2.5} concentrations and how those relate to ambient PM_{2.5}
15 concentrations in areas that meet the primary PM_{2.5} standards. As such, we focus on 2-hour
16 exposures (the exposure window most often utilized) and consider the degree to which 2-hour
17 ambient PM_{2.5} concentrations in locations meeting the current primary standards are likely to
18 exceed the 2-hour exposure concentrations at which statistically significant effects are reported
19 in multiple studies for one or more indicators of cardiovascular function. To this end, we refer to
20 Figure 2-19 (Chapter 2, section 2.3.2.2.3), which presents the frequency distribution of 2-hour
21 average PM_{2.5} concentrations from all FEM PM_{2.5} monitors in the U.S. for 2017-2019. At sites
22 meeting the current primary PM_{2.5} standards, most 2-hour concentrations are below 10 µg/m³,
23 and almost never exceed 30 µg/m³. The extreme upper end of the distribution of 2-hour PM_{2.5}
24 concentrations is shifted higher during the warmer months (April to September, denoted by red
25 bars in Figure 2-19), generally corresponding to the period of peak wildfire frequency in the U.S.
26 At sites meeting the current primary standards, the highest 2-hour concentrations measured
27 almost never occur outside of the period of peak wildfire frequency (i.e., 99.9th percentile of 2-
28 hour concentrations is 62 µg/m³ during the warm season). Most of the sites measuring these very

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

1 high concentrations are in the northwestern U.S. and California (see Appendix A, Figure A-1),
2 where wildfires have been relatively common in recent years. When the typical fire season is
3 excluded from the analysis (blue in Figure 2-19), the extreme upper end of the distribution is
4 reduced (i.e., 99.9th percentile of 2-hour concentrations is 55 $\mu\text{g}/\text{m}^3$).¹⁹ Given these results, we
5 conclude that PM_{2.5} exposure concentrations evaluated in most of these controlled human
6 exposure studies are well-above the 2-hour ambient PM_{2.5} concentrations typically measured in
7 locations meeting the current primary standards.

8 Animal Toxicology Studies

9 The 2019 ISA relies on animal toxicology studies to support the plausibility of a wide
10 range of PM_{2.5}-related health effects. While animal toxicology studies often examine more
11 severe health outcomes and longer exposure durations than controlled human exposure studies,
12 there is uncertainty in extrapolating the effects seen in animals, and the PM_{2.5} exposures and
13 doses that cause those effects, to human populations. We consider these uncertainties when
14 evaluating what the available animal toxicology studies may indicate with regard to the current
15 primary PM_{2.5} standards.

16 Most of the animal toxicology studies assessed in the 2019 ISA have generally examined
17 short-term exposures to PM_{2.5} concentrations from 100 to >1,000 $\mu\text{g}/\text{m}^3$ and long-term exposures
18 to concentrations from 66 to >400 $\mu\text{g}/\text{m}^3$ (e.g., see U.S. EPA, 2019, Table 1-2). Two exceptions
19 are a study reporting impaired lung development following long-term exposures (i.e., 24 hours
20 per day for several months prenatally and postnatally) to an average PM_{2.5} concentration of 16.8
21 $\mu\text{g}/\text{m}^3$ (Mauad et al., 2008) and a study reporting increased carcinogenic potential following
22 long-term exposures (i.e., 2 months) to an average PM_{2.5} concentration of 17.7 $\mu\text{g}/\text{m}^3$ (Cangerana
23 Pereira et al., 2011). These two studies demonstrate serious effects following long-term
24 exposures to PM_{2.5} concentrations similar to the ambient concentrations reported in some PM_{2.5}
25 epidemiologic studies (U.S. EPA, 2019, Table 1-2), though still above the ambient
26 concentrations likely to occur in areas meeting the current primary standards. However, noting
27 uncertainty in extrapolating the effects seen in animals, and the PM_{2.5} exposures and doses that
28 cause those effects to human populations, animal toxicology studies are of limited utility in
29 informing decisions on the public health protection provided by the current or alternative
30 primary PM_{2.5} standards. As such, the animal toxicological studies are most useful in providing
31 further evidence to support the biological mechanisms and plausibility of various adverse effects.

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

3.3.3.2 Ambient PM Concentrations in Locations of Epidemiologic Studies

As summarized in section 3.1.1 above, epidemiologic studies examining associations between daily or annual average PM_{2.5} exposures and mortality or morbidity represent a large part of the evidence base supporting several of the 2019 ISA’s “causal” and “likely to be causal” determinations and provide further support for these associations as assessed in the draft ISA Supplement. In this section, we consider the ambient PM_{2.5} concentrations present in areas where epidemiologic studies have evaluated associations with mortality or morbidity, and what such concentrations may indicate regarding the primary PM_{2.5} standards. As noted in section 3.2, the use of information from epidemiologic studies to inform conclusions on the primary PM_{2.5} standards is complicated by the fact that such studies evaluate associations between distributions of ambient PM_{2.5} and health outcomes, and do not identify the specific exposures that can lead to the reported effects. Rather, health effects can occur over the entire distribution of ambient PM_{2.5} concentrations evaluated, and epidemiologic studies do not identify a population-level threshold below which it can be concluded with confidence that PM-associated health effects do not occur (U.S. EPA, 2019, section 1.5.3). To address these issues, we consider the following question:

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

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

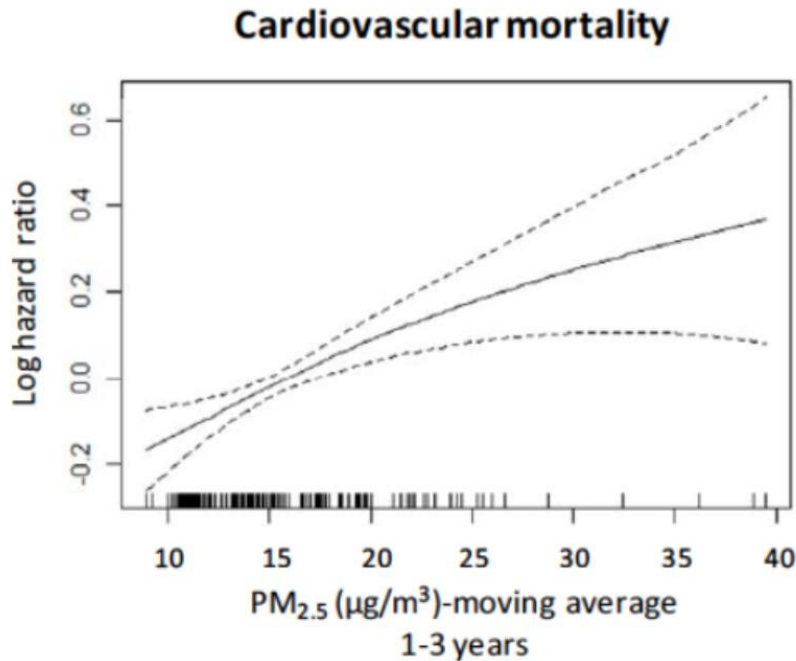
3.3.3.2.1 PM_{2.5} Air Quality Distributions Associated with Mortality or Morbidity in Key Epidemiologic Studies

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

1 observed associations for the part of the air quality distribution corresponding to where the bulk
2 of the health events in each study have been observed, generally at or around the mean
3 concentration. This is the case both for studies of daily PM_{2.5} exposures and for studies of annual
4 average PM_{2.5} exposures.

5 Studies of daily PM_{2.5} exposures examine associations between day-to-day variation in
6 PM_{2.5} concentrations and health outcomes, often over several years. While there can be
7 considerable variability in daily exposures over a multi-year study period, most of the estimated
8 exposures reflect days with ambient PM_{2.5} concentrations around the middle of the air quality
9 distributions examined (i.e., “typical” days rather than days with extremely high or extremely
10 low concentrations). Similarly, for studies of annual PM_{2.5} exposures, most of the estimated
11 exposures reflect annual average PM_{2.5} concentrations around the middle of the air quality
12 distributions examined. In both cases, epidemiologic studies provide the strongest support for
13 reported health effect associations for this middle portion of the PM_{2.5} air quality distribution,
14 which corresponds to the bulk of the underlying data, rather than the extreme upper or lower
15 ends of the distribution. Consistent with this, as noted above in section 3.3.1.1, several
16 epidemiologic studies report that associations persist in analyses that exclude the upper portions
17 of the distributions of estimated PM_{2.5} exposures, indicating that “peak” PM_{2.5} exposures are not
18 disproportionately responsible for reported health effect associations.

19 An example of the relationship between data density and reported health effect
20 associations is illustrated in Figure 3-2 below (from Lepeule et al., 2012, Figure 1 in
21 supplemental material; U.S. EPA, 2019, Figure 6-26). For the years 1974 to 2009, Lepeule et al.
22 (2012) report a positive and statistically significant association between estimated long-term
23 PM_{2.5} exposures and cardiovascular mortality in six U.S. cities. Based on a visual inspection of
24 the concentration-response function reported in this study (i.e., presented in Figure 3-2), 95%
25 confidence intervals are narrowest for long-term PM_{2.5} concentrations near the overall mean
26 concentration reported in the study (i.e., 15.9 µg/m³). Confidence intervals widen at lower and
27 higher long-term PM_{2.5} concentrations, particularly at concentrations ≤ ~10 µg/m³ and ≥ ~20
28 µg/m³. This widening in the confidence intervals is likely due in part to the comparative lack of
29 data at concentrations approaching the lower and upper ends of the air quality distribution (i.e.,
30 exposure estimates are indicated by hash marks on the horizontal axis).



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

5
 6 Similar to the information presented in Figure 3-2, other studies have also reported that
 7 confidence intervals around concentration-response functions are relatively narrow at PM_{2.5}
 8 concentrations around the overall mean concentrations reported by those studies, likely reflecting
 9 high data density in the middle portions of the distributions (e.g., Crouse et al., 2015; Villeneuve
 10 et al., 2015; Shi et al., 2016 as discussed in U.S. EPA, 2019, section 11.2.4). Thus, consistent
 11 with the approaches in the 2012 and 2020 reviews (78 FR 3161, January 15, 2013; U.S. EPA,
 12 2011, sections 2.1.3 and 2.3.4.1; 85 FR 82716-82717, December 18, 2020; U.S. EPA, 2020,
 13 sections 3.1.2 and 3.2.3), in this reconsideration, we use study-reported means (or medians) of
 14 daily and annual average PM_{2.5} concentrations over the entire study period as proxies for the
 15 middle portions of the air quality distributions, over which studies generally provide strong
 16 support for reported associations. As described further below, when considering the PM_{2.5} air
 17 quality distributions in epidemiologic studies in this section, we focus on PM_{2.5} concentrations
 18 around these overall means (including concentrations somewhat below the means (e.g., 25th and
 19 10th percentiles)).

20 In evaluating the overall study-reported means, the focus is on the form, averaging time
 21 and level of the current annual PM_{2.5} standard. Consistent with the approaches used in the 2012
 22 and 2020 reviews (78 FR 3161-3162, January 15, 2013; 85 FR 82716-82717, December 18,
 23 2020), this is because the annual standard has been utilized as the primary means of providing

1 public health protection against the bulk of the distribution of short- and long-term PM_{2.5}
2 exposures. Thus, the evaluation of the study-reported mean concentrations from key
3 epidemiologic studies lends itself best to evaluating the adequacy of the annual PM_{2.5} standard
4 (rather than the 24-hour standard with its 98th percentile form). This is true for the study-reported
5 means from both long-term and short-term epidemiologic studies, recognizing that the overall
6 mean PM_{2.5} concentrations reported in studies of short-term (24-hour) exposures reflect averages
7 across the study population and over the years of the study. Thus, mean concentrations from
8 short-term studies reflect long-term averages of 24-hour PM_{2.5} exposure estimates. In this way,
9 our examination aims to evaluate the protection provided by the annual PM_{2.5} standard against
10 the exposures that provide strong support for associations with mortality and morbidity in key
11 epidemiologic studies. We note that the protection provided by the annual standard is evaluated
12 in partnership with that provided by the 24-hour standard, with its 98th percentile form, which
13 aims to provide supplemental protection against the short-term exposures to peak PM_{2.5}
14 concentrations that can occur in areas with strong contributions from local or seasonal sources,
15 even when overall mean PM_{2.5} concentrations remain relatively low.

16 As in past reviews, application of a decision framework based on assessing means of key
17 epidemiologic studies must also consider how the study means were computed and how these
18 values compare to the annual standard metric (including the level, averaging time and form) and
19 the use of the monitor with the highest PM_{2.5} design value in an area for compliance. In the 2012
20 review, it was recognized that the key epidemiologic studies computed the study mean using an
21 average across monitor-based PM_{2.5} concentrations. As such, the Agency noted that this decision
22 framework applied an approach of using maximum monitor concentrations to determine
23 compliance with the standard, while selecting the standard level based on consideration of
24 composite monitor concentrations. Further, the Agency included analyses (Hassett-Sipple et al.,
25 2010; Frank, 2012) that examined the differences in these two metrics (i.e., maximum monitor
26 concentrations and composite monitor concentrations) across the U.S. and in areas included in
27 the key epidemiologic studies and found that the maximum design value in an area was generally
28 higher than the monitor average across that area, with that amount varying based on location and
29 concentration. This information was taken into account in the Administrator's final decision in
30 selecting a level for the primary annual PM_{2.5} standard the 2012 review and discussed more
31 specifically in her considerations on adequate margin of safety.

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

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

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

25 To evaluate the PM_{2.5} air quality distributions in key studies in this draft PA
26 reconsideration, we first identify the epidemiologic studies assessed in the 2019 ISA and draft
27 ISA Supplement that have the potential to be most informative in reaching preliminary
28 conclusions on the primary PM_{2.5} standards. As with the experimental studies discussed above,
29 we focus on epidemiologic studies that provide strong support for “causal” or “likely to be
30 causal” relationships with PM_{2.5} exposures in the 2019 ISA. We focus on the health effect
31 associations that are determined in the 2019 ISA and draft ISA Supplement to be consistent
32 across studies, coherent with the broader body of evidence (e.g., including animal and controlled
33 human exposure studies), and robust to potential confounding by co-occurring pollutants and
34 other factors. We emphasize multicity/multistate studies that examine health effect associations
35 in the U.S. or Canada, as such studies examine potential associations over large geographic areas
36 with diverse atmospheric conditions and population demographics. Additionally, studies

1 examining associations outside the U.S. or Canada reflect air quality and exposure patterns that
2 may be less typical of the U.S., and thus less likely to be informative for purposes of reviewing
3 the NAAQS.²⁰ We note that, while we consider studies from Canada in our evaluation of the
4 epidemiologic evidence, there are considerable differences between studies conducted in the
5 U.S. and in Canada, particularly those related to population densities, PM_{2.5} concentration
6 gradients, and source distributions in the two countries. As a result, while we consider the
7 information from studies conducted in Canada, we generally place a greater emphasis on U.S.-
8 based studies.

9 Figure 3-3 to Figure 3-6 below summarize information from U.S. and Canadian studies
10 that are assessed in the 2019 ISA and draft ISA Supplement and that meet these criteria. For each
11 study, Figure 3-3 to Figure 3-6 present the cohort and/or geographic area examined, the approach
12 used to estimate PM_{2.5} exposures (i.e., monitored or predicted with hybrid modeling methods²¹),
13 the study years during which health events occurred, the years of PM_{2.5} air quality data used to
14 estimate exposures, and the effect estimate²² with 95% confidence intervals (per 5 µg/m³ for
15 long-term exposures; 10 µg/m³ for short-term exposures). When available, these figures also
16 include the overall means (or medians if means are not available) of the short- or long-term
17 PM_{2.5} exposure estimates reported by the study. Figure 3-3 and Figure 3-4 summarize
18 information from studies of long-term PM_{2.5} exposures. Figure 3-5 and Figure 3-6 summarize
19 information from studies of short-term PM_{2.5} exposures.

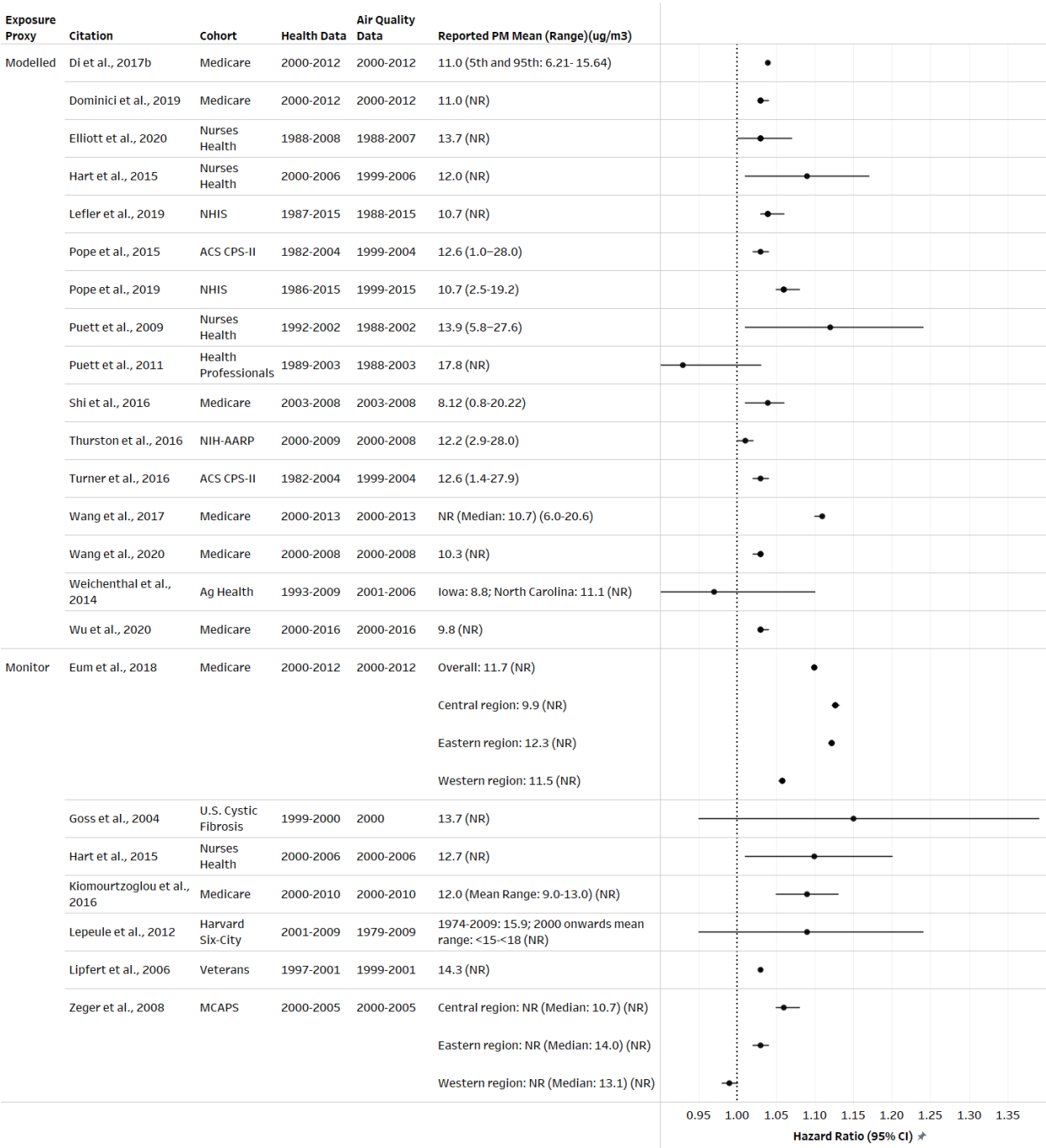
20
21
22

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

²¹ As discussed further below, and in Chapter 2, hybrid methods incorporate data from several sources, often including satellites and models, in addition to ground-based monitors.

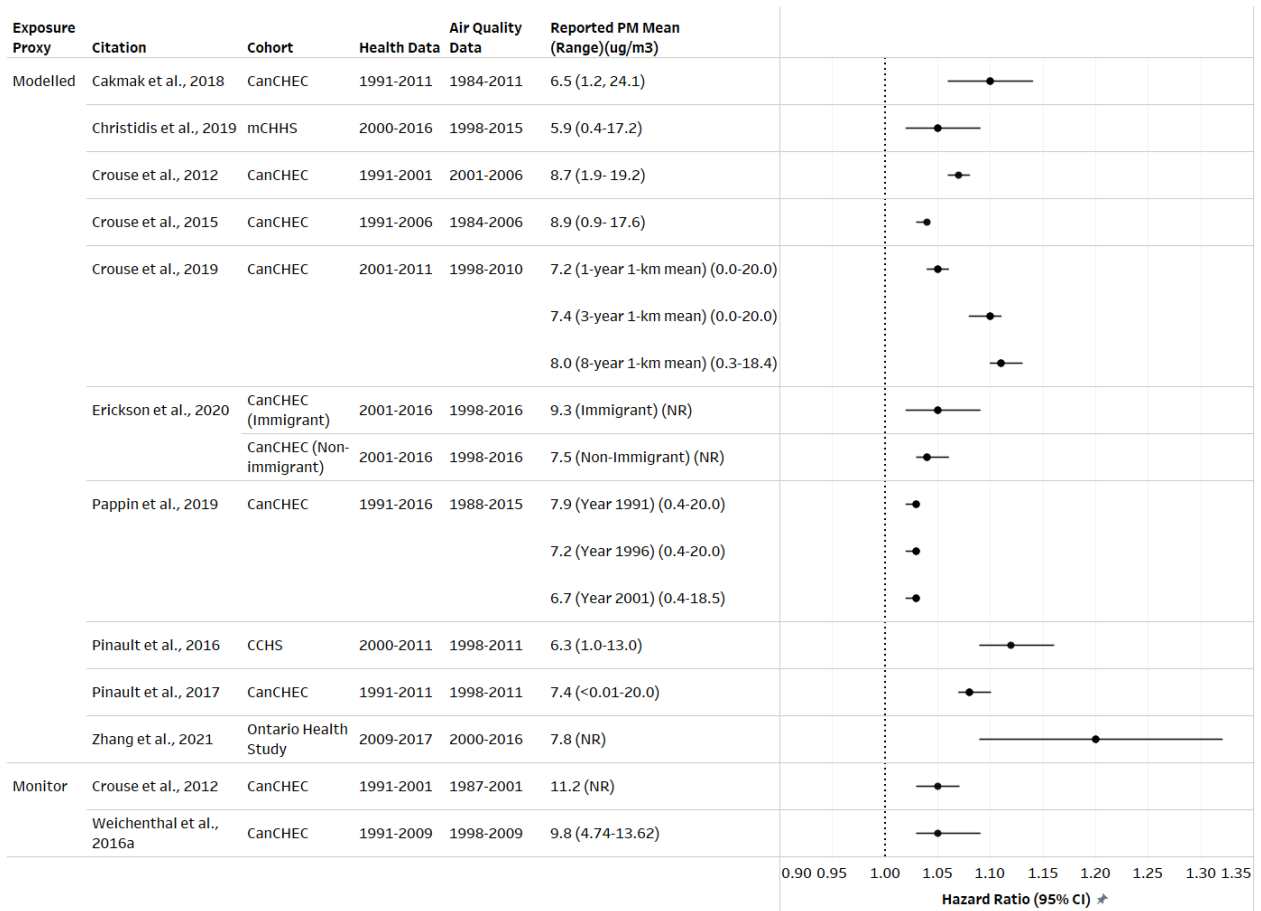
²² The effect estimates presented in the forest plot figures (Figure 3-3 to Figure 3-6) show the associations of long- or short-term PM_{2.5} exposures with health endpoints presented either as hazard ratio or odds ratio or relative risk (for which the bold dotted vertical line is at 1), or as per unit or percent change (for which the bold dotted vertical line is at 0).

All-cause mortality (U.S.)



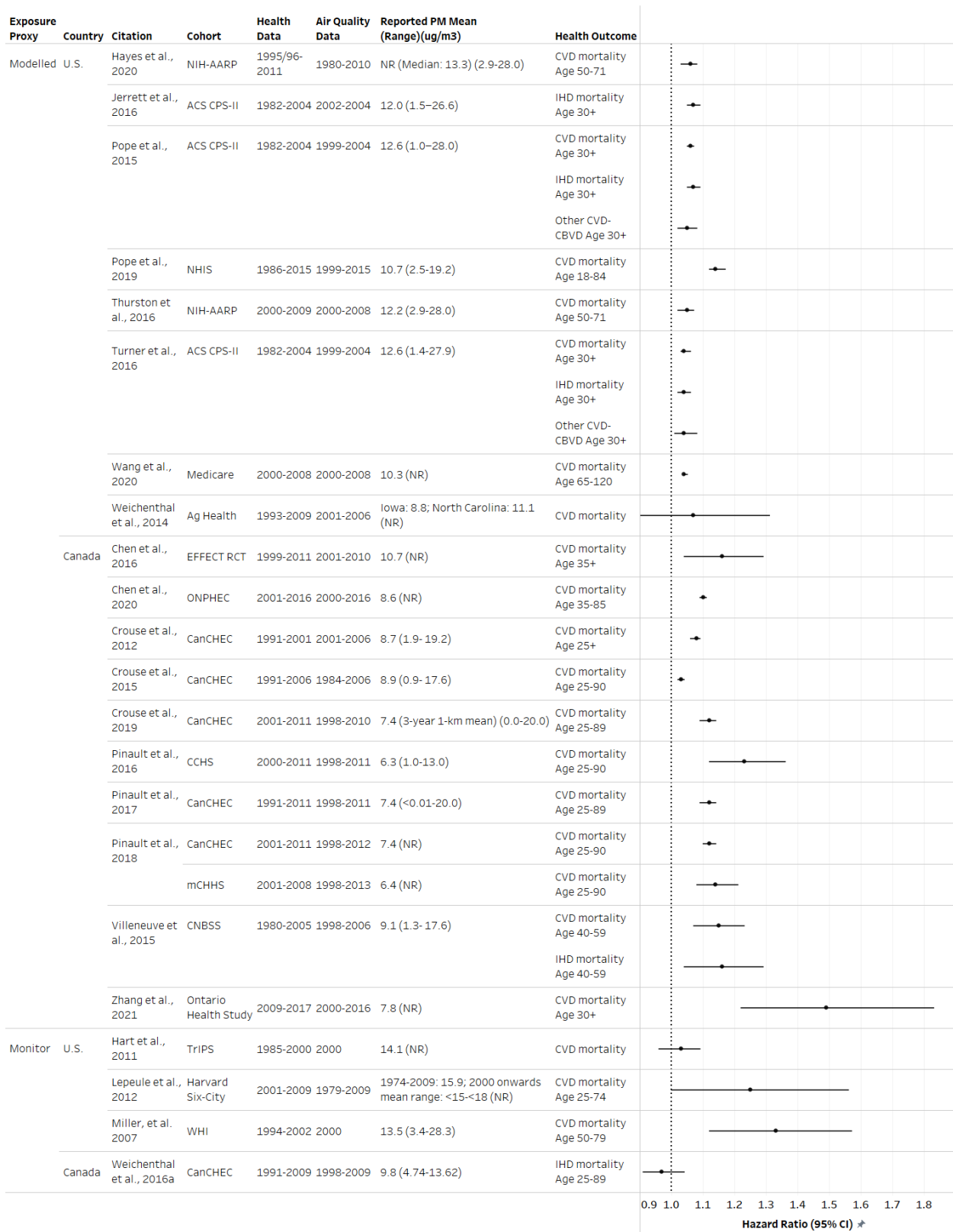
1

All-cause mortality (Canada)

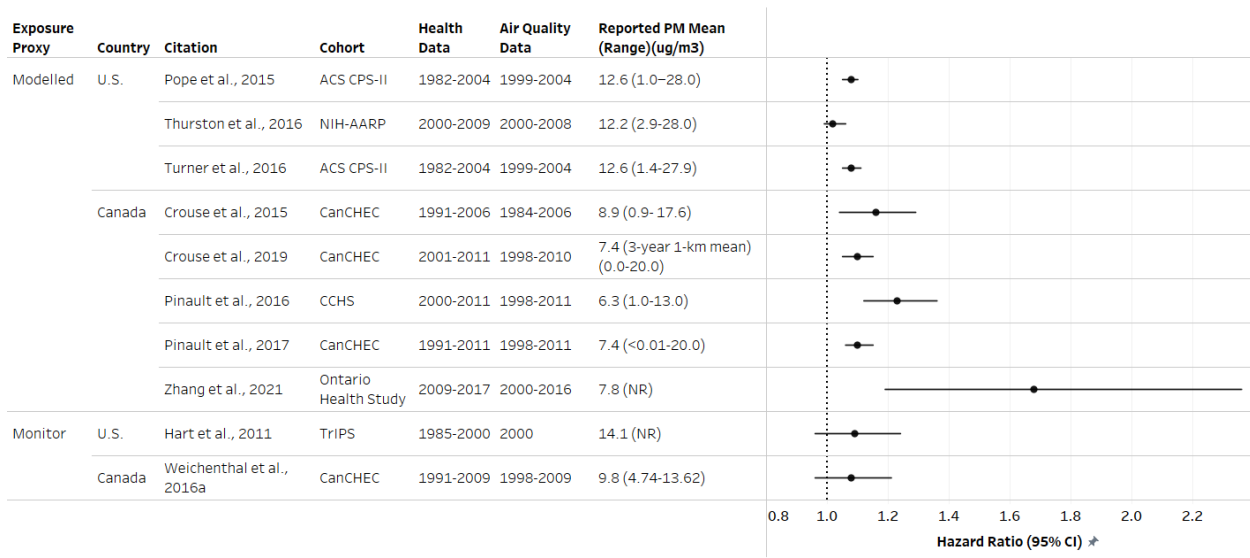


1

CVD mortality

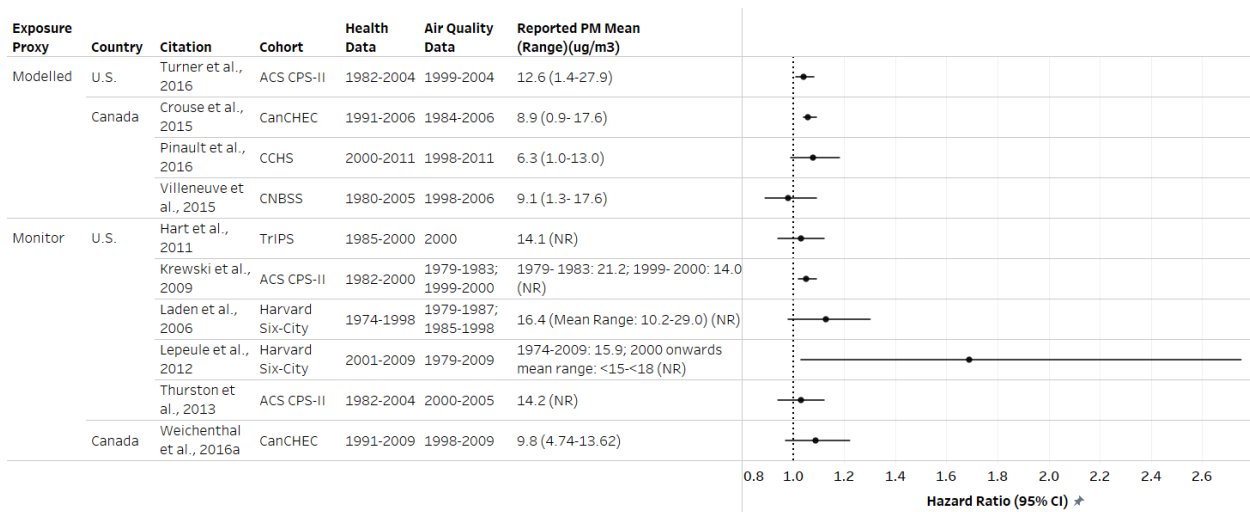


Respiratory mortality



1

Lung cancer mortality



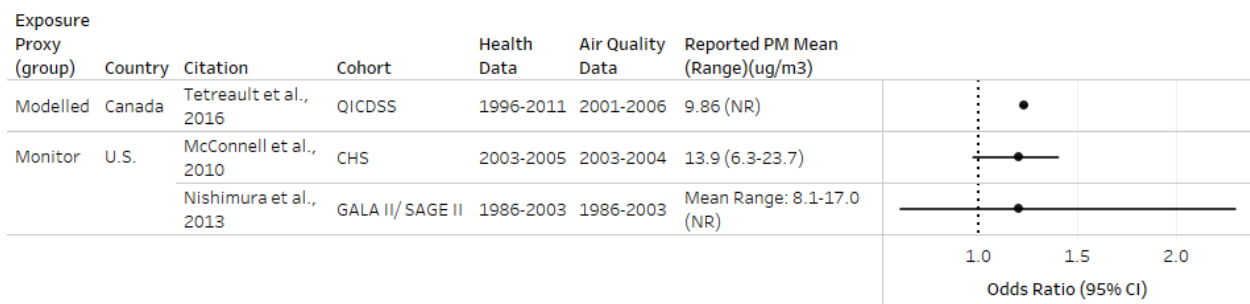
2

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

5

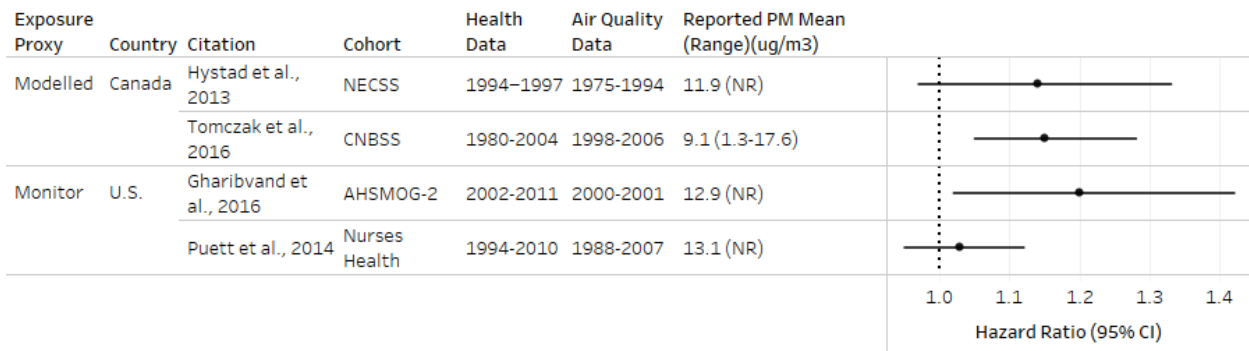
6

Asthma incidence



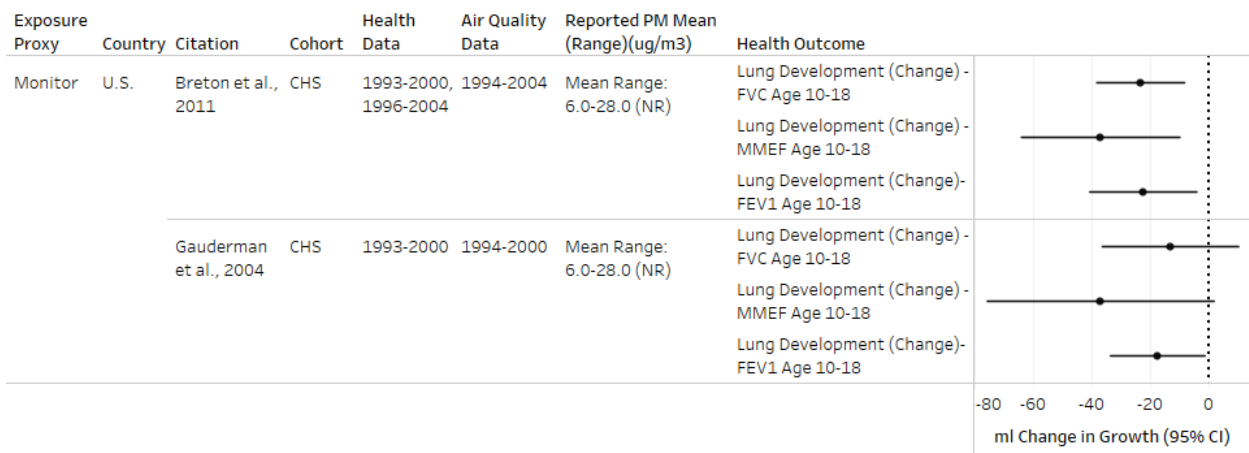
7

Lung cancer incidence



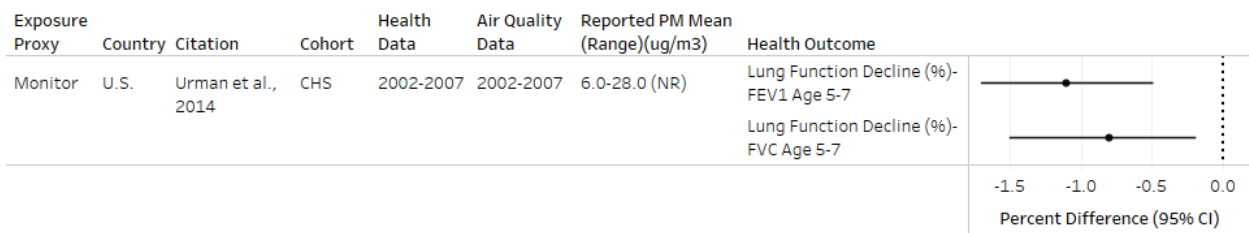
1

Lung development



2

Lung function

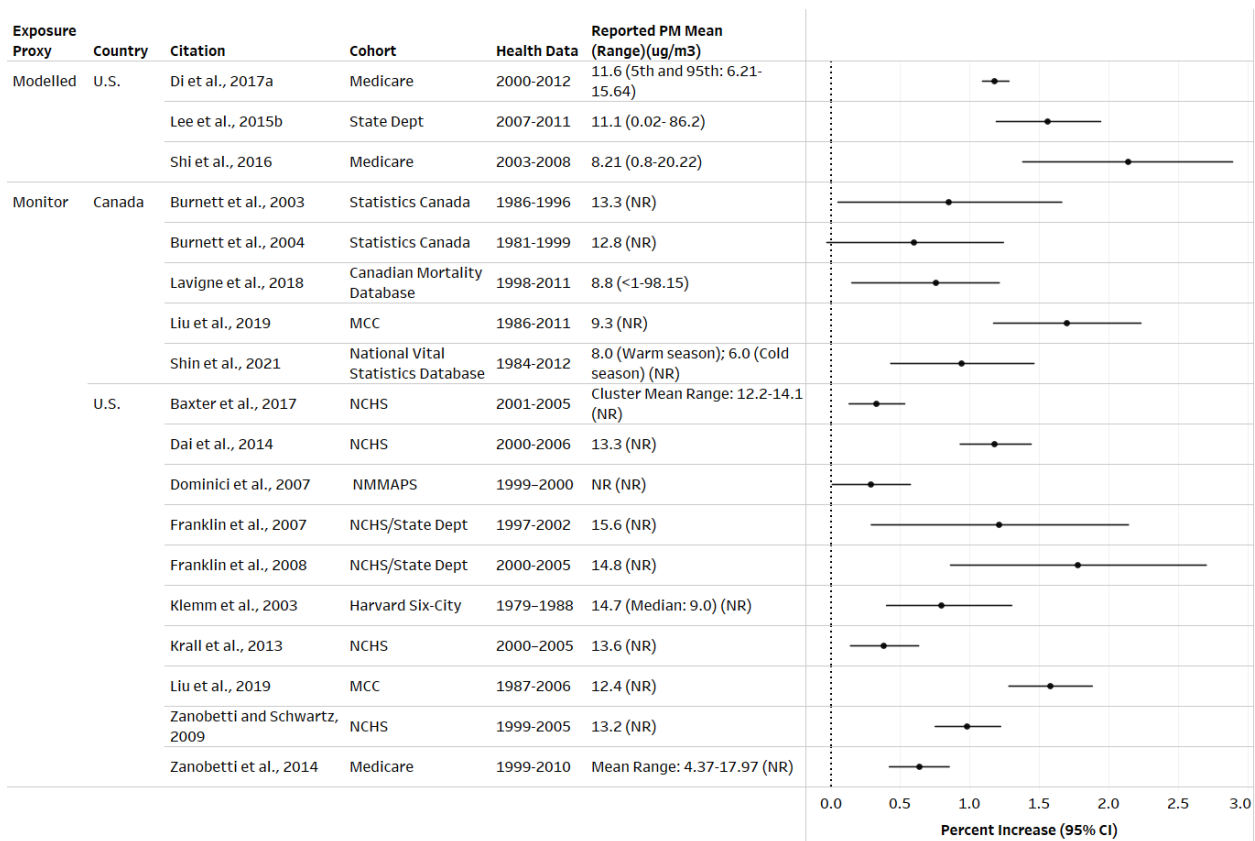


3

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

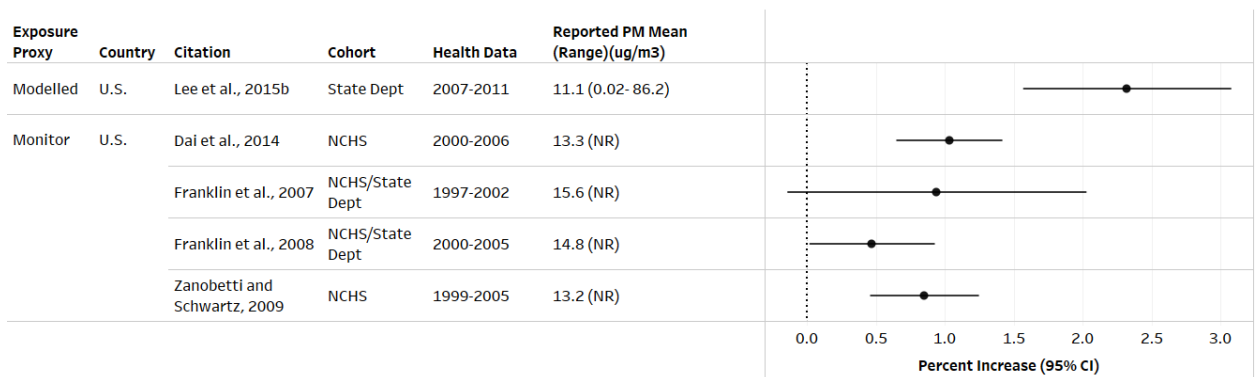
6

All-cause mortality



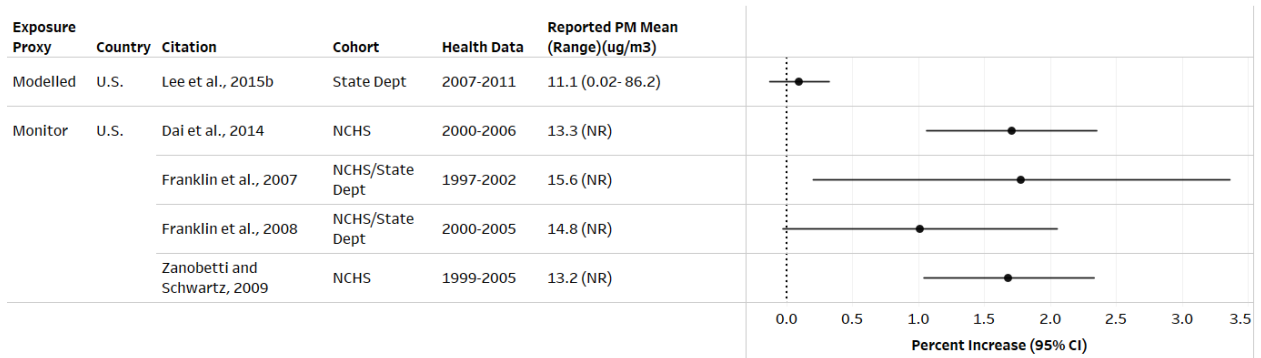
1

CVD mortality



2

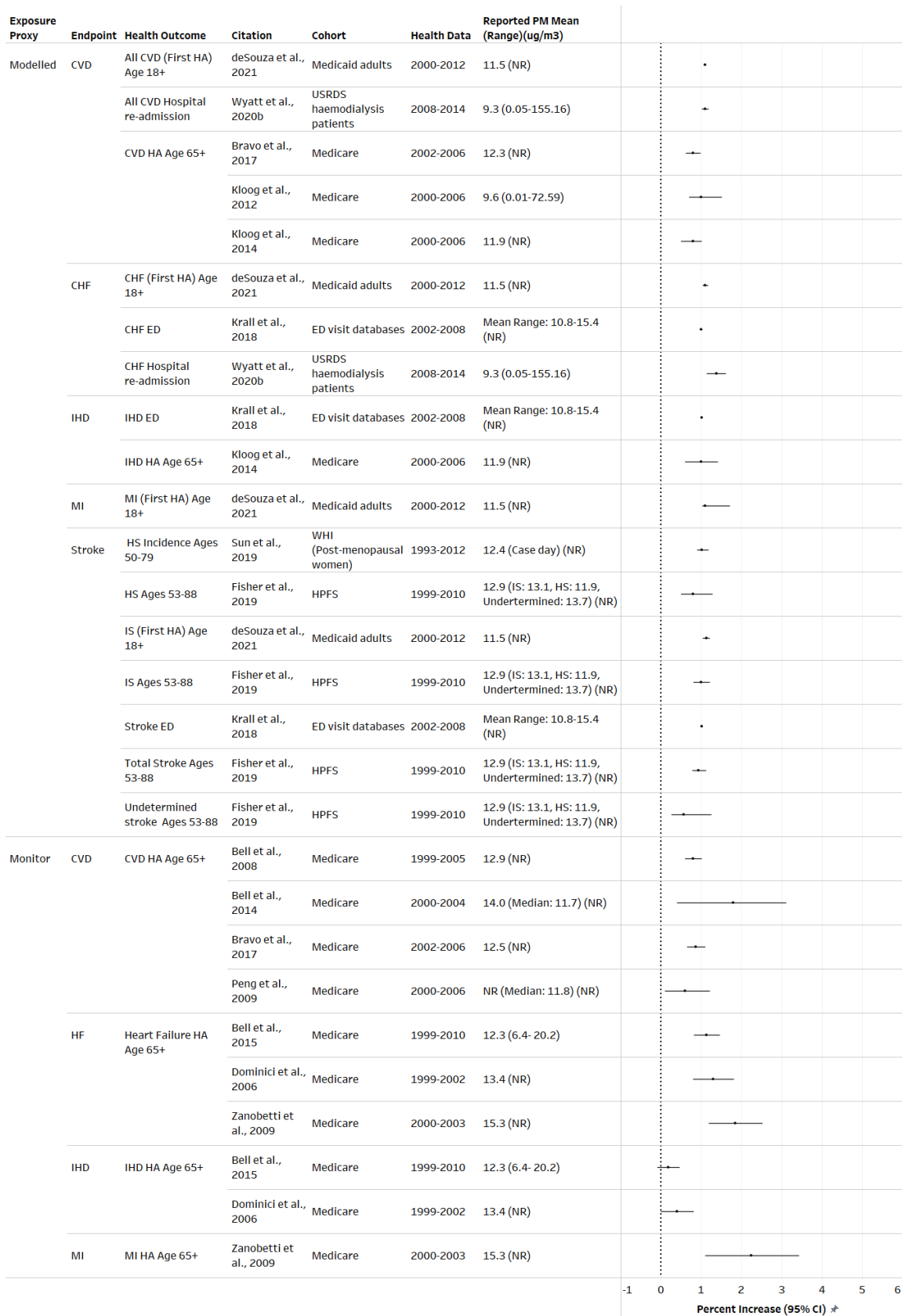
Respiratory mortality



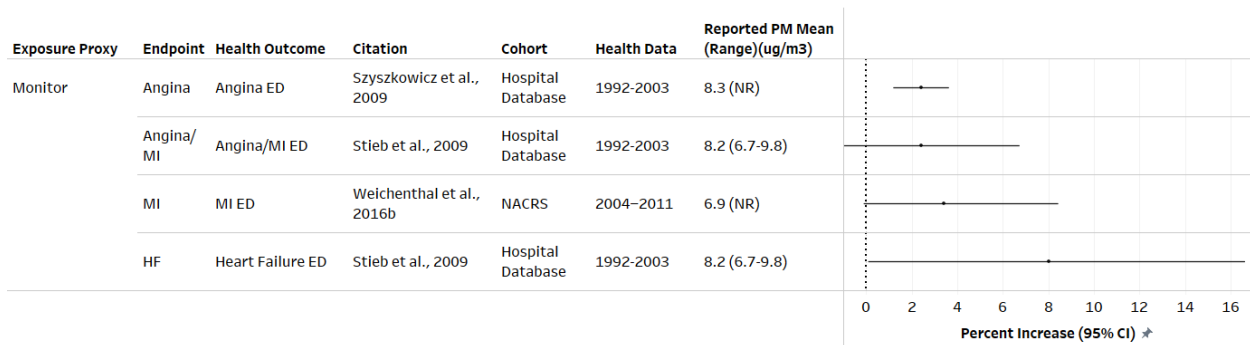
1
2 **Figure 3-5. Epidemiologic studies examining associations between short-term PM_{2.5}**
3 **exposures and mortality.**²³

²³ As noted above, the overall mean PM_{2.5} concentrations reported in studies of short-term (24-hour) exposures reflect averages across the study population and over the years of the study. Thus, mean concentrations reflect long-term averages of 24-hour PM_{2.5} exposure estimates.

CVD morbidity (U.S.)

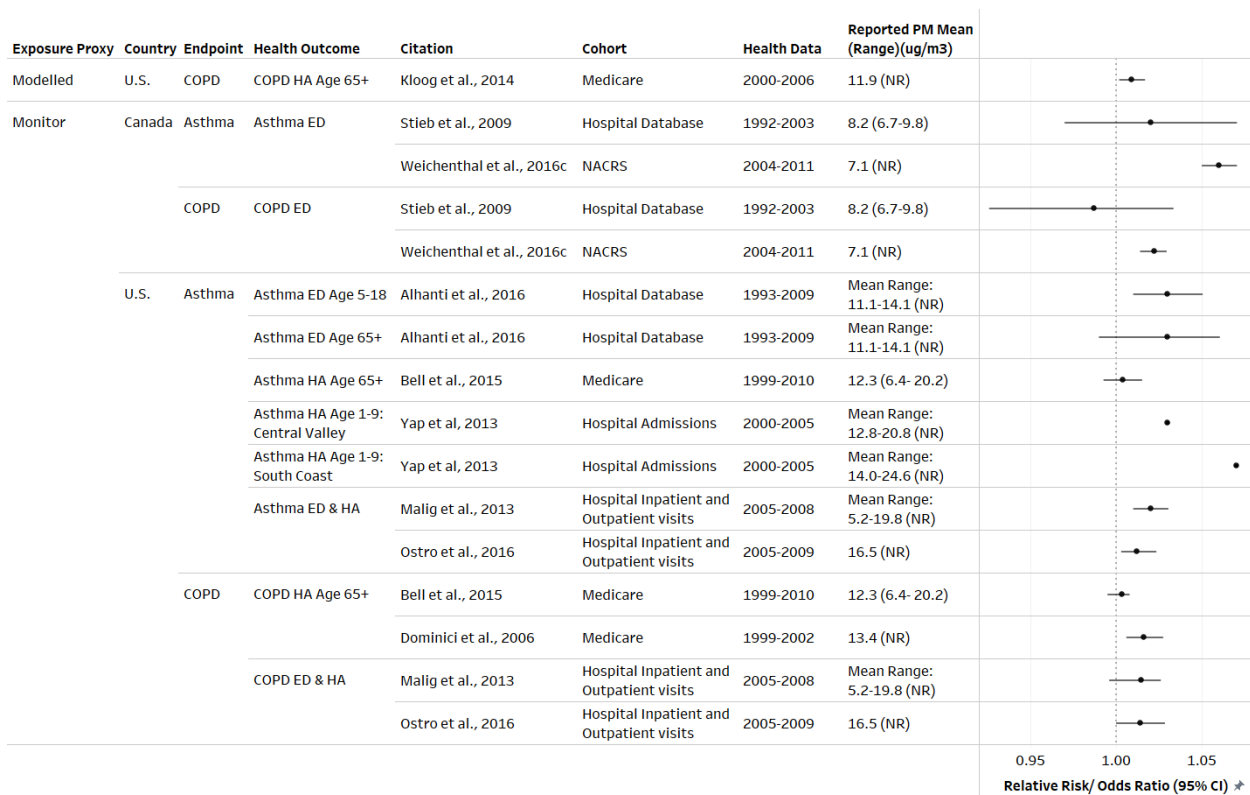


CVD morbidity (Canada)



1

Respiratory morbidity



2

3

4

5

6

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

7

8

9

10

11

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

1 Based on the information in Figure 3-3 to Figure 3-6, key epidemiologic studies indicate
2 generally positive and statistically significant associations between estimated PM_{2.5} exposures
3 (short- or long-term) and mortality or morbidity across a range of ambient PM_{2.5} concentrations.
4 Drawing from the multicity studies in Figure 3-3 to Figure 3-6, we identify the key
5 epidemiologic studies most informative to our understanding to evaluate the PM_{2.5} air quality
6 distributions in key studies in this reconsideration. Key epidemiologic studies are those that
7 report overall mean (or median) PM_{2.5} concentrations and for which the years of PM_{2.5} air quality
8 data used to estimate exposures overlap entirely with the years during which health events are
9 reported. For some studies of long-term PM_{2.5} exposures, exposure is estimated from air quality
10 data corresponding to only part of the study period, often including only the later years of the
11 health data, and are not likely to reflect the full ranges of ambient PM_{2.5} concentrations that
12 contributed to reported associations.²⁴ While this approach can be reasonable in the context of an
13 epidemiologic study that is evaluating health effect associations with long-term PM_{2.5} exposures,
14 under the assumption that spatial patterns in PM_{2.5} concentrations are not appreciably different
15 during time periods for which air quality information is not available (e.g., Chen et al., 2016),
16 our interest is in understanding the distribution of ambient PM_{2.5} concentrations that could have
17 contributed to reported health outcomes. Therefore, we identify studies as key epidemiologic
18 studies when the years of air quality data and health data overlap in their entirety.

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

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

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

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

3 While all of the key epidemiologic studies in the 2012 review relied on ground-based
4 monitoring information to characterize PM_{2.5} exposure concentrations, as at the time of the 2020
5 review, a number of the more recent epidemiologic studies in Figure 3-3 to Figure 3-6 utilized
6 various “hybrid modeling” approaches that include fusion techniques that combine ground-based
7 monitored data with air quality modeled estimates and/or information from satellites to estimate
8 PM_{2.5} exposures. Furthermore, some studies use various mathematical approaches (e.g.,
9 population weighting, trimmed mean²⁶) to compute the study-reported mean from the estimated
10 PM_{2.5} exposure concentrations. The fact that there are more and different techniques utilized to
11 characterize exposure in the key epidemiologic studies in this reconsideration highlights the
12 importance of understanding those techniques and how they compare to each other and to
13 consider how those differences translate into comparisons between the mean PM_{2.5}
14 concentrations reported in the studies and the level of the primary annual PM_{2.5} standard.

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

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

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

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

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

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

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

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

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

1
2
3

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

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

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

1
2
3
4
5
6

1 In the studies that use hybrid modeling approaches to estimate long- or short-term PM_{2.5}
2 concentrations, data can be incorporated from several different sources, including satellites and
3 air quality models, in addition to ground-based monitors, as described in section 2.3.3 above.
4 Compared to ground-based monitors alone, hybrid modeling methods have the potential to
5 improve the characterization of PM_{2.5} concentrations in areas with relatively sparse monitoring
6 networks. These approaches also tend to have lower study-reported mean PM_{2.5} concentrations
7 since they often include estimates of PM_{2.5} concentrations in less populated areas compared to
8 those methods using only ground-based monitored. Studies that use hybrid modeling approaches
9 can estimate PM_{2.5} concentrations at different spatial resolutions, including at 1 km x 1 km grid
10 cells (i.e., Di et al., 2017b and Di et al., 2017a), at 10 km x 10 km grid cells (i.e., Kloog et al.,
11 2014), or at the census tract level (i.e., Bravo et al., 2017). Estimated PM_{2.5} concentrations are
12 then generally averaged up to a larger spatial resolution that corresponds to the spatial resolution
13 for which health data exists (e.g., ZIP code level). These values are then averaged across all
14 study locations at the larger spatial resolution (e.g., averaged across all ZIP codes in the study)
15 over the study period, resulting in the study-reported mean 24-hour average or annual average
16 PM_{2.5} concentration. Table 3-7 and Table 3-8 list the key U.S. and Canadian epidemiologic
17 studies, respectively, that use hybrid modeling approaches to estimate exposure and give the
18 reported study mean and describes the method used to calculate the mean. Studies included in
19 these tables are those that report overall mean (or median) PM_{2.5} concentrations and for which
20 the years of PM_{2.5} air quality data used to estimate exposures overlap entirely with the years
21 during which health events are reported. In addition, studies included in Table 3-7 and Table 3-8
22 are those for which recent methods and models were used (e.g., recent versions and
23 configurations of the air quality models); studies that are fused with PM_{2.5} data from national
24 monitoring networks (i.e., FRM/FEM data); and studies that reported a thorough model
25 performance evaluation for core years of the study.

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

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

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

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

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

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

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

1

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

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

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

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

Citation ¹	Health Endpoint	Geographic Area	Study Design	Years, Model Type, and Method Used to Calculate Study-reported Mean PM _{2.5} Concentrations	Reported Mean (other percentiles) µg/m ³
Shin et al., 2019	AF and Stroke (1 st HA)	Ontario	Cohort study (ONPHEC)	<p>1998-2012</p> <p>Fused surface (AOD, GEOS-Chem & geographically weighted regression)</p> <p>Annual PM_{2.5} estimates from the postal code and assigned to study participants based on the postal code for residence were used to calculate 5-year moving average based on the location and year of follow-up. The average PM_{2.5} concentrations were then used to calculate overall mean PM_{2.5} concentration for the study period.</p>	9.8 (25 th : 8)
Zhang et al., 2021	Non-accidental, CVD, and respiratory mortality	Ontario	Cohort study (Ontario Health Study)	<p>Modeled from AOD satellite retrievals 2000-2016</p> <p>Annual PM_{2.5} estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year and 5-year moving averages based on the location and year of follow-up. The 5-year average PM_{2.5} concentrations were then used to calculate overall mean PM_{2.5} concentration for the baseline year.</p>	Baseline: 7.8 (Median: 8.0; 25 th : 6.7)
¹ None of the studies presented in this table were evaluated in the 2012 review.					

1

1 As noted above, the key epidemiologic studies use differing approaches to estimate mean
2 $PM_{2.5}$ concentrations. Approaches differ not only between monitor-based studies and model-
3 based studies, but also between studies using the same types of air quality information. It is
4 important to recognize the differences between the techniques used for estimating mean $PM_{2.5}$
5 concentrations in epidemiologic studies, in particular when comparing the results across the
6 studies and considering what the study reported means represent and how that information
7 informs our consideration of the form, averaging time and level of the current annual $PM_{2.5}$
8 standard. To further understand these differences, we seek to answer the following question:

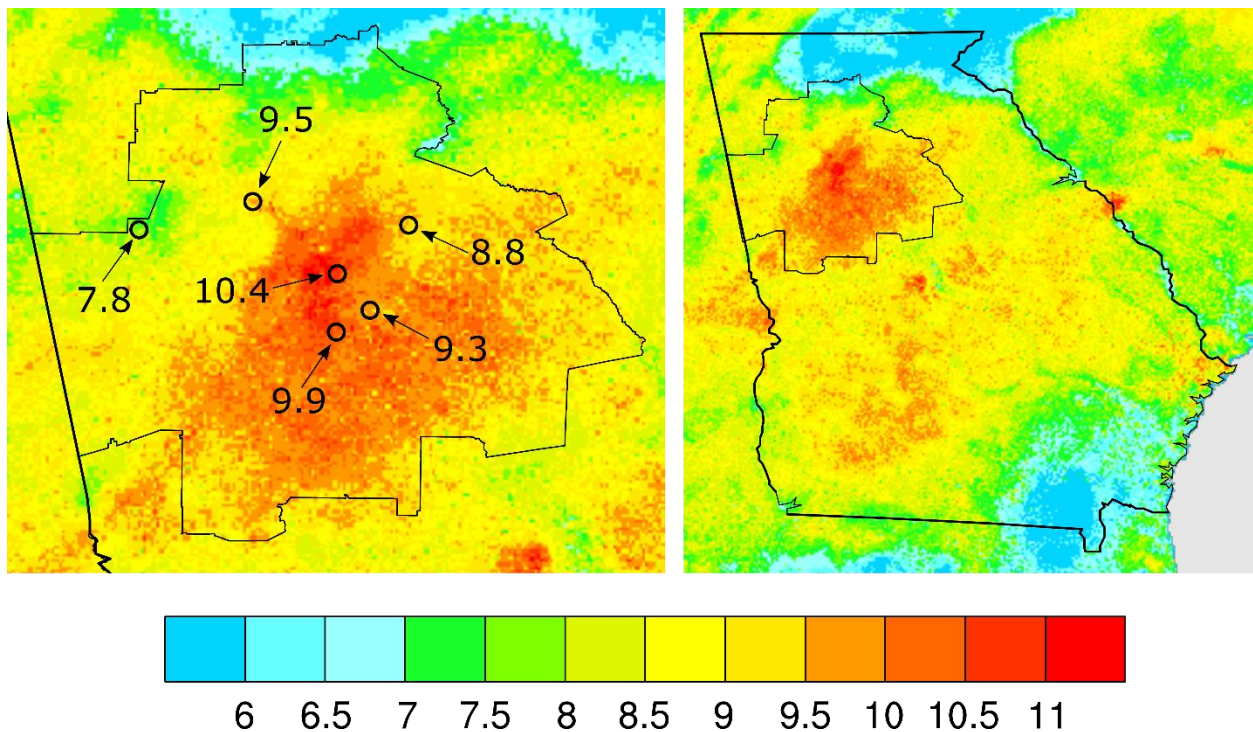
- 9 • **How can the approaches used in key epidemiologic studies to estimate exposure**
10 **affect the study-reported mean $PM_{2.5}$ concentrations? How do these approaches and**
11 **the resulting means compare to one another?**

12 In answering this question, we first utilize a simplified example to show differences in
13 the mean concentrations depending on the methods used to estimate exposure. In Figure 3-7
14 below, we exhibit the state of Georgia and the CBSA of Atlanta-Sandy Springs-Roswell. In this
15 Figure, the gradient of $PM_{2.5}$ concentrations are shown for 1 km x 1 km grid cells using one of
16 the hybrid approaches described in more detail in Chapter 2, referred to as the DI2019²⁷ hybrid
17 approach, from 2014-2016, as well as the monitor locations within the Atlanta-Sandy Springs-
18 Roswell CBSA and their annual $PM_{2.5}$ design values for 2016. Using these data, several metrics
19 were calculated and shown in Table 3-9 below. For all monitors within the CBSA, the average
20 $PM_{2.5}$ concentration is $9.3 \mu\text{g}/\text{m}^3$, while the design value (based on the highest monitored $PM_{2.5}$
21 concentration in the area) is $10.4 \mu\text{g}/\text{m}^3$. This comparison helps to illustrate the fact that
22 composite monitor values tend to be somewhat lower than the highest area monitor values,
23 consistent with the key points made in the 2012 review. This example also communicates how
24 monitors are sited to represent the higher concentrations within the area and that the area's
25 annual design value, which is used for compliance with the standard, is calculated based on the
26 highest monitor in the area.

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

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

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



16
 17 **Figure 3-7. Estimated $\text{PM}_{2.5}$ concentrations using the DI2019 hybrid approach and**
 18 **monitoring locations and design values for the state of Georgia and the Atlanta-Sandy**
 19 **Springs-Roswell, Georgia CBSA.** (Note: Additional information on the DI2019 hybrid
 20 approach is described in section 2.3.3.1.4 and in Di et al., 2019a.)

21

1 **Table 3-9. PM_{2.5} Concentrations Metrics from Monitor and Modeled Data**²⁸

Description of Metric	PM _{2.5} Concentrations (µg/m ³)
Atlanta highest monitor	10.4
Atlanta monitored average	9.3
Atlanta spatial average	9.2
Atlanta population-weighted average	9.6
Georgia spatial average	8.3
Georgia population-weighted average	9.1

2

3 To expand upon this example in answering our question, we look to the analyses in
 4 Chapter 2 which compared area annual design values, composite monitor PM_{2.5} concentrations
 5 and mean concentrations from two hybrid approaches. The analyses also included population-
 6 weighted mean metrics. In the air quality analyses comparing composite monitored PM_{2.5}
 7 concentrations with annual PM_{2.5} design values in U.S. CBSAs, maximum annual PM_{2.5} design
 8 values were approximately 10% to 20% higher than annual average concentrations (i.e.,
 9 averaged across multiple monitors in the same CBSA) (section 2.3.3.1, Figure 2-28 and Table 2-
 10 2). The difference between the maximum annual design value and average concentration in an
 11 area can be smaller or larger than this range, depending on factors such as the number of
 12 monitors, monitor siting characteristics, and the distribution of ambient PM_{2.5} concentrations.²⁹
 13 Such ratios may also depend on how the average concentrations are calculated (i.e., averaged
 14 across monitors versus across modeled grid cells). Compared to annual design values, Figure 2-
 15 29 indicates a more variable relationship between maximum 24-hour PM_{2.5} design values and
 16 annual average concentrations.

17 In addition, the air quality analyses in Chapter 2 looked at data from two hybrid modeling
 18 approaches. While hybrid modeling approaches are not universal and the various hybrid
 19 approaches all have their different nuances, the analysis in Chapter 2 focused on the DI2019 and
 20 HA2020 approaches, which have been used in several of the key epidemiologic studies in Table
 21 3-7 and Table 3-8. Section 2.3.3.2.4 details a comparison of PM_{2.5} fields in estimating exposure
 22 relative to design values using these two hybrid modeling surfaces. PM_{2.5} concentrations are

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

²⁹ Given that higher PM_{2.5} concentrations have been reported at some near-road monitoring sites, relative to the surrounding area (section 2.3.2.2.2), recent requirements for PM_{2.5} monitoring at near-road locations in large urban areas (section 2.2.3.3) may increase the ratios of maximum annual design values to averaged concentrations in some areas. In the Georgia example above, a near-road monitor was not included in our analysis. The near-road monitor was not added until 2015, and data related to DI2019 ended in 2016. For purposes of developing three-year average concentrations using the most recent data for which we had monitored and modeled data, 2014-2016 data was selected for monitors as well, for which data from 2014-2016 was not available for the near-road monitor.

1 estimated per year at a 1 km x 1 km spatial resolution. As exhibited in Figure 2-37, the means
2 vary when one estimates PM_{2.5} exposures in urban areas only (CBSAs) versus when the averages
3 used all or most grid cells nationwide. This is likely indicative of the fact that areas included
4 outside of CBSAs tend to be more rural and have lower estimated PM_{2.5} concentrations. This is
5 important to note since, which study area is included in the calculation of the mean (Table 3-7
6 and Table 3-8 above), and more specifically whether a study is focused on nationwide, regional,
7 or urban areas, will affect the calculation of the study mean based on how many rural areas are
8 included with lower estimated PM_{2.5} concentrations. While the determination of what spatial
9 scale to use to estimate PM_{2.5} concentrations does not inherently affect the quality of the
10 epidemiologic study, the spatial scale can affect the calculation of the long-term mean
11 concentration across the study area and period. As exhibited in Table 2-4, regardless of the
12 hybrid modeling approach assessed, the annual average PM_{2.5} concentrations in CBSA-only
13 analyses are 4-8% higher than for nationwide analyses, likely as a result of higher PM_{2.5}
14 concentrations in more densely populated areas. When evaluating comparisons between surfaces
15 that estimate exposure using population-weighting versus surfaces that do not calculate means
16 using population-weighting, surfaces that calculate long-term mean PM_{2.5} concentrations with
17 population-weighted averages have higher average annual PM_{2.5} concentrations, ranging from
18 8.2-10.2 µg/m³, compared to annual PM_{2.5} concentrations that range from 7.0-8.6 µg/m³ in
19 analyses that do not apply population weighting. Average maximum annual design values, on the
20 other hand, exhibit a range from 9.5 to 11.7 µg/m³. Analyses exhibit that average maximum
21 annual design values are 40 to 50% higher when compared to annual average PM_{2.5}
22 concentrations estimated without population-weighting and are 15% to 18% higher when
23 compared to average annual PM_{2.5} concentrations with population weighting applied.

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

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

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

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

21 To further expand our evaluation of study-reported mean PM_{2.5} concentrations, we
22 specifically consider the following questions:

- 23 • **What are the overall mean PM_{2.5} concentrations reported by key epidemiologic**
24 **studies? For studies with available information on the broader distributions of**
25 **exposure estimates and/or health events, what are the PM_{2.5} concentrations**
26 **corresponding to the lower percentiles of those data (e.g., 25th and/or 10th)?**

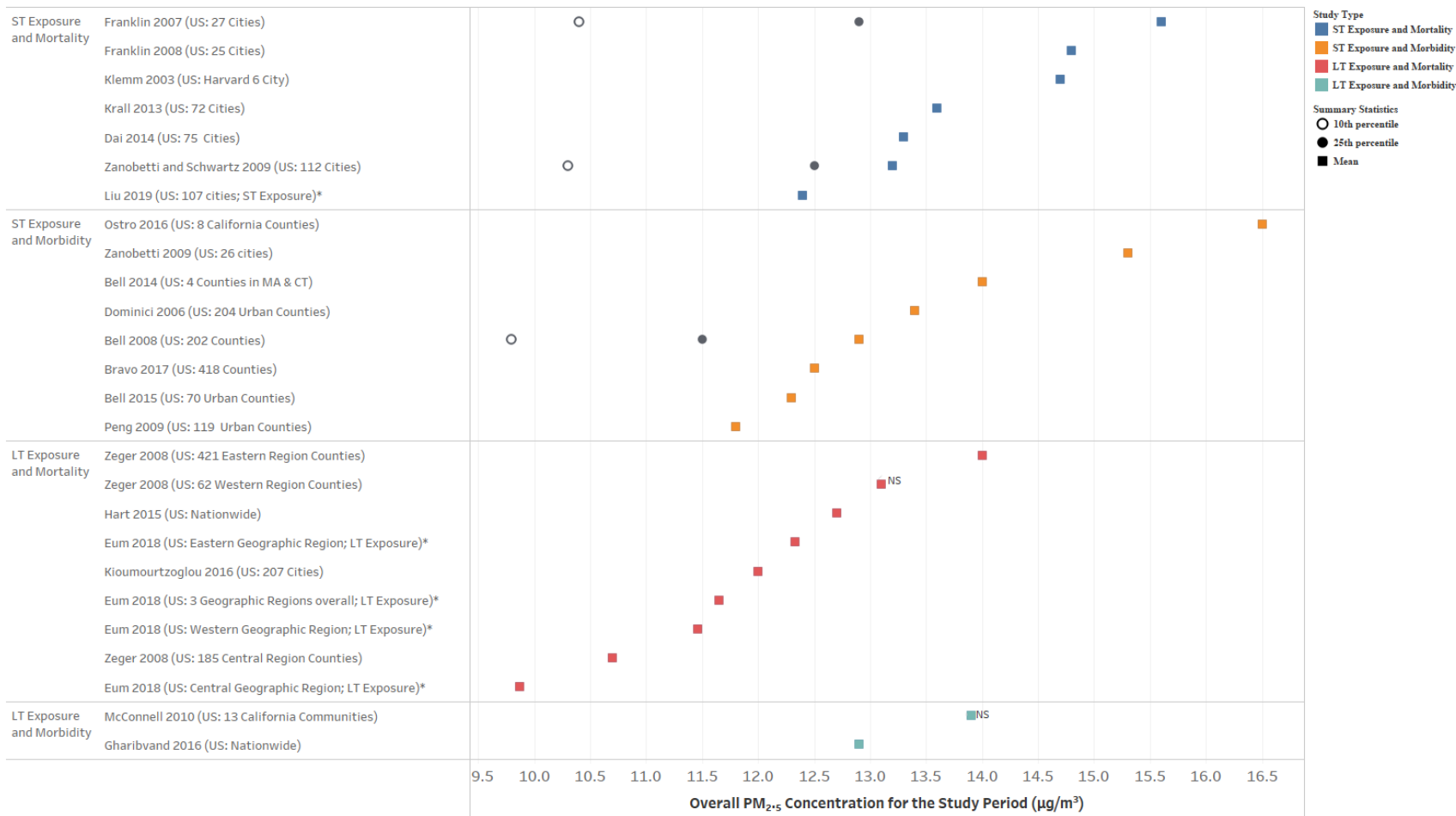
27 Figure 3-8 and Figure 3-9 highlight the overall mean (or median) PM_{2.5} concentrations
28 reported in key U.S. and Canadian studies, respectively, that use ground-based monitors alone to
29 estimate long- or short-term PM_{2.5} exposures. For the small subset of studies with available
30 information on the broader distributions of underlying data, Figure 3-8 and Figure 3-9 also
31 identify the study-period mean PM_{2.5} concentrations corresponding to the 25th and 10th
32 percentiles of health events³⁰ (see Appendix B, Section B.2 for more information).

³⁰ That is, 25% of the total health events occurred in study locations with mean PM_{2.5} concentrations (i.e., averaged over the study period) below the 25th percentiles identified in Figure 3-8 and Figure 3-9 and 10% of the total health events occurred in study locations with mean PM_{2.5} concentrations below the 10th percentiles identified.

1 Figure 3-10 and Figure 3-11 present overall means of predicted PM_{2.5} concentrations for
2 key U.S. and Canadian model-based epidemiologic studies, respectively, and the concentrations
3 corresponding to the 25th and 10th percentiles of estimated exposures or health events³¹ when
4 available (see Appendix B, section B.3 for additional information).

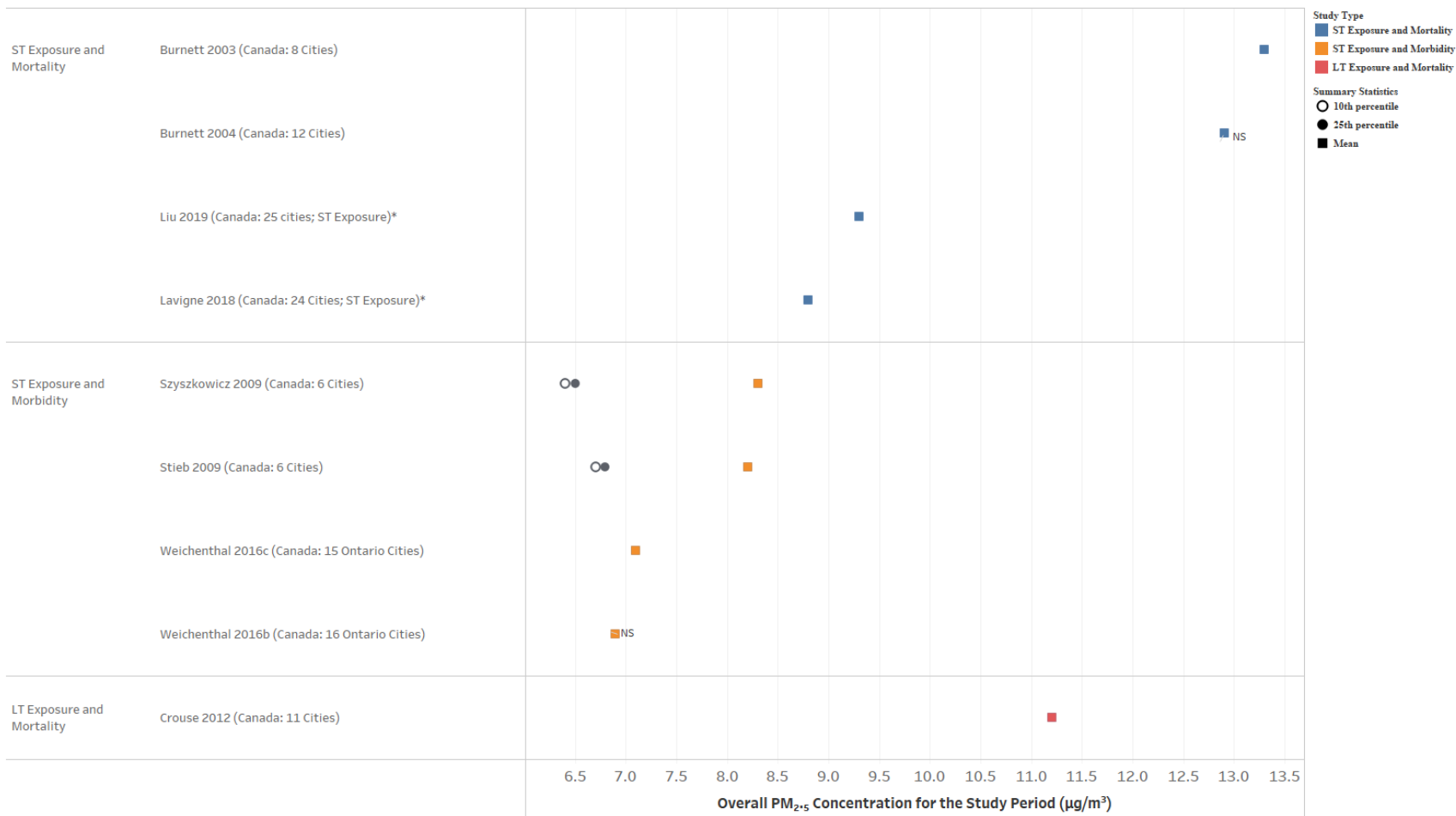
³¹ For most studies in Figure 3-10 and Figure 3-11, 25th percentiles of exposure estimates are presented. The exception is Di et al., 2017b, for which Figure 3-10 presents the short-term PM_{2.5} exposure estimates corresponding to the 25th and 10th percentiles of deaths in the study population (i.e., 25% and 10% of deaths occurred at concentrations below these concentrations). In addition, the authors of Di et al., 2017b provided population-weighted exposure values (Chan, 2019). The 10th and 25th percentiles of these population-weighted exposure estimates are 7.9 and 9.5 µg/m³, respectively.

1

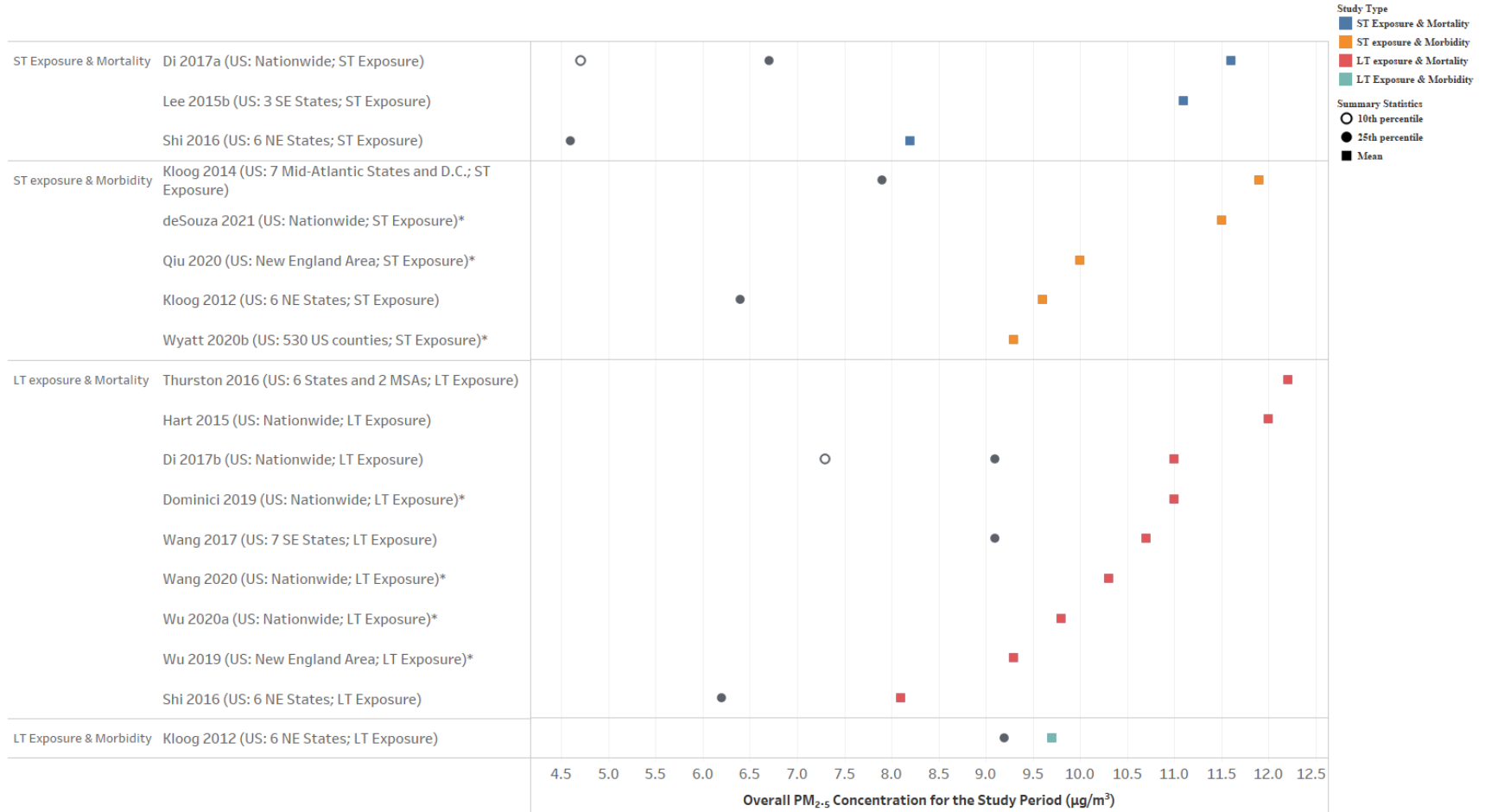


2
3
4
5

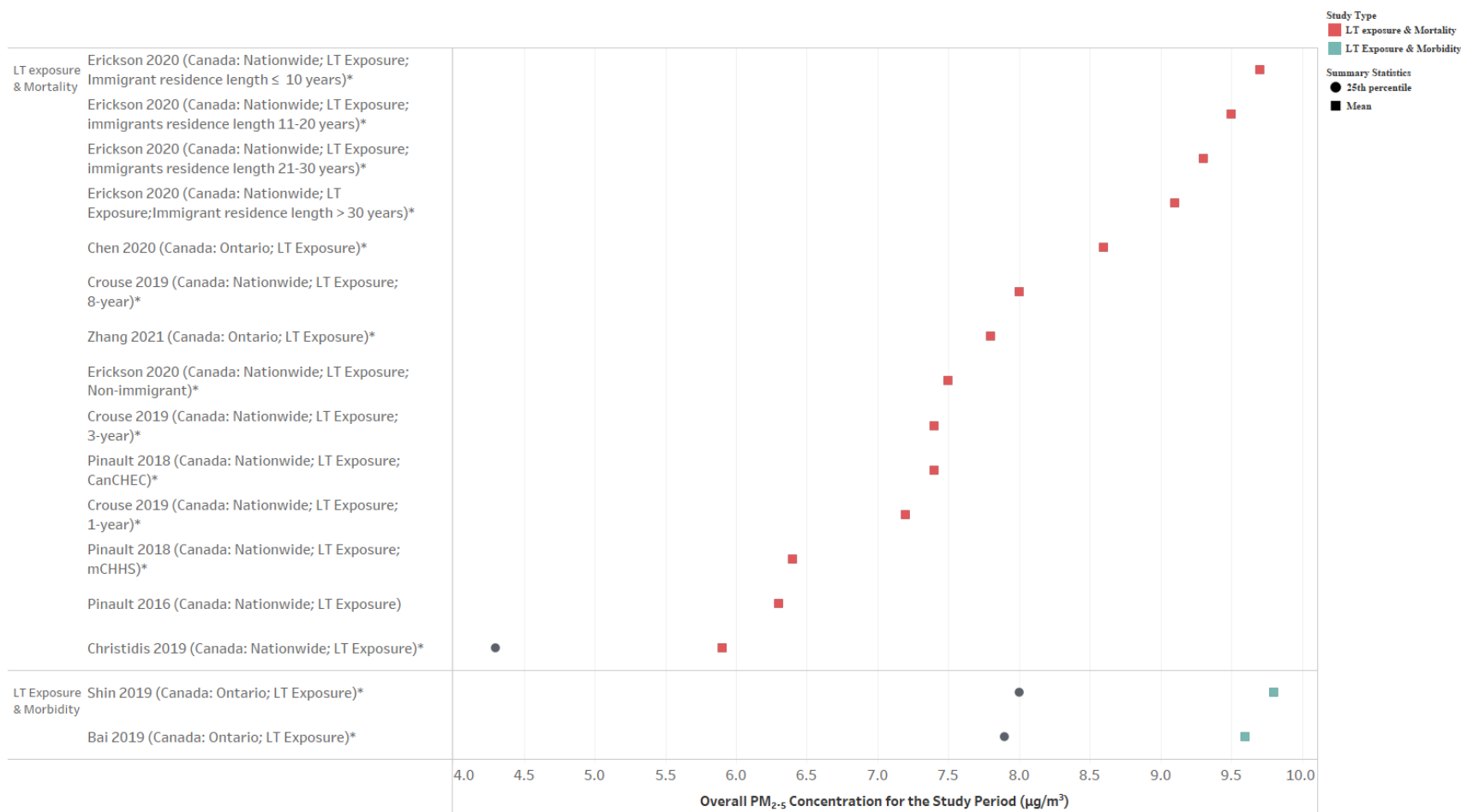
Figure 3-8. Monitor-based PM_{2.5} concentrations in key U.S. epidemiologic studies. (Asterisks denote studies included in the draft ISA Supplement).



1
2 **Figure 3-9. Monitor-based PM_{2.5} concentrations in key Canadian epidemiologic studies.** (Asterisks denote studies included in the
3 draft ISA Supplement).



1
 2 **Figure 3-10. Hybrid model-predicted PM_{2.5} concentrations in key U.S. epidemiologic studies.** (Asterisks denote studies included
 3 in the draft ISA Supplement).



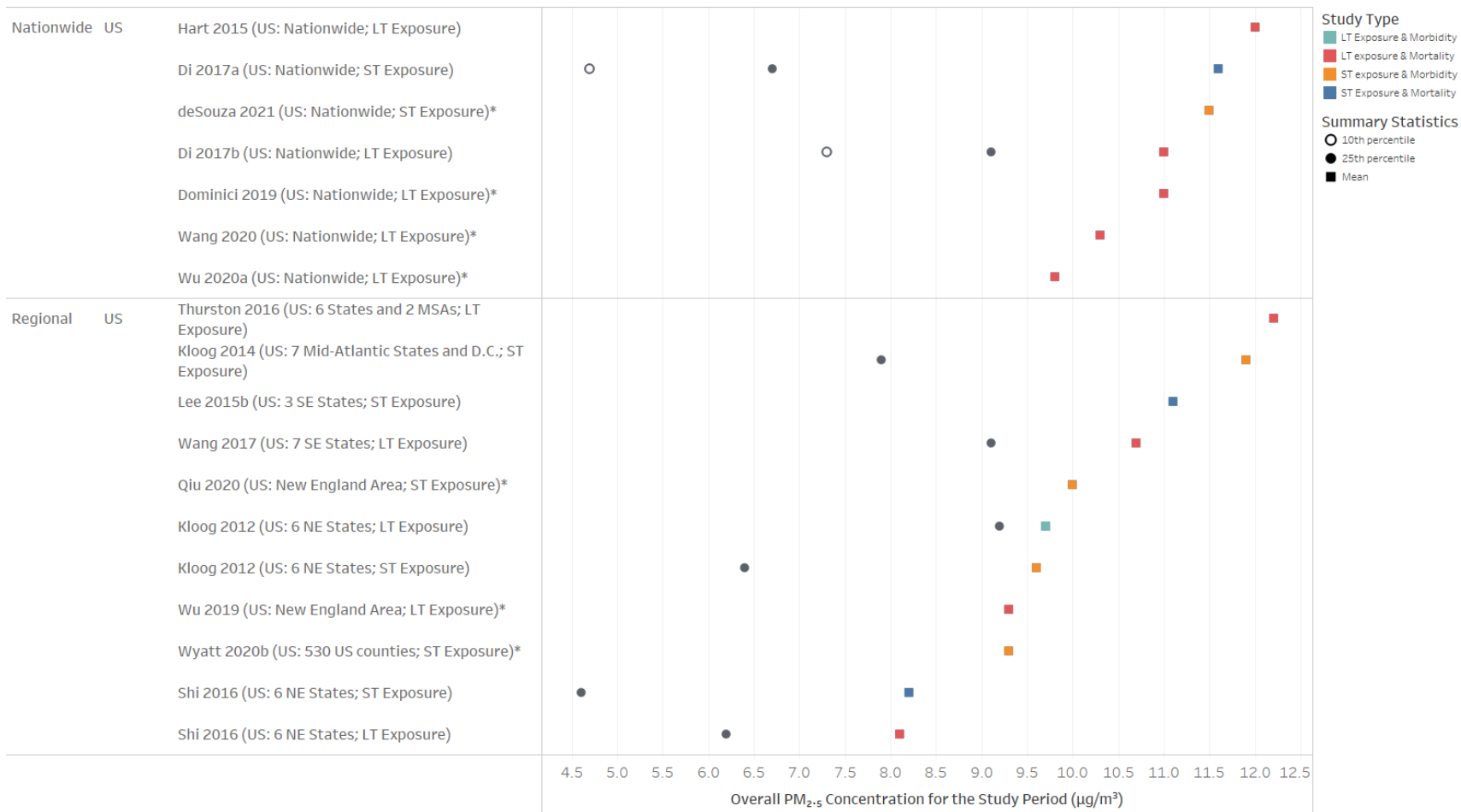
1
 2 **Figure 3-11. Hybrid model-predicted PM_{2.5} concentrations in key Canadian epidemiologic studies.** (Asterisks denote studies
 3 included in the draft ISA Supplement).

1 In further examining these data, we also ask:

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

5 Figure 3-12 and Figure 3-13 present the same key model-based epidemiologic studies
6 from the figures above but focus on the U.S. studies and group them based on their approach to
7 calculating the study-reported mean. For Figure 3-12, the studies are grouped by the
8 geographical spatial scale at which the modeling was conducted (i.e., nationwide, regional,
9 rural). Figure 3-13 presents the same key U.S. model-based epidemiologic studies, but subset by
10 the method used to average grid cells in study-reported long-term mean PM_{2.5} concentrations.
11 For the key U.S. model-based epidemiologic studies, the various methods include the average of
12 all grid cells; grid cells averaged up to ZIP code, postal code or census tract; or population-
13 weighted grid cell averaged up to ZIP code or census tract. Lastly, Figure 3-14 subsets the key
14 U.S. epidemiologic studies that used hybrid exposure models by both spatial scale and the
15 method used to average grid cells in study-reported long-term mean PM_{2.5} concentrations.
16 Grouping the key epidemiologic studies in such ways allows for visual comparisons of the study-
17 reported mean PM_{2.5} concentrations across the different spatial scales and methods of averaging
18 the grid cells.
19

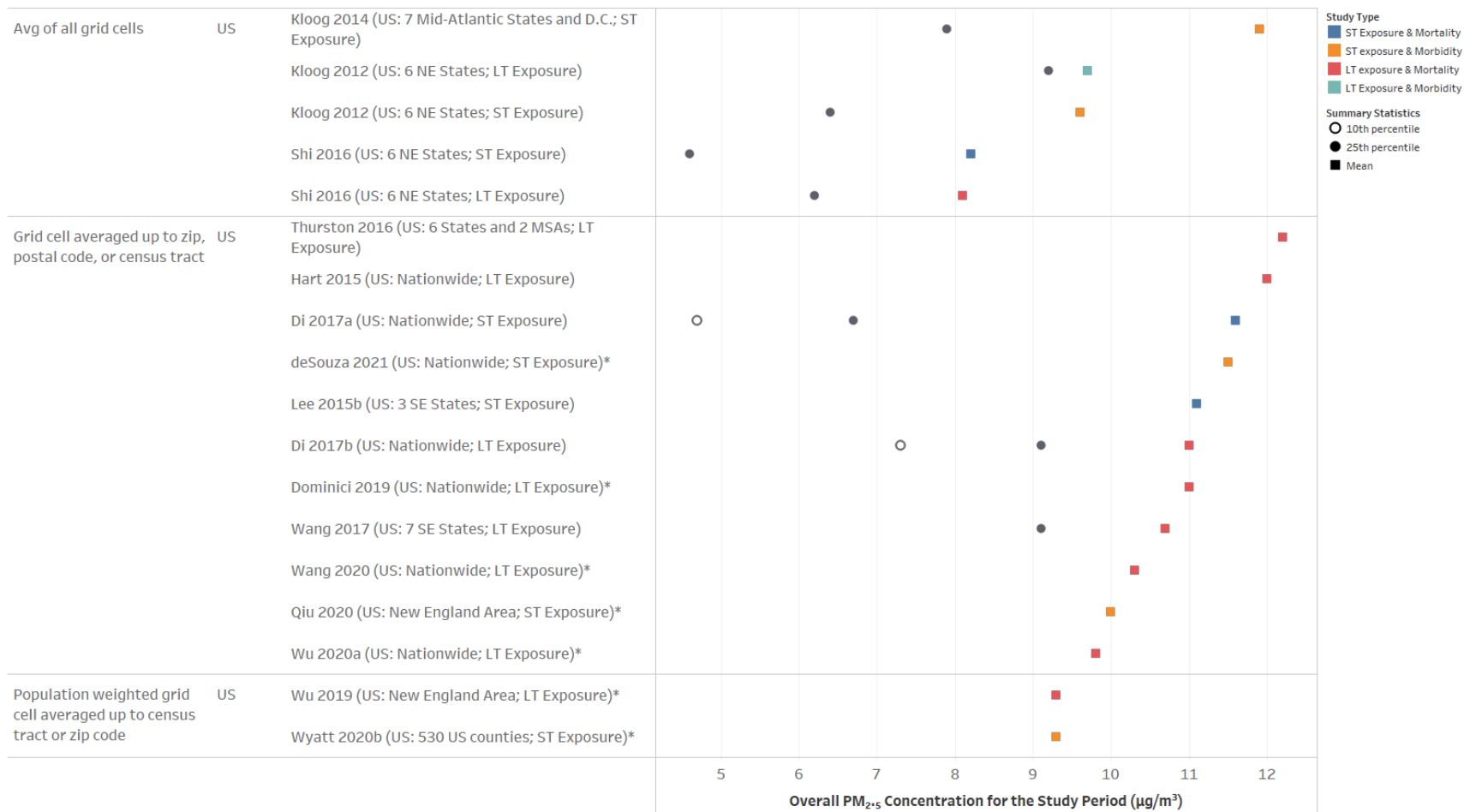
1



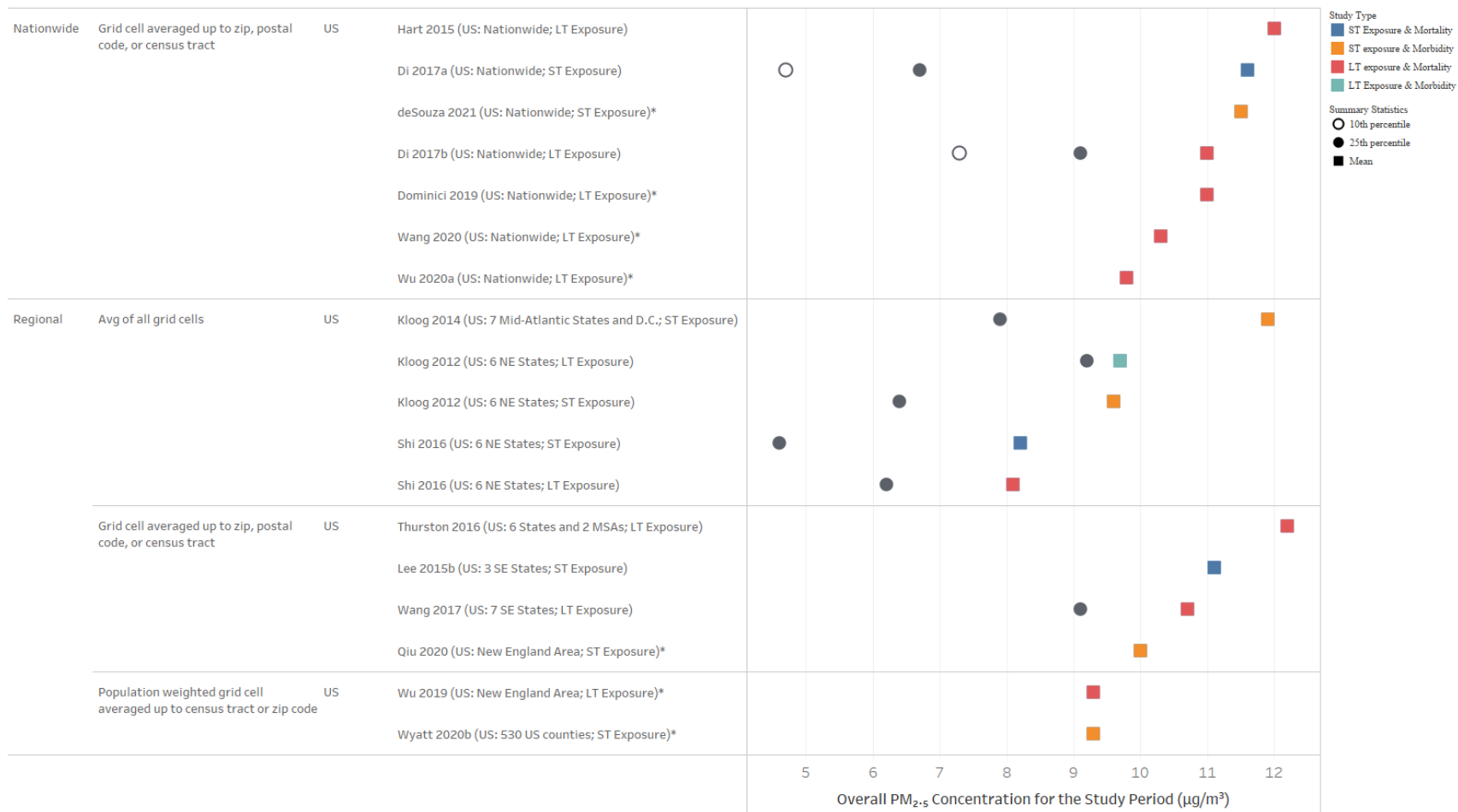
2

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

5



1
2 **Figure 3-13. Hybrid model-predicted PM_{2.5} concentrations in key U.S. epidemiologic studies, subset by method used to average**
3 **grid cells in study-reported long-term mean PM_{2.5} concentrations.** (Asterisks denote studies included in the draft ISA
4 Supplement).
5



1
 2 **Figure 3-14. Hybrid model-predicted PM_{2.5} concentrations in key U.S. epidemiologic studies, subset by spatial scale and**
 3 **method used to average grid cells in study-reported long-term mean PM_{2.5} concentrations.** (Asterisks denote studies included
 4 in the draft ISA Supplement).
 5

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

- 3 • For key U.S. epidemiologic studies that use monitors to estimate PM_{2.5} exposures (Figure 3-
4 8), overall mean PM_{2.5} concentrations are generally at or above 9.9 µg/m³.³² Based on our air
5 quality analyses, we would generally expect these values to be 10-20% lower than the
6 corresponding area annual design value.
- 7 • For key U.S. epidemiologic studies that use hybrid model-predicted exposure (Figure 3-10),
8 mean PM_{2.5} concentrations range from just above 8.0 µg/m³ to just above 12.0 µg/m³. The
9 majority of these studies estimate PM_{2.5} exposure by averaging up from the grid cell spatial
10 resolution used in the modeling approach to the spatial resolution of health study data (e.g.,
11 ZIP code or census tract). This incorporates an aspect of population weighting in the
12 calculation of the mean. Based on our air quality analyses, we would expect these
13 epidemiologic studies to report means similar to those from monitor-based studies and to
14 generally be about 14-18% less than the area annual design value.
 - 15 - In studies that average up from the grid cell level to the ZIP code, postal code,
16 or census tract level, mean PM_{2.5} concentrations range from 9.8 µg/m³ to 12.2
17 µg/m³.
 - 18 - The one study that population weighted the grid cell prior to averaging up to
19 the ZIP code or census tract level report mean PM_{2.5} concentrations of 9.3
20 µg/m³.
- 21 • The other set of key U.S. epidemiologic studies averaged up from the grid cell spatial
22 resolution across the entire study area, whether that be the nation or a region of the country.
23 Based on our air quality analyses (i.e., suggesting these means are 40-50% lower than the
24 area annual design value), we would expect these epidemiologic studies to report some of the
25 lowest mean values.
 - 26 - For these studies, the reported mean PM_{2.5} concentrations range from 8.1
27 µg/m³ to 11.9 µg/m³.
- 28 • Of the key epidemiologic studies evaluated in the 2019 ISA and draft ISA Supplement, a
29 subset of studies report PM_{2.5} concentrations corresponding to the 25th and 10th percentiles of
30 health data or exposure estimates to provide insight into the concentrations that comprise the
31 lower quartiles of the air quality distributions.
 - 32 - In key U.S. epidemiologic studies that use monitors to estimate PM_{2.5}
33 exposures, 25th percentiles of health events correspond to mean PM_{2.5}
34 concentrations (i.e., averaged over the study period for each study city) at or
35 above 11.5 µg/m³ and 10th percentiles of health events correspond to mean
36 PM_{2.5} concentrations at or above 9.8 µg/m³ (i.e., 25% and 10% of health
37 events, respectively, occur in study locations with mean PM_{2.5} concentrations
38 below these values).

³² This is generally consistent with, but slightly below, the lowest study-reported mean PM_{2.5} concentration from monitor-based studies available in the 2020 PA, which was 10.7 µg/m³ (U.S. EPA, 2020, Figure 3-7).

- 1 - Of the key U.S. epidemiologic studies that use hybrid modeling approaches to
2 estimate long-term PM_{2.5} exposures, the ambient PM_{2.5} concentrations
3 corresponding to 25th percentiles of estimated exposures are 6.2 and 9.1
4 µg/m³.
- 5 - In key U.S. epidemiologic studies that use hybrid modeling approaches to
6 estimate short-term PM_{2.5} exposures, the ambient concentrations
7 corresponding to 25th percentiles of estimated exposures, or health events, are
8 generally at or above 6.4 µg/m³. In the one study with lower concentrations,
9 the ambient PM_{2.5} concentration corresponding to the 25th percentile of
10 estimated exposures is 4.7 µg/m³.³³ In the one study with information
11 available on the 10th percentile of health events, the ambient PM_{2.5}
12 concentration corresponding to that 10th percentile is 4.7 µg/m³.
- 13 • Generally, the study-reported mean concentrations in Canadian studies are lower than those
14 reported in the U.S. studies for both monitor-based and hybrid model methods. However,
15 based on our lack of information about how best to compare air quality gradients between
16 the two countries, it is unclear how to view these Canadian study mean values in the context
17 of a level of the annual standard in the U.S.
 - 18 - For the majority of key Canadian epidemiologic studies that use monitor-
19 based exposure (Figure 3-9), mean PM_{2.5} concentrations generally ranged
20 from 7.0 µg/m³ to 9.0 µg/m³. For these studies, 25th percentiles of health
21 events correspond to mean PM_{2.5} concentrations at or above 6.5 µg/m³ and
22 10th percentiles of health events correspond to mean PM_{2.5} concentrations at
23 or above 6.4 µg/m³.
 - 24 - For the key Canadian epidemiologic studies that use hybrid model-predicted
25 exposure (Figure 3-11), the mean PM_{2.5} concentrations are generally lower
26 than in U.S. model-based studies (Figure 3-10), ranging from approximately
27 6.0 µg/m³ to just below 10.0 µg/m³.
 - 28 - The majority of the key Canadian epidemiologic studies that used hybrid
29 modeling were completed at the nationwide scale, while four studies were
30 completed at the regional geographic spatial scale. In addition, all the key
31 Canadian epidemiologic studies, average up from the grid cell level to the
32 spatial resolution of health study data (e.g., postal code).
 - 33 - The majority of studies estimating exposure nationwide range between just
34 below 6.0 µg/m³ to 8.0 µg/m³. One study (Erickson et al. (2020)) presents an
35 analysis related immigrant status and length of residence in Canada versus
36 non-immigrant populations, which accounts for the four highest mean PM_{2.5}
37 concentrations in Figure 3-11, ranging between 9.0 µg/m³ and 10.0 µg/m³.
 - 38 - The four studies that estimate exposure at the regional scale report mean PM_{2.5}
39 concentrations that range from 7.8 µg/m³ to 9.8 µg/m³.

³³ As noted above, in this study (Shi et al., 2016), the authors report that most deaths occurred at or above the 75th percentile of annual exposure estimates (i.e., 10 µg/m³). The short-term exposure estimates accounting for most deaths are not presented in the published study.

1 - In two Canadian studies with information available on the 25th percentile of
2 health events, the ambient PM_{2.5} concentration corresponding to that 25th
3 percentile is approximately 8.0 µg/m³ in two studies, and 4.3 µg/m³ in a third
4 study.

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

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

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

17 The first type are studies that examine health effect associations in analyses with the
18 highest exposures excluded, restricting analyses to daily exposures less than the 24-hour primary
19 PM_{2.5} standard and annual exposures less than the annual PM_{2.5} standard. The restricted analyses
20 can be informative in assessing the nature of the association between long-term exposures (e.g.,
21 < 12.0 µg/m³) or short-term exposures (e.g., < 35 µg/m³) when looking only at exposures to
22 lower concentrations, including whether the association persists in such restricted analyses
23 compared to the same analyses for all exposures, as well as whether the association is stronger,
24 in terms of magnitude and precision, than when completing the same analysis for all exposures.
25 These studies, as assessed in the 2019 ISA and draft ISA Supplement, are summarized in Table
26 3-10 below.

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

Citation	Study Area (health endpoint)	Years of PM _{2.5} Air Quality (monitored)	AQ in restricted analysis (µg/m ³)	Study-reported Mean in restricted analysis (µg/m ³)	Study-reported Mean in main analysis (µg/m ³)	Effect Estimate in restricted analysis (95% CI)	Effect Estimate in main analysis (95% CI)
U.S.-based Studies and Long-term Exposure (per 5 µg/m³)							
Di et al., 2017b	Nationwide (All-cause mortality 65+)	2000-2012	< 12.0	9.6	11.0	1.07 (1.06-1.07)	1.04 (1.04-1.04)
Dominici et al., 2019	Nationwide (All-cause mortality)	2000-2012	< 12	9.6	11.0	1.06 (1.06-1.07)	1.03 (1.03-1.04)
Shi et al., 2016	6 NE States	2003-2008	< 10.0	NR	8.1	1.04 (1.00, 1.09)	1.04 (1.01, 1.06)
Yazdi et al., 2019	7 SE States (CVD morbidity)	2000-2012	< 12	NR	NR	Stroke: 1.29 (1.27-1.31) MI: 1.18 (1.16-1.20) HF: 1.44 (1.43-1.46)	Stroke: 1.16 (1.16-1.17) MI: 1.14 (1.13-1.15) HF: 1.29 (1.29-1.30)
Canadian Studies and Long-term Exposure (per 5 µg/m³)							
Zhang et al., 2021	Ontario (Non-accidental and CVD mortality)	2000-2016	< 10.0 and < 8.8	NR	7.8	Non-accidental mortality: < 10.0: 1.22 (1.10-1.36); and < 8.8: 1.04 (0.91-1.17) CVD mortality: < 10.0: 1.38 (1.10-1.73); and < 8.8: 1.05 (0.80-1.38)	Non-accidental mortality: 1.20 (1.09-1.32) CVD mortality: 1.49 (1.22-1.83)
U.S. Studies and Short-term Exposure (per 10 µg/m³)							
deSouza et al., 2021	Nationwide (First CVD HA)	2000-2012	≤ 25	NR	11.5	1.3% (0.9-1.6 %)	0.9% (0.6-1.1 %)

Di et al., 2017a	Nationwide (All-cause mortality 65+)	2000-2012	<25.0	NR	11.6	1.61 (1.48-1.74)	1.18 (1.09-1.28)
Lee et al., 2015 ¹	3 SE States (Non-accidental)	2007-2011	In ZIP codes where annual average <12.0 and only on days < 35.0	NR	11.1	Non-accidental: 2.08% (1.99-2.17) %	Non-accidental: 1.56% (1.19-1.94%)
Lee et al., 2015 ²	3 SE States (Non-accidental)	2007-2011	In ZIP codes where annual average < 12.0	NR	11.1	Non-accidental: 2.06% (1.97-2.15%)	Non-accidental: 1.56% (1.19-1.94%)
Shi et al., 2016	6 NE States	2003-2008	< 30.0	NR	8.2	2.14% (1.34-2.95%)	2.14% (1.38, 2.89%)
Wei et al., 2019	Nationwide (CVD HA)	2000-2012	≤ 25 (WHO air quality guideline value for daily PM _{2.5})	NR	NR	Relative increase in risk for HA with 1 µg/m ³ increase in lag0-1 PM _{2.5} : MI: 0.16 (0.09, 0.24) CHF: 0.16 (0.11, 0.22)	Relative increase in risk for HA with 1 µg/m ³ increase in lag0-1 PM _{2.5} : MI: 0.11 (0.07, 0.16) CHF: 0.14 (0.10, 0.17)
¹ First, restricted ZIP code areas to where the annual average of predicted PM _{2.5} is < 12 µg/m ³ to assess the acute effect of PM _{2.5} on mortality only areas with annual average concentrations < 12 µg/m ³ . ² In terms of daily standard, conducted analysis on the days < 35 µg/m ³ and only in ZIP codes with annual average concentrations < 12 µg/m ³ .							

1

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

- 7 • In the four U.S. studies that estimate effects associated with long-term exposure to PM_{2.5}, the
8 effect estimates are greater in the restricted analyses than in the main analyses.
 - 9 ○ Di et al. (2017a) and Dominici et al. (2019) report positive and statistically significant
10 associations in analyses restricted to concentrations less than 12.0 µg/m³ for all-cause
11 mortality Di et al. (2017b) and stroke, MI, and HF Dominici et al. (2019), and effect
12 estimates are greater in the restricted analyses than effect estimates reported in main
13 analyses. In addition, both studies report mean PM_{2.5} concentrations of 9.6 µg/m³
 - 14 ○ Shi et al. (2016) and Yazdi et al. (2019) report positive and statistically significant
15 associations in analyses restricted to concentrations less than 10.0 µg/m³ and 12.0
16 µg/m³, respectively. Shi et al. (2016) does not report overall mean PM_{2.5}
17 concentrations in restricted analyses, though such means are presumably somewhat
18 below the main analysis reported mean of 8.1 µg/m³. Yazdi et al. (2019) does not
19 report the overall mean PM_{2.5} concentration in either the restricted analysis or main
20 analysis, but the effect estimates for stroke, MI, and HF are all higher in the restricted
21 analyses compared to main analyses.
- 22 • While none of the U.S. studies of short-term exposure present mean PM_{2.5} concentrations for
23 the restricted analyses, these studies generally have mean 24-hour average PM_{2.5}
24 concentrations in the main analyses below 12.0 µg/m³, and report increases in the effect
25 estimates in the restricted analyses compared to the main analyses.
 - 26 ○ With the exception of Wei et al. (2019), short-term exposure studies report mean 24-
27 hour average PM_{2.5} concentration in main analyses all below 12.0 µg/m³, and ranging
28 from 8.2 µg/m³ Shi et al. (2016) to 11.6 (Di et al. (2017a).
 - 29 ○ These studies, except for Shi et al. (2016), report increases in effect estimates in
30 restricted analyses compared to main analyses. Shi et al. (2016) reports the same effect
31 estimates for both the restricted and main analyses.
- 32 • In the one Canadian study of long-term PM_{2.5} exposure, Zhang et al. (2021) conducted
33 analyses where annual PM_{2.5} concentrations were restricted to concentrations below 10.0
34 µg/m³ and 8.8 µg/m³, which presumably have lower mean concentrations than the mean of
35 7.8 µg/m³ reported in the main analyses, though restricted analysis mean PM_{2.5}
36 concentrations are not reported.
 - 37 ○ Effect estimates for non-accidental mortality are greater in analyses restricted to PM_{2.5}
38 concentrations less than 10.0 µg/m³, but less in analyses restricted to < 8.8 µg/m³.
39 Effect estimates for CVD mortality are lower in restricted analyses than the main
40 analysis.

1
2 Overall, these studies provide additional information on the nature of the association
3 between long- or short-term exposures when analyses are restricted to lower PM_{2.5}
4 concentrations. Further, these studies indicate that effect estimates are generally greater in
5 magnitude in the restricted analyses for long- and short-term PM_{2.5} exposure compared to the
6 main analyses.

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

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

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

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

	GPS adjustment					GPS adjustment: HR = 1.231 (95% CI: 1.180,1.284)
Yazdi et al., 2021	Doubly Robust Additive Model (DRAM)	U.S. Nationwide	2000-2016 (modeled)	LT Cardiovascular hospitalization outcomes (65+)	10.21	% increase in the risk with 1 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$: MI: 0.002; Stroke: 0.009; AI: 0.006
Yitshak-Sade et al., 2019	DID	Northeastern and mid-Atlantic States (14 U.S. States)	2000-2013	LT mortality (65+)	Range: 6.5-14.5	4.04% (95% CI: 3.49,4.59) increase in mortality rates for an IQA (3 $\mu\text{g}/\text{m}^3$) increase in annual $\text{PM}_{2.5}$ concentrations
¹ GPS: generalized propensity score; IPW: inverse probability weighting; DID: Difference-in-difference; HR: hazard ratio; IRR: incidence rate ratio; IPTW: inverse probability treatment weighting; IV: instrument variable; OLS: Ordinary Least Squares; RC: regression calibration						

1

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

- These studies reported consistent results among large study populations across the U.S. The results from studies that use causal modeling methods further inform the relationship between long- and short-term PM_{2.5} exposure and total mortality.
- Studies that employ causal methods to assess the association between long-term exposure to PM_{2.5} and mortality provide additional support for the associations reported in the broader body of cohort studies that examined long-term PM_{2.5} exposure and mortality.
 - For example, Wu et al., 2020a used three different causal modeling statistical approaches, in addition to two more traditional statistical method methods (Cox proportional hazards modeling and Poisson time-series regression model), finding consistent positive and statistically significant results between the five statistical methods and with HRs per a 10 µg/m³ increase in PM_{2.5} ranging from 1.062 (95% CI: 1.055,1.069) using the poisson statistical method to 1.076 (95% CI: 1.065, 1.088) with the GPS matching statistical method.

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

Table 3-12. Epidemiologic studies examining the health impacts of long-term reductions in ambient PM_{2.5} concentrations.

Study Reference	Study Area	Years of PM _{2.5} Air Quality (monitored)	Starting Mean PM _{2.5} Concentration (µg/m ³)	Ending Mean PM _{2.5} Concentration (µg/m ³)	Study Results
Pope et al. (2009)	211 U.S. counties	1979-1983 compared to 1999-2000	20.6	14.1	Statistically significant association between declining ambient PM _{2.5} and increasing life expectancy
Correia et al. (2013)	545 U.S. counties	2000 compared to 2007	13.2	11.6	Statistically significant association between declining ambient PM _{2.5} and

					increasing life expectancy
Berhane et al. (2016)	4,602 children in 8 California communities	1992-2000; 1995-2003; 2002-2011	20.5	14.4	Statistically significant decrease in bronchitic symptoms in 10-year old children with and without asthma
Gauderman et al. (2015)	2,120 children in 5 California communities	1994-1997; 1997-2000; 2007-2010	21.3-31.5	11.9-17.8	Statistically significant improvements in 4-year growth of lung function
Wyatt et al., 2020b	2132 counties in the U.S. (population $\geq 20,000$)	1990-2010	NR	NR	The annual change in cardiovascular mortality rate ranged from 6.5-7.6 fewer deaths/year (per 100,000 person-years) per 1 $\mu\text{g}/\text{m}^3$ decrease in $\text{PM}_{2.5}$ over time.
Bennett et al., 2019	U.S. Nationwide and 1339 U.S. counties	1999-2015	13.6 (Pop-weighted mean)	8.0 (Population-weighted mean; Mean range in counties: 2.8-13.2)	Reductions in $\text{PM}_{2.5}$ since 1999 have increased life expectancy in men and women in all but 14 counties where $\text{PM}_{2.5}$ increased slightly
Corrigan et al., 2018	619 U.S. counties	2000-2010	2000-2004: 12.0	2005-2010: 10.8	Fewer CV deaths per year for each 1 $\mu\text{g}/\text{m}^3$ decrease in $\text{PM}_{2.5}$.
Henneman et al., 2019	Multiple U.S. states	2005-2012	2005: 10.0	2012: 7.2	Reduced exposure to total $\text{PM}_{2.5}$ and coal emissions led to reduced rates total mortality and CVD HA.
Sanders et al., 2020	600-700 U.S. counties	2000-2013	Before 2006: Non-attainment: 15.3 and Attainment: 11.0	After 2006: Non-attainment: 12.0 Attainment: 9.3	By 2005 $\text{PM}_{2.5}$ designation status (attainment or non-attainment), $\text{PM}_{2.5}$ levels and corresponding mortality rates
Fan and Wang, 2020	Eastern US	1999-2013	NR	NR	Fewer CVD deaths per year for each 1

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

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37

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

- Of the new studies evaluated in the 2019 ISA and draft ISA Supplement, Corrigan et al. (2018), Henneman et al. (2019) and Sanders et al. (2020) present analyses with starting concentrations below 12.0 µg/m³.
 - Henneman et al. (2019) explored the changes in modeled PM_{2.5} concentrations following the retirement of coal fired power plants in the U.S., and found that reductions from mean annual PM_{2.5} concentrations of 10.0 µg/m³ in 2005 to mean annual PM_{2.5} concentrations of 7.2 µg/m³ in 2012 from coal-fueled power plants resulted in corresponding reductions in the number of cardiovascular-related hospital admissions and total mortality in those aged 65 and older.
 - Corrigan et al. (2018) examined whether there was a change in the cardiovascular mortality rate before (2000-2004) and after (2005-2010) implementation of the first annual PM_{2.5} NAAQS implementation based on mortality data from the National Center for Health Statistics. They reported 1.10 (95% CI: 0.37, 1.82) fewer cardiovascular deaths per year per 100,000 people for each 1 µg/m³ reduction in annual PM_{2.5} concentrations. When comparing whether counties met the annual PM_{2.5} standard, there were 1.96 (95% CI: 0.77, 3.15) fewer cardiovascular deaths for each 1 µg/m³ reduction in annual PM_{2.5} concentrations between the two periods for attainment counties, whereas for non-attainment counties, there were 0.59 (95% CI: -0.54, 1.71) fewer cardiovascular deaths between the two periods.
 - Sanders et al. (2020) examined whether policy actions (i.e., the first annual PM_{2.5} NAAQS implementation rule in 2005 for the 1997 annual PM_{2.5} standard with a 3-year annual average of 15 µg/m³) reduced PM_{2.5} concentrations and mortality rates in Medicare beneficiaries between 2000-2013. They found evidence of changes in associations with mortality (a decreased mortality rate of ~ 0.5 per 1,000 in attainment and non-attainment areas) due to changes in annual PM_{2.5} concentrations in both attainment and non-attainment areas, which had starting concentrations below 12.0 µg/m³ following implementation of the annual PM_{2.5} NAAQS in 2005. In addition, following implementation of the annual PM_{2.5} NAAQS, annual PM_{2.5}

1 concentrations decreased by 1.59 $\mu\text{g}/\text{m}^3$ (95% CI: 1.39, 1.80) which
2 corresponded to a reduction in mortality rates among individuals 65 years and
3 older (0.93% [95% CI: 0.10%, 1.77%]) in non-attainment counties relative to
4 attainment counties.

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

11 The information in Table 3-10, Table 3-11, and Table 3-12 provide additional support to
12 inform the relationship between long- and short-term $\text{PM}_{2.5}$ exposure and total mortality.
13 Analyses that are restricted only to concentrations at or below the levels of the current primary
14 $\text{PM}_{2.5}$ standards find positive and significant associations with exposure to $\text{PM}_{2.5}$ and health
15 outcomes. These restricted analyses often report greater effect estimates compared to effect
16 estimates in the main analysis that uses the full distribution of $\text{PM}_{2.5}$ concentrations. Studies that
17 use causal modeling methods to assess the relationship between $\text{PM}_{2.5}$ and health outcomes
18 provide additional support for the associations reported in other epidemiologic studies. Finally,
19 new studies assessed in the draft ISA Supplement evaluate the relationship between declines in
20 ambient $\text{PM}_{2.5}$ concentrations over time and the potential for improvements in public health, and
21 support the conclusion in the 2020 PA; improvements in air quality are associated with
22 improvements in public health. Some of these new studies have lower starting concentrations
23 than similar studies included in the 2019 ISA.

24 **3.3.4 Uncertainties in the Health Effects Evidence**

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

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

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

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

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

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

1 metric that describe the air quality status of a given area relative to the NAAQS.³⁴ As discussed
2 above, the overall mean PM_{2.5} concentrations reported by key epidemiologic studies reflect
3 averaging of short- or long-term PM_{2.5} exposure estimates across location (i.e., across multiple
4 monitors or across modeled grid cells) and over time (i.e., over several years). For monitor-based
5 studies, the comparison is somewhat more straightforward than for studies that use hybrid
6 modeling methods, as the monitors used to estimate exposure in the epidemiologic studies are
7 generally the same monitors that are used to calculate design values for a given area. It is
8 expected that areas meeting a PM_{2.5} standard with a particular level would be expected to have
9 average PM_{2.5} concentrations (i.e., averaged across space and over time in the area) somewhat
10 below that standard level. Analyses of recent air quality in U.S. CBSAs indicate that maximum
11 annual PM_{2.5} design values for a given three-year period are often 10% to 20% higher than
12 average monitored concentrations (i.e., averaged across multiple monitors in the same CBSA
13 (U.S. EPA, 2020, Appendix B, section B.7). The difference between the maximum annual design
14 value and average concentration in an area can be smaller or larger than this range, likely
15 depending on factors such as the number of monitors, monitor siting characteristics, and the
16 distribution of ambient PM_{2.5} concentrations. For studies that use hybrid modeling methods to
17 estimate PM_{2.5} concentrations, the comparison between study-reported mean PM_{2.5}
18 concentrations and design values is more complicated given the variability in the modeling
19 methods, temporal scales (i.e., daily versus annual), and spatial scales (i.e., nationwide versus
20 urban) across studies. A recent comparison between two hybrid modeling surfaces explored the
21 impact of these factors on the resulting mean PM_{2.5} concentrations and provided additional
22 information about the relationship between mean concentrations from studies using hybrid
23 modeling methods and design values (see section 2.3.3.1.4). However, the results of those
24 analyses only reflect two surfaces and two types of approaches, so uncertainty remains in
25 understanding the relationship between estimated modeled PM_{2.5} concentrations and design
26 values more broadly across hybrid modeling studies. Moreover, this analysis was completed
27 using two hybrid modeling methods that estimate PM_{2.5} concentrations in the U.S., thus an
28 additional uncertainty includes understanding the relationship between modeled PM_{2.5}
29 concentrations and design values reported in Canada.

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

³⁴ For the annual PM_{2.5} standard, design values are calculated as the annual arithmetic mean PM_{2.5} concentration, averaged over 3 years. For the 24-hour standard, design values are calculated as the 98th percentile of the annual distribution of 24-hour PM_{2.5} concentrations, averaged over three years (Appendix N of 40 CFR Part 50).

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

19 Another important limitation associated with the evidence is that, while epidemiologic
20 studies indicate associations between PM_{2.5} and health effects, they do not identify particular
21 PM_{2.5} exposures that cause effects. Rather, health effects can occur over the entire distribution of
22 ambient PM_{2.5} concentrations evaluated, and epidemiologic studies do not identify a population-
23 level threshold below which it can be concluded with confidence that PM_{2.5}-related effects do not
24 occur. Overall, evidence assessed in the 2019 ISA and draft ISA Supplement continues to
25 indicate a linear, no-threshold concentration-response relationship for long- and short-term PM_{2.5}
26 exposure and all-cause or cause specific mortality. There is less certainty in the shape of the
27 concentration-response curve at mean annual PM_{2.5} concentrations generally below 8 µg/m³,
28 although some studies characterize the concentration-response function with certainty in the
29 linear relationship below 8 µg/m³ and down to as low as 5 µg/m³ (U.S. EPA, 2019, section
30 11.2.4; U.S. EPA, 2021a, section 2.2.3.2).

31 **3.4 RISK INFORMATION**

32 To inform conclusions regarding the primary PM_{2.5} standards that are “requisite” to
33 protect public health (i.e., neither more nor less stringent than necessary; section 1.2), it is
34 important to consider the health risks that would be allowed under those standards. For the
35 current standards, this means evaluating PM_{2.5}-related health risks in locations with three-year

1 annual PM_{2.5} design values of 12.0 µg/m³ and/or three-year 24-hour design values of 35 µg/m³
2 (i.e., neither above nor below the levels of the current standards). Therefore, in addition to our
3 evaluation of PM_{2.5} concentrations in locations of key epidemiologic studies (which are based on
4 existing air quality; section 3.3.3.2), we assess PM_{2.5}-attributable risk associated with either:

- 5 • PM_{2.5} air quality that has been adjusted to simulate “just meeting” the current standards (i.e.,
6 design values equal to 12.0 µg/m³ and/or 35 µg/m³) or lower alternative annual and/or 24-
7 hour standards.
- 8 • The change in risk associated with moving from PM_{2.5} air quality “just meeting” the current
9 standards to “just meeting” alternative annual and/or 24-hour standards.

10 These risk estimates, when considered alongside analyses of the evidence discussed in
11 section 3.3.3, are meant to inform conclusions on the primary standards that would be requisite
12 to protect the public health against long- and short-term PM_{2.5} exposures. Our consideration of
13 estimated risks focuses on addressing the following policy-relevant questions:

- 14 • **What are the estimated PM_{2.5}-associated health risks for air quality just meeting the**
15 **current primary PM_{2.5} standards?**
- 16 • **To what extent are risks estimated to decline when air quality is adjusted to just**
17 **meet potential alternative standards with lower levels?**
- 18 • **What are the uncertainties and limitations in these risk estimates?**

19 The sections below summarize our approach to estimating risks (section 3.4.1) and the
20 results of the risk assessment (section 3.4.1.8). Additional detail on the risk assessment is
21 provided in Appendix C.

22 3.4.1 Risk Assessment Overview

23 Risk assessments combine data from multiple sources and involve various assumptions
24 and uncertainties. Below we summarize key aspects of the risk modeling approach. Input data for
25 these analyses includes concentration-response functions from epidemiologic studies (section
26 3.4.1.1) for each health outcome (section 3.4.1.2) and ambient annual or 24-hour PM_{2.5}
27 concentrations (sections 3.4.1.3 and 3.4.1.4) for the study areas (section 3.4.1.5) utilized in the
28 risk assessment. Quantitative and qualitative methods used to characterize variability and
29 uncertainty in the risk estimates are discussed in section 3.4.1.7.

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

1 **3.4.1.1 Concentration-Response Functions**

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

11 **3.4.1.2 Health Outcomes**

12 Consistent with the overall approach for this reconsideration, this risk assessment has a
 13 targeted scope that focuses on all-cause or nonaccidental mortality associated with long-term and
 14 short-term PM_{2.5} exposures (Table 3-13 and Appendix C, section C.1.1).³⁵ Evidence for these
 15 outcomes supports the determination of a “causal relationship” in the 2019 ISA (U.S. EPA,
 16 2019).³⁶

17 **Table 3-13. Epidemiologic studies used to estimate PM_{2.5}-associated risk.**

Epidemiology Study	Study Population ^a	Age Range (years)	Mortality Categories Covered
<i>Long-term mortality studies</i>			
Di et al., 2017b	Medicare	65+	All-cause
Turner et al., 2016	ACS	30+	All-cause
<i>Short-term mortality</i>			
Baxter et al., 2017	77 cities	All ages	Non-accidental
Ito et al., 2013	NPACT	All ages	All cause
Zanobetti et al., 2014	121 communities	65+	All cause
^a ACS (American Cancer Survey), NPACT (National Particle Components Toxicity). See Appendix C Table C-1 for additional study details.			

18

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

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

3.4.1.3 Air Quality Scenarios

We first estimate health risks associated with air quality adjusted to simulate “just meeting” the current primary PM_{2.5} standards (i.e., the annual standard with its level of 12.0 μg/m³ and the 24-hour standard with its level of 35 μg/m³). We then use air quality modeling to simulate air quality just meeting an alternative standard with a level of 10.0 μg/m³ (annual) and 30 μg/m³ (24-hour). In addition to the model-based approach, for the subset of 30 areas controlled by the annual standard we also employ linear interpolation and extrapolation to simulate just meeting alternative annual standards with levels of 11.0 (interpolated between 12.0 and 10.0 μg/m³), 9.0 μg/m³, and 8.0 μg/m³ (both extrapolated from 12.0 and 10.0 μg/m³).³⁷ Figure 3-15 provides an example of the interpolation and extrapolation calculations performed for a single grid cell. In this example grid cell, modeled annual PM_{2.5} concentrations are 11.23 when the corresponding design value monitor just meets the current annual standard and 9.87 when the corresponding design value monitor just meets the alternative annual standard of 10.0 μg/m³. The interpolated and extrapolated values for the example grid cells are provided in green and blue text, respectively.³⁸

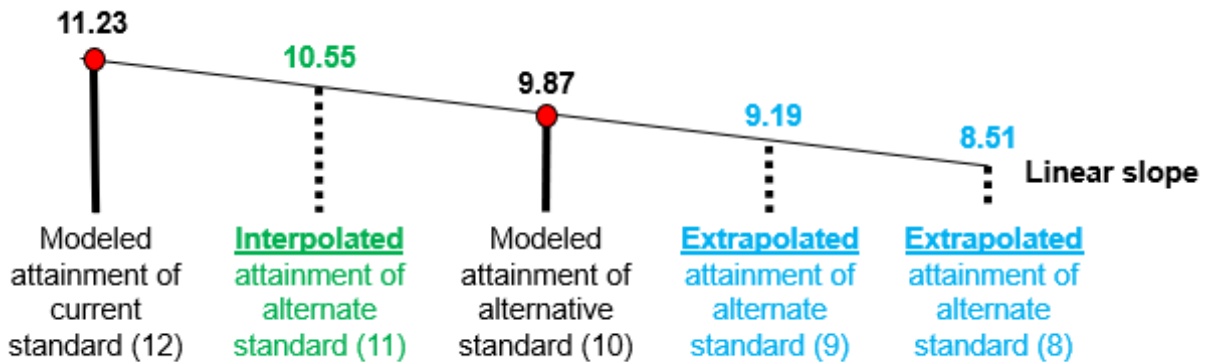


Figure 3-15. Illustration of approach to adjusting air quality to simulate just meeting annual standards with levels of 11.0, 9.0, and 8.0 μg/m³.

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

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

1 There is greater uncertainty regarding whether a revised 24-hour standard (i.e., with a
2 lower level) is needed to further limit “peak” PM_{2.5} concentration exposure³⁹ and whether a
3 lower 24-hour standard level would most effectively reduce PM_{2.5}-associated health risks
4 associated with “typical” daily exposures. However, we do estimate health risks associated with
5 air quality adjusted to meet a revised 24-hour standard with a level of 30 µg/m³, in conjunction
6 with estimating the health risks associated with meeting a revised annual standard with a level of
7 10 µg/m³.^{40,41}

8 **3.4.1.4 Model-Based Approaches to Adjusting Air Quality**

9 Air quality modeling was used to develop 12 km gridded PM_{2.5} concentration fields for
10 the risk assessment in the 2020 PM PA, and the same air quality simulations used in that
11 assessment are used here (U.S. EPA, 2020). A PM_{2.5} concentration field for 2015 was developed
12 using a Bayesian statistical model (Downscaler) that calibrates chemical transport model (CTM)
13 predictions of PM_{2.5} to surface measurements (section 2.3.3). The 2015 PM_{2.5} concentration field
14 was then adjusted using response factors developed from CTM modeling with emission changes
15 relative to 2015. The modeling approach applies realistic spatial response patterns from CTM
16 modeling to a concentration field, similar to those used in a number of recent epidemiologic
17 studies, to characterize PM_{2.5} concentration fields at 12 km resolution for study areas. The
18 adjusted concentration fields correspond to:

- 19 (1) Just meeting the existing annual and 24-hour standards of 12.0 µg/m³ and 35 µg/m³, and
20 (2) Just meeting potential alternative annual and 24-hour standards of 10.0 µg/m³ and 30 µg/m³.

21 The adjustments to simulate just meeting the current standards and alternative standards
22 are approximations of these air quality scenarios. In reality, changes in PM_{2.5} in an area will
23 depend on what emissions changes occur and the concentration gradients of PM_{2.5} will vary
24 across an area accordingly. In this risk assessment, two different adjustment approaches were
25 applied to provide two outcomes that could represent potential bounding scenarios of PM_{2.5}

³⁹ As noted in section 3.3.2.1, while controlled human exposure studies provided consistent evidence for cardiovascular effects following PM_{2.5} exposures for less than 24 hours (i.e., < 30 minutes to 5 hours), exposure concentrations in the studies were well-above the ambient concentrations typically measured in locations meeting the existing standards.

⁴⁰ The simulated air quality surface, which just meets both an alternative annual standard of 10.0 µg/m³ and alternative 24-hour standard of 30 µg/m³, was subset into areas that are controlled by either the alternative annual standard of 10.0 µg/m³ or 24-hour standard of 30 µg/m³ to assess risk associated with just meeting each alternative standard.

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

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

- 3 • *Reductions in primary PM_{2.5} (Pri-PM)*: This modeling approach simulates air quality
4 scenarios of interest by preferentially adjusting direct/primary PM emissions. As such, the
5 changes in PM_{2.5} tend to be more localized near the direct emissions sources of PM.⁴²
- 6 • *Reductions in secondary PM_{2.5} (Sec-PM)*: This modeling approach simulates air quality
7 scenarios of interest by preferentially adjusting SO₂ and NO_x precursor emissions to simulate
8 changes in secondary PM_{2.5}. In this case, the reductions in PM_{2.5} tend to be more evenly
9 spread across a study area.⁴³

10 The air quality surfaces generated using these two approaches are not additive. Rather,
11 they should be viewed as reflecting two different broad strategies for adjusting ambient PM_{2.5}
12 concentrations.

13 3.4.1.5 Study Area Selection

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

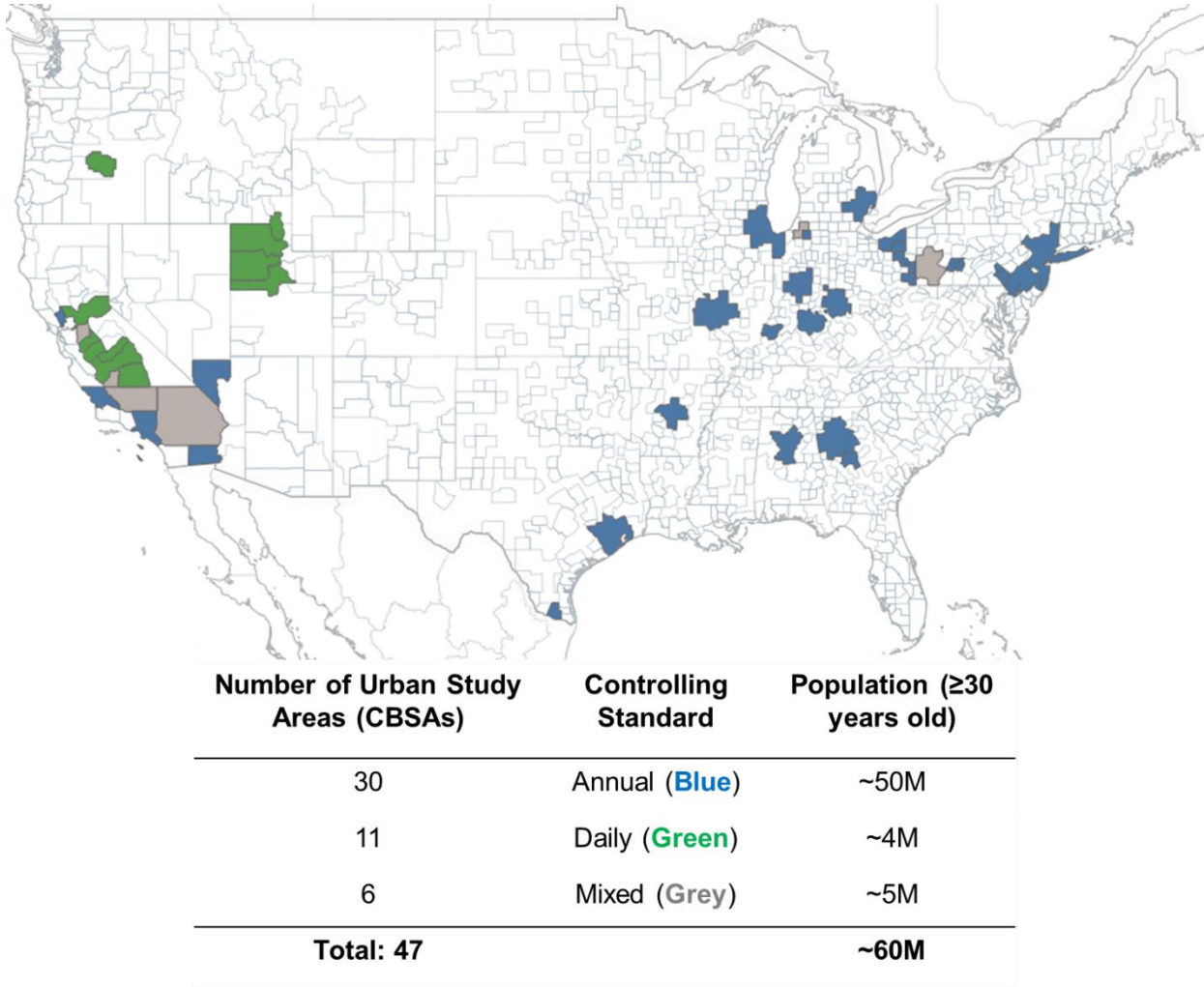
- 16 • *Available Ambient Monitors*: We have greater confidence in estimating and simulating air
17 quality concentrations over areas with relatively dense ambient monitoring networks, as the
18 modeled air quality surfaces can be compared with monitored concentrations (additional
19 detail available in Appendix C, section C.1.4).
- 20 • *Geographical Diversity*: Risk assessments including areas that represent a variety of regions
21 across the U.S. and a substantial portion of the U.S. population can be more representative.
- 22 • *Ambient PM_{2.5} Air Quality Concentrations*: Based on 2014-2016 design values, only 16
23 CBSAs⁴⁴, also called urban study areas here, exceeded either or both the current annual and
24 24-hour PM_{2.5} NAAQS. To include a larger portion of the U.S. in this risk assessment, we
25 also identified CBSAs with ambient PM_{2.5} concentrations below, but near, the current annual
26 and/or 24-hour PM_{2.5} NAAQS. Inclusion of such areas in the risk assessment necessitates an
27 upward adjustment to PM_{2.5} air quality concentrations in order to simulate just meeting the
28 current standards. Given uncertainty in how such increases could potentially occur, we select
29 areas requiring a relatively modest upward adjustment (i.e., no more than 2.0 µg/m³ for the
30 annual standard and 5 µg/m³ for the 24-hour standard, based on the 2014-2016 design value
31 period). Areas that appeared to be strongly influenced by exceptional events were also
32 excluded (section C.1.4). Using these criteria, 47 urban study areas were identified, which

⁴² In locations for which air quality scenarios cannot be simulated by adjusting modeled directly emitted PM alone, modeled SO₂ and NO_x precursor emissions are additionally adjusted to simulate changes in secondarily formed PM_{2.5} (Appendix C, section C.1.4).

⁴³ In locations for which air quality scenarios cannot be simulated by adjusting modeled precursor emissions alone, a proportional adjustment of air quality is subsequently applied. This behavior occurs in areas where emission changes in addition to NO_x and SO₂ would be needed to adjust design values to just meet the standard. (Appendix C, Figure C-19).

⁴⁴ CBSAs (core-based statistical areas) can include one or more counties. Each CBSA selected included at least one monitor with valid design values and several CBSAs had more than 10 monitors. See Table C-3 in Appendix C.

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



7
 8 **Figure 3-16. Map of 47 urban study areas included in risk modeling.**

9

⁴⁵ For these areas, the annual standard is the “controlling standard” because when air quality is adjusted to simulate just meeting the current or potential alternative annual standards, that air quality also would meet the 24-hour standard being evaluated.

⁴⁶ For these areas, the 24-hour standard is the controlling standard because when air quality is adjusted to simulate just meeting the current or potential alternative 24-hour standards, that air quality also would meet the annual standard being evaluated. Some areas classified as being controlled by the 24-hour standard also violate the annual standard.

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

3.4.1.6 At-Risk Analysis

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

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

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

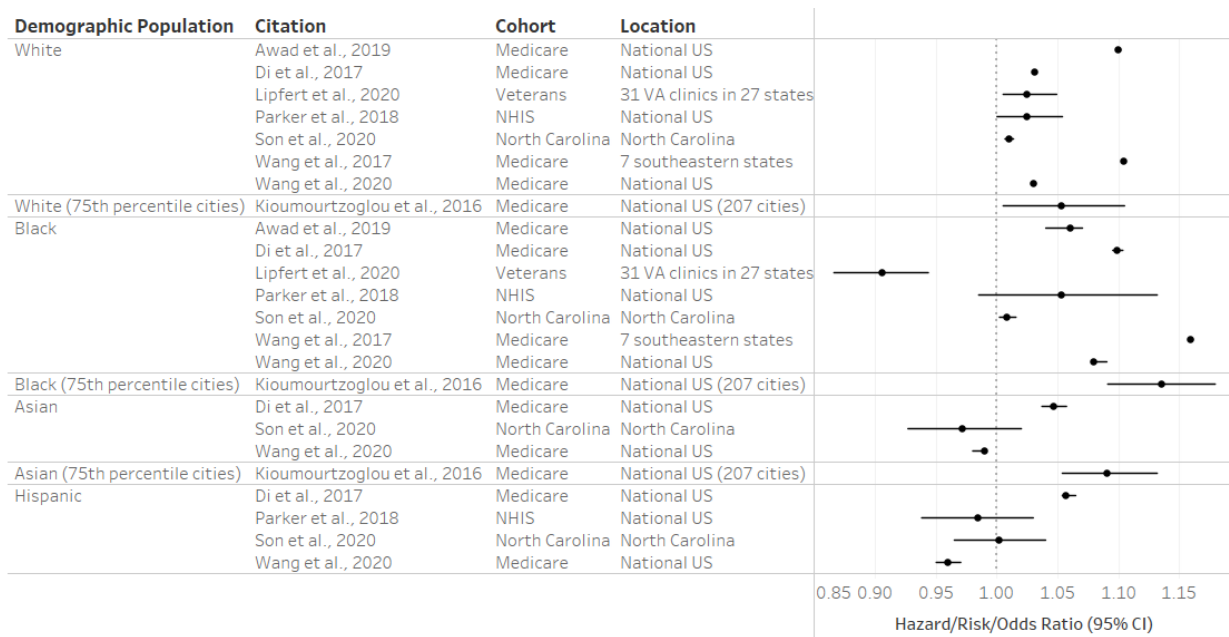
Concentration-response functions used in this at-risk analysis are from large, multicity U.S. epidemiologic studies that evaluate the relationship between PM_{2.5} exposures and mortality. Eight epidemiologic long-term exposure studies of PM_{2.5} exposure and all-cause, nonaccidental, or total mortality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

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

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

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

9



10

11 **Figure 3-17. Available epidemiologic associations between long-term PM_{2.5} exposure and**
 12 **mortality outcomes in demographic populations.⁴⁹**

13 **3.4.1.7 Characterization of Variability and Uncertainty in the Risk Assessment**

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

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

⁴⁹ All studies estimated median or average long-term PM_{2.5} exposures between 10-12 µg/m³, other than Lipfert and Wyzga (2020), which reported an approximate average exposure concentration of 14 µg/m³. Kioumourtzoglou et al., 2016 reported associations in cities ranking at or about the 75th percentile proportionally with regards to demographic population only. VA, Veterans Affairs; NHIS, National Health Insurance Service.

- 1 • *95th percentile confidence intervals*: We use an iterative Monte Carlo simulation that samples
2 from the standard error associated with each epidemiologic concentration-response function.
3 We present the resulting 2.5th and 97.5th percentile values from this distribution as a 95th
4 percentile confidence interval around the risk estimate. Monte Carlo methods are a well-
5 established means of characterizing random sampling error associated with concentration-
6 response functions.
- 7 • *Health endpoint sensitivity analyses*: We include multiple concentration-response functions
8 reflecting epidemiology studies differing in various ways, such as the population (e.g.,
9 geographic locations and demographics), exposure estimation methods (e.g., monitor-based
10 or hybrid techniques), and potential confounders included in the epidemiologic model (e.g.,
11 ozone).⁵⁰
- 12 • *Air quality adjustment sensitivity analyses*: We simulate just meeting the current and
13 alternative standards using two approaches, which represent potential bounding scenarios of
14 PM_{2.5} concentration changes across the study areas. The Pri-PM adjustment method
15 preferentially adjusts direct (i.e., primary, directly-emitted) PM_{2.5} emissions, whereas the
16 Sec-PM method preferentially adjusts SO₂ and NO_x precursor emissions to simulate changes
17 in secondarily formed PM_{2.5}.
- 18 • *Qualitative uncertainty assessment*: We perform additional qualitative evaluations of the
19 potential for key sources of uncertainty to impact the magnitude and direction of risk
20 estimates (Appendix C, section C.3.2).

21 **3.4.1.8 Characterization of Variability and Uncertainty in the At-Risk Analysis**

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

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

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

1 primary PM sources⁵¹. In contrast, Sec-PM air quality adjustments may be strongly associated
2 with sources located outside of the study areas. Since the at-risk analyses are performed for
3 population groups within the 47 areas alone, the Pri-PM adjustment case (in which air quality
4 adjustments are primarily associated with emission sources within the 47 areas) is most
5 appropriate for this at-risk analysis. However, limiting the analysis to a single simulation
6 decreases the potential representativeness of simulated PM_{2.5} concentrations changes across the
7 study area.

8 **3.4.2 Results of the Risk Assessment**

9 This section presents estimates of PM_{2.5}-associated mortality risks for populations in the
10 identified urban study areas (additional results available in Appendix C, section C.2). Results are
11 shown as point estimates with 95th percentile confidence intervals for air quality adjusted to
12 simulate just meeting the current, and potential alternative, standards. We provide tables that
13 include the total mortality risk associated with air quality just meeting the current or potential
14 alternative standards, the change in mortality risk (also called delta risk) when moving from air
15 quality just meeting the current standard to just meeting potential alternative standards, and the
16 percent risk reduction when moving from air quality just meeting the current standard to just
17 meeting potential alternative standards.⁵² We also quantify the percent of baseline incidence,
18 which estimates the percent of total incidence that is associated with ambient PM_{2.5} exposure
19 (e.g., percent of mortality attributable to PM_{2.5} exposure out of all deaths in the specified
20 population).⁵³ In addition to tables, we provide figures to illustrate how risks are distributed
21 across annual average ambient PM_{2.5} concentrations. Figures present results for all-cause
22 mortality associated with long-term PM_{2.5} exposures, based on a key epidemiologic study by
23 (Turner et al., 2016). Additional results are presented in Appendix C (section C.2).

24 The sections below present risk estimates for the full set of 47 urban study areas (section
25 3.4.2.1), the subset of 30 areas for which the annual PM_{2.5} standard is controlling (section
26 3.4.2.2), and the subset of 11 areas for which the 24-hour PM_{2.5} standard is controlling (section
27 3.4.2.3). Risk estimates from populations potentially at increased risk of PM-related effects are

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

⁵² *Total risk* refers to risk associated with the full increment of exposure associated with each air quality scenario. Both *delta risk* and *percent risk reduction* reflect the change in risk in going from the current standard to a specific alternative standard, with delta risk referring to the change in incidence (i.e., premature PM_{2.5}-attributable mortality) and percent risk reduction referring to the percent change when comparing risk under the current standard to risk under simulation of an alternative standard. Percent risk reduction is calculated by dividing the delta risk by the total risk.

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

1 available in section 3.4.2.4. Uncertainties in the risk assessment are summarized in section
2 3.4.2.5.

3 **3.4.2.1 Summary of Risk Estimates for the Full Set of 47 Urban Study Areas**

4 Risk estimates for the 47 urban study areas are presented in Table 3-14 and Table 3-15.
5 Table 3-14 presents all-cause and non-accidental mortality risk estimates attributable to PM_{2.5}
6 when just meeting the current primary PM_{2.5} standards and just meeting either an alternative
7 modeled annual standard of 10.0 µg/m³ or an alternative modeled 24-hour standard of 30 µg/m³.
8 Table 3-14 also provides the percent of total all-cause mortality attributable to PM_{2.5} in 2015
9 estimated by each epidemiologic concentration-response function.

10 Table 3-15 presents the reduction in estimated risk when moving from air quality
11 scenarios just meeting the current standard to air quality just meeting alternative standards. Areas
12 are again subset into those just meeting either an alternative annual standard of 10.0 µg/m³ or an
13 alternative 24-hour standard of 30 µg/m³, based on which standard is controlling in that study
14 area. Smaller reductions estimated for the alternative 24-hour standard reflect the reduced
15 number of study areas controlled by the 24-hour standard and the lesser population in those
16 areas.

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

- 19 • Substantially larger risk reductions are associated with lowering the annual standard than
20 with lowering the 24-hour standard (Table 3-15). Impacts are estimated to decrease by 13-
21 17% when air quality is adjusted to just meet an alternative annual standard with a level of
22 10.0 µg/m³ or by 1-2% when adjusted to just meet an alternative 24-hour standard with a
23 level of 30 µg/m³. This corresponds to up to 7,440 (5,040-9,830) fewer deaths per year
24 attributable to long-term PM_{2.5} exposures.⁵⁴
- 25 • Up to 45,100 deaths in 2015 are attributable to long-term PM_{2.5} exposures associated with air
26 quality just meeting the current annual and 24-hour PM_{2.5} standards, with a 95th percentile
27 confidence interval of 30,800-59,000. This constitutes up to 8% of total baseline mortality in
28 adults age 30-99 (Table 3-14).

⁵⁴In most study areas, the risk reductions presented for an annual standard with a level of 10.0 µg/m³ reflect the difference between air quality with a maximum three-year annual PM_{2.5} design value of 12.0 µg/m³ and air quality with a maximum three-year annual PM_{2.5} design value of 10.0 µg/m³. Similarly, in most study areas, the risk reduction presented for a 24-hour standard with a level of 30 µg/m³ reflects the difference between air quality with a maximum three-year 24-hour PM_{2.5} design value of 35 µg/m³ and air quality with a maximum three-year 24-hour PM_{2.5} design value of 30 µg/m³. However, in a small number of study areas, the “starting concentration” for the annual standard are below 12.0 µg/m³ (four study areas: Riverside-San Bernardino-Ontario, CA; Stockton-Lodi, CA; Bakersfield, CA; and Hanford-Corcoran, CA) or the starting concentration for the 24-hour standard are below 35 µg/m³ (two study areas Pittsburgh, PA and South Bend-Mishawaka, IN-MI:). This is because, in these areas, the controlling standard for air quality adjusted to just meet the current standards is different from the controlling standard for air quality adjusted to simulate just meeting the alternative standards evaluated.

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

4

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

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

7

8

9

1 **Table 3-15. Estimated reduction in PM_{2.5}-associated mortality for alternative annual and**
 2 **24-hour standards (47 urban study areas).**

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

3
4
5 **3.4.2.2 Summary of Risk Estimates for the 30 Areas Controlled by the Annual**
 6 **Standard**

7 This section presents the results for the range of alternative annual standard levels for the
 8 30 urban study areas for which the annual standard is controlling under all air quality scenarios
 9 evaluated.^{55,56} Table 3-16 presents total all-cause and non-accidental mortality risk estimates
 10 attributable to PM_{2.5} when just meeting the current standard of 12.0 µg/m³ and just meeting
 11 potential alternative annual standards with levels of 11.0, 10.0, 9.0, and 8.0 µg/m³. It also
 12 provides the percent of baseline risk attributable to PM_{2.5} when just meeting the current annual
 13 standard. Table 3-17 presents the reduction in estimated mortality incidence and percent of risk
 14 reduction when moving from air quality scenarios just meeting the current annual standard to air
 15 quality just meeting the various alternative annual standards.

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

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

⁵⁶ Alternative annual air quality surfaces in addition to the modeled surface just meeting 10.0 µg/m³ were developed using interpolation and extrapolation of modeled PM_{2.5} concentrations (section 3.4.1.4 and Appendix C section C.1.4).

1 Turner et al. (2016) to provide additional insight into the distribution of health impacts across
2 long-term ambient PM_{2.5} concentrations.⁵⁷ Figure 3-18 presents distributions of total risk
3 attributable to annual PM_{2.5} concentration bins of 1 µg/m³ when just meeting the current and
4 alternative annual standards.⁵⁸ Figure 3-19 presents distributions as a heat map, again binned in 1
5 µg/m³ increments, associated with moving from just meeting the current standard to just meeting
6 each alternative annual standard.⁵⁹

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

- 10 • There is a potential for significant public health impacts in locations just meeting the current
11 primary PM_{2.5} standards. The majority of PM_{2.5}-associated deaths fall well-within the range
12 of long-term average concentrations over which key epidemiologic studies provide strong
13 support for reported positive and statistically significant PM_{2.5} health effect associations.
- 14 • Compared to the current annual standards, air quality adjusted to meet alternative annual
15 standards with lower levels is associated with reductions in estimated all-cause mortality
16 impacts (i.e., 7-9% reduction for an alternative annual level of 11.0 µg/m³, 15-19% reduction
17 for a level of 10.0 µg/m³, 22-28% reduction for a level of 9.0 µg/m³, and 30-37% reduction
18 for a level of 8.0 µg/m³) (Table 3-17 and Figure 3-18).
- 19 • The magnitude of estimated risk reduction increases as alternative annual standards with
20 lower levels are simulated, and these estimated risk reductions are associated with lower
21 ambient PM_{2.5} concentrations. Specifically, for air quality adjusted to simulate just meeting
22 an alternative annual standard, the majority of risk reduction occurs in grid cells with
23 ambient PM_{2.5} concentrations between the alternative standard and 2 µg/m³ lower (e.g., for
24 air quality adjusted to simulate just meeting an annual standard with a level of 8.0 µg/m³, the

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

⁵⁸ Bins correspond to the lower whole number and include up to, but not including the next whole number. For example, the bin for 8 µg/m³, includes all risk occurring at PM_{2.5} concentrations from 8.00 µg/m³ to 8.99 µg/m³. Previously this data was presented as a line graph, which can be found in Appendix C, Figure C-30.

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

1 majority of risk reduction occurs in grid cells with ambient PM_{2.5} concentrations between 6
 2 and 8 µg/m³) (Figure 3-18 and Figure 3-19).⁶⁰

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

6 **Table 3-16. Estimates of PM_{2.5}-associated mortality for the current and potential**
 7 **alternative annual standards in the 30 study areas where the annual standard is**
 8 **controlling.**

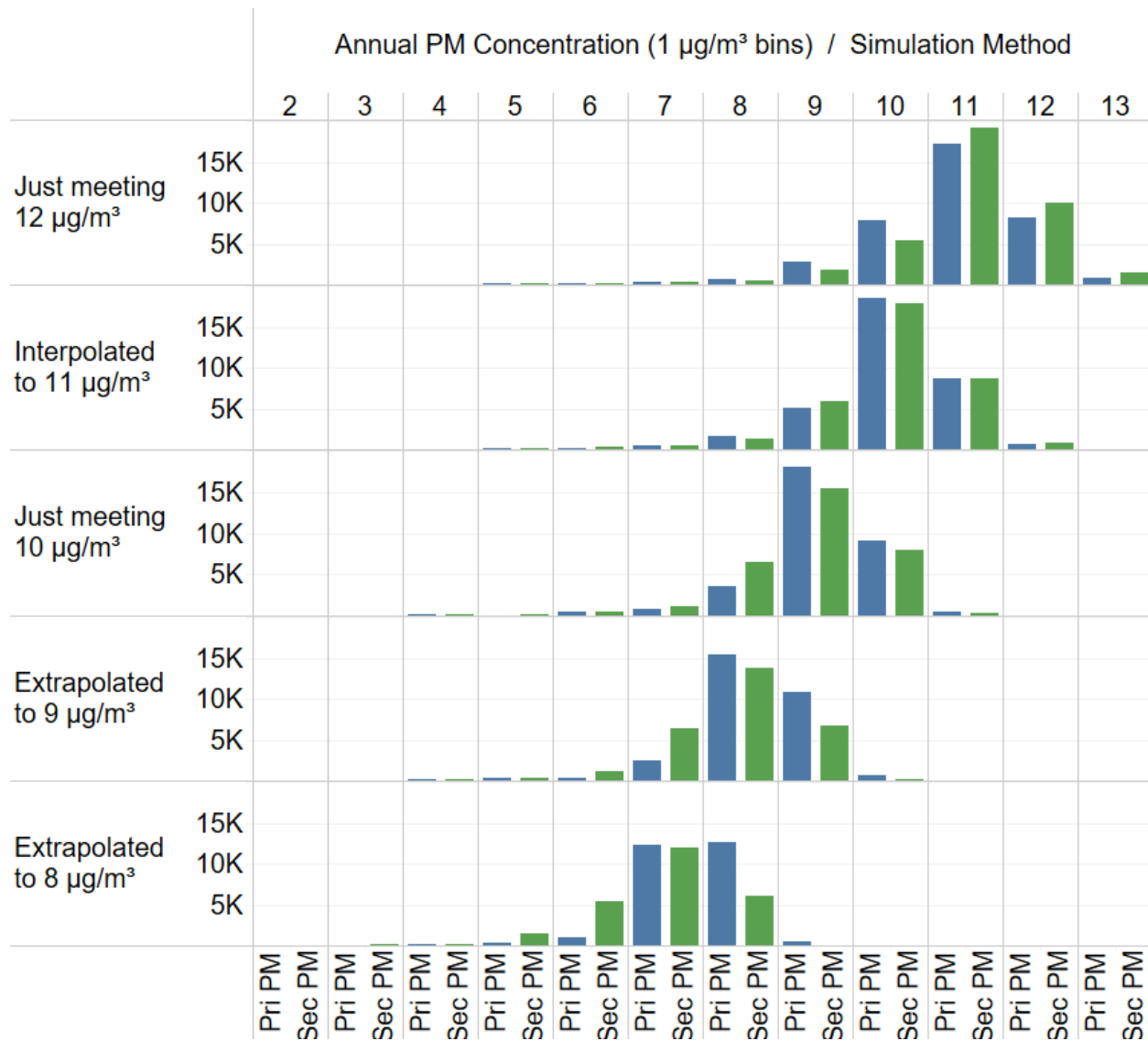
Exposure	Study & Ages	Simulation Method	Total Risk Under the Current Standard (12/35-0)	% of Baseline Risk Attributable to the Current Standard	Total Risk Under an Alternative Annual Standard (11-0)	Total Risk Under an Alternative Annual Standard (10-0)	Total Risk Under an Alternative Annual Standard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

⁶⁰ Compared to adjusting primary PM_{2.5} emissions, adjustment of PM precursor emissions resulted in substantially larger estimated risk reductions at 7 µg/m³.

1 **Table 3-17. Estimated delta and percent reduction in PM_{2.5}-associated mortality for the**
 2 **current and potential alternative annual standards in the 30 study areas where the**
 3 **annual standard is controlling.**

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

4
5



1
2
3
4
5
6
7

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

⁶¹ Risk is estimated in this figure using Turner et al., 2016. Risk estimates are rounded toward zero into whole PM_{2.5} concentration values (e.g., risk estimate at 10 µg/m³ includes risk occurring at 10.0-10.9 µg/m³). For each standard, a small amount of risk is estimated at concentrations higher than the level of the annual standard (e.g., some risk is estimated at an average concentration of 13 µg/m³ when air quality is adjusted to just meet the current standard). This can result because risk estimates are for a single year (i.e., 2015) within the 3-year design value period (i.e., 2014 to 2016). While the three-year average design value is 12.0 µg/m³, a single year can have grid cells with annual average concentrations above or below 12.0 µg/m³.

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

Figure 3-19. Distribution of the difference in risk estimates between the current annual standard (level of 12.0 $\mu\text{g}/\text{m}^3$) and alternative annual standards with levels of 11.0, 10.0, 9.0, and 8.0 $\mu\text{g}/\text{m}^3$ for the subset of 30 urban study areas where the annual standard is controlling.⁶²

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

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

⁶² Risks are presented as integers rounded to three significant digits and aggregated into 1 $\mu\text{g}/\text{m}^3$ bins. Bins begin at the whole number value indicated and include values up to, but not including the next whole number (e.g., risk occurring at PM concentrations of 6.00 to 6.99 are shown in the bin at 6). Risk is estimated in this figure using Turner et al., 2016.

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

1 **Table 3-18. Estimates of PM_{2.5}-associated mortality for the current 24-hour standard, and**
 2 **an alternative, in the 11 study areas where the 24-hour standard is controlling.**

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

3
4
5 **3.4.2.4 Summary of Risk Estimates for At-Risk Populations**

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

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

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

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

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

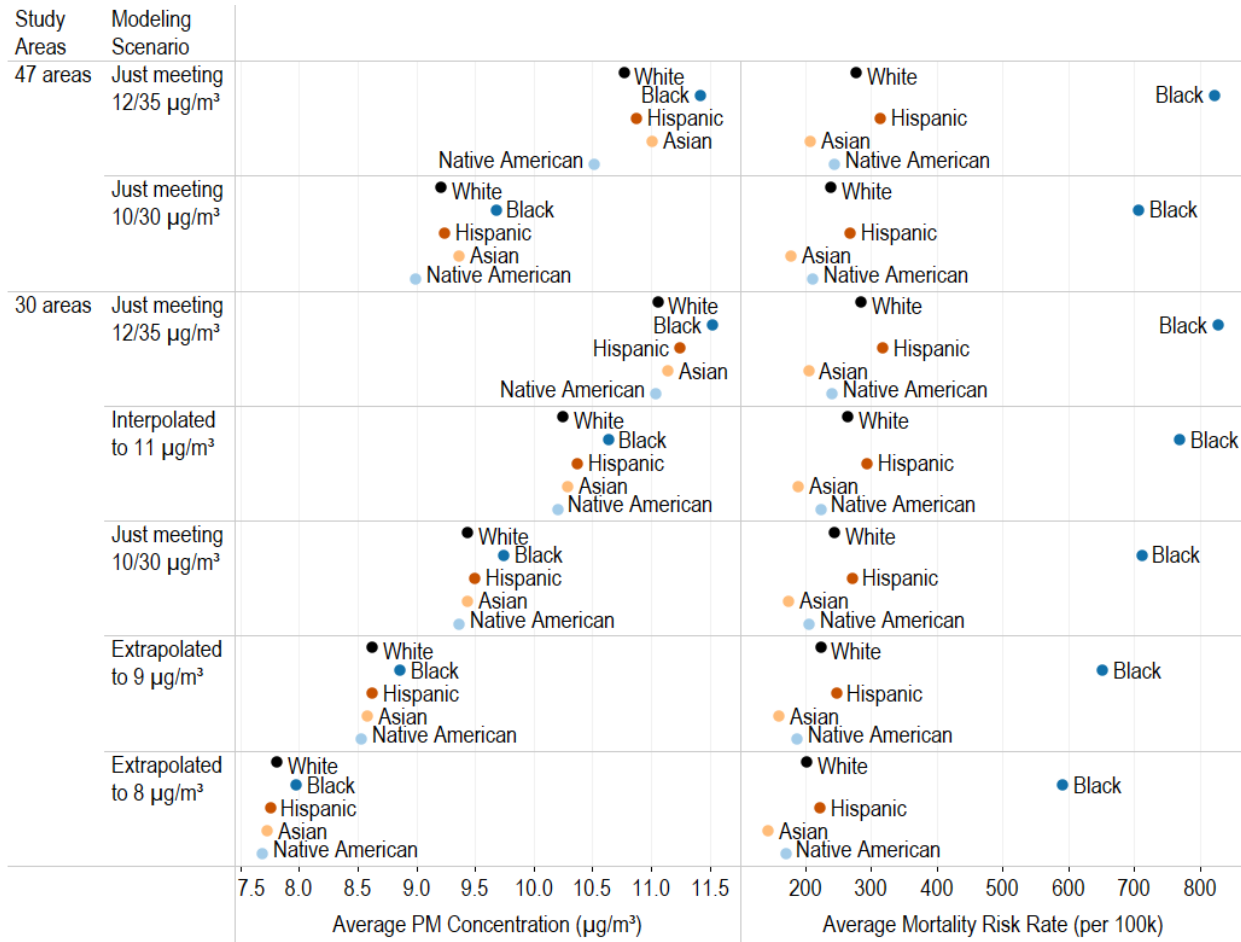
- 19 • The number of people affected by the air pollution reduction. In this instance, the population
20 is further divided by demographic group.
- 21 • The relationship between exposure and health impact baseline incidence rates, or more
22 specifically, the percentage change in the risk of an adverse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

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

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

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

1 baseline incidence rates, to more accurately estimate risk within each demographic group.⁶⁷
 2 Population-normalized mortality risk occurring within each demographic population is available
 3 on the right side of Figure 3-20. Across all scenarios and demographic groups evaluated, Black
 4 populations are associated with the largest PM_{2.5}-attributable mortality risk rate per 100,000
 5 people. An example of the 95th percentile confidence interval is available in Appendix Figure C-
 6 32.

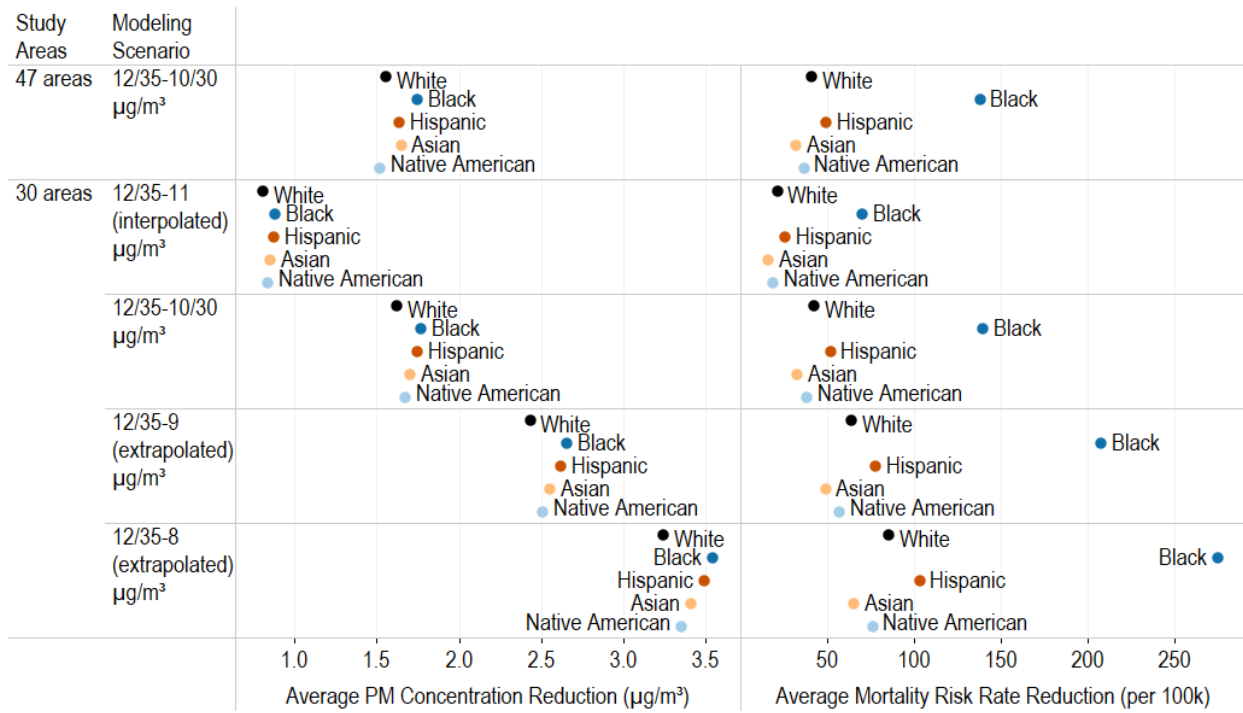


7
 8 **Figure 3-20. Average PM_{2.5} exposure concentration and PM_{2.5}-attributable risk estimates**
 9 **by demographic population when just meeting current or alternative PM_{2.5} standards.**

10
 11 We next estimate demographic-specific average exposure and risk changes when
 12 modeled air quality shifts from just meeting the current annual standard to just meeting potential

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

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



6
 7 **Figure 3-21. Average change in PM_{2.5} exposure concentration and PM_{2.5}-attributable**
 8 **mortality risk estimates by demographic population when moving from the current to**
 9 **alternative PM_{2.5} standards.**

10
 11 We also directly compare the reductions in average national PM_{2.5} concentrations and
 12 risk rates within each demographic population. Table 3-19 and Table 3-20 provide the percent of
 13 national average PM_{2.5}-attributable exposures and risk reductions, when shifting from the current
 14 annual PM_{2.5} standard (12.0 µg/m³) to potential alternative annual PM_{2.5} standards (11.0 µg/m³,
 15 10.0 µg/m³, 9.0 µg/m³, and 8.0 µg/m³). The percent PM_{2.5} and risk reductions are greater in the
 16 Black population than in the White population for each alternative standard evaluated for both
 17 the full set of study areas and the subset controlled by the annual standard. Additionally, the
 18 difference in percent risk reduction increases more in Blacks than in Whites as the potential
 19 alternative annual standard decreases. In other words, Blacks will experience proportionally
 20 greater benefit from successively lower annual standards, although even at an annual standard of
 21 8 µg/m³ Blacks will experience higher rates of premature mortality risk from PM_{2.5} exposure
 22 than Whites.

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

Ethnicity & Race	% PM Reduction from 12 µg/m ³ to 11 (interpolated) µg/m ³	% PM Reduction from 12 µg/m ³ to 10 µg/m ³		% PM Reduction from 12 µg/m ³ to 9 (extrapolated) µg/m ³	% PM Reduction from 12 µg/m ³ to 8 (extrapolated) µg/m ³
	30 areas	47 areas	30 areas	30 areas	30 areas
White	7	14	15	22	29
Black	8	15	15	23	31
Hispanic	8	15	16	23	31
Asian	8	15	15	23	31
Native American	8	14	15	23	30

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

Ethnicity & Race	% Risk Reduction from 12 µg/m ³ to 11 (interpolated) µg/m ³	% Risk Reduction from 12 µg/m ³ to 10 µg/m ³		% Risk Reduction from 12 µg/m ³ to 9 (extrapolated) µg/m ³	% Risk Reduction from 12 µg/m ³ to 8 (extrapolated) µg/m ³
	30 areas	47 areas	30 areas	30 areas	30 areas
White	8	15	15	23	30
Black	9	17	17	25	33
Hispanic	8	16	16	25	33
Asian	8	16	16	24	32
Native American	8	15	16	24	32

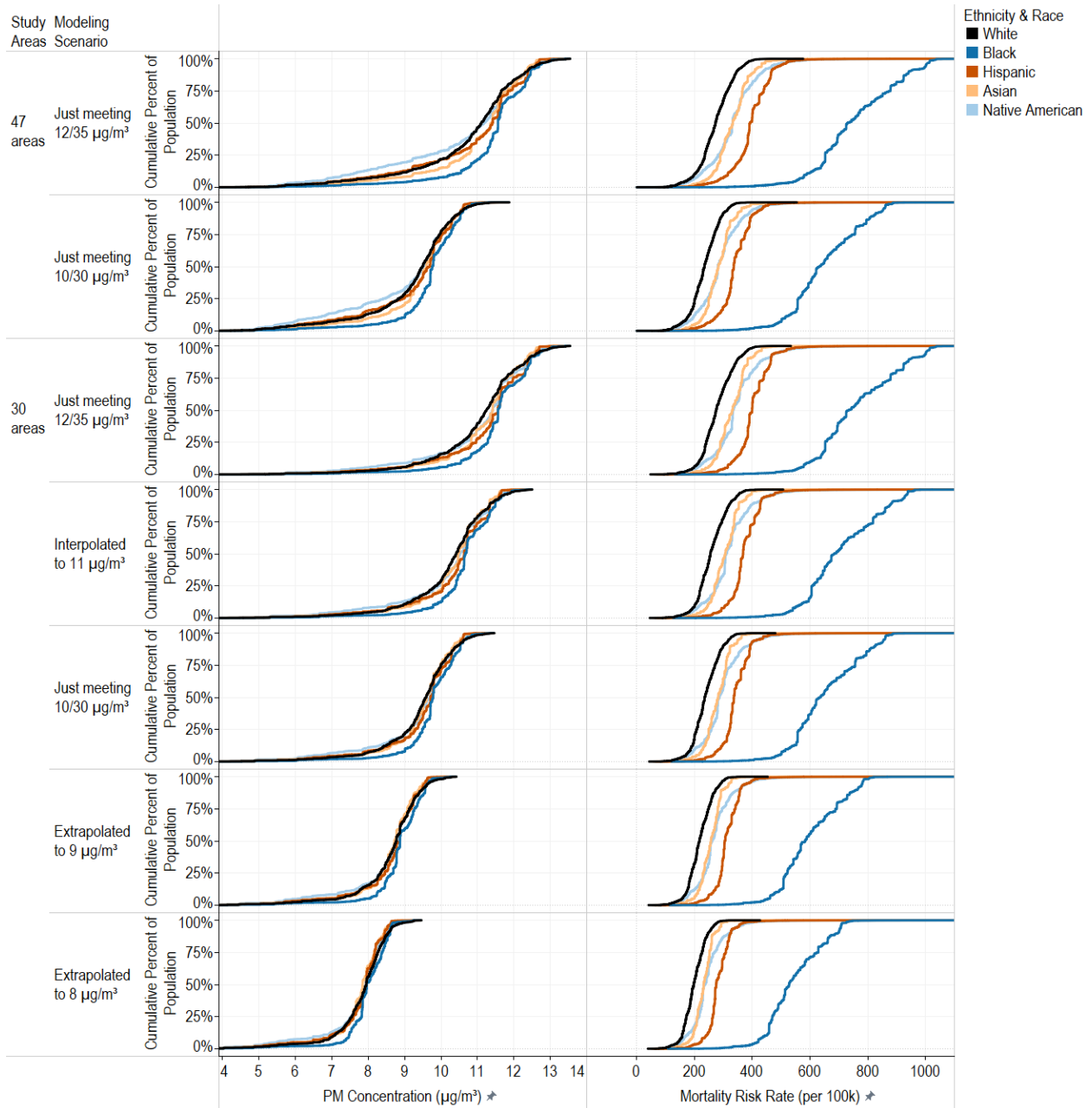
While average exposure concentrations and risk estimates across demographic populations can convey some insight regarding whether certain populations may be disproportionately impacted, distributional information, while more complex, can provide a more comprehensive understanding of the analytical results. As such, we compare both estimated PM_{2.5} exposures and mortality risk rates per 100k individuals to the running sum of each demographic population. To permit the direct comparison of demographic populations with different absolute numbers, populations are expressed as a percentage in Figure 3-22 and Figure 3-23.⁶⁸

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

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

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

5

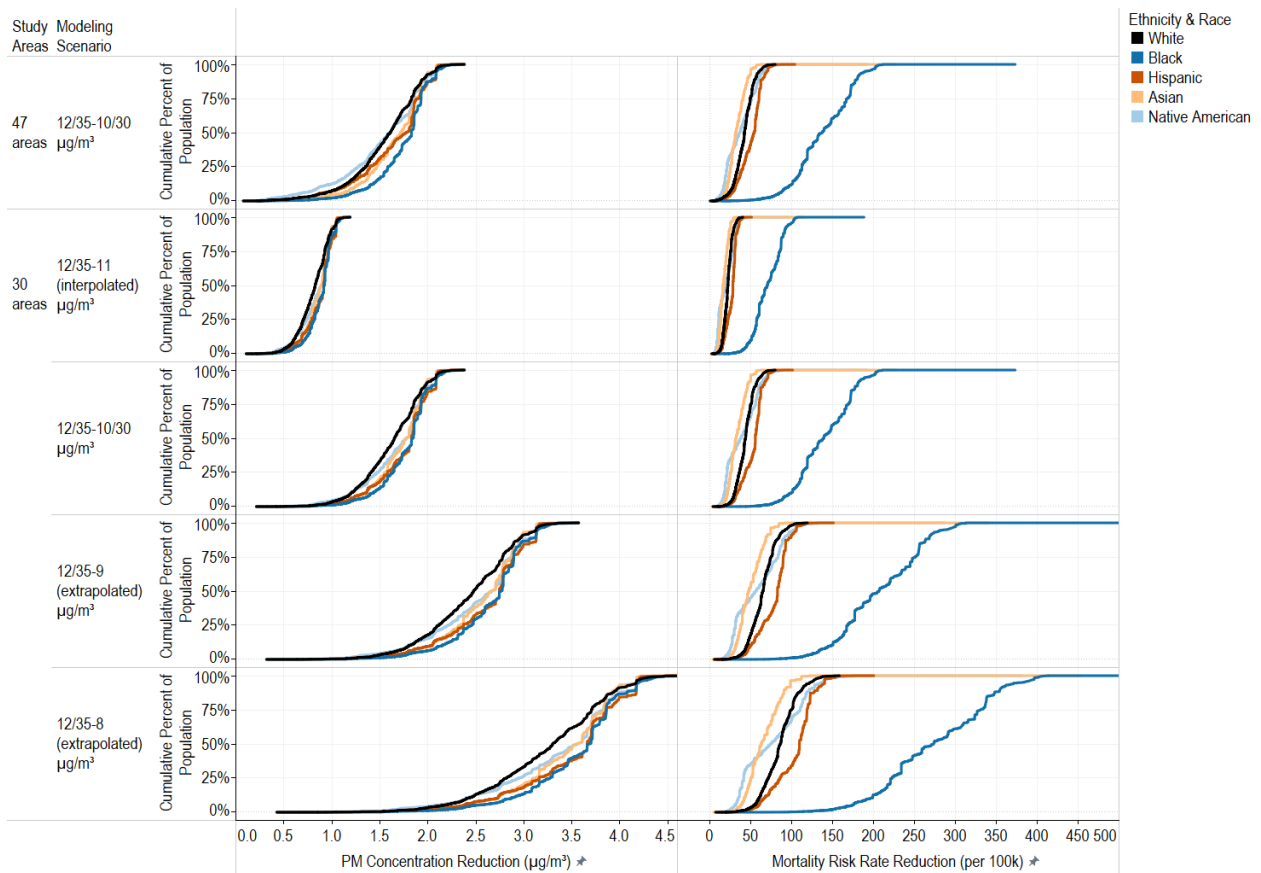


6

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

9

10



1
2 **Figure 3-23. Change in PM_{2.5} exposure concentrations and PM_{2.5}-attributable mortality**
3 **risk estimates by demographic population when moving from the current to alternative**
4 **PM_{2.5} standards.**

5
6 **3.4.2.5 Variability and Uncertainty in Risk Estimates**

7 We characterize variability and uncertainty associated with risk estimates using several
8 quantitative and qualitative approaches, as described in detail in Appendix C (section C.3).

9 Approaches to addressing key uncertainties include the following:

- 10 • Evaluating multiple concentration-response functions for the same health endpoint: The
11 degree to which different concentration-response functions result in different risk estimates
12 could reflect differences in study design and/or study populations evaluated, as well as other
13 factors. In most instances in this risk assessment, the concentration-response function used
14 has only a small impact on risk estimates.
- 15 • Evaluating multiple methods for simulating air quality scenarios: The approach used to adjust
16 air quality (i.e., Pri-PM and Sec-PM adjustments) has some impact on overall estimates of
17 risk (e.g., Table 3-14). However, the adjustment approach has a larger impact on the
18 distribution of risk reductions, particularly for alternative annual levels of 9.0 and 8.0 μg/m³
19 (Figure 3-19).

- 1 • Characterizing the 95th percentile confidence intervals associated with risk estimates: There
2 is considerable variation in the range of confidence intervals associated with the point
3 estimates generated for this analysis (Table 3-14), with some concentration-response
4 functions displaying substantially greater variability than others (e.g., short-term PM_{2.5}
5 exposure and all-cause mortality based on effect estimates from Ito et al. (2013) versus long-
6 term PM_{2.5} exposure all-cause mortality estimates based on Turner et al., 2016. There are a
7 number of factors potentially responsible for the varying degrees of statistical precision in
8 effect estimates, including sample size, exposure measurement error, degree of control for
9 confounders/effect modifiers, and variability in PM_{2.5} concentrations evaluated in the original
10 epidemiologic study.
- 11 • Qualitative assessment of additional sources of uncertainty: Based in part on WHO (2008)
12 guidance and on guidance documents developed by the EPA (U.S. EPA, 2001, U.S. EPA,
13 2004), we also completed a qualitative characterization of sources of uncertainty including an
14 assessment of both the magnitude and direction of impact of those uncertainties on risk
15 estimates. The classification of the magnitude of impact for sources of uncertainty includes
16 three levels: (a) low (unlikely to produce a sufficient impact on risk estimates to affect their
17 interpretation), (b) medium (potential to have a sufficient impact to affect interpretation), and
18 (c) high (likely to have an impact sufficient to affect interpretation). For several of the
19 sources, we provide a classification between these levels (e.g., low-medium, medium-high).⁶⁹
20 The below uncertainties, as well as various additional sources of uncertainty, are detailed in
21 the *Estimating PM_{2.5} and Ozone-Attributable Health Benefits TSD* (U.S. EPA,
22 2021b). Sources of uncertainty with at least a low classification as to the magnitude of
23 potential impacts include the following (from Appendix C, Table C-32):⁷⁰
- 24 - Use of air quality modeling to adjust PM_{2.5} concentrations: The baseline and
25 adjusted air quality concentration fields were developed using modeling to fill
26 spatial and temporal gaps in monitoring and explore “what if” scenarios.
27 State-of-the-science modeling methods were used, but modeling-related biases
28 and errors introduce uncertainty into the PM_{2.5} concentration estimates. In
29 addition, due to the national scale of the assessment, scenarios are based on
30 changing modeled emissions of primary PM_{2.5} or NO_x and SO₂ from all
31 anthropogenic sources throughout the U.S. by fixed percentages. Although
32 this approach tends to target key emission sources in each study area, it does
33 not tailor emission changes to specific sources. The two adjustment cases span
34 a wide range of emission conditions, but these cases are necessarily a subset
35 of the full set of possible emission scenarios that could be used to adjust PM_{2.5}
36 concentrations to simulate “just meeting” standards.

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

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

- 1 - Use of linear interpolation/extrapolation to adjust air quality: The use of
2 interpolation and extrapolation to simulate just meeting annual standards with
3 levels of 11.0, 9.0, and 8.0 $\mu\text{g}/\text{m}^3$ does not fully capture potential non-
4 linearities associated with real-world changes in air quality.
- 5 - Potential confounding of the $\text{PM}_{2.5}$ -mortality effect: Factors are considered
6 potential confounders if demonstrated in the scientific literature to be related
7 to the health effect and correlated with $\text{PM}_{2.5}$. Omitting potential confounders
8 from analyses could either increase or decrease the magnitude of $\text{PM}_{2.5}$ effect
9 estimates (e.g., Di et al., 2017b, supplemental Figure S2). Thus, not
10 accounting for confounders can introduce uncertainty into effect estimates
11 and, consequently, into the estimated impacts generated using those effect
12 estimates. Confounders vary according to study design, exposure duration,
13 and health effect. For studies of short-term exposures, confounders may
14 include meteorology (e.g., temperature, humidity), day of week, season,
15 medication use, allergen exposure, and long-term temporal trends. For studies
16 of long-term exposures, confounders may include socioeconomic status, race,
17 age, medication use, smoking status, stress, noise, and occupational
18 exposures. While various approaches to control for potential confounders have
19 been adopted across the studies used in the risk assessment, and across the
20 broader body of $\text{PM}_{2.5}$ epidemiologic studies assessed in the 2019 ISA, no
21 individual study adjusts for all potential confounders (U.S. EPA, 2019, Table
22 A-1).
- 23 - Potential for exposure error: Epidemiologic studies have employed a variety
24 of approaches to estimate population-level $\text{PM}_{2.5}$ exposures (e.g., stationary
25 monitors and hybrid modeling approaches). These approaches are based on
26 using measured and/or predicted ambient $\text{PM}_{2.5}$ concentrations as surrogates
27 for population exposures. As such, exposure estimates in epidemiologic
28 studies are subject to exposure error. The 2019 ISA notes that, while bias in
29 either direction can occur, exposure error tends to result in underestimation of
30 health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019,
31 section 3.5). Consistent with this, Hart et al. (2015) reports that correction for
32 $\text{PM}_{2.5}$ exposure error using personal exposure information results in a
33 moderately larger effect estimate for long-term $\text{PM}_{2.5}$ exposure and mortality,
34 though with wider confidence intervals. Error in the underlying epidemiologic
35 studies contributes to uncertainty in the risk estimates based on concentration-
36 response relationships in those studies. Beyond the exposure error in
37 concentration-response functions, the use of a different approach to represent
38 exposures in the risk assessment (i.e., 12 x 12 km gridded surface based on
39 modeling) could introduce additional error into risk estimates.
- 40 - Shape of the concentration-response relationship at low ambient PM
41 concentrations: Interpreting the shapes of concentration-response
42 relationships, particularly at $\text{PM}_{2.5}$ concentrations near the lower end of the air
43 quality distribution, can be complicated by relatively low data density in the
44 lower concentration range, the possible influence of exposure measurement
45 error, and variability among individuals with respect to air pollution health
46 effects. These sources of variability and uncertainty tend to smooth and

1 “linearize” population-level concentration-response functions, and thus could
2 obscure the existence of a threshold or nonlinear relationship (U.S. EPA,
3 2015b, section 6.c).
4

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

9 **3.4.3 Conclusions of the Risk Assessment**

10 Although limitations in the underlying data and approaches lead to some uncertainty
11 regarding estimates of PM_{2.5}-associated risk (summarized in section 3.4.1.7), the risk assessment
12 estimates that the current primary PM_{2.5} standards could allow a substantial number of PM_{2.5}-
13 associated deaths in the U.S. For example, when air quality in the 47 study areas is adjusted to
14 simulate just meeting the current standards, the risk assessment estimates 40,600-45,100 long-
15 term PM_{2.5} exposure-related deaths in a single year, with confidence intervals ranging from
16 30,300-59,000 deaths (Table 3-14). Additionally, the at-risk assessment estimated that Black
17 populations may experience disproportionately higher exposures and risk under simulated air
18 quality conditions just meeting the current primary PM_{2.5} annual standard as compared to White
19 populations (section 3.4.2.4).⁷¹

20 Compared to the current annual standard, meeting a revised annual standard with a lower
21 level is estimated to reduce PM_{2.5}-associated health risks in the 30 annually-controlled study
22 areas by about 7-9% for a level of 11.0 µg/m³, 15-19% for a level of 10.0 µg/m³, 22-28% for a
23 level of 9.0 µg/m³, and 30-37% for a level of 8.0 µg/m³. (Table 3-17)⁷² Meeting a revised annual
24 standard with a lower level may also reduce exposure and risk in Black populations slightly more
25 so than in White populations in simulated scenarios just meeting alternative annual standards
26 (section 3.4.2.4).

27 Revising the level of the 24-hour standard to 30 µg/m³ is estimated to lower PM_{2.5}-
28 associated risks across a more limited population and number of areas than revising the annual
29 standard (section 3.4.2.3). Risk reduction predictions are largely confined to areas located in the

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

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

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

3 **3.5 KEY CONSIDERATIONS REGARDING THE ADEQUACY OF THE** 4 **PRIMARY PM_{2.5} STANDARDS**

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

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

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

17 **3.5.1 Evidence-based Considerations**

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

26 **3.5.1.1 Health Effects Associated with Exposure to PM_{2.5}**

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

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

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

1 concentrations in ambient air, and potential for health effects. While some studies evaluate the
2 health effects of particular sources of fine particles, or of particular fine particle components,
3 evidence from these studies does not identify any one source or component that is a better
4 predictor of health effects than PM_{2.5} mass (U.S. EPA, 2019, section 1.5.4). The 2019 ISA
5 specifically notes that “results of these studies confirm and further support the conclusion of the
6 2009 ISA that many PM_{2.5} components and sources are associated with many health effects and
7 that the evidence does not indicate that any one source or component is consistently more
8 strongly related with health effects than PM_{2.5} mass” (U.S. EPA, 2019, section 1.5.4). In
9 addition, the evidence for health effects following exposures specifically to the ultrafine fraction
10 of fine particles continues to be far more limited than the evidence for PM_{2.5} mass as a whole. As
11 discussed in the 2019 ISA, the lack of a consistent UFP definition in health studies and across
12 disciplines, together with a variety of approaches to administering and measuring UFP in those
13 studies, contribute to such limitations (U.S. EPA, 2019, section 1.4.3). Thus, as was the case for
14 previous reviews, the evidence base for health effects of fine particles does not support
15 consideration of other PM characteristics, such as components, or size fractions. For these
16 reasons, we continue to focus on the health effects associated with PM_{2.5} mass.

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

19 The scientific evidence, including that assessed in the 2019 ISA and draft ISA
20 Supplement, is consistent with the conclusion reached in the previous reviews regarding health
21 effects and PM exposures where a causal relationship was concluded. Specifically, as in prior
22 reviews, it was concluded that there is a causal relationship between short- and long-term PM_{2.5}
23 exposures and mortality and cardiovascular effects (U.S. EPA, 2019, sections 11.1, 11.2, 6.1,
24 6.2; U.S. EPA, 2021a, sections 3.2.1, 3.2.2, 3.1.1, and 3.1.2). Further, a likely to be causal
25 relationship was concluded for short- and long-term PM_{2.5} exposures and respiratory effects
26 (U.S. EPA, 2019, sections 5.1 and 5.2). Additionally, conclusions reached in the 2019 ISA differ
27 with regard to cancer and nervous systems effects and long-term PM_{2.5} exposure, based on
28 evidence assessed in the 2019 ISA and it was concluded that there is a likely to be causal
29 relationship (U.S. EPA, 2019, sections 10.2 and 8.2). The evidence base is concluded to be
30 suggestive of, but not sufficient to infer, causal relationships between short- and long-term PM_{2.5}
31 exposures and metabolic effects (U.S. EPA, 2019, sections 7.1 and 7.2), reproduction and
32 fertility (U.S. EPA, 2019, section 9.1.1), and pregnancy and birth outcomes (U.S. EPA, 2019,
33 section 9.1.2). In addition, effects associated with short-term exposure to UFP and cardiovascular
34 (U.S. EPA, 2019, section 6.5), respiratory (U.S. EPA, 2019, section 5.5), and nervous system
35 effects (U.S. EPA, 2019, section 8.5), as well as long-term exposure to UFP and nervous system
36 effects (U.S. EPA, 2019, section 8.6) are concluded to be suggest of, but not sufficient to infer,

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

4 **3.5.1.2 Populations At-Risk of PM_{2.5}-related Health Effects**

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

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

14 The current evidence does not alter our understanding of which populations are
15 potentially at greater risk from health effects of PM_{2.5} exposures. As in previous reviews, the
16 2019 ISA continues to provide support that factors that may contribute to increased risk of
17 PM_{2.5}-related health effects include lifestage (children and older adults), pre-existing diseases
18 (cardiovascular disease and respiratory disease), race/ethnicity, and socioeconomic status. Other
19 factors that have the potential to contribute to increased risk, but for which the evidence is less
20 clear, include obesity, diabetes, genetic factors, smoking status, sex, diet, and residential location
21 (U.S. EPA, 2019, chapter 12).

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

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

3.5.1.3 Exposure Concentrations Associated with Health Effects

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

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

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

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

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

3 To provide some insight into what these studies may indicate regarding the primary PM_{2.5}
4 standards, air quality analyses examine monitored 2-hour PM_{2.5} concentrations at sites meeting
5 the current primary PM_{2.5} standards (as described in section 2.3.2 and section A.3 of Appendix
6 A).⁷³ The 2-hour PM_{2.5} concentrations to which individuals were exposed in most of these
7 studies are well-above the ambient concentrations typically measured in locations meeting the
8 current primary standards. For example, at air quality monitoring sites meeting the current
9 primary PM_{2.5} standards (i.e., the 24-hour standard and the annual standard), the 2-hour
10 concentrations generally remain below 10 µg/m³, and virtually never exceed 30 µg/m³. Two-hour
11 concentrations are higher at monitoring sites violating the current standards, but generally remain
12 below 16 µg/m³ and virtually never exceeding 80 µg/m³. Thus, while controlled human exposure
13 studies provide support for the biological mechanisms and plausibility of the serious
14 cardiovascular effects associated with ambient PM_{2.5} exposures in epidemiologic studies (U.S.
15 EPA, 2019, chapter 6), the exposures evaluated in most of these studies are well-above the
16 ambient concentrations typically measured in locations meeting the current primary standards,
17 and the results are variable across some of the controlled human exposure studies evaluated at
18 near ambient PM_{2.5} concentrations.

19 While controlled human exposure studies provide insight on the exposure concentrations
20 that directly elicit health effects in humans, uncertainty exists in translating the observations in
21 animal toxicology studies to potential adverse health effects in humans. The interpretation of the
22 animal toxicology studies with regard to the potential implications for human health is
23 complicated by the fact that the concentrations of PM_{2.5} in animal toxicologic studies are much
24 higher than those shown to elicit effects in human populations, and there are also significant
25 anatomical and physiological differences between animal models and humans. Most of the
26 animal toxicology studies have generally examined short-term exposures to PM_{2.5} concentrations
27 from 100 to >1,000 µg/m³ and long-term exposures to concentrations from 66 to >400 µg/m³
28 (e.g., see U.S. EPA, 2019, Table 1-2). Two exceptions are a study reporting impaired lung
29 development following long-term exposures (i.e., 24 hours per day for several months prenatally
30 and postnatally) to an average PM_{2.5} concentration of 16.8 µg/m³ (Mauad et al., 2008) and a
31 study reporting increased carcinogenic potential following long-term exposures (i.e., 2 months)
32 to an average PM_{2.5} concentration of 17.7 µg/m³ (Cangerana Pereira et al., 2011). These two
33 studies report serious effects following long-term exposures to PM_{2.5} concentrations close to the

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

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

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

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

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

9 Consistent findings from the broad body of epidemiologic studies are also supported by
10 an emerging body of studies employing causal modeling methods to further inform the causal
11 nature of the relationship between long- or short-term term PM_{2.5} exposure and mortality (U.S.
12 EPA, 2019, sections 11.1.2.1, 11.2.2.4, U.S. EPA, 2021a, sections 3.1.1.3, 3.1.2.3, 3.2.1.3, and
13 3.2.2.3). These studies, summarized above in Table 3-11, used a variety of statistical methods to
14 control for confounding bias and consistently report positive associations, which support the
15 positive and significant effects seen in cohort studies associated with short- and long-term
16 exposure to PM_{2.5} and mortality.

17 In addition to broadening our understanding of the health effects that can result from
18 exposures to PM_{2.5} and strengthening support for some key effects (e.g., nervous system effects,
19 cancer), recent epidemiologic studies strengthen support for health effect associations at
20 relatively low ambient PM_{2.5} concentrations. Studies that examine the shapes of concentration-
21 response functions over the full distribution of ambient PM_{2.5} concentrations have not identified
22 a threshold concentration, below which associations no longer exist (U.S. EPA, 2019, section
23 1.5.3, U.S. EPA, 2021a, sections 2.2.3.1 and 2.2.3.2). While such analyses are complicated by
24 the relatively sparse data available at the lower end of the air quality distribution (U.S. EPA,
25 2019, section 1.5.3), analyses that assess the concentration-response relationship support a linear,
26 no-threshold effect down to 5.0 µg/m³, though uncertainties increase at concentrations of less
27 than 8.0 µg/m³.

28 There are a number of U.S. and Canadian studies that examine health effect associations
29 in analyses with the highest exposures excluded and report positive and statistically significant
30 associations in analyses restricted to annual average PM_{2.5} exposures at or below 12 µg/m³ and
31 or to daily exposures below 35 µg/m³ (Table 3-10). While mean PM_{2.5} concentrations for these
32 restricted analyses may not be reported in most studies, we can presume that the mean PM_{2.5}
33 concentrations in the restricted analyses are less than the study-reported mean PM_{2.5}
34 concentrations in the main analyses, which range from 8.1 µg/m³ to 11.6 µg/m³ in the U.S., and
35 was 7.8 µg/m³ for the one study in Canada that included restricted analysis. It is important to
36 note that even if we had information on PM_{2.5} mean concentrations reported in restricted

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

8 Finally, accountability studies evaluate whether changes in air quality are associated with
9 improvements in public health and a number of recent studies are evaluated in the draft ISA
10 Supplement (summarized in Table 3-12 above). These studies exhibit positive and significant
11 associations, including some studies that report starting PM_{2.5} concentrations below 12.0 µg/m³,
12 indicating that public health improvements may occur following PM_{2.5} reductions in areas that
13 already meet the current annual PM_{2.5} standard. For example, studies by Corrigan et al. (2018)
14 and Sanders et al. (2020) both found improvements in mortality rates due to improvements in air
15 quality in both attainment and nonattainment areas following implementation of the 1997
16 primary annual PM_{2.5} NAAQS.⁷⁴ Other recent studies additionally report that declines in ambient
17 PM_{2.5} concentrations over a period of years have been associated with decreases in mortality
18 rates and increases in life expectancy, improvements in respiratory development, and decreased
19 incidence of respiratory disease in children, further supporting the robustness of PM_{2.5} health
20 effect associations reported in the epidemiologic evidence.

21 Consistent with previous reviews, we note that the use of information from epidemiologic
22 studies to inform conclusions on the primary PM_{2.5} standards is complicated by the fact that such
23 studies evaluate associations between distributions of ambient PM_{2.5} and health outcomes, and
24 do not identify the specific exposures that can lead to the reported effects. Rather, health effects
25 can occur over the entire distribution of ambient PM_{2.5} concentrations evaluated, and
26 epidemiologic studies do not identify a population-level threshold below which it can be
27 concluded with confidence that PM-associated health effects do not occur (U.S. EPA, 2019,
28 section 1.5.3). However, the study-reported ambient PM_{2.5} concentrations reflecting estimated
29 exposure in the middle portion of the PM_{2.5} air quality distribution, which corresponds to the
30 bulk of the underlying data, which provide the strongest support for reported health effect
31 associations and can inform our preliminary conclusions on the current and potential alternative
32 standards. In using this information to inform our preliminary conclusions, we recognize that the
33 mean PM_{2.5} concentrations reported by key epidemiologic studies differ in how mean

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

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

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

8 In the 2012 review, the Administrator recognized that evidence of an association between PM_{2.5}-
9 related health effects and long- and short-term exposures in the epidemiologic studies were
10 strongest at and around the long-term average where the data in the study are most concentrated.
11 In so doing, she noted that the long-term mean PM_{2.5} concentrations were available for the
12 studies considered and represented the most robust data set to inform decisions on appropriate
13 levels for the annual primary PM_{2.5} standard, while also recognizing that this approach did not
14 provide a bright line for reaching this decision (78 FR 3140, January 15, 2013). As detailed in
15 section 3.3.3.2.1, the reported mean PM_{2.5} concentrations derived from monitored observations
16 are not the same as the mean PM_{2.5} concentrations estimated using hybrid modeling methods,
17 which are also not the same as design values used to determine whether an area meets or exceeds
18 the PM_{2.5} NAAQS. Additional analyses, new in this draft PA though similar to those in the 2012
19 review, examine how the calculation of the study mean varies across studies and how these
20 metrics compare to the annual design value. The analysis indicates that study means from
21 methods that use hybrid models to estimate exposures are generally lower in areas where urban
22 and rural PM_{2.5} concentrations are estimated, compared to hybrid modeled PM_{2.5} concentrations
23 in urban areas or concentrations that have been population-weighted. Moreover, the analysis
24 indicates that hybrid modeling mean estimates are generally lower than the average of monitored
25 PM_{2.5} concentrations, which are both below the concentration measured at the highest monitor
26 (i.e., the approach used to calculate the design value). In the national-scale analysis, where air
27 quality analyses compared composite monitored PM_{2.5} concentrations with annual PM_{2.5} design
28 values in the U.S., annual PM_{2.5} design values were approximately 10% to 20% higher than
29 concentrations averaged across multiple monitors in the same CBSA (section 2.3.3.1, Figure 2-
30 28 and Table 2-2).

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

1 exposure relative to design values using the DI2019 and HA2020⁷⁵ surfaces, which are two air
2 quality surfaces included in several of the key epidemiologic studies. This analysis illustrates that
3 population-weighting the PM_{2.5} concentrations in the hybrid modeling approaches has an effect
4 on the resulting study-reported mean. Specifically, the analysis shows that area annual design
5 values are 40% to 50% higher compared to the study-reported means when population-weighting
6 is not employed. Additionally, when population-weighting is applied in studies using hybrid
7 modeling approaches, average annual PM_{2.5} design values are only 15% to 18% higher than the
8 study-reported means. This suggests that whether a study using a hybrid modeling approach
9 incorporated population-weighting is very important for understanding how to interpret the
10 estimated PM_{2.5} exposure concentrations, particularly for purposes of comparing those estimated
11 concentrations to actual design values.

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

- 22 • Range of monitor-based mean PM_{2.5} concentrations is from 9.9 µg/m³ to 16.5 µg/m³ (range in
23 2020 PA: 10.7 µg/m³ to 16.5 µg/m³)
- 24 • Range of mean PM_{2.5} concentrations in studies that use hybrid modeling and apply
25 population-weighting: 9.3 µg/m³ to 12.3 µg/m³
- 26 • Range of mean PM_{2.5} concentrations in studies that use hybrid modeling and do not apply
27 population-weighting: 8.1 µg/m³ to 11.9 µg/m³

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

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

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

- 5 • Range of monitor-based mean PM_{2.5} concentrations: 6.9 µg/m³ to 13.3 µg/m³
- 6 • Range of mean PM_{2.5} concentrations in studies that use hybrid modeling (all of which
7 average up to postal codes and thus include some aspects of population-weighting): 5.9
8 µg/m³ to 9.8 µg/m³

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

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

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

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

1 the distribution to guide the decision on the appropriate standard level (78 FR 3161, January 15,
2 2013). Given the potential for consideration of this information in this reconsideration with
3 regard to the adequacy of the standard level, we note that of the key epidemiologic studies
4 evaluated in the 2019 ISA and draft ISA Supplement, a subset of studies report PM_{2.5}
5 concentrations corresponding to the 25th and 10th percentiles of health data or exposure estimates
6 to provide insight into the concentrations that comprise the lower quartiles of the air quality
7 distributions. In the key U.S. epidemiologic studies that report the 25th and 10th percentiles of
8 health events corresponding to mean PM_{2.5} concentrations (i.e., averaged over the study period
9 for each study city), we note:

- 10 • Monitor-based 25th percentiles of health events correspond to mean PM_{2.5} concentrations
11 (i.e., averaged over the study period for each study city): at or above 11.5 µg/m³
- 12 • Monitor-based 10th percentiles of health events correspond to mean PM_{2.5} concentrations: at
13 or above 9.8 µg/m³
- 14 • PM_{2.5} concentrations corresponding to 25th percentiles of estimated exposures that use hybrid
15 modeling approaches to estimate long-term PM_{2.5} exposures range from 6.2 to 9.2 µg/m³
- 16 • PM_{2.5} concentrations corresponding to 25th percentiles of estimated exposures in studies that
17 uses hybrid modeling to estimate short-term exposures: at or above 6.4 µg/m³
- 18 • PM_{2.5} concentration corresponding to the 25th percentile of estimated exposures in one study
19 with lower concentrations is 4.6 µg/m³
- 20 • PM_{2.5} concentration corresponding to the 10th percentile in the two studies with available
21 information on this percentile range from 4.7 µg/m³ to 7.3 µg/m³.

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

28 **3.5.1.4 Uncertainties in the Health Effects Evidence**

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

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

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

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

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

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

3 Additionally, studies that examine the shapes of concentration-response functions over
4 the full distribution of ambient PM_{2.5} concentrations have not identified a threshold
5 concentration, below which associations no longer exist (U.S. EPA, 2019, section 1.5.3, U.S.
6 EPA, 2021a, sections 2.2.3.1 and 2.2.3.2). While such analyses are complicated by the relatively
7 sparse data available at the lower end of the air quality distribution (U.S. EPA, 2019, section
8 1.5.3), analyses that assess the concentration-response relationship support a linear, no-threshold
9 effect down to 5.0 µg/m³, though uncertainties increase at concentrations of less than 8.0 µg/m³.

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

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

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

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

8 **3.5.2 Risk-based Considerations**

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

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

25 **3.5.2.1 Risk Assessment Analyses**

26 In the risk assessment conducted for this reconsideration, described in detail in section
27 3.4 above and Appendix C, we have estimated PM_{2.5} health risks associated with air quality
28 conditions that just meet the current primary PM_{2.5} standards and potential alternative standard
29 levels. These analyses inform our understanding of the health risks for all-cause or nonaccidental
30 mortality associated with long- and short-term PM_{2.5} exposures. These analyses estimate
31 exposure and risk for populations in 47 urban study areas, as well as subsets of those study areas
32 depending on which of the primary PM_{2.5} standards is controlling in a given study area.

33 The 47 urban study areas were identified as they required relatively small adjustments
34 (<20%) to just meet the current primary PM_{2.5} standards and present a variety of circumstances
35 with regard to risk associated with long- and short-term exposures to PM_{2.5} in ambient air. This

1 set of study areas and the associated populations are intended to be informative to the EPA's
2 consideration of potential risks that may be associated with the air quality conditions that meet
3 the current and potential alternative primary PM_{2.5} standards. The 47 study areas include nearly
4 60 million people ages 30 years or older and illustrate the differences likely to occur across
5 various locations with such air quality as a result of area-specific differences in emissions,
6 meteorological, and population characteristics. While the same conceptual air quality scenarios
7 are simulated in all study areas (i.e., conditions that just meet the existing or alternate standards),
8 source, meteorological and population characteristics in the study areas contribute to variability
9 in the estimated magnitude of risk across study areas.

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

19 In the risk assessment, air quality modeling was used to develop a PM_{2.5} concentration
20 field for 2015 (described in more detail in section 3.4.1.4 and Appendix C). The 2015 PM_{2.5}
21 concentration field was adjusted to simulate just meeting the existing annual and 24-hour
22 standards of 12.0 µg/m³ and 35 µg/m³ and to just meeting potential alternative annual and 24-
23 hour standards of 10.0 µg/m³ and 30 µg/m³. The adjustments made to the PM_{2.5} concentration
24 field are based on assumptions. Changes in PM_{2.5}, in reality, require specific information
25 regarding emissions changes, with concentration gradients of PM_{2.5} varying accordingly across
26 an area. The risk assessment used two adjustment approaches to serve as bounding scenarios for
27 the various ways an alternative standard may be met: (1) preferentially adjusting direct/primary
28 PM emissions, for which changes in PM_{2.5} tend to be more localized near the direct emissions
29 sources of PM (Pri-PM), and (2) preferentially adjusting SO₂ and NO_x precursor emissions to
30 simulate changes in secondarily formed PM_{2.5}, for which reductions in PM_{2.5} tend to be more
31 evenly spread across a study area (Sec-PM). In addition to the air quality modeling approach,
32 linear interpolation and extrapolation were used to simulate just meeting alternative annual
33 standards with levels of 11.0 (interpolated between 12.0 and 10.0 µg/m³), 9.0 µg/m³, and 8.0
34 µg/m³ (both extrapolated from 12.0 and 10.0 µg/m³) in the subset of study areas controlled by
35 the annual standard.

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

19 **3.5.2.2 Estimating Risk under the Current and Alternative Primary PM_{2.5} Standards**

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

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

24 In considering the risk results, we focus first on estimates for the full set of 47 urban
25 study areas. The risk assessment estimates that the current primary PM_{2.5} standards could allow a
26 substantial number of deaths in the U.S., with the large majority of those deaths associated with
27 long-term PM_{2.5} exposures. For example, when air quality in the 47 study areas is adjusted to just
28 meet the current standards, the risk assessment estimates about 41,000 to 45,000 deaths from all-
29 cause mortality in a single year (i.e., for long-term exposures; confidence intervals range from
30 about 30,000 to 59,000) (section 3.4.2.1). For the 30 study areas⁷⁶ where just meeting the current

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

1 standards is controlled by the annual standard,⁷⁷ long-term PM_{2.5} exposures are estimated to be
2 associated with as many as 39,000 (confidence intervals range from about 26,000 to 51,000)
3 deaths from all-cause mortality in a single year (section 3.4.2.2). For the 11 study areas⁷⁸ where
4 just meeting the current standards is controlled by the daily standard,⁷⁹ long-term PM_{2.5}
5 exposures are estimated to be associated with as many as 2,600 (confidence intervals ranging
6 from 1,700 to 3,400) deaths in a single year (section 3.4.2.3). The risk assessment estimates far
7 fewer deaths in a single year for short-term PM_{2.5} exposures as compared to long-term PM_{2.5}
8 exposures, across all of the study area subsets.

9 While the absolute numbers of estimated deaths vary across exposure durations,
10 populations, and concentration-response functions, the general magnitude of risk estimates
11 supports the potential for significant public health impacts in locations meeting the current
12 primary PM_{2.5} standards. This is particularly the case given that the large majority of PM_{2.5}-
13 associated deaths for air quality just meeting the current standards are estimated at annual
14 average PM_{2.5} concentrations from about 10 to 12 µg/m³. These annual average PM_{2.5}
15 concentrations fall within the range of long-term average concentrations over which key
16 epidemiologic studies provide strong support for reported positive and statistically significant
17 health effect associations.

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

20 In the 47 urban study areas, when air quality is simulated to just meet alternative
21 standards, there are substantially larger risk reductions associated with lowering the annual
22 standard then with lowering the 24-hour standard. Risks are estimated to decrease by 13-17%
23 when air quality is adjusted to just meet an alternative annual standard with a level of 10.0 µg/m³
24 or by 1-2% when adjusted to just meet an alternative 24-hour standard with a level of 30 µg/m³
25 (section 3.4.2.1). The percentage decrease when just meet an alternative annual standard with a
26 level of 10.0 µg/m³ corresponds to approximately 7,400 fewer deaths per year (confidence
27 intervals ranging from about 4,100 to 9,800) attributable to long-term PM_{2.5} exposures.

⁷⁷ For these areas, the annual standard is the “controlling standard” because when air quality is adjusted to simulate just meeting the current or potential alternative annual standards, that air quality also would meet the 24-hour standard being evaluated.

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

⁷⁹ For these areas, the 24-hour standard is the controlling standard because when air quality is adjusted to simulate just meeting the current or potential alternative 24-hour standards, that air quality also would meet the annual standard being evaluated. Some areas classified as being controlled by the 24-hour standard also violate the annual standard.

1 In the 30 study areas where just meeting the current and alternative standards is
2 controlled by the annual standard, air quality adjusted to meet alternative annual standards with
3 lower levels is associated with reductions in estimated all-cause mortality risk. These reductions
4 in risk for alternative annual levels are as follows: 7-9% reduction for an alternative annual level
5 of 11.0 $\mu\text{g}/\text{m}^3$, 15-19% reduction for a level of 10.0 $\mu\text{g}/\text{m}^3$, 22-28% reduction for a level of 9.0
6 $\mu\text{g}/\text{m}^3$, and 30-37% reduction for a level of 8.0 $\mu\text{g}/\text{m}^3$ (section 3.4.2.2). For each of these
7 standards, most of the risk remaining is estimated at annual average $\text{PM}_{2.5}$ concentrations that
8 fall somewhat below the alternative standard levels.

9 3.5.2.3 At-Risk Analyses

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

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

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

27 Risk estimates can provide additional information beyond the exposure information to
28 inform our understanding of potentially disproportionate impacts, in this instance by including
29 demographic-specific information on baseline incidence and the relationship between exposure
30 and health effect. Across all air quality scenarios and demographic groups evaluated, Black
31 populations are associated with the largest $\text{PM}_{2.5}$ -attributable mortality risk rate per 100,000
32 people, while White populations are associated with the smallest $\text{PM}_{2.5}$ -attributable mortality
33 risk rate (section 3.4.2.4, Figure 3-20). Generally, as the levels of potential alternative annual
34 $\text{PM}_{2.5}$ standards decrease in the 30 areas controlled by the annual standard, the average reduction
35 in $\text{PM}_{2.5}$ concentration and mortality risk rates increase across all demographic populations
36 (section 3.4.2.4, Figure 3-21).

1 In comparing the reductions in average national PM_{2.5} concentrations and risk rates
2 within each demographic population, we note that the average percent PM_{2.5} concentrations and
3 risk reductions are slightly greater in the Black population than in the White population for each
4 alternative standard evaluated (11.0 µg/m³, 10.0 µg/m³, 9.0 µg/m³, and 8.0 µg/m³), when shifting
5 from the current annual PM_{2.5} standard (12.0 µg/m³) in the full set of 47 areas and the subset of
6 30 areas controlled by the annual standard. We further note that the difference in average percent
7 risk reductions increases slightly more in Blacks than in Whites as the level of the potential
8 alternative annual standard decreases (section 3.4.2.4, Table 3-19 and Table 3-20).

9 **3.5.2.4 Uncertainties**

10 In this section, we consider uncertainties associated with the quantitative estimates of risk
11 in the overall risk assessment and from risk rates and exposure estimates in the at-risk analysis
12 (sections 3.4.2.5, 3.4.1.7, and 3.4.1.8). Variability and uncertainty associated with the risk
13 estimates are assessed using several quantitative and qualitative approaches, as described in more
14 detail in section C.3 of Appendix C. Generally, the quantitative uncertainty characterization
15 approaches include the following: (1) evaluating multiple concentration-response functions for
16 the same health endpoint; (2) evaluating multiple methods for simulating air quality scenarios;
17 and (3) characterizing the 95% confidence intervals associated with risk estimates. The
18 qualitative uncertainty characterization approach is based on WHO (2008) guidance and on
19 guidance documents developed by the EPA (U.S. EPA, 2001, U.S. EPA, 2004). This qualitative
20 approach includes an assessment of both the magnitude and direction of impact of those
21 uncertainties on risk estimates, including three levels of classification for the magnitude: low,
22 medium, and high.⁸⁰

- 23 • **What are the key uncertainties associated with the risk estimates and at-risk**
24 **analysis, including those of particular significance with regard to drawing**
25 **conclusions as to the adequacy of the protection afforded by the current primary**
26 **PM_{2.5} standards?**

27 Based on the uncertainty characterization and associated analyses in the risk assessment
28 and consideration of associated policy implications, we recognize several areas of uncertainty as
29 particularly important in our consideration of the risk estimates, as was also the case in previous
30 reviews, and in the risk rates and exposure and risk reductions in the at-risk analysis.

⁸⁰ The classification of the magnitude of impact for sources of uncertainty includes three levels: (a) low (unlikely to produce a sufficient impact on risk estimates to affect their interpretation), (b) medium (potential to have a sufficient impact to affect interpretation), and (c) high (likely to have an impact sufficient to affect interpretation). For several of the sources, a classification was provided between these levels (e.g., low-medium, medium-high). More information is available in Appendix C, section C.3.

1 With regard to the concentration-response relationships, we recognize that the degree to
2 which different concentration-response functions result in different risk estimates could reflect
3 differences in study design and/or populations evaluated, as well as other factors. We also note
4 uncertainty in the risk assessment associated with the interpretation of the shapes of
5 concentration-response relationships, particularly at PM_{2.5} concentrations near the lower end of
6 the air quality distribution. This interpretation is complicated by relatively low data density in the
7 lower concentration range, the possible influence of exposure measurement error, and variability
8 among individuals with respect to air pollution health effects. These sources of variability and
9 uncertainty tend to smooth and “linearize” population-level concentration-response functions,
10 and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015b,
11 section 6.c). As described in section 3.3.1, the 2019 ISA concludes and the draft ISA Supplement
12 provides further support that the majority of evidence of long-term PM_{2.5} exposure and mortality
13 supports a linear, no-threshold concentration-response relationship, though there is initial
14 evidence indicating that the slope of the concentration-response curve may be steeper at lower
15 concentrations for cardiovascular mortality (U.S. EPA, 2019, section 1.5.3.2; U.S. EPA, 2021a,
16 section 3.2.2.2). The 2019 ISA and draft ISA Supplement note that there is less certainty in the
17 shape of the concentration-response curve at mean annual PM_{2.5} concentrations generally below
18 8 µg/m³ because data density is reduced below this concentration (U.S. EPA, 2019, section
19 11.2.4; U.S. EPA, 2021a, section 3.2.2.2.7). As described in more detail in section 3.4.2.5 above
20 and Appendix C, a portion of risk modeling in the risk assessment does include locations with
21 annual ambient PM_{2.5} concentrations adjusted to below 8 ug/m³, so there is the potential for
22 significant uncertainty being introduced into the risk assessment (particularly for that portion of
23 risk modeled at or below 8 ug/m³). With regard to short-term PM_{2.5} exposure and mortality, the
24 2019 ISA concludes and the draft ISA Supplement provides additional support that, while
25 difficulties remain in assessing the shape of the PM_{2.5}-mortality concentration-response
26 relationship and studies have not conducted systematic evaluations of alternatives to linearity,
27 recent studies continue to provide evidence of a no-threshold linear relationship, with less
28 confidence at concentrations lower than 5 µg/m³ (U.S. EPA, 2021a, section 3.2.1.2.6). However,
29 we note that in most instances in the risk assessment for this reconsideration, the concentration-
30 response function used had only a small impact on the risk estimates.

31 With regard to the method for simulating air quality scenarios, the approach used to
32 adjust air quality (i.e., adjusting primary PM emissions or secondary PM emission precursors)
33 had some impact on the overall risk estimates. We also note that there may be uncertainty
34 associated with the methods used to simulate air quality scenarios just meeting the current and
35 potential alternative primary PM_{2.5} standards. The model-based methods for simulating air
36 quality scenarios that just meet the current and alternative standards could contribute to

1 uncertainties associated with the PM_{2.5} concentration estimates used in the risk assessment and
2 at-risk analyses. While state-of-the-science modeling methods were used to fill in the spatial and
3 temporal gaps in monitoring data, model-related biases and errors can introduce uncertainties.
4 Additionally, the modeling scenarios are based on “across-the-board” changes in primary PM_{2.5}
5 or NO_x and SO₂ emissions from all anthropogenic sources throughout the U.S. by fixed
6 percentages. While this approach tends to target the key sources in each area, emission changes
7 are not tailored to specific periods or sources. Furthermore, while the two adjustment approaches
8 that were applied span a wide range of emissions conditions, they represent a subset of the
9 possible emissions cases that could be used to adjust PM_{2.5} concentrations. In addition, when
10 simulating air quality scenarios that just meet potential alternative annual PM_{2.5} standards using
11 linear extrapolation/interpolation, we recognize that this approach does not fully capture the
12 potential non-linearities associated with real-world changes in air quality. However, it is
13 important to note that the adjustment approach had a larger impact on the distribution of risk
14 reductions, particularly for potential alternative annual standard levels of 9.0 and 8.0 µg/m³.

15 It is important to note that the air quality adjustment approaches applied in the risk
16 assessment differ from the development and modeling of emission control strategies that would
17 occur in implementing a standard. In implementing a standard, an appropriately defined
18 nonattainment area would reduce emissions of primary PM and/or PM precursors selected
19 through analysis of site-specific conditions to meet a standard that is exceeded. In the risk
20 assessment, gridded concentration fields over CBSAs were adjusted to higher or lower
21 concentrations to correspond to just meet standards based on emission changes applied
22 throughout the U.S. Two emission adjustment cases (primary PM and NO_x and SO₂) were used
23 to provide concentration fields that span a wide range of realistic spatial patterns, but the air
24 quality modeling for the risk assessment is not designed to reflect emission changes that might
25 occur in implementing a standard. The Regulatory Impact Analysis (RIA) associated with
26 NAAQS revisions provides illustrative estimates of emission changes needed to meet potential
27 alternative standards and more closely reflects implementation considerations (U.S. EPA, 2013,
28 U.S. EPA, 2015a).

29 We further note that there is considerable variation in the range of confidence intervals
30 associated with the point estimates generated in the risk assessment, with some concentration-
31 response functions displaying greater variability than others. A number of factors could
32 potentially influence the varying degrees of statistical precision in effect estimates, including
33 sample size, exposure measurement error, degree of control for confounders/effect modifiers,
34 and variability in PM_{2.5} concentrations evaluated in the original epidemiologic study.

35 There may also be uncertainty associated with the potential confounding of the PM_{2.5}-
36 mortality effect and the omission of potential confounders from analyses could either increase or

1 decrease the magnitude of PM_{2.5} effect estimates. Not accounting for confounders can introduce
2 uncertainty into the effect estimates, and thereby introduce uncertainty into the risk estimates that
3 are generated using those effect estimates. While various approaches to control for potential
4 confounders have been adopted across the epidemiologic studies assessed in the 2019 ISA and
5 draft ISA Supplement, and those used in the risk assessment, no individual study adjusts for all
6 potential confounders.

7 In addition to the uncertainty associated with the risk assessment estimates, additional
8 uncertainties are associated with the risk rates, exposure estimate, and risk reductions in the at-
9 risk analysis. As an initial matter, we note that this analysis is based on race- and ethnicity-
10 stratified concentration-response functions only for ages 65 and over (Di et al., 2017b). The use
11 of one study in such an analysis introduces uncertainties and limitations in the broad applicability
12 of such results in the context of the national U.S. population across demographic groups and age
13 ranges. In addition, each non-White demographic group analyzed in the study comprised a
14 smaller percentage of the full study population, which reduces analytical power. Finally, the risk
15 and exposure assessment focuses on urban areas. This means that demographic groups that
16 preferentially reside in rural areas, such as Native Americans, are underrepresented in this
17 analysis. Additionally, average exposure concentrations estimated for demographic groups with
18 substantial rural populations, such as Whites, may be overestimated in this urban analysis.

19 In summary, here we recognize several particularly important uncertainties that affect the
20 quantitative estimates of risk rates and exposure in the at-risk analysis and their interpretation in
21 the context of considering the current primary PM_{2.5} standards. These include uncertainties
22 related to the modeling and adjustment methods for simulating air quality scenarios; the potential
23 influence of confounders on the relationship between PM_{2.5} exposure and mortality; the
24 interpretation of the shapes of concentration-response functions, particularly at lower
25 concentrations; and limited availability of studies to inform the at-risk analysis.

26 **3.5.2.5 Potential Public Health Implications**

27 In considering the public health implications of the quantitative risk assessment and at-
28 risk analysis that may inform the Administrator's judgments in this area, this section discusses
29 the information pertaining to the following questions.

- 30 • **To what extent are the estimates of risk important from a public health perspective?**
31 **What does the information available in this reconsideration indicate with regard to**
32 **the size of the at-risk populations?**

33 Several factors are important to consideration of public health implications. These
34 include the magnitude or severity of the effects associated with the estimated exposures, as well
35 as their adversity at the individual and population scales. Other important considerations include

1 the size of the population estimated to experience such effects or to experience exposures
2 associated with such effects. Thus, the discussion here reflects consideration of the risk-based
3 evidence in the context of potential health implications in previous NAAQS decisions.

4 With regard to PM_{2.5} concentrations in ambient air, the public health implications and
5 potential public health impacts of interest in this reconsideration relate to those effects where a
6 causal relationship with PM_{2.5} exposure was concluded. These are mortality and cardiovascular
7 effects related to both long- and short-term exposures, as summarized in section 3.3.1 above.
8 Such effects, including more serious effects such as mortality, can be considered severe from a
9 public health perspective.

10 In considering public health implications, it is important to consider impacts on
11 population groups of differing susceptibility. The size of the at-risk populations (children, older
12 adults, those with pre-existing cardiovascular or respiratory diseases) in the U.S. is substantial.
13 As summarized in section 3.3.2, more than 22% of the population are children (<18 years old;
14 approximately 73 million people) and about 16% are older adults (65+ years old; approximately
15 54 million people). For adults in the U.S. 18 years old and older, cardiovascular diseases are
16 most prevalent in adult populations over the age of 65, with 29% of this age group reporting
17 some type of heart disease (Table 3-3 above). Similarly, adults over the age of 65 also have a
18 greater prevalence of respiratory diseases, particularly COPD reported as chronic bronchitis or
19 emphysema, while the asthma prevalence is generally consistent across all adult age groups for
20 those 18 years or older (Table 3-3). It is important to note that for older adults, the increased risk
21 in this lifestage can likely be attributed to the gradual decline in physiological processes that
22 occurs with aging, and some overlap exists between populations considered to be at-risk because
23 of pre-existing disease and lifestage (U.S. EPA, 2019, p. 12-25).

24 Another factor that may contribute to differences PM_{2.5} exposures and PM_{2.5}-related
25 health risk is race/ethnicity. As described above in section 3.3.2 and in the 2019 ISA and draft
26 ISA Supplement, there is strong evidence demonstrating that Black and Hispanic populations, in
27 particular, have higher PM_{2.5} exposures and health risk disparities compared to non-Hispanic
28 White populations. In the U.S., more than 12% of the U.S. population (more than 40.5 million
29 people) are Blacks and more than 18% are Hispanics (more than 60 million people), while 60%
30 of the population (nearly 197 million people) are non-Hispanic Whites (Table 3-2). Black and
31 Hispanic individuals of all ages make up a substantial portion of the population.

32 In considering the public health implications of the risk estimates across the study areas,
33 we note the purpose for the study areas is to illustrate circumstances that may occur in areas that
34 just meet the current or potential alternative standards, and not to estimate risk associated with
35 conditions occurring in those specific locations currently. We note that some areas across the U.S.
36 have air quality for PM_{2.5} that is near or above the existing standards. Thus, the air quality and

1 exposure circumstances assessed in the study areas in the risk assessment are of particular
2 importance in considering whether the currently available information calls into question the
3 adequacy of the public health protection afforded by the current standards.

4 The risk estimates for the study areas assessed in this reconsideration reflect differences
5 in exposure circumstances among those areas and illustrate the exposures and risks that might be
6 expected to occur in other areas with such circumstances under air quality conditions that just
7 meet the current standards or the alternative standards assessed. Thus, the exposure and risk
8 estimates indicate the magnitude of exposure and risk that might be expected in many areas of
9 the U.S. with PM_{2.5} concentrations at or near the current or alternative standards. Although the
10 methodologies and data used to estimate risks in this reconsideration differ in several ways from
11 what was used in the 2020 review, the findings and considerations summarized here present a
12 pattern of exposure and risk that is generally similar to that considered in the 2020 review, and
13 indicate a level of protection generally consistent with that described in the 2020 PA.

14 In summary, the considerations raised here are important to conclusions regarding the
15 public health significance of the risk assessment results. Specifically, we note that available
16 evidence and information suggests that both long- and short-term PM_{2.5} exposures are associated
17 with adverse health effects, including more severe effects such as mortality. In addition, we note
18 that such effects impact large segments of the U.S. population, including those populations that
19 may have other factors that influence risk (i.e., lifestage, pre-existing cardiovascular and
20 respiratory diseases, race/ethnicity), as well as disparities in PM_{2.5} exposures and health risks
21 based on race and ethnicity. Therefore, we recognize that the air quality allowed by the current
22 primary PM_{2.5} standards could be judged to be associated with significant public health risk. We
23 recognize that such conclusions also depend in part on public health policy judgments that will
24 weigh in the Administrator's decision in this reconsideration with regard to the adequacy of
25 protection afforded by the current standards. Such judgments that are common to NAAQS
26 decisions include those related to public health implications of effects of differing severity. Such
27 judgments also include those concerning the public health significance of effects at exposures for
28 which evidence is limited or lacking, such as effects at lower concentrations than those
29 demonstrated in the key epidemiologic studies and in those population groups for which
30 population-specific information, such as concentration-response functions, are not available from
31 the epidemiologic literature.

32 **3.5.3 Preliminary Conclusions**

33 This section describes our preliminary conclusions for the Administrator's consideration
34 in this reconsideration of the primary PM_{2.5} standards. These preliminary conclusions are based
35 on considerations described in the sections above, and in the discussion below regarding the

1 scientific evidence (as summarized in the 2019 ISA (U.S. EPA, 2019) and the draft ISA
2 Supplement (U.S. EPA, 2021a)), the quantitative assessments of PM_{2.5}-associated health risks,
3 and analyses of PM_{2.5} air quality.

4 **3.5.3.1 Current Standards**

5 In taking into consideration the discussions responding to specific questions above in this
6 chapter, this section addresses the following overarching policy question.

- 7 • **Does the currently available scientific evidence and risk-based information support**
8 **or call into question the adequacy of the public health protection afforded by the**
9 **current annual and 24-hour PM_{2.5} standards?**

10 In considering this question, we recognize that, as is the case with NAAQS reviews in
11 general, the extent to which the current primary PM_{2.5} standards are judged to be adequate will
12 depend on a variety of factors, including science policy judgments and public health policy
13 judgments to be made by the Administrator. These factors include public health policy
14 judgments concerning the appropriate PM_{2.5} concentrations on which to place weight, as well as
15 judgments on the public health significance of the effects that have been observed at the
16 exposures evaluated in the health effects evidence. The factors relevant to judging the adequacy
17 of the standards also include the interpretation of, and decisions as to the weight to place on,
18 different aspects of the results of the risk assessment for the study areas included and the
19 associated uncertainties. Thus, we recognize that the Administrator's conclusions regarding the
20 adequacy of the current standards will depend in part on judgments regarding aspects of the
21 evidence and risk estimates, and judgments about the degree of protection that is requisite to
22 protect public health with an adequate margin of safety.

23 Our response to the overarching question above takes into consideration the discussions
24 that address the specific policy-relevant questions in prior sections of this document (sections
25 3.3, 3.4, 3.5.1, and 3.5.2) and builds on the approach from previous reviews (summarized in
26 section 3.1 above). We focus first on consideration of the evidence, including that assessed in the
27 2019 ISA and the draft ISA Supplement, and the extent to which it alters key conclusions
28 supporting the current standards. We then turn to consideration of the quantitative estimates of
29 risk developed in this reconsideration, including associated uncertainties and limitations, and the
30 extent to which they indicate differing conclusions regarding the magnitude of risk, as well as
31 level of protection from adverse effects, associated with the current standards. We additionally
32 consider the key aspects of the evidence and risk estimates emphasized in establishing the
33 current standards, and the associated public health policy judgments and judgments about the
34 uncertainties inherent in the scientific evidence and quantitative analyses that are integral to
35 decisions on the adequacy of the current primary PM_{2.5} standards.

1 We first note that our approach recognizes that the current annual standard (based on
2 arithmetic mean concentrations) and 24-hour standard (based on 98th percentile concentrations),
3 together, are intended to provide public health protection against the full distribution of short-
4 and long-term PM_{2.5} exposures. In general, the annual standard is most effective at controlling
5 exposures to “typical” daily PM_{2.5} concentrations that are experienced over the year, while the
6 24-hour standard, with its 98th percentile form, is most effective at limiting peak daily or 24-
7 hour PM_{2.5} concentrations. In considering the combined effects of these standards, we recognize
8 that changes in PM_{2.5} air quality designed to meet an annual standard would likely result not only
9 in lower short- and long-term PM_{2.5} concentrations near the middle of the air quality distribution,
10 but also in fewer and lower short-term peak PM_{2.5} concentrations. Additionally, changes
11 designed to meet a lower 24-hour standard, with a 98th percentile form, would most effectively
12 result in fewer and lower peak 24-hour PM_{2.5} concentrations, but also have an effect on lowering
13 the annual average PM_{2.5} concentrations. Thus, our focus in evaluating the current primary
14 standards is on the protection provided by the combination of the annual and 24-hour standards
15 against the distribution of both short- and long-term PM_{2.5} exposures.

16 As an initial matter, we note the longstanding body of health evidence supporting
17 relationships between PM_{2.5} exposures (short- and long-term) and mortality or serious morbidity
18 effects. The evidence available in this reconsideration (i.e., assessed in U.S. EPA, 2019 and U.S.
19 EPA, 2021a) and summarized above in section 3.3.1 and section 3.5.1) reaffirms, and in some
20 cases strengthens, the conclusions from the 2009 ISA regarding the health effects of PM_{2.5}
21 exposures (U.S. EPA, 2009). As noted above, epidemiologic studies conducted in North
22 America, Europe, or Asia demonstrate generally positive, and often statistically significant,
23 PM_{2.5} health effect associations. Such studies report associations between estimated PM_{2.5}
24 exposures and non-accidental, cardiovascular, or respiratory mortality; cardiovascular or
25 respiratory hospitalizations or emergency room visits; and other mortality/morbidity outcomes
26 (e.g., lung cancer mortality or incidence, asthma development). Recent experimental evidence, as
27 well as evidence from panel studies, strengthens support for potential biological pathways
28 through which PM_{2.5} exposures could lead to the serious effects reported in many population-
29 level epidemiologic studies, including support for pathways that could lead to cardiovascular,
30 respiratory, nervous system, and cancer-related effects.

31 Epidemiologic studies in the U.S. report health effect associations with mortality and/or
32 morbidity across multiple cities and in diverse populations, including in studies examining
33 populations and lifestages that may be at comparatively higher risk of experiencing a PM_{2.5}-
34 related health effect (e.g., older adults, children). Further, these studies use a variety of statistical
35 designs, and employ a variety of methods to examine exposure measurement error as well as to
36 control for confounding effects, including more recent causal modeling studies. Results of these

1 analyses support the robustness of the reported associations. Additional findings from an
2 expanded body of studies that employ causal modeling and accountability methods further
3 inform the causal nature of the relationship between long- or short-term term PM_{2.5} exposure and
4 mortality (U.S. EPA, 2019, sections 11.1.2.1, 11.2.2.4,U.S. EPA, 2021a, sections 3.1.1.3, 3.1.2.3,
5 3.2.1.3, and 3.2.2.3). These studies, summarized above in Table 3-11 and Table 3-12, examine
6 both short- and long-term PM_{2.5} exposure and cardiovascular effects and mortality, and using a
7 variety of statistical methods to control for confounding bias, consistently report positive
8 associations, which further supports the broader body of epidemiologic evidence for both
9 cardiovascular effects and mortality. Moreover, recent epidemiologic studies strengthen support
10 for health effect associations at relatively low ambient PM_{2.5} concentrations. Studies that
11 examine the shapes of concentration-response relationships over the full distribution of ambient
12 PM_{2.5} concentrations have not identified a threshold concentration, below which associations no
13 longer exist (U.S. EPA, 2019, section 1.5.3, U.S. EPA, 2021a, sections 2.1.1.5.1 and 2.1.1.5.2).
14 While such analyses are complicated by the relatively sparse data available at the lower end of
15 the air quality distribution (U.S. EPA, 2019, section 1.5.3), several studies report positive and
16 statistically significant associations in additional analyses restricted to annual average PM_{2.5}
17 exposures below 12 µg/m³ or to daily exposures below 35 µg/m³ as exhibited in Table 3-10.

18 These and other recent studies provide support for health effect associations at lower
19 ambient PM_{2.5} concentrations than in previous reviews. In this reconsideration, a large number of
20 key studies report positive and statistically significant associations for air quality distributions
21 with lower overall mean PM_{2.5} concentrations (i.e., Figure 3-8, Figure 3-9, Figure 3-10, Figure 3-
22 11). Consistent with the 2012 review, it is important to consider the manner in which PM_{2.5} mean
23 concentrations are estimated (e.g., monitored concentrations versus modeled concentrations) and
24 the method by which means are calculated and reported as the overall mean PM_{2.5} concentration
25 (e.g., averaging across all grid cells in an urban area versus population-weighting). Additional
26 analyses, new in this draft PA though similar to those in the 2012 review, suggest that the area
27 annual design value is generally greater than the study mean by 10-20% (monitor-based studies),
28 14-18% (hybrid modeling with population-weighting) or 40-50% (hybrid modeling without
29 population weighting). We note this information relative to the overall mean PM_{2.5}
30 concentrations in key U.S. epidemiologic studies which are: 9.9 µg/m³ to 16.5 µg/m³ for monitor-
31 based studies; 9.3 µg/m³ to 12.3 µg/m³ for studies that use hybrid modeling and apply
32 population-weighting; and 8.1 µg/m³ to 11.9 µg/m³ for studies that use hybrid modeling and do
33 not apply population-weighting. The study reported mean concentrations in Canadian studies are
34 more difficult to compare to the area annual standard design value but are lower than those
35 reported in the U.S. studies for both monitor-based and hybrid model methods, ranging from 7.0
36 µg/m³ to 9.0 µg/m³ in monitor-based studies, and 6.0 µg/m³ to 10.0 µg/m³ in model-based

1 studies. These mean values are consistent with the mean PM_{2.5} concentrations reported in studies
2 available at the time of the 2020 review (U.S. EPA, 2020, Figure 3-8).

3 In assessing the adequacy of the current standard, we examine a subset of studies, many
4 of which are newly available in this reconsideration, that employ causal modeling methods to
5 control for confounding bias (Table 3-11), which report positive and significant associations for
6 a variety of health outcomes and support the positive and significant associations in analyses
7 identified as key epidemiologic studies above. We also evaluate what the accountability studies
8 may indicate with respect to improvements in public health with improvements in air quality. In
9 so doing, we take note of two accountability studies (Sanders et al., 2020 and Corrigan et al.,
10 2018) newly available in this reconsideration with starting concentrations at or below 12.0 µg/m³
11 that indicate positive and significant associations with mortality and reductions in ambient PM_{2.5}
12 (Table 3-12). We further evaluate studies with analyses that restrict annual or daily PM_{2.5}
13 concentrations to values below the annual or daily PM_{2.5} standard, respectively (Table 3-10).
14 These restricted analyses indicate positive and significant associations, including mean PM_{2.5}
15 concentrations presumably below the mean reported PM_{2.5} in the main cohort, where long-term
16 mean PM_{2.5} concentrations range from 8.2 µg/m³ to 11.5 µg/m³, as well as effect estimates that
17 are generally greater in magnitude than effect estimates seen in main analyses.

18 In addition to the epidemiologic evidence, we examine experimental studies, including
19 controlled human exposure studies and animal toxicological studies. As detailed in above in
20 section 3.3.3.1 and section 3.5.1.3, these studies provide support for the effects of exposure to
21 PM_{2.5}, and support for biologically plausible mechanisms through which adverse human health
22 outcomes could occur. Exposures in controlled human exposure studies last from less than one
23 hour and up to five hours, and indicate that the most consistent evidence is associated with
24 cardiovascular effects, and more specifically, impaired vascular function. PM_{2.5} exposures
25 evaluated in most of these studies are well-above the ambient concentrations typically measured
26 in locations meeting the current primary standards. For example, at air quality monitoring sites
27 meeting the current primary PM_{2.5} standards (i.e., the 24-hour standard and the annual standard),
28 the 2-hour concentrations generally remain below 10 µg/m³, and virtually never exceed 30
29 µg/m³. Two-hour concentrations are higher at monitoring sites violating the current standards,
30 but generally remain below 16 µg/m³ and virtually never exceed 80 µg/m³. In addition, as noted
31 earlier in section 3.3.3.1, chronic vascular dysfunction can be judged to be a biomarker of an
32 adverse health effect from air pollution, but the health relevance of acute reductions in vascular
33 function are less certain (Thurston et al., 2017). Thus, while these studies are important in
34 establishing biological plausibility, it is unclear how the results alone and the importance of the
35 effects observed in these studies, particularly in studies conducted at near-ambient PM_{2.5}
36 concentrations, should be interpreted with respect to adversity to public health.

1 In addition to the evidence above, we also consider what the risk assessment indicates
2 with regard to the adequacy of the current primary PM_{2.5} standards. The risk assessment
3 estimates that the current primary PM_{2.5} standards could allow a substantial number of deaths in
4 the U.S., with the large majority of those deaths associated with long-term PM_{2.5} exposures. For
5 example, when air quality in the 47 study areas is adjusted to simulate just meeting the current
6 standards, the risk assessment estimates 40,600-45,100 long-term PM_{2.5} exposure-related deaths
7 in a single year, with confidence intervals ranging from 30,300-59,000. While the absolute
8 numbers of estimated deaths vary across exposure durations, populations, and concentration-
9 response functions, the general magnitude of risk estimates supports the potential for significant
10 public health impacts in locations meeting the current primary PM_{2.5} standards. This is
11 particularly the case given that the large majority of PM_{2.5}-associated deaths for air quality just
12 meeting the current standards are estimated at annual average PM_{2.5} concentrations from about
13 10 to 12 µg/m³. These annual average PM_{2.5} concentrations fall well-within the range of long-
14 term average concentrations over which key epidemiologic studies provide strong support for
15 reported positive and statistically significant PM_{2.5} health effect associations.

16 Based on the information summarized above, and discussed in more detail in sections 3.3,
17 3.4, and 3.5 of this draft PA, we particularly note the following in reaching preliminary
18 conclusions on the current primary PM_{2.5} standards:

- 19 • There is a long-standing body of strong health evidence demonstrating relationships between
20 long- or short-term PM_{2.5} exposures and a variety of outcomes, including mortality and
21 serious morbidity effects. Studies assessed in the 2019 ISA and the draft ISA Supplement
22 have reduced key uncertainties and broadened our understanding of the health effects that
23 can result from exposures to PM_{2.5}.
 - 24 - Recent U.S. and Canadian epidemiologic studies provide support for generally
25 positive and statistically significant health effect associations across a broad
26 range of ambient PM_{2.5} concentrations, including for air quality distributions
27 with overall mean concentrations lower than in the previous reviews.
 - 28 - Controlled human exposure studies and animal toxicological studies provide
29 support for the effects of exposure to PM_{2.5}, and support for biologically
30 plausible mechanisms through which adverse human health outcomes could
31 occur.
 - 32 - Epidemiologic studies that use causal modeling methods have expanded since
33 the 2020 PA and further inform the causal nature of the relationship between
34 short- and long-term exposure to PM_{2.5} and mortality and cardiovascular
35 effects. These studies use a variety of statistical methods to reduce
36 uncertainties with respect to confounding bias.
- 37 • Recent U.S. accountability studies provide support for improvements in public health,
38 including reductions in mortality in studies with starting PM_{2.5} concentrations at or below the
39 current primary PM_{2.5} annual standard. Some epidemiologic studies (Corrigan et al., 2018
40 and Sanders et al., 2020) that employ accountability methods using monitored data evaluate

1 the effect of the implementation of the 1997 annual PM_{2.5} standard, finding evidence of
2 reductions in mortality in areas with starting PM_{2.5} concentrations at or below 12.0 µg/m³.

- 3 • Studies that restrict analyses to air quality below the current daily or annual PM_{2.5} standard
4 exhibit positive and significant associations, which are often greater in magnitude than main
5 analyses. Di et al. (2017b) and Dominici et al. (2019) report positive and statistically
6 significant associations that are greater in analyses restricted below 12.0 µg/m³ and report
7 mean concentrations of 9.6 µg/m³. In studies that restrict analyses < 35.0 µg/m³ or lower,
8 mean PM_{2.5} concentrations are not reported, though such means are presumably somewhat
9 below those based on the overall cohort, which range from 8.2 µg/m³ to 11.5 µg/m³, and
10 effect estimates are generally great than those in the overall cohort. More specifically, one
11 U.S. study by Shi et al. (2016) reports positive and statistically significant associations in
12 analyses restricted to relatively low annual or 24-hour PM_{2.5} exposure estimates.
- 13 • Exposures in controlled human exposure studies last from less than one hour and up to five
14 hours and indicate that the most consistent evidence is associated with cardiovascular effects,
15 and more specifically, impaired vascular function. Further, air quality analyses suggest that
16 the ambient concentrations in these studies typically do not occur in locations meeting the
17 current primary standards, thus suggesting that the current primary PM_{2.5} standards provide
18 protection against these “peak” concentrations.
- 19 • We note the decision framework used in previous reviews that places significant weight on
20 key epidemiologic studies and consider whether the mean concentrations in these studies
21 would be allowed in areas meeting the current primary standard.
 - 22 - Such a decision framework placed significant weight on epidemiologic studies
23 that assessed associations between PM_{2.5} exposure and health outcomes that
24 were most strongly supported by the body of scientific evidence and
25 recognized there is significantly greater confidence in the magnitude and
26 significance of observed associations for the part of the air quality distribution
27 corresponding to where the bulk of the health events in each study have been
28 observed, generally at or around the mean concentration.
 - 29 - Additional analyses, new in this draft PA though similar to analyses in the
30 2012 review, suggest that the area annual design value is greater than the
31 study reported mean values by 10-20% (monitor-based studies), 14-18%
32 (hybrid modeling with population-weighting) or 40-50% (hybrid modeling
33 without population weighting).
 - 34 - Focusing on the key epidemiologic studies available in this reconsideration,
35 the overall mean PM_{2.5} concentrations in key U.S. epidemiologic studies are
36 as follows:
 - 37 ○ Range of monitor-based mean PM_{2.5} concentrations is from 9.9 µg/m³
38 to 16.5 µg/m³ (range in 2020 PA: 10.7 µg/m³ to 16.5 µg/m³)
 - 39 ○ Range of mean PM_{2.5} concentrations in studies that use hybrid
40 modeling and apply population-weighting: 9.3 µg/m³ to 12.3 µg/m³
 - 41 ○ Range of mean PM_{2.5} concentrations in studies that use hybrid
42 modeling and do not apply population-weighting: 8.1 µg/m³ to 11.9
43 µg/m³

- 1 - Though the Canadian studies are more difficult to utilize for comparison to the
2 annual design value used to determine compliance in the U.S., the overall
3 mean PM_{2.5} concentrations in key Canadian epidemiologic studies are within
4 the range, though somewhat lower than those from the U.S. studies, and are as
5 follows:
- 6 ○ Range of monitor-based mean PM_{2.5} concentrations is from 6.9 µg/m³
7 to 13.3 µg/m³
 - 8 ○ Range of mean PM_{2.5} concentrations in studies that use hybrid
9 modeling (all of which average up to postal codes and thus include
10 some aspects of population-weighting) is 5.9 µg/m³ to 9.8 µg/m³
- 11 - Past decision frameworks also placed some weight on considering the annual
12 standard level relative to the 25th and 10th percentile of health events while
13 also noting that epidemiologic studies provide more limited support for health
14 effect associations based on air quality distributions at these lower PM_{2.5}
15 concentration percentiles.
- 16 ○ In key U.S. epidemiologic studies that use monitors to estimate PM_{2.5}
17 exposures, 25th percentiles of health events correspond to mean PM_{2.5}
18 concentrations (i.e., averaged over the study period for each study city)
19 at or above 11.5 µg/m³ and 10th percentiles of health events correspond
20 to mean PM_{2.5} concentrations at or above 9.8 µg/m³
 - 21 ○ Of the key U.S. epidemiologic studies that use hybrid modeling
22 approaches to estimate long-term PM_{2.5} exposures and do not apply
23 population-weighting, the ambient PM_{2.5} concentrations corresponding
24 to 25th percentiles of estimated exposures range from 4.6 µg/m³ to 9.2
25 µg/m³, while in studies that do apply population-weighting, 25th
26 percentiles range from 6.7 µg/m³ to 9.1 µg/m³. In the two studies (each
27 apply population-weighting) with information available on the 10th
28 percentile of health events, the ambient PM_{2.5} concentrations
29 corresponding to the 10th percentile are 4.7 µg/m³ and 7.3 µg/m³.
- 30 ● The risk assessment estimates that the current primary PM_{2.5} standards could allow a
31 substantial number of PM_{2.5}-associated deaths in the U.S. The large majority of these
32 estimated deaths are associated with the annual average PM_{2.5} concentrations near (and above
33 in some cases) the average concentrations in key epidemiologic studies reporting positive and
34 statistically significant health effect associations. Further, the risk assessment estimated that
35 Black populations may experience disproportionately higher exposures and risk under
36 simulated air quality conditions just meeting the current primary PM_{2.5} annual standard as
37 compared to White populations.

38 When taken together, we reach the conclusion that the available scientific evidence, air quality
39 analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling
40 into question the adequacy of the public health protection afforded by the combination of the
41 current annual and 24-hour primary PM_{2.5} standards. In particular, we note the information and
42 analyses new to this reconsideration (and discussed in detail above) in reaching this conclusion.

3.5.3.2 Potential Alternative Standards

In this section, we consider the potential alternative primary PM_{2.5} standards that could be supported by the evidence and quantitative information available in this reconsideration. These considerations are framed by the following overarching policy-relevant question, posed at the beginning of this chapter:

- **What is the range of potential alternative standards that could be supported by the available scientific evidence and risk-based information to increase public health protection against short- and long-term fine particle exposures?**

In answering this question, we consider each of the elements of the annual and 24-hour PM_{2.5} standards: indicator, averaging time, form, and level. The sections below discuss our consideration of these elements, and our conclusions that (1) it is appropriate to consider revising the level of the current annual standard, in conjunction with retaining the current indicator, averaging time, and form of that standard, to increase public health protection against fine particle exposures and (2) depending on the decision made on the annual standard, consideration could be given to either retaining or revising the level of the 24-hour PM_{2.5} standard.

3.5.3.2.1 Indicator

In initially setting standards for fine particles in 1997, the EPA concluded it was appropriate to control fine particles as a group, rather than singling out any particular component or class of fine particles. The Agency noted that community health studies had found significant health effect associations using various indicators of fine particles, and that health effects in a large number of areas had significant mass contributions from differing components or sources of fine particles. In addition, a number of toxicological and controlled human exposure studies had reported health effects following exposures to high concentrations of numerous fine particle components (62 FR 38667, July 18, 1997). In establishing a size-based indicator in 1997 to distinguish fine particles from particles in the coarse mode, the EPA noted that the available epidemiologic studies of fine particles were based largely on PM_{2.5} mass. The selection of a 2.5 µm size cut additionally reflected the regulatory importance of defining an indicator that would more completely capture fine particles under all conditions likely to be encountered across the U.S. and the monitoring technology that was generally available (62 FR 38666 to 38668, July 18, 1997).

Since the 1997 review, studies that evaluate fine particle-related health effects continue to provide strong support for such effects using PM_{2.5} mass as the metric for fine particle exposures. Subsequent reviews have recognized the strength of this evidence, concluding that it has continued to support a PM_{2.5} mass-based indicator for a standard meant to protect against fine particle exposures. In the 2012 review, some studies had additionally examined health effects of

1 exposures to particular sources or components of fine particles, or to the ultrafine fraction of fine
2 particles. Based on limitations in such studies, together with the continued strong support for
3 effects of PM_{2.5} exposures, the Agency retained PM_{2.5} mass as the indicator for fine particles and
4 did not supplement the PM_{2.5} standards with standards based on particle composition or on the
5 ultrafine fraction (78 FR 3123, January 15, 2013).

6 As in the 2012 review, studies assessed the 2019 ISA continue to provide strong support
7 for health effects following long- and short-term PM_{2.5} exposures (U.S. EPA, 2019). While some
8 studies evaluate the health effects of particular sources of fine particles, or of particular fine
9 particle components, evidence from these studies does not identify any one source or component
10 that is a better predictor of health effects than PM_{2.5} mass (U.S. EPA, 2019, section 1.5.4). As
11 summarized in section 3.5.1 above, the 2019 ISA the evidence confirms and further supports that
12 many PM_{2.5} components and sources are associated with health effects, and does not indicate that
13 any one source or component is consistently more strongly related with health effects than PM_{2.5}
14 mass (U.S. EPA, 2019, section 1.5.4). Further, the evidence for health effects following
15 exposures specifically to the ultrafine fraction of fine particles continues to be far more limited
16 than the evidence for PM_{2.5} mass, and the varying definitions of UFP, as well as differences in
17 approaches to administering and measuring UFP, contribute to such limitations (U.S. EPA, 2019,
18 section 1.4.3). Thus, for reasons similar to those discussed in the 2020 review (85 FR 82715,
19 December 18, 2020), we reach the preliminary conclusion that the available information
20 continues to support the PM_{2.5} mass-based indicator and remains too limited to support a distinct
21 standard for any specific PM_{2.5} component or group of components, and too limited to support a
22 distinct standard for the ultrafine fraction.

23 **3.5.3.2.2 Averaging Time**

24 In 1997, the EPA initially set an annual PM_{2.5} standard to protect against health effects
25 associated with both long- and short-term PM_{2.5} exposures, and a 24-hour standard to supplement
26 the protection afforded by the annual standard (62 FR 38667 to 38668, July 18, 1997). In
27 subsequent reviews, the EPA retained both annual and 24-hour averaging times, largely
28 reflecting the strong evidence for health effects associated with annual and daily PM_{2.5} exposure
29 estimates (71 FR 61164, October 17, 2006; 78 FR 3123 to 3124, January 15, 2013).

30 In this reconsideration, epidemiologic and controlled human exposure studies have
31 examined a variety of PM_{2.5} exposure durations. Epidemiologic studies continue to provide
32 strong support for health effects associated with both long- and short-term PM_{2.5} exposures based
33 on annual (or multiyear) and 24-hour PM_{2.5} averaging periods, respectively.

34 With regard to short-term exposures in particular, a smaller number of epidemiologic
35 studies examine associations between sub-daily PM_{2.5} exposures and respiratory effects,

1 cardiovascular effects, or mortality. Compared to 24-hour PM_{2.5} exposure estimates, associations
2 with sub-daily estimates are less consistent and, in some cases, smaller in magnitude (U.S. EPA,
3 2019, section 1.5.2.1). In addition, studies of sub-daily exposures typically examine subclinical
4 effects, rather than the more serious population-level effects that have been reported to be
5 associated with 24-hour exposures (e.g., mortality, hospitalizations). Taken together, the 2019
6 ISA concludes that epidemiologic studies do not indicate sub-daily averaging periods are more
7 closely associated with health effects than the 24-hour average exposure metric (U.S. EPA, 2019,
8 section 1.5.2.1).

9 Additionally, while recent controlled human exposure studies provide consistent evidence
10 for cardiovascular effects following PM_{2.5} exposures for less than 24 hours (i.e., < 30 minutes to
11 5 hours), exposure concentrations in these studies are well-above the ambient concentrations
12 typically measured in locations meeting the current standards (section 3.3.3.1). Thus, these
13 studies also do not suggest the need for additional protection against sub-daily PM_{2.5} exposures,
14 beyond that provided by the current primary standards.

15 Drawing from the evidence assessed in the 2019 ISA, and the observations noted above,
16 we reach the conclusion that the available evidence continues to provide strong support for
17 consideration of retaining the current annual and 24-hour averaging times. The available
18 evidence suggests that PM_{2.5} standards with these averaging times, when coupled with
19 appropriate forms and levels, can protect against the range of long- and short-term PM_{2.5}
20 exposures that have been associated with health effects. Thus, as in the 2020 review (78 FR
21 82715, December 18, 2020), we reach the preliminary conclusion that the currently available
22 evidence does not support considering alternatives to the annual and 24-hour averaging times for
23 standards meant to protect against long- and short-term PM_{2.5} exposures.

24 **3.5.3.2.3 Form**

25 The form of a standard defines the air quality statistic that is to be compared to the level
26 in determining whether an area attains that standard. As in other recent reviews, our foremost
27 consideration in reaching preliminary conclusions on form is the adequacy of the public health
28 protection provided by the combination of the form and the other elements of the standard.

29 As noted above, in 1997 the EPA initially set an annual PM_{2.5} standard to protect against
30 health effects associated with both long- and short-term PM_{2.5} exposures and a 24-hour standard
31 to provide supplemental protection, particularly against the short-term exposures to “peak” PM_{2.5}
32 concentrations that can occur in some areas (62 FR 38667 to 38668, July 18, 1997). The EPA
33 established the form of the annual PM_{2.5} standard as an annual arithmetic mean, averaged over 3
34 years, from single or multiple community-oriented monitors. That is, the level of the annual
35 standard was to be compared to measurements made at each community-oriented monitoring site

1 or, if specific criteria were met, measurements from multiple community-oriented monitoring
2 sites could be averaged together (i.e., spatial averaging) (62 FR 38671 to 38672, July 18, 1997).
3 In the 1997 review, the EPA also established the form of the 24-hour PM_{2.5} standard as the 98th
4 percentile of 24-hour concentrations at each monitor within an area (i.e., no spatial averaging),
5 averaged over three years (62 FR at 38671 to 38674, July 18, 1997). In the 2006 review, the EPA
6 retained these standard forms but tightened the criteria for using spatial averaging with the
7 annual standard (71 FR 61117, October 17, 2006).⁸¹

8 In the 2012 review, the EPA’s consideration of the form of the annual PM_{2.5} standard
9 again included a focus on the issue of spatial averaging. An analysis of air quality and population
10 demographic information indicated that the highest PM_{2.5} concentrations in a given area tended
11 to be measured at monitors in locations where the surrounding populations were more likely to
12 live below the poverty line and to include larger percentages of racial and ethnic minorities (U.S.
13 EPA, 2011, p. 2-60). Based on this analysis, the 2011 PA concluded that spatial averaging could
14 result in disproportionate impacts in minority populations and populations with lower SES. The
15 Administrator concluded that public health would not be protected with an adequate margin of
16 safety in all locations, as required by law, if disproportionately higher PM_{2.5} concentrations in
17 low income and minority communities were averaged together with lower concentrations
18 measured at other sites in a large urban area. Therefore, she concluded that the form of the
19 annual PM_{2.5} standard should be revised to eliminate spatial averaging provisions (78 FR 3124,
20 January 15, 2013).

21 In the 2012 review, the EPA also considered the form of the 24-hour PM_{2.5} standard. The
22 Agency recognized that the existing 98th percentile form for the 24-hour standard was originally
23 selected to provide a balance between limiting the occurrence of peak 24-hour PM_{2.5}
24 concentrations and identifying a stable target for risk management programs. Updated air quality
25 analyses in the 2012 review provided additional support for the increased stability of the 98th
26 percentile PM_{2.5} concentration, compared to the 99th percentile (U.S. EPA, 2011, Figure 2-2, p.
27 2-62). Thus, the Administrator concluded that it was appropriate to retain the 98th percentile form
28 for the 24-hour PM_{2.5} standard (78 FR 3127, January 15, 2013).

29 In the 2020 review, the Administrator noted that the scientific evidence continued to
30 provide strong support for health effect associations for both long-term (e.g., annual or multi-
31 year) and short-term (e.g., mostly 24-hour) exposures to PM_{2.5} and judged that the evidence did
32 not support considering alternative averaging times (85 FR 82715, December 18, 2020). For

⁸¹ Specifically, the Administrator revised spatial averaging criteria such that “(1) [t]he annual mean concentration at each site shall be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.9 for each calendar quarter (71 FR 61167, October 17, 2006).

1 reasons consistent with those in the 2012 review, the Administrator judged that the current
2 annual and 24-hour averaging times remained appropriate.

3 The information available in this reconsideration continues to support the current forms
4 of the annual and 24-hour PM_{2.5} standards. As discussed above (section 3.3.1), epidemiologic
5 studies continue to provide strong support for health effect associations with both long-term
6 (e.g., annual or multi-year) and short-term (e.g., mostly 24-hour) PM_{2.5} exposures. These studies
7 provide the strongest support for such associations for the part of the air quality distribution
8 corresponding to the bulk of the underlying data, typically around the overall mean
9 concentrations reported (section 3.3.3.2.1). The form of the current annual standard (i.e.,
10 arithmetic mean, averaged over three years) remains appropriate for targeting “typical” daily and
11 annual exposures around these means of the PM_{2.5} air quality distribution. In addition, controlled
12 human exposure studies provide evidence for health effects following single short-term PM_{2.5}
13 exposures near the peak concentrations measured in the ambient air (section 3.3.3.1). Thus, the
14 evidence also supports retaining a standard focused on providing supplemental protection against
15 short-term peak exposures. The information available in this reconsideration continues to support
16 the decision to use a 98th percentile form for a 24-hour standard that is meant to provide a
17 balance between limiting the occurrence of such peak 24-hour PM_{2.5} concentrations and
18 identifying a stable target for risk management programs. Thus, when the information
19 summarized above is taken together, we reach the preliminary conclusion that it is appropriate to
20 consider retaining the forms of the current annual and 24-hour PM_{2.5} standards, in conjunction
21 with a revised level as discussed below.

22 **3.5.3.2.4 Level**

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

- 24 • **For primary PM_{2.5} standards defined in terms of the current averaging times and**
25 **forms, what potential alternative levels are appropriate to consider in order to**
26 **increase public health protection against long- and short-term exposures to PM_{2.5} in**
27 **ambient air?**

28 In answering this question, we consider key epidemiologic studies that evaluate associations
29 between PM_{2.5} air quality distributions and mortality or morbidity, controlled human exposure
30 studies examining effects following short-term PM_{2.5} exposures, air quality analyses that help to
31 place these studies into a policy-relevant context, and the risk assessment estimates of PM_{2.5}-
32 associated mortality under various alternative standard scenarios.

33 Consideration of the evidence and analyses, as summarized in this chapter, informs our
34 evaluation of the public health protection that could be provided by alternative annual and 24-
35 hour standards with revised levels. There are various ways to combine an annual standard (based
36 on arithmetic mean concentrations) and a 24-hour standard (based on 98th percentile

1 concentrations), to achieve an appropriate degree of public health protection. In particular, we
2 recognize that changes in PM_{2.5} air quality designed to meet an annual standard would likely
3 result not only in lower short- and long-term PM_{2.5} concentrations near the middle of the air
4 quality distribution (i.e., around the mean of the distribution), but also in fewer and lower short-
5 term peak PM_{2.5} concentrations. Additionally, changes designed to meet a 24-hour standard, with
6 a 98th percentile form, would result not only in fewer and lower peak 24-hour PM_{2.5}
7 concentrations, but also in lower average PM_{2.5} concentrations.

8 However, while either standard could be viewed as providing some measure of protection
9 against both average exposures and peak exposures, the 24-hour and annual standards are not
10 expected to be equally effective at limiting both types of exposures. Specifically, the 24-hour
11 standard (with its 98th percentile form) is more directly tied to short-term peak PM_{2.5}
12 concentrations, and thus more likely to appropriately limit exposures to such concentrations, than
13 the more typical concentrations that make up the middle portion of the air quality distribution.
14 Therefore, compared to a standard that is directly tied to the middle of the air quality distribution,
15 the 24-hour standard is less likely to appropriately limit the “typical” daily and annual exposures
16 that are most strongly associated with the health effects observed in epidemiologic studies. In
17 contrast, the annual standard, with its form based on the arithmetic mean concentration, is more
18 likely to effectively limit the PM_{2.5} concentrations that comprise the middle portion of the air
19 quality distribution, affording protection against the daily and annual PM_{2.5} exposures that
20 strongly support associations with the most serious PM_{2.5}-related effects in epidemiologic studies
21 (e.g., mortality, hospitalizations).

22 For these reasons, we focus on alternative levels of the annual PM_{2.5} standard as the
23 principle means of providing increased public health protection against the bulk of the
24 distribution of short- and long-term PM_{2.5} exposures, and thus protecting against the exposures
25 that provide strong support for associations with mortality and morbidity in key epidemiologic
26 studies. We additionally consider the 24-hour standard, with its 98th percentile form, primarily as
27 a means of providing supplemental protection against the short-term exposures to peak PM_{2.5}
28 concentrations that can occur in some areas (e.g., those with strong contributions from local or
29 seasonal sources), even when overall mean PM_{2.5} concentrations remain relatively low.

30 To inform our consideration of potential alternative annual and 24-hour standard levels,
31 we specifically note the key observations in section 3.5.3.1 (rather than repeating them here) and
32 note more specifically, related to those observations that:

33
34 *Mean PM_{2.5} Concentrations in Key Epidemiologic Studies and Relationships between Mean*
35 *PM_{2.5} Concentrations and Annual Design Values*

- 1 • Areas meeting a particular annual PM_{2.5} standard would be expected to have *average* PM_{2.5}
2 concentrations (i.e., averaged across the area and over time) somewhat below the level of that
3 standard (which is measured at the peak monitor). This is supported by analyses of
4 monitoring data in CBSAs across the U.S., which show that maximum annual PM_{2.5} design
5 values are often 10% to 20% higher than long-term mean PM_{2.5} concentrations in an area
6 (section 2.3.3.1, Figure 2-28; Table 2-2). Additional analyses also support differences
7 between annual PM_{2.5} design values and long-term mean PM_{2.5} concentrations in hybrid
8 modeling studies, with the extent of the difference depending on the methods used to
9 estimate mean PM_{2.5} concentrations. These analyses suggest that the area annual design
10 values are generally higher than the study mean by 14-18% (hybrid modeling with
11 population-weighting) or 40-50% higher (hybrid modeling without population-weighting)
12 (section 2.3.3.2.4, Table 2-4).
- 13 • Most key U.S. epidemiologic studies indicate consistently positive and statistically
14 significant health effect associations based on air quality distributions with overall mean
15 PM_{2.5} concentrations at or above 9.3 µg/m³ (9.9 µg/m³ based on U.S. studies that use
16 monitors to estimate PM_{2.5} exposures). Other key epidemiologic studies (which do not
17 incorporate population-weighting into their calculation of the study mean) report mean PM_{2.5}
18 concentrations to be as low as 8.1 µg/m³ with the air quality analyses suggesting that areas
19 included in these studies would have corresponding area annual design values generally 40-
20 50% higher than the study reported mean concentrations.
- 21 • Though the mean PM_{2.5} concentrations from Canadian studies are more difficult to directly
22 compare to the annual design value used to determine compliance in the U.S., the overall
23 mean PM_{2.5} concentrations in key Canadian epidemiologic studies are close to, though
24 somewhat lower than, those from the U.S. studies. The range of monitor-based mean PM_{2.5}
25 concentrations is from 6.9 µg/m³ to 13.3 µg/m³ while the range of mean PM_{2.5} concentrations
26 in studies that use hybrid modeling (all of which average up to postal codes and thus include
27 some aspects of population-weighting) is 5.9 µg/m³ to 9.8 µg/m³.
- 28 • Epidemiologic studies provide more limited support for health effect associations based on
29 air quality distributions at lower PM_{2.5} percentile concentrations. In assessing the 25th
30 percentile of data, PM_{2.5} concentrations in key U.S. epidemiologic studies that use hybrid
31 modeling methods and do not apply some aspects of population-weighting range from 4.6
32 µg/m³ to 9.2 µg/m³, while those that apply some aspects of population weighting range from
33 6.7 µg/m³ to 9.1 µg/m³. In U.S. studies that use monitored values have 25th percentiles
34 ranging from 11.5 µg/m³ to just below 13.0 µg/m³. In Canada two monitored studies report
35 25th percentile concentrations around 6.5 µg/m³, while hybrid modeled studies in Canada, all
36 of which average up to postal codes and thus include some aspects of population-weighting,
37 report 25th percentile concentrations around 8.0 µg/m³ in two studies, and 4.3 µg/m³ in one
38 study.

39

40 *Scientific Evidence and Associated Uncertainties Supporting Associations at Lower* 41 *Concentrations*

- 42 • Recent evidence further demonstrates that associations with mortality remain robust in
43 copollutants analyses (U.S. EPA, 2019, section 11.2.3), and that associations persist in

1 analyses restricted to long-term exposures below 12 $\mu\text{g}/\text{m}^3$ (Di et al., 2017b) or 10 $\mu\text{g}/\text{m}^3$
2 (Shi et al., 2016) (i.e., indicating that risks are not disproportionately driven by the upper
3 portions of the air quality distribution).

- 4 • Studies that examine the shapes of concentration-response functions over the full distribution
5 of ambient $\text{PM}_{2.5}$ concentrations have not identified a threshold concentration, below which
6 associations no longer exist (U.S. EPA, 2019, section 1.5.3, U.S. EPA, 2021a, section 2.2.3.1
7 and 2.2.3.2). While such analyses are complicated by the relatively sparse data available at
8 the lower end of the air quality distribution (U.S. EPA, 2019, section 1.5.3), analyses that
9 assess the concentration-response relationship support a linear, no-threshold effect down to
10 5.0 $\mu\text{g}/\text{m}^3$, though uncertainties increase at concentrations of less than 8.0 $\mu\text{g}/\text{m}^3$.
- 11 • While there is no specific point in the air quality distribution of any epidemiologic study that
12 represents a “bright line” at and above which effects have been observed and below which
13 effects have not been observed, there is significantly greater confidence in the magnitude and
14 significance of observed associations for the part of the air quality distribution corresponding
15 to where the bulk of the health events in each study have been observed, generally at or
16 around the mean concentration, with more limited support for health effect associations
17 based on air quality distributions at lower $\text{PM}_{2.5}$ percentile concentrations.
- 18 • Controlled human exposure studies demonstrate consistent evidence of effects at higher
19 concentrations (e.g., > 120 $\mu\text{g}/\text{m}^3$) and provide support for biological plausibility for more
20 serious effects (e.g., hospital admissions) (U.S. EPA, 2019, Figure 6-1).

21 *Scientific Evidence on Short-term Exposures and $\text{PM}_{2.5}$ Exposures Shown to Cause Effects*

- 22 • While controlled human exposure studies support the plausibility of the serious
23 cardiovascular effects that have been linked with ambient $\text{PM}_{2.5}$ exposures (U.S. EPA, 2019,
24 chapter 6), the $\text{PM}_{2.5}$ exposure concentrations evaluated in most of these studies are well-
25 above the ambient concentrations typically measured in locations meeting the current
26 primary standards (and thus well-above those likely to be measured in locations that would
27 meet revised standards with lower annual or 24-hour levels) (Figure 2-19, Figure A-2, Figure
28 A-3).

30 *$\text{PM}_{2.5}$ -Associated Risk Estimates*

- 31 • The risk assessment estimates that, compared to the current standards, potential alternative
32 annual standards with levels from 11.0 down to 8.0 $\mu\text{g}/\text{m}^3$ could reduce $\text{PM}_{2.5}$ -associated
33 mortality broadly across the United States. Meeting a revised annual standard with a lower
34 level is estimated to reduce $\text{PM}_{2.5}$ -associated health risks in the 30 annually-controlled study
35 areas by about 7-9% for a level of 11.0 $\mu\text{g}/\text{m}^3$, 15-19% for a level of 10.0 $\mu\text{g}/\text{m}^3$, 22-28% for
36 a level of 9.0 $\mu\text{g}/\text{m}^3$, and 30-37% for a level of 8.0 $\mu\text{g}/\text{m}^3$, compared to the current annual
37 standard.
- 38 • Revising the level of the 24-hour standard to 30 $\mu\text{g}/\text{m}^3$ is estimated to lower $\text{PM}_{2.5}$ -associated
39 risks across a more limited population and number of areas than revising the annual standard
40 (section 3.4.2.3). Risk reduction predictions are largely confined to areas located in the
41 western U.S., several of which are also likely to experience risk reductions upon meeting a
42 revised annual standard.

- 1 • The at-risk assessment estimated that Black populations may experience disproportionately
2 higher exposures and risk under simulated air quality conditions just meeting the current
3 primary PM_{2.5} annual standard as compared to White populations. Meeting a revised annual
4 standard with a lower level may also proportionally reduce exposure and risk in Black
5 populations slightly more so than in White populations in simulated scenarios just meeting
6 alternative annual standards.
- 7 • Uncertainties in risk estimates (e.g., in the size of risk estimates) can result from a number of
8 factors, including assumptions about the shape of the C-R relationship with mortality at low
9 ambient PM concentrations, the potential for confounding and/or exposure measurement
10 error in the underlying epidemiologic studies, and the methods used to adjust PM_{2.5} air
11 quality. In considering such uncertainties, we recognize that the risk estimates can help to
12 place the evidence for specific effects into a broader public health context, but should be
13 considered along with the inherent uncertainties and limitations of such analyses when
14 informing judgments about the potential for additional public health protection associated
15 with PM_{2.5} exposure and related health effects.

16 The information summarized in these key observations could support various decisions on
17 the levels of the annual and 24-hour PM_{2.5} standards, depending on the weight given to different
18 aspects of the evidence, air quality and risk information, including its uncertainties. In this draft
19 PA we seek to provide as broad an array of policy options as is supportable by the available
20 evidence and quantitative information, recognizing that the selection of a specific approach to
21 reaching final decisions on the primary PM_{2.5} standards will reflect the judgments of the
22 Administrator as to what weight to place on the various types of evidence and information, and
23 on associated uncertainties. Potential approaches to considering support for particular alternative
24 annual and 24-hour standard levels are discussed below.

25 26 *Alternative Annual Standard Levels*

27 As discussed above, the degree to which particular alternative annual standard levels
28 below 12.0 µg/m³ are supported will depend on the weight placed on various aspects of the
29 scientific evidence, air quality and risk information, and its associated uncertainties. In selecting
30 a particular level from 10.0 µg/m³ to < 12.0 µg/m³, consideration of the evidence could take into
31 account individual study characteristics such as study design and statistical approaches, precision
32 of reported associations, study size and location, and uncertainties in the study itself or in our
33 analyses of study area air quality. For example, a level below 12 µg/m³ and as low as about 10.0
34 µg/m³ could be supported to the extent weight is placed on the following:

- 35 • Setting a standard expected to maintain the PM_{2.5} air quality distributions below those
36 present in most key epidemiologic studies, recognizing the general relationships
37 demonstrated in the air quality analyses between study mean calculation and the annual
38 standard and noting the values of the study reported means as listed below:

- 1 ○ The monitor-based key epidemiologic studies report mean PM_{2.5} concentrations from
2 9.9 µg/m³ to 16.5 µg/m³;
- 3 ○ The key epidemiologic studies that incorporate hybrid modeling and population-
4 weight study mean PM_{2.5} concentrations report means from 9.3 µg/m³ to 12.2 µg/m³.
- 5 • Noting that given the differences between population densities, PM_{2.5} concentration
6 gradients, and source distributions between the U.S. and Canada, it may be inappropriate to
7 draw a direct comparison between the Canadian study means and the annual design value
8 metric used for compliance in the U.S., but also noting that the study reported means from
9 the Canadian studies are similar, though somewhat lower, than those in the U.S.
- 10 • Setting a standard level within the starting range of the mean PM_{2.5} concentrations evaluated
11 in accountability studies, recognizing that some of the studies that report public health
12 improvements with improvements to air quality have starting concentrations that range
13 between 10.0 µg/m³ to 12.0 µg/m³ (Table 3-12).
- 14 • Setting a standard estimated to reduce PM_{2.5}-associated health risks, such that a substantial
15 portion of the risk reduction that would be accomplished is estimated at annual average PM_{2.5}
16 concentrations within the range of overall means for which key epidemiologic studies
17 indicate consistently positive and statistically significant health effect associations (≥ about 8
18 µg/m³) while also noting important uncertainties inherent in the risk assessment as described
19 in detail in sections 3.4.1.7 and 3.4.1.8. Further, the at-risk analyses indicate that the average
20 percent reduction in PM_{2.5} concentrations and risk are slightly greater in the Black population
21 than in the White population for each alternative standard evaluated (11.0 µg/m³ and 10.0
22 µg/m³), when shifting from the current annual PM_{2.5} standard (12.0 µg/m³) in the full set of
23 47 areas and the subset of 30 areas controlled by the annual standard (section 3.4).
- 24 • Noting a number of uncertainties associated with the scientific evidence and risk information
25 including: (1) there are few key epidemiologic studies (and only one key U.S. study) that
26 report positive and statistically significant health effect associations for PM_{2.5} air quality
27 distributions with overall mean concentrations below 9.6 µg/m³, and areas meeting a standard
28 with a level of 10.0 µg/m³ would generally be expected to have lower long-term mean
29 PM_{2.5} concentrations (and potentially around 8.0 µg/m³ in some areas) (section 3.3.3.2.1); (2)
30 there is increasing uncertainty in PM_{2.5} exposure estimates in some of the largest key studies
31 at lower ambient concentrations (i.e., those that use hybrid model predictions to estimate
32 exposures), given the more limited information available to develop and validate model
33 predictions (sections 2.3.3 and 3.3.3.2.1); and (3) there is increasing uncertainty in
34 quantitative estimates of PM_{2.5}-associated mortality risk for standard levels below 10.0 µg/m³,
35 given that a substantial proportion of the risk reductions estimated for lower standard
36 levels occur at annual average PM_{2.5} concentrations below 8 µg/m³, and thus below the lower
37 end of the range of overall mean PM_{2.5} concentrations in key epidemiologic studies that
38 consistently report positive and statistically significant associations (section 3.4.1.7).

39 In contrast, an annual standard with a level below 10.0 µg/m³ and as low as 8.0 µg/m³,
40 could be supported to the extent greater weight is placed on the potential public health
41 improvements that could result from additional reductions in ambient PM_{2.5} concentrations (i.e.,
42 beyond those achieved by a standard with a level of 10.0 µg/m³) and less weight is placed on the

- 1 limitations in the evidence that contribute to greater uncertainty at lower concentrations. For
2 example, a level below $10.0 \mu\text{g}/\text{m}^3$ could be supported to the extent greater weight is placed on
3 the following:
- 4 • Setting the annual standard at or below most or all of the study reported means, including
5 means of hybrid modeling studies that did not use population weighted approaches, such that
6 the standard would be expected to maintain the $\text{PM}_{2.5}$ air quality distributions further below
7 those present in most key epidemiologic studies and noting that the relationships between
8 study mean calculation and the annual standard in the draft PA analyses are approximations
9 and less weight should be placed on them and the mathematical approach used to calculate
10 the mean.
 - 11 • Results of the key Canadian epidemiologic studies, which report mean $\text{PM}_{2.5}$ concentrations
12 that are lower than those reported in U.S. studies and for which the $\text{PM}_{2.5}$ concentrations
13 generally range from $7.0 \mu\text{g}/\text{m}^3$ to $9.0 \mu\text{g}/\text{m}^3$ (monitor-based) and $6.0 \mu\text{g}/\text{m}^3$ to $10.0 \mu\text{g}/\text{m}^3$
14 (hybrid model-based and all of which apply some aspects of population-weighting) (section
15 3.3.3.2.1);
 - 16 • Consideration of the air quality distribution below the mean for which key epidemiologic
17 studies have reported associations with health effects. The ambient $\text{PM}_{2.5}$ concentrations
18 around the 25th percentile of underlying data, which range from $11.5 \mu\text{g}/\text{m}^3$ to $12.9 \mu\text{g}/\text{m}^3$ in
19 U.S. monitor-based studies, from $6.5 \mu\text{g}/\text{m}^3$ to $6.8 \mu\text{g}/\text{m}^3$ in Canadian monitor-based studies,
20 from $4.6 \mu\text{g}/\text{m}^3$ to $9.2 \mu\text{g}/\text{m}^3$. In key U.S. epidemiologic studies that use hybrid modeling
21 methods and do not apply some aspects of population-weighting range from $4.6 \mu\text{g}/\text{m}^3$ to 9.2
22 $\mu\text{g}/\text{m}^3$, while those that apply some aspects of population weighting range from $6.7 \mu\text{g}/\text{m}^3$ to
23 $9.1 \mu\text{g}/\text{m}^3$ while hybrid modeled studies in Canada, all of which average up to postal codes
24 and thus include some aspects of population-weighting, report 25th percentile concentrations
25 around $8.0 \mu\text{g}/\text{m}^3$ in two studies, and $4.3 \mu\text{g}/\text{m}^3$ in one study (section 3.3.3.2.1);
 - 26 • Noting studies that examined the shapes of concentration-response functions over the full
27 distribution of ambient $\text{PM}_{2.5}$ concentrations and concluded that while the concentration-
28 response relationship support a linear, no-threshold effect down to $5.0 \mu\text{g}/\text{m}^3$, uncertainties
29 increase at concentrations of less than $8.0 \mu\text{g}/\text{m}^3$; and also noting that the $\text{PM}_{2.5}$ exposure
30 concentrations in an area with a design value of less than $8.0 \mu\text{g}/\text{m}^3$ would reflect a
31 distribution of air quality that would be mostly associated with average daily concentrations
32 below $8.0 \mu\text{g}/\text{m}^3$.
 - 33 • The potential for continued public health improvements with improvements in air quality
34 below the lowest starting concentration evaluated in accountability studies, which was
35 approximately $10.0 \mu\text{g}/\text{m}^3$ (Table 3-12);
 - 36 • Studies that restrict analyses to air quality associated with levels below the current annual
37 standard and report positive and significant associations, often with effect estimates that are
38 greater in magnitude than those reported in the main analysis. Although the mean of the
39 restricted analyses are generally not reported, in one key U.S. epidemiologic study, the mean
40 concentration when restricting annual average $\text{PM}_{2.5}$ concentrations to below $12.0 \mu\text{g}/\text{m}^3$ was
41 presumably lower than the overall mean concentration of $8.1 \mu\text{g}/\text{m}^3$ reported in the main
42 analysis (Shi et al., 2016) (Table 3-10);

- 1 • The potential public health importance of the additional reductions in PM_{2.5}-associated health
2 risks estimated for a level below 10.0 µg/m³ µg/m³ and the potential for continued
3 improvements below the lowest level examined in the risk assessment (8.0 µg/m³). Further,
4 the at-risk analyses indicate that the average percent reduction in PM_{2.5} concentrations and
5 risk are slightly greater in the Black population than in the White population for each
6 alternative standard evaluated (9.0 µg/m³ and 8.0 µg/m³), when shifting from the current
7 annual PM_{2.5} standard (12.0 µg/m³) in the full set of 47 areas and the subset of 30 areas
8 controlled by the annual standard (section 3.4).

9 *Alternative 24-Hour Standard Levels*

10 We additionally evaluate the degree to which the evidence supports considering potential
11 alternative levels for the 24-hour PM_{2.5} standard, in conjunction with the current 98th percentile
12 form of that standard. With respect to current and recent air quality relationships, we note that
13 the risk assessment indicates that the annual standard is the controlling standard across most of
14 the urban study areas evaluated and revising the level of the 24-hour standard to 30 µg/m³ would
15 be estimated to lower PM_{2.5}-associated risks, compared to the current standards, largely in a few
16 study areas located in the western U.S. (several of which are also likely to experience risk
17 reductions upon meeting a revised annual standard). Additionally, recent air quality analyses
18 indicate that almost all CBSAs with maximum annual PM_{2.5} design values at or below 12.0
19 µg/m³ also have maximum 24-hour PM_{2.5} design values below 35 µg/m³ (and below 30 µg/m³ in
20 most areas) (chapter 2, Figure 2-18). The exceptions are a few CBSAs in the western U.S.

21 As in previous reviews, we recognize that the annual standard would generally be the
22 controlling standard across much of the U.S., except for certain areas where there are high
23 seasonal emissions (e.g., wood smoke) and conducive meteorology (e.g., temperature inversions)
24 or where there are more unique source-oriented influences (e.g., near manufacturing sources). In
25 such areas, the 24-hour standard is the generally controlling standard, though the number of these
26 areas in the U.S. is small. Thus, as was the approach in multiple recent reviews, we focus on the
27 annual standard as the principle means of limiting both long- and short-term PM_{2.5}
28 concentrations, recognizing that the 24-hour standard, with its 98th percentile form, would
29 provide supplemental protection against short-term peak exposures, particularly for areas with
30 high peak-to-mean ratios (e.g., areas with strong seasonal sources). Compared to the annual
31 standard, we recognize that the 24-hour standard is less likely to appropriately limit the more
32 typical PM_{2.5} exposures (i.e., corresponding to the middle portion of the air quality distribution)
33 that are most strongly associated with the health effects observed in epidemiologic studies. Thus,
34 as in previous reviews (78 FR 3161-3162, January 15, 2013; 85 FR 82715, December 18, 2020),
35 we focus on the 24-hour standard as a means of providing supplemental protection against the
36 short-term exposures to “peak” PM_{2.5} concentrations, such as can occur in areas with strong
37 contributions from local or seasonal sources.

1 Taking into account this approach, an important consideration is whether additional
2 protection is needed against short-term exposures to peak PM_{2.5} concentrations in areas meeting
3 both the current 24-hour standard and the current, or a revised, annual standard. To the extent
4 that the evidence indicates that such exposures can lead to adverse health effects, it would be
5 appropriate to consider alternative levels for the 24-hour standard. In considering this issue, we
6 evaluate the evidence from key health studies. With regard to these studies, we particularly note
7 the following:

- 8 • Controlled human exposure studies provide evidence for health effects following single,
9 short-term PM_{2.5} exposures to concentrations that typically correspond to upper end of the
10 PM_{2.5} air quality distribution in the U.S. (i.e., “peak” concentrations). In the studies evaluated
11 at near ambient PM_{2.5} concentrations, results are mixed but they do report statistically
12 significant effects on one or more indicators of cardiovascular function following 2-hour
13 exposures to PM_{2.5} concentrations at and above 120 µg/m³ (at and above 149 µg/m³ for
14 vascular impairment, the effect shown to be most consistent across studies).
- 15 • Animal toxicologic studies provide evidence of effects related to short-term exposures to
16 PM_{2.5} at concentrations ranging from 100 to > 1,000 µg/m³ and providing further evidence to
17 support the biological mechanisms and plausibility of various adverse effects associated with
18 short-term exposures.
- 19 • The body of epidemiologic evidence provides limited support for judging adequacy of the
20 level of the 24-hour standard. As discussed in detail above (section 3.3.3.2.1), epidemiologic
21 studies provide the strongest support for reported health effect associations for the part of the
22 air quality distribution corresponding to the bulk of the underlying data (i.e., estimated
23 exposures and/or health events), often around the overall mean concentrations evaluated
24 rather than near the upper end of the distribution. Additionally, the magnitudes of the
25 associations in restricted analyses are similar to or larger than the magnitudes of the
26 associations based on the full cohorts (Table 3-10), suggesting that, at a minimum, short-term
27 exposures to peak PM_{2.5} concentrations are not disproportionately responsible for reported
28 health effect associations.

29 Based on the evidence above, we assessed the protection provided by the current
30 standards against the concentrations seen in the human exposure studies. The air quality analyses
31 included in this draft PA show that 2-hour ambient concentrations of PM_{2.5} at monitoring sites
32 meeting the current standards almost never exceed 30 µg/m³ (Figure 2-19). In fact, even the
33 extreme upper end of the distribution of 2-hour PM_{2.5} concentrations at sites meeting the current
34 standards remain well-below the PM_{2.5} exposure concentrations consistently shown to elicit
35 effects (i.e., 99.9th percentile of 2-hour concentrations at these sites is 62 µg/m³ during the warm
36 season). We also note some caution in placing too much weigh on the need to provide protection
37 against any of the exposures observed in the clinical studies given that it is unclear how the
38 results alone and the importance of the effects observed in these studies, particularly in the
39 studies conducted at near-ambient PM_{2.5} concentrations, should be interpreted with respect to
40 adversity to public health.

1 When the information summarized above is considered in the context of the 24-hour
2 standard, we reach the preliminary conclusion that, in conjunction with a lower annual standard
3 level intended to increase protection against average short- and long-term PM_{2.5} exposures across
4 the U.S., the evidence does not support the need for additional protection against short-term
5 exposures to peak PM_{2.5} concentrations. In particular, while the epidemiologic studies do support
6 the need to consider increasing protection against the typical daily and annual PM_{2.5} exposures
7 that provide strong support for reported health effect associations, these studies do not provide
8 the same support for a need for increasing protection against short-term, peak exposures. Further,
9 the epidemiologic studies do not indicate that the reported health effect associations in these
10 studies are strongly influenced by exposures to the peak concentrations in the air quality
11 distribution. Also, while animal toxicologic studies provide evidence to support the biological
12 mechanisms and plausibility of various adverse effects associated with short-term exposures,
13 they provide limited support for judging adequacy of the level of the 24-hour standard. Human
14 clinical studies support the occurrence of effects following single short-term exposures to PM_{2.5}
15 concentrations that correspond to the peak of the air quality distribution, though these
16 concentrations are well above those typically measured in areas meeting the current standards,
17 suggesting that the current standards are providing protection against these exposures. As such,
18 the available evidence supports the need for the current 24-hour standard to protect against peak
19 concentrations but does not clearly support the need for a lower level of that standard. Thus, in
20 the context of a 24-hour standard that is meant to provide supplemental protection (i.e., beyond
21 that provided by the annual standard alone) against short-term exposures to peak PM_{2.5}
22 concentrations, the evidence supports consideration of retaining the current 24-hour standard
23 with its level of 35 µg/m³.

24 However, we also recognize that a different policy approach than that described above
25 could be applied to considering the level of the 24-hour standard. For example, consideration
26 could be given to lower 24-hour standard levels in order to increase protection across the U.S.
27 against the broader PM_{2.5} air quality distribution. If such an approach is evaluated in this
28 reconsideration, consideration of 24-hour standard levels as low as 30 µg/m³ could be supported
29 (either alone or in conjunction with a lower annual standard level). The risk assessment estimates
30 that a level of 30 µg/m³ would increase protection compared to the current standards, though
31 only in a small number of study areas largely confined to the western U.S. (section 3.4.2).

32 If this alternative approach to revising the primary PM_{2.5} standards is adopted, the
33 uncertainty inherent in using the 24-hour standard to increase protection against the broad
34 distribution of PM_{2.5} air quality should be carefully considered. Specifically, the degree of
35 protection provided by any particular 24-hour standard against the typical PM_{2.5} exposures
36 corresponding to the middle portion of the air quality distribution will vary across locations and

1 over time, depending on the relationship between those typical concentrations and the short-term
2 peak PM_{2.5} concentrations that are directly targeted by the 24-hour standard (i.e., with its 98th
3 percentile form). Thus, lowering the level of the 24-hour standard is likely to have a more
4 variable impact on public health than lowering the level of the annual standard. Depending on
5 the 24-hour standard level set, some areas could experience reductions that are greater than
6 warranted, based on the evidence, while others could experience reductions that are less than
7 warranted. Therefore, the rationale supporting this approach would need to recognize and
8 account for the uncertainty inherent in using 24-hour standard, with a 98th percentile form, to
9 increase protection against the broad distribution of PM_{2.5} air quality.

10 **3.6 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

11 In this section, we identify key areas for additional research and data collection for fine
12 particles, based on the uncertainties and limitations that remain in the evidence and technical
13 information. Additional research in these areas could reduce uncertainties and limitations in
14 future reviews of the primary PM_{2.5} standards. Important areas for future research include the
15 following:

- 16 • Further elucidating the physiological pathways through which exposures to the PM_{2.5}
17 concentrations present in the ambient air across much of the U.S. could be causing mortality
18 and the morbidity effects shown in many epidemiologic studies. This could include the
19 following:
 - 20 - Controlled human exposure studies that examine exposures near ambient
21 PM_{2.5} concentrations (e.g., Wyatt et al. (2020a) longer exposure periods (e.g.,
22 24-hour as in Bräuner et al. (2008); 5-hour as in Hemmingsen et al. (2015b)),
23 or repeated exposures, to concentrations typically measured in the ambient air
24 across the U.S.
 - 25 - Studies that evaluate the health impacts of decreasing PM_{2.5} exposures (e.g.,
26 due to changes in policies or behavior, shifts in important emissions sources,
27 or targeted interventions).
 - 28 - Additional animal toxicological studies that evaluate exposures to near
29 ambient PM_{2.5} concentrations.
- 30 • Additional research into “causal inference” methods in epidemiologic studies to evaluate the
31 causal nature of relationships between PM_{2.5} exposure and mortality or morbidity.
- 32 • Additional research into “accountability” or “quasi-experimental” epidemiologic studies with
33 ‘starting PM_{2.5} concentrations’ below 12.0 µg/m³.
- 34 • Improving our understanding of the PM_{2.5} concentration-response relationships near the
35 lower end of the PM_{2.5} air quality distribution, including the shapes of concentration-
36 response functions and the uncertainties around estimated functions for various health
37 outcomes and populations (e.g., older adults, people with pre-existing diseases, children).

- 1 • Understanding of the potential for particle characteristics, other than size-fractionated mass,
2 to influence PM toxicity (e.g., composition, oxidative potential, etc.) and the PM health
3 effect associations observed in epidemiologic studies.
- 4 • Improving our understanding of the uncertainties inherent in the various approaches used to
5 estimate PM_{2.5} exposures in epidemiologic studies, including how those uncertainties may
6 vary across space and time, and over the PM_{2.5} air quality distribution. Approaches to
7 incorporating these uncertainties into quantitative estimates of PM_{2.5} concentration-response
8 relationships should also be explored.
- 9 • Additional health research on ultrafine particles, with a focus on consistently defining UFPs
10 across studies and across disciplines (i.e., animal, controlled human exposure, and
11 epidemiologic studies), on using consistent exposure approaches in experimental studies, and
12 on improving exposure characterizations in epidemiologic studies. Also, further examine the
13 potential for translocation of ultrafine particles from the respiratory tract into other
14 compartments (i.e., blood) and organs (e.g., heart, brain), with particular emphasis on studies
15 conducted in humans.
- 16 • Additional work to measure ultrafine particle emissions and the composition of ultrafine
17 particles, using comparable methods to measure emissions from various types of sources
18 (e.g., mobile sources, fires, etc.).
- 19 • Further evaluate the potential for some groups to be at higher risk of PM_{2.5}-related effects
20 than the general population and the potential for PM_{2.5} exposures to contribute to the
21 development of underlying conditions that may then confer higher risk of PM_{2.5}-related
22 effects. For example, research to address this latter need could include efforts to understand
23 the potential for long-term PM exposures to contribute to the development and progression of
24 atherosclerosis in adults and/or asthma in children. It could also include research to
25 understand the potential role of PM exposures in developmental outcomes (e.g.,
26 neurodevelopmental effects, reproductive and birth outcomes).
- 27 • Research to further evaluate the combination of factors that contribute to differences in risk
28 estimates between cities, potentially including differences in exposures, demographics,
29 particle characteristics.
- 30 • Research to improve our understanding of variability in PM_{2.5} exposures within and across
31 various populations (e.g., defined by life stage, pre-existing condition, etc.), the most health-
32 relevant exposure durations, as well as the temporal and spatial variability in ambient PM_{2.5}
33 that is not captured by existing ambient monitors.
- 34 • Future research to examine PM_{2.5} exposure and associated effects in pregnant women, and
35 birth outcomes, as well as future research and data collection to examine developmental
36 outcomes and different life stages

37 In addition to research and data collection, additional information that could be reported
38 in epidemiologic studies may help to reduce uncertainties and limitations in future reviews of the
39 primary PM_{2.5} standards. This information includes:

- 40 • Descriptive statistics of PM_{2.5} concentrations that are used in epidemiologic studies to
41 evaluate associations between PM_{2.5} and health effects (e.g., minimum, maximum, 10th
42 percentile, 25th percentile, mean, median, 75th percentile).

- 1 • More detailed information on the methods used to calculate the mean PM_{2.5} concentrations
2 that are reported in the study (e.g., whether population-weighting was applied, how the PM_{2.5}
3 concentrations estimated from hybrid modeling are averaged prior to being assigned to health
4 events).
- 5 - Noting whether the mean PM_{2.5} concentration reported is the concentration
6 across the area evaluated or if the mean PM_{2.5} concentration reported is based
7 only PM_{2.5} concentrations used in analyses to assess the association between
8 health outcomes and PM_{2.5}.
- 9 • In analyses restrict PM_{2.5} concentrations below specific concentrations (e.g., below annual
10 averages of 12.0 µg/m³ or below daily averages of 35 µg/m³) reporting of the Mean PM_{2.5}
11 concentrations in the restricted analysis could be helpful.

1 REFERENCES

- 2 Alhanti, BA, Chang, HH, Winqvist, A, Mulholland, JA, Darrow, LA and Sarnat, SE (2016).
3 Ambient air pollution and emergency department visits for asthma: A multi-city
4 assessment of effect modification by age. *Journal of Exposure Science and*
5 *Environmental Epidemiology* 26(2): 180-188.
- 6 ATS (2000). What Constitutes an Adverse Health Effect of Air Pollution? *American Journal of*
7 *Respiratory and Critical Care Medicine* 161(2): 665-673.
- 8 Awad, YA, Di, Q, Wang, Y, Choirat, C, Coull, BA, Zanobetti, A and Schwartz, J (2019).
9 Change in PM_{2.5} exposure and mortality among Medicare recipients: Combining a semi-
10 randomized approach and inverse probability weights in a low exposure population.
11 *Environmental Epidemiology* 3(4): e054.
- 12 Bai, L, Shin, S, Burnett, RT, Kwong, JC, Hystad, P, van Donkelaar, A, Goldberg, MS, Lavigne,
13 E, Copes, R, Martin, RV, Kopp, A and Chen, H (2019). Exposure to ambient air pollution
14 and the incidence of congestive heart failure and acute myocardial infarction: A
15 population-based study of 5.1 million Canadian adults living in Ontario. *Environment*
16 *International* 132: 105004.
- 17 Baxter, LK, Crooks, JL and Sacks, JD (2017). Influence of exposure differences on city-to-city
18 heterogeneity in PM_{2.5}-mortality associations in US cities. *Environmental Health: A*
19 *Global Access Science Source* 16(1): 1.
- 20 Behbod, B, Urch, B, Speck, M, Scott, JA, Liu, L, Poon, R, Coull, B, Schwartz, J, Koutrakis, P,
21 Silverman, F and Gold, DR (2013). Endotoxin in concentrated coarse and fine ambient
22 particles induces acute systemic inflammation in controlled human exposures.
23 *Occupational and Environmental Medicine* 70(11): 761-767.
- 24 Bell, ML, Ebisu, K, Leaderer, BP, Gent, JF, Lee, HJ, Koutrakis, P, Wang, Y, Dominici, F and
25 Peng, RD (2014). Associations of PM_{2.5} constituents and sources with hospital
26 admissions: analysis of four counties in Connecticut and Massachusetts (USA) for
27 persons ≥ 65 years of age. *Environmental Health Perspectives* 122(2): 138-144.
- 28 Bell, ML, Ebisu, K, Peng, RD, Walker, J, Samet, JM, Zeger, SL and Dominic, F (2008).
29 Seasonal and regional short-term effects of fine particles on hospital admissions in 202
30 U.S. counties, 1999-2005. *American Journal of Epidemiology* 168(11): 1301-1310.
- 31 Bell, ML, Son, JY, Peng, RD, Wang, Y and Dominici, F (2015). Brief report: Ambient PM_{2.5} and
32 risk of hospital admissions: do risks differ for men and women? *Epidemiology* 26(4):
33 575-579.
- 34 Bellavia, A, Urch, B, Speck, M, Brook, RD, Scott, JA, Albeti, B, Behbod, B, North, M, Valeri,
35 L, Bertazzi, PA, Silverman, F, Gold, D and Baccarelli, AA (2013). DNA
36 hypomethylation, ambient particulate matter, and increased blood pressure: Findings

- 1 from controlled human exposure experiments. *Journal of the American Heart Association*
2 2(3): e000212.
- 3 Bennett, JE, Tamura-Wicks, H, Parks, RM, Burnett, RT, Pope, CA, Bechle, MJ, Marshall, JD,
4 Danaei, G and Ezzati, M (2019). Particulate matter air pollution and national and county
5 life expectancy loss in the USA: A spatiotemporal analysis. *PLoS Medicine* 16(7):
6 e1002856.
- 7 Berhane, K, Chang, CC, McConnell, R, Gauderman, WJ, Avol, E, Rapaport, E, Urman, R,
8 Lurmann, F and Gilliland, F (2016). Association of changes in air quality with bronchitic
9 symptoms in children in California, 1993-2012. *JAMA: Journal of the American Medical*
10 *Association* 315(14): 1491-1501.
- 11 Bevan, GH, Freedman, DA, Lee, EK, Rajagopalan, S and Al-Kindi, SG (2021). Association
12 between ambient air pollution and county-level cardiovascular mortality in the United
13 States by social deprivation index. *American Heart Journal* 235: 125-131.
- 14 Bourdrel, T, Annesi-Maesano, I, Alahmad, B, Maesano, CN and Bind, MA (2021). The impact
15 of outdoor air pollution on COVID-19: a review of evidence from in vitro, animal, and
16 human studies. 30(159).
- 17 Bräuner, EV, Møller, P, Barregard, L, Dragsted, LO, Glasius, M, Wåhlin, P, Vinzents, P,
18 Raaschou-Nielsen, O and Loft, S (2008). Exposure to ambient concentrations of
19 particulate air pollution does not influence vascular function or inflammatory pathways in
20 young healthy individuals. *Particle and Fibre Toxicology* 5: 13.
- 21 Bravo, MA, Ebisu, K, Dominici, F, Wang, Y, Peng, RD and Bell, ML (2017). Airborne fine
22 particles and risk of hospital admissions for understudied populations: Effects by
23 urbanicity and short-term cumulative exposures in 708 U.S. counties. *Environmental*
24 *Health Perspectives* 125(4): 594-601.
- 25 Breton, CV, Salam, MT, Vora, H, Gauderman, WJ and Gilliland, FD (2011). Genetic variation in
26 the glutathione synthesis pathway, air pollution, and children's lung function growth.
27 *American Journal of Respiratory and Critical Care Medicine* 183(2): 243-248.
- 28 Brook, RD, Urch, B, Dvorchak, JT, Bard, RL, Speck, M, Keeler, G, Morishita, M, Marsik, FJ,
29 Kamal, AS, Kaciroti, N, Harkema, J, Corey, P, Silverman, F, Gold, DR, Wellenius, G,
30 Mittleman, MA, Rajagopalan, S and Brook, JR (2009). Insights into the mechanisms and
31 mediators of the effects of air pollution exposure on blood pressure and vascular function
32 in healthy humans. *Hypertension* 54(3): 659-667.
- 33 Burnett, RT and Goldberg, MS (2003). Size-fractionated particulate mass and daily mortality in
34 eight Canadian cities. *Health Effects Institute*. Boston, MA.
- 35 Burnett, RT, Stieb, D, Brook, JR, Cakmak, S, Dales, R, Raizenne, M, Vincent, R and Dann, T
36 (2004). Associations between short-term changes in nitrogen dioxide and mortality in
37 Canadian cities. *Archives of Environmental and Occupational Health* 59(5): 228-236.

- 1 Cakmak, S, Hebborn, C, Pinault, L, Lavigne, E, Vanos, J, Crouse, DL and Tjepkema, M (2018).
2 Associations between long-term PM2.5 and ozone exposure and mortality in the
3 Canadian Census Health and Environment Cohort (CANCHEC), by spatial synoptic
4 classification zone. *Environment International* 111: 200-211.
- 5 Cangerana Pereira, FA, Lemos, M, Mauad, T, de Assuncao, JV and Nascimento Saldiva, PH
6 (2011). Urban, traffic-related particles and lung tumors in urethane treated mice. *Clinics*
7 66(6): 1051-1054.
- 8 Chan, E. (2019). Email and attachment from Elizabeth Chan, EPA, Question re: Request for
9 additional PM2.5 information from Di et al 2017. Available in PM NAAQS Review
10 Docket (EPA-HQ-OAR-2015-0072). December 3, 2019. Office of Air Quality Planning
11 and Standards Research Triangle Park, NC.
- 12 Chen, H, Burnett, RT, Copes, R, Kwong, JC, Villeneuve, PJ, Goldberg, MS, Brook, RD, van
13 Donkelaar, A, Jerrett, M, Martin, RV, Brook, JR, Kopp, A and Tu, JV (2016). Ambient
14 fine particulate matter and mortality among survivors of myocardial infarction:
15 population-based cohort study. *Environmental Health Perspectives* 124(9): 1421-1428.
- 16 Chen, H, Zhang, ZL, van Donkelaar, A, Bai, L, Martin, RV, Lavigne, E, Kwong, JC and Burnett,
17 RT (2020). Understanding the Joint Impacts of Fine Particulate Matter Concentration and
18 Composition on the Incidence and Mortality of Cardiovascular Disease: A Component-
19 Adjusted Approach. *Environmental Science and Technology* 54(7): 4388-4399.
- 20 Christidis, T, Erickson, AC, Pappin, AJ, Crouse, DL, Pinault, LL, Weichenthal, SA, Brook, JR,
21 van Donkelaar, A, Hystad, P, Martin, RV, Tjepkema, M, Burnett, RT and Brauer, M
22 (2019). Low concentrations of fine particle air pollution and mortality in the Canadian
23 Community Health Survey cohort. *Environmental Health: A Global Access Science*
24 *Source* 18(1): 84.
- 25 Correia, AW, Pope, CA, III, Dockery, DW, Wang, Y, Ezzati, M and Dominici, F (2013). Effect
26 of air pollution control on life expectancy in the United States: an analysis of 545 U.S.
27 counties for the period from 2000 to 2007. *Epidemiology* 24(1): 23-31.
- 28 Corrigan, AE, Becker, MM, Neas, LM, Cascio, WE and Rappold, AG (2018). Fine particulate
29 matters: The impact of air quality standards on cardiovascular mortality. *Environmental*
30 *Research* 161: 364-369.
- 31 Cox, LA. (2019). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory
32 Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's
33 *Policy Assessment for the Review of the National Ambient Air Quality Standards for*
34 *Particulate Matter (External Review Draft - September 2019)*. December 16, 2019. EPA-
35 CASAC-20-001. Office of the Administrator, Science Advisory Board U.S. EPA HQ,
36 Washington DC. Available at:
37 [https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F](https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf)
38 [6C71737201612852584D20069DFB1/\\$File/EPA-CASAC-20-001.pdf](https://yosemite.epa.gov/sab/sabproduct.nsf/264cb1227d55e02c85257402007446a4/E2F6C71737201612852584D20069DFB1/$File/EPA-CASAC-20-001.pdf).

- 1 Crouse, DL, Peters, PA, Hystad, P, Brook, JR, van Donkelaar, A, Martin, RV, Villeneuve, PJ,
2 Jerrett, M, Goldberg, MS, Pope, CA, Brauer, M, Brook, RD, Robichaud, A, Menard, R
3 and Burnett, RT (2015). Ambient PM 2.5, O₃, and NO₂ exposures and associations with
4 mortality over 16 years of follow-up in the Canadian Census Health and Environment
5 Cohort (CanCHEC). *Environmental Health Perspectives* 123(11): 1180-1186.
- 6 Crouse, DL, Peters, PA, van Donkelaar, A, Goldberg, MS, Villeneuve, PJ, Brion, O, Khan, S,
7 Atari, DO, Jerrett, M, Pope, CA, Brauer, M, Brook, JR, Martin, RV, Stieb, D and
8 Burnett, RT (2012). Risk of nonaccidental and cardiovascular mortality in relation to
9 long-term exposure to low concentrations of fine particulate matter: a Canadian national-
10 level cohort study. *Environmental Health Perspectives* 120(5): 708-714.
- 11 Crouse, DL, Pinault, L, Balram, A, Brauer, M, Burnett, RT, Martin, RV, van Donkelaar, A,
12 Villeneuve, PJ and Weichenthal, S (2019). Complex relationships between greenness, air
13 pollution, and mortality in a population-based Canadian cohort. *Environment*
14 *International* 128: 292-300.
- 15 Dai, L, Zanobetti, A, Koutrakis, P and Schwartz, JD (2014). Associations of fine particulate
16 matter species with mortality in the United States: a multicity time-series analysis.
17 *Environmental Health Perspectives* 122(8): 837-842.
- 18 deSouza, P, Braun, D, Parks, RM, Schwartz, J, Dominici, F and Kioumourtzoglou, MA (2021).
19 Nationwide Study of Short-term Exposure to Fine Particulate Matter and Cardiovascular
20 Hospitalizations Among Medicaid Enrollees. *Epidemiology* 32(1): 6-13.
- 21 Devlin, RB, Ghio, AJ, Kehrl, H, Sanders, G and Cascio, W (2003). Elderly humans exposed to
22 concentrated air pollution particles have decreased heart rate variability. *European*
23 *Respiratory Journal* 40: 76S-80S.
- 24 Di, Q, Amini, H, Shi, L, Kloog, I, Silvern, R, Kelly, J, Sabath, MB, Choirat, C, Koutrakis, P and
25 Lyapustin, A (2019a). An ensemble-based model of PM_{2.5} concentration across the
26 contiguous United States with high spatiotemporal resolution. *Environment International*
27 130: 104909.
- 28 Di, Q, Amini, H, Shi, L, Kloog, I, Silvern, R, Kelly, J, Sabath, MB, Choirat, C, Koutrakis, P,
29 Lyapustin, A, Wang, Y, Mickley, LJ and Schwartz, J (2019b). An ensemble-based model
30 of PM_{2.5} concentration across the contiguous United States with high spatiotemporal
31 resolution. *Environment International* 130: 104909.
- 32 Di, Q, Dai, L, Wang, Y, Zanobetti, A, Choirat, C, Schwartz, JD and Dominici, F (2017a).
33 Association of short-term exposure to air pollution with mortality in older adults. *JAMA:*
34 *Journal of the American Medical Association* 318(24): 2446-2456.
- 35 Di, Q, Kloog, I, Koutrakis, P, Lyapustin, A, Wang, Y and Schwartz, J (2016). Assessing PM_{2.5}
36 exposures with high spatiotemporal resolution across the Continental United States.
37 *Environmental Science and Technology* 50(9): 4712-4721.

- 1 Di, Q, Wang, Y, Zanobetti, A, Wang, Y, Koutrakis, P, Choirat, C, Dominici, F and Schwartz, JD
2 (2017b). Air pollution and mortality in the Medicare population. *New England Journal of*
3 *Medicine* 376(26): 2513-2522.
- 4 Dominici, F, Peng, RD, Bell, ML, Pham, L, McDermott, A, Zeger, SL and Samet, JL (2006).
5 Fine particulate air pollution and hospital admission for cardiovascular and respiratory
6 diseases. *JAMA: Journal of the American Medical Association* 295(10): 1127-1134.
- 7 Dominici, F, Peng, RD, Zeger, SL, White, RH and Samet, JM (2007). Particulate air pollution
8 and mortality in the United States: did the risks change from 1987 to 2000? *American*
9 *Journal of Epidemiology* 166(8): 880-888.
- 10 Dominici, F, Schwartz, J, Di, Q, Braun, D, Choirat, C and Zanobetti, A (2019). Assessing
11 adverse health effects of long-term exposure to low levels of ambient air pollution: Phase
12 1. Health Effects Institute. Boston, MA.
13 <https://www.healtheffects.org/system/files/dominici-rr-200-report.pdf>.
- 14 Elliott, EG, Laden, F, James, P, Rimm, EB, Rexrode, KM and Hart, JE (2020). Interaction
15 between Long-Term Exposure to Fine Particulate Matter and Physical Activity, and Risk
16 of Cardiovascular Disease and Overall Mortality in US Women. *Environmental Health*
17 *Perspectives* 128(12): 127012.
- 18 Erickson, AC, Christidis, T, Pappin, A, Brook, JR, Crouse, DL, Hystad, P, Li, C, Martin, RV,
19 Meng, J, Pinault, L, von Donkelaar, A, Weichenthal, S, Tjepkema, M, Burnett, RT and
20 Brauer, M (2020). Disease assimilation: The mortality impacts of fine particulate matter
21 on immigrants to Canada. *Health Reports* 31(3): 14-26.
- 22 Eum, K, Suh, HH, Pun, V and Manjourides, J (2018). Impact of long-term temporal trends in
23 fine particulate matter (PM_{2.5}) on association of annual PM_{2.5} exposure and mortality:
24 an analysis of over 20 million Medicare beneficiaries. *Environmental Epidemiology* 2(2):
25 e009.
- 26 Fan, M and Wang, Y (2020). The impact of PM_{2.5} on mortality in older adults: evidence from
27 retirement of coal-fired power plants in the United States. *Environmental Health: A*
28 *Global Access Science Source* 19(1): 28.
- 29 Fisher, JA, Puett, RC, Laden, F, Wellenius, GA, Sapkota, A, Liao, D, Yanosky, JD, Carter-
30 Pokras, O, He, X and Hart, JE (2019). Case-crossover analysis of short-term particulate
31 matter exposures and stroke in the health professionals follow-up study. *Environment*
32 *International* 124: 153-160.
- 33 Frank, N. (2012). Memorandum to PM NAAQS Review Docket (EPA-HQ-OAR-2007-0492)
34 regarding the Differences between maximum and composite monitor annual PM_{2.5}
35 design values by CBSA. . Dec 14, 2012. Docket ID No. EPA-HQ-OAR-2007-0492.
36 Office of Air Quality Planning and Standards Research Triangle Park, NC. Available at:
37 <https://www.regulations.gov/document/EPA-HQ-OAR-2007-0492-10099>.

- 1 Franklin, M, Koutrakis, P and Schwartz, J (2008). The role of particle composition on the
2 association between PM_{2.5} and mortality. *Epidemiology* 19(5): 680-689.
- 3 Franklin, M, Zeka, A and Schwartz, J (2007). Association between PM_{2.5} and all-cause and
4 specific-cause mortality in 27 US communities. *Journal of Exposure Science and*
5 *Environmental Epidemiology* 17(3): 279-287.
- 6 Gauderman, WJ, Avol, E, Gilliland, F, Vora, H, Thomas, D, Berhane, K, McConnell, R,
7 Kuenzli, N, Lurmann, F, Rappaport, E, Margolis, H, Bates, D and Peters, J (2004). The
8 effect of air pollution on lung development from 10 to 18 years of age. *New England*
9 *Journal of Medicine* 351(11): 1057-1067.
- 10 Gauderman, WJ, Urman, R, Avol, E, Berhane, K, McConnell, R, Rappaport, E, Chang, R,
11 Lurmann, F and Gilliland, F (2015). Association of improved air quality with lung
12 development in children. *New England Journal of Medicine* 372(10): 905-913.
- 13 Gharibvand, L, Shavlik, D, Ghamsary, M, Beeson, WL, Soret, S, Knutsen, R and Knutsen, SF
14 (2016). The association between ambient fine particulate air pollution and lung cancer
15 incidence: Results from the AHSMOG-2 study. *Environmental Health Perspectives*
16 125(3): 378-384.
- 17 Ghio, AJ, Hall, A, Bassett, MA, Cascio, WE and Devlin, RB (2003). Exposure to concentrated
18 ambient air particles alters hematologic indices in humans. *Inhalation Toxicology* 15(14):
19 1465-1478.
- 20 Ghio, AJ, Kim, C and Devlin, RB (2000). Concentrated ambient air particles induce mild
21 pulmonary inflammation in healthy human volunteers. *American Journal of Respiratory*
22 *and Critical Care Medicine* 162(3): 981-988.
- 23 Gong, H, Jr., Linn, WS, Sioutas, C, Terrell, SL, Clark, KW, Anderson, KR and Terrell, LL
24 (2003). Controlled exposures of healthy and asthmatic volunteers to concentrated
25 ambient fine particles in Los Angeles. *Inhalation Toxicology* 15(4): 305-325.
- 26 Gong, H, Jr., Linn, WS, Terrell, SL, Anderson, KR, Clark, KW, Sioutas, C, Cascio, WE, Alexis,
27 N and Devlin, RB (2004). Exposures of elderly volunteers with and without chronic
28 obstructive pulmonary disease (COPD) to concentrated ambient fine particulate pollution.
29 *Inhalation Toxicology* 16(11-12): 731-744.
- 30 Goss, CH, Newsom, SA, Schildcrout, JS, Sheppard, L and Kaufman, JD (2004). Effect of
31 ambient air pollution on pulmonary exacerbations and lung function in cystic fibrosis.
32 *American Journal of Respiratory and Critical Care Medicine* 169(7): 816-821.
- 33 Greven, S, Dominici, F and Zeger, S (2011). An Approach to the Estimation of Chronic Air
34 Pollution Effects Using Spatio-Temporal Information. *Journal of the American Statistical*
35 *Association* 106(494): 396-406.
- 36 Hammer, MS, van Donkelaar, A, Li, C, Lyapustin, A, Sayer, AM, Hsu, NC, Levy, RC, Garay,
37 MJ, Kalashnikova, OV and Kahn, RA (2020). Global estimates and long-term trends of

- 1 fine particulate matter concentrations (1998–2018). *Environmental Science &*
2 *Technology* 54(13): 7879-7890.
- 3 Hart, JE, Garshick, E, Dockery, DW, Smith, TJ, Ryan, L and Laden, F (2011). Long-term
4 ambient multi-pollutant exposures and mortality. *American Journal of Respiratory and*
5 *Critical Care Medicine* 183(1): 73-78.
- 6 Hart, JE, Liao, X, Hong, B, Puett, RC, Yanosky, JD, Suh, H, Kioumourtzoglou, MA,
7 Spiegelman, D and Laden, F (2015). The association of long-term exposure to PM2.5 on
8 all-cause mortality in the Nurses' Health Study and the impact of measurement-error
9 correction. *Environmental Health: A Global Access Science Source* 14: 38.
- 10 Hassett-Sipple, B, Schmidt, M and Rajan, P. (2010). Memorandum to PM NAAQS Review
11 Docket (EPA-HQ-OAR-2007-0492). Analysis of PM2.5 (Particulate Matter Smaller than
12 2.5 Micrometers in Diameter). Mar 30, 2010. Docket ID No. EPA-HQ-OAR-2007-0492.
13 Office of Air Quality Planning and Standards Research Triangle Park, NC. Available at: .
- 14 Hayes, RB, Lim, C, Zhang, YL, Cromar, K, Shao, YZ, Reynolds, HR, Silverman, DT, Jones,
15 RR, Park, Y, Jerrett, M, Ahn, J and Thurston, GD (2020). PM2.5 air pollution and cause-
16 specific cardiovascular disease mortality. *International Journal of Epidemiology* 49(1):
17 25-35.
- 18 Hazucha, MJ, Bromberg, PA, Lay, JC, Bennett, W, Zeman, K, Alexis, NE, Kehrl, H, Rappold,
19 A, Cascio, WE and Devlin, RB (2013). Pulmonary responses in current smokers and ex-
20 smokers following a two hour exposure at rest to clean air and fine ambient air particles.
21 *Particle and Fibre Toxicology* 10(1): 58.
- 22 Hemmingsen, JG, Jantzen, K, Møller, P and Loft, S (2015a). No oxidative stress or DNA
23 damage in peripheral blood mononuclear cells after exposure to particles from urban
24 street air in overweight elderly. *Mutagenesis* 30(5): 635-642.
- 25 Hemmingsen, JG, Rissler, J, Lykkesfeldt, J, Sallsten, G, Kristiansen, J, P, PM and Loft, S
26 (2015b). Controlled exposure to particulate matter from urban street air is associated with
27 decreased vasodilation and heart rate variability in overweight and older adults. *Particle*
28 *and Fibre Toxicology* 12(1): 6.
- 29 Henneman, LRF, Choirat, C and Zigler, ACM (2019). Accountability assessment of health
30 improvements in the United States associated with reduced coal emissions between 2005
31 and 2012. *Epidemiology* 30(4): 477-485.
- 32 Higbee, JD, Lefler, JS, Burnett, RT, Ezzati, M, Marshall, JD, Kim, SY, Bechle, M, Robinson,
33 AL and Pope, CA (2020). Estimating long-term pollution exposure effects through
34 inverse probability weighting methods with Cox proportional hazards models.
35 *Environmental Epidemiology* 4(2): e085.
- 36 Honda, T, Eliot, MN, Eaton, CB, Whitsel, E, Stewart, JD, Mu, L, Suh, H, Szpiro, A, Kaufman,
37 JD, Vedal, S and Wellenius, GA (2017). Long-term exposure to residential ambient fine

- 1 and coarse particulate matter and incident hypertension in post-menopausal women.
2 Environment International 105: 79-85.
- 3 Huang, YC, Rappold, AG, Graff, DW, Ghio, AJ and Devlin, RB (2012). Synergistic effects of
4 exposure to concentrated ambient fine pollution particles and nitrogen dioxide in humans.
5 Inhalation Toxicology 24(12): 790-797.
- 6 Hystad, P, Demers, PA, Johnson, KC, Brook, J, van Donkelaar, A, Lamsal, L, Martin, R and
7 Brauer, M (2012). Spatiotemporal air pollution exposure assessment for a Canadian
8 population-based lung cancer case-control study. Environmental Health: A Global Access
9 Science Source 11: 22.
- 10 Hystad, P, Demers, PA, Johnson, KC, Carpiano, RM and Brauer, M (2013). Long-term
11 residential exposure to air pollution and lung cancer risk. Epidemiology 24(5): 762-772.
- 12 Ito, K, Ross, Z, Zhou, J, Nádas, A, Lippmann, M and Thurston, GD (2013). National Particle
13 Component Toxicity (NPACT) initiative: Study 3. Time-series analysis of mortality,
14 hospitalizations, and ambient PM_{2.5} and its components. 177. Health Effects Institute.
15 Boston, MA. [https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-
16 AppendixG_0.pdf](https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-AppendixG_0.pdf).
- 17 Jenkins, S. (2016). Email from Scott Jenkins to Robert Devlin. Re: Average PM_{2.5} concentration
18 in Devlin et al. (2003). Available in PM NAAQS Review Docket (EPA-HQ-OAR-2015-
19 0072-0021). March 2, 2016. Office of Air Quality Planning and Standards Research
20 Triangle Park, NC.
- 21 Kioumourtzoglou, MA, Schwartz, J, James, P, Dominici, F and Zanobetti, A (2016). PM_{2.5} and
22 mortality in 207 us cities: Modification by temperature and city characteristics.
23 Epidemiology 27(2): 221-227.
- 24 Klemm, RJ and Mason, R (2003). *HEI Special Report: Revised Analyses of Time-Series Studies
25 of Air Pollution and Health, Part II Replication of reanalysis of Harvard Six-City
26 mortality study*. Health Effects Institute. Boston, MA.
- 27 Kloog, I, Coull, BA, Zanobetti, A, Koutrakis, P and Schwartz, JD (2012). Acute and chronic
28 effects of particles on hospital admissions in New-England. PLoS ONE 7(4): e34664.
- 29 Kloog, I, Nordio, F, Zanobetti, A, Coull, BA, Koutrakis, P and Schwartz, JD (2014). Short term
30 effects of particle exposure on hospital admissions in the Mid-Atlantic states: a
31 population estimate. PLoS ONE 9(2): e88578.
- 32 Kloog, I, Ridgway, B, Koutrakis, P, Coull, BA and Schwartz, JD (2013). Long- and short-term
33 exposure to PM_{2.5} and mortality: Using novel exposure models. Epidemiology 24(4):
34 555-561.
- 35 Krall, JR, Anderson, GB, Dominici, F, Bell, ML and Peng, RD (2013). Short-term exposure to
36 particulate matter constituents and mortality in a national study of U.S. urban
37 communities. Environmental Health Perspectives 121(10): 1148-1153.

- 1 Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, III,
2 Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, Deluca, P, Finkelstein, N, Ito, K,
3 Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H and Tempalski, B (2009).
4 Extended follow-up and spatial analysis of the American Cancer Society study linking
5 particulate air pollution and mortality. ISSN 1041-5505, HEI Research Report 140.
6 Health Effects Institute. Boston, MA.
7 <https://www.healtheffects.org/system/files/Krewski140Statement.pdf>.
- 8 Kusha, M, Masse, S, Farid, T, Urch, B, Silverman, FS, Brook, RD, Gold, DR, Mangat, I, Speck,
9 M, Nair, K, Poku, K, Meyer, C, Mittleman, MA, Wellenius, GA and Nanthakumar, K
10 (2012). Controlled exposure study of air pollution and t wave alternans in volunteers
11 without cardiovascular disease. Environmental Health Perspectives 120(8): 1157-1161.
- 12 Laden, F, Schwartz, J, Speizer, FE and Dockery, DW (2006). Reduction in fine particulate air
13 pollution and mortality: extended follow-up of the Harvard Six Cities study. American
14 Journal of Respiratory and Critical Care Medicine 173(6): 667-672.
- 15 Lavigne, E, Burnett, RT and Weichenthal, S (2018). Association of short-term exposure to fine
16 particulate air pollution and mortality: effect modification by oxidant gases. Scientific
17 Reports 8(1): 16097.
- 18 Lee, M, Koutrakis, P, Coull, B, Kloog, I and Schwartz, J (2015). Acute effect of fine particulate
19 matter on mortality in three Southeastern states from 2007-2011. Journal of Exposure
20 Science and Environmental Epidemiology 26(2): 173-179.
- 21 Lefler, JS, Higbee, JD, Burnett, RT, Ezzati, M, Coleman, NC, Mann, DD, Marshall, JD, Bechle,
22 M, Wang, Y, Robinson, AL and Arden Pope, C (2019). Air pollution and mortality in a
23 large, representative U.S. cohort: multiple-pollutant analyses, and spatial and temporal
24 decompositions. Environmental Health: A Global Access Science Source 18(1): 101.
- 25 Lepeule, J, Laden, F, Dockery, D and Schwartz, J (2012). Chronic exposure to fine particles and
26 mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009.
27 Environmental Health Perspectives 120(7): 965-970.
- 28 Lipfert, FW and Wyzga, RE (2020). Environmental predictors of survival in a cohort of U.S.
29 military veterans: A multi-level spatio-temporal analysis stratified by race.
30 Environmental Research 183(Elsevier): 108842.
- 31 Lipfert, FW, Wyzga, RE, Baty, JD and Miller, JP (2006). Traffic density as a surrogate measure
32 of environmental exposures in studies of air pollution health effects: Long-term mortality
33 in a cohort of US veterans. Atmospheric Environment 40(1): 154-169.
- 34 Lippmann, M, Chen, LC, Gordon, T, Ito, K and Thurston, GD (2013). National Particle
35 Component Toxicity (NPACT) Initiative: Integrated epidemiologic and toxicologic
36 studies of the health effects of particulate matter components: Investigators' Report. 177.
37 Health Effects Institute. Boston, MA.

- 1 Liu, C, Chen, R, Sera, F, Vicedo-Cabrera, AM, Guo, Y, Tong, S, Coelho, M, Saldiva, PHN,
2 Lavigne, E, Matus, P, Valdes Ortega, N, Osorio Garcia, S, Pascal, M, Stafoggia, M,
3 Scortichini, M, Hashizume, M, Honda, Y, Hurtado-Díaz, M, Cruz, J, Nunes, B, Teixeira,
4 JP, Kim, H, Tobias, A, Íñiguez, C, Forsberg, B, Åström, C, Ragettli, MS, Guo, YL, Chen,
5 BY, Bell, ML, Wright, CY, Scovronick, N, Garland, RM, Milojevic, A, Kysely, J, Urban,
6 A, Orru, H, Indermitte, E, Jaakkola, JJK, Rytí, NRI, Katsouyanni, K, Analitis, A,
7 Zanobetti, A, Schwartz, J, Chen, J, Wu, T, Cohen, A, Gasparrini, A and Kan, H (2019).
8 Ambient Particulate Air Pollution and Daily Mortality in 652 Cities. *New England*
9 *Journal of Medicine* 381(8): 705-715.
- 10 Liu, L, Urch, B, Poon, R, Szyszkowicz, M, Speck, M, Gold, DR, Wheeler, AJ, Scott, JA, Brook,
11 JR, Thorne, PS and Silverman, FS (2015). Effects of ambient coarse, fine, and ultrafine
12 particles and their biological constituents on systemic biomarkers: A controlled human
13 exposure study. *Environmental Health Perspectives* 123(6): 534-540.
- 14 Loop, MS, McClure, LA, Levitan, EB, Al-Hamdan, MZ, Crosson, WL and Safford, MM (2018).
15 Fine particulate matter and incident coronary heart disease in the REGARDS cohort.
16 *American Heart Journal* 197: 94-102.
- 17 Lucking, AJ, Lundbäck, M, Barath, SL, Mills, NL, Sidhu, MK, Langrish, JP, Boon, NA,
18 Pourazar, J, Badimon, JJ, Gerlofs-Nijland, ME, Cassee, FR, Boman, C, Donaldson, K,
19 Sandstrom, T, Newby, DE and Blomberg, A (2011). Particle traps prevent adverse
20 vascular and prothrombotic effects of diesel engine exhaust inhalation in men.
21 *Circulation* 123(16): 1721-1728.
- 22 Malig, BJ, Green, S, Basu, R and Broadwin, R (2013). Coarse particles and respiratory
23 emergency department visits in California. *American Journal of Epidemiology* 178(1):
24 58-69.
- 25 Mauad, T, Rivero, DH, de Oliveira, RC, Lichtenfels, AJ, Guimaraes, ET, de Andre, PA,
26 Kasahara, DI, Bueno, HM and Saldiva, PH (2008). Chronic exposure to ambient levels of
27 urban particles affects mouse lung development. *American Journal of Respiratory and*
28 *Critical Care Medicine* 178(7): 721-728.
- 29 McClure, LA, Loop, MS, Crosson, W, Kleindorfer, D, Kissela, B and Al-Hamdan, M (2017).
30 Fine Particulate Matter (PM_{2.5}) and the Risk of Stroke in the REGARDS Cohort. *Journal*
31 *of Stroke & Cerebrovascular Diseases* 26(8): 1739-1744.
- 32 McConnell, R, Islam, T, Shankardass, K, Jerrett, M, Lurmann, F, Gilliland, F, Gauderman, J,
33 Avol, E, Künzli, N, Yao, L, Peters, J and Berhane, K (2010). Childhood incident asthma
34 and traffic-related air pollution at home and school. *Environmental Health Perspectives*
35 118(7): 1021-1026.
- 36 Miller, KA, Siscovick, DS, Sheppard, L, Shepherd, K, Sullivan, JH, Anderson, GL and
37 Kaufman, JD (2007). Long-term exposure to air pollution and incidence of
38 cardiovascular events in women. *New England Journal of Medicine* 356(5): 447-458.

- 1 NHLBI (2017). "NHLBI fact book, fiscal year 2012: Disease statistics." Retrieved August 23,
2 2017, from <https://www.nhlbi.nih.gov/about/documents/factbook/2012/chapter4>.
- 3 Nishimura, KK, Galanter, JM, Roth, LA, Oh, SS, Thakur, N, Nguyen, EA, Thyne, S, Farber, HJ,
4 Serebrisky, D, Kumar, R, Brigino-Buenaventura, E, Davis, A, Lenoir, MA, Meade, K,
5 Rodriguez-Cintron, W, Avila, PC, Borrell, LN, Bibbins-Domingo, K, Rodriguez-Santana,
6 JR, Sen, S, Lurmann, F, Balmes, JR and Burchard, EG (2013). Early-life air pollution and
7 asthma risk in minority children: The GALA II and SAGE II studies. *American Journal*
8 *of Respiratory and Critical Care Medicine* 188(3): 309-318.
- 9 Ostro, B, Malig, B, Hasheminassab, S, Berger, K, Chang, E and Sioutas, C (2016). Associations
10 of source-specific fine particulate matter with emergency department visits in California.
11 *American Journal of Epidemiology* 184(6): 450-459.
- 12 Pappin, AJ, Christidis, T, Pinault, LL, Crouse, DL, Brook, JR, Erickson, A, Hystad, P, Li, C,
13 Martin, RV, Meng, J, Weichenthal, S, van Donkelaar, A, Tjepkema, M, Brauer, M and
14 Burnett, RT (2019). Examining the Shape of the Association between Low Levels of Fine
15 Particulate Matter and Mortality across Three Cycles of the Canadian Census Health and
16 Environment Cohort. *Environmental Health Perspectives* 127(10): 107008.
- 17 Parker, JD, Kravets, N and Vaidyanathan, A (2018). Particulate matter air pollution exposure and
18 heart disease mortality risks by race and ethnicity in the United States: 1997-2009 NHIS
19 with mortality followup through 2011. *Circulation* 137(16): 1688-1697.
- 20 Peng, R, Bell, M, Geyh, A, McDermott, A, Zeger, S, Samet, J and Dominici, F (2009a).
21 Emergency admissions for cardiovascular and respiratory diseases and the chemical
22 composition of fine particle air pollution. *Environmental Health Perspectives* 117(6):
23 957-963.
- 24 Peng, RD, Dominici, F and Welty, LJ (2009b). A Bayesian hierarchical distributed lag model for
25 estimating the time course of risk of hospitalization associated with particulate matter air
26 pollution. *J Roy Stat Soc Ser C (Appl Stat)* 58: 3-24.
- 27 Peterson, GCL, Hogrefe, C, Corrigan, AE, Neas, LM, Mathur, R and Rappold, AG (2020).
28 Impact of Reductions in Emissions from Major Source Sectors on Fine Particulate
29 Matter-Related Cardiovascular Mortality. *Environmental Health Perspectives* 128(1):
30 17005.
- 31 Pinault, L, Brauer, M, Crouse, DL, Weichenthal, S, Erickson, A, van Donkelaar, A, Martin, RV,
32 Charbonneau, S, Hystad, P, Brook, JR, Tjepkema, M, Christidis, T, Ménard, R,
33 Robichaud, A and Burnett, RT (2018). Diabetes Status and Susceptibility to the Effects of
34 PM_{2.5} Exposure on Cardiovascular Mortality in a National Canadian Cohort.
35 *Epidemiology* 29(6): 784-794.
- 36 Pinault, LL, Weichenthal, S, Crouse, DL, Brauer, M, Erickson, A, Donkelaar, AV, Martin, RV,
37 Hystad, P, Chen, H, Finès, P, Brook, JR, Tjepkema, M and Burnett, RT (2017).
38 Associations between fine particulate matter and mortality in the 2001 Canadian Census
39 Health and Environment Cohort. *Environmental Research* 159: 406-415.

- 1 Pope, CA, III, , I, Burnett, RT, Thurston, GD, Thun, MJ, Calle, EE, Krewski, D and Godleski, JJ
2 (2004). Cardiovascular mortality and long-term exposure to particulate air pollution:
3 epidemiological evidence of general pathophysiological pathways of disease. *Circulation*
4 109(1): 71-77.
- 5 Pope, CA, III, Ezzati, M and Dockery, DW (2009). Fine-particulate air pollution and life
6 expectancy in the United States. *New England Journal of Medicine* 360(4): 376-386.
- 7 Pope, CA, Lefler, JS, Ezzati, M, Higbee, JD, Marshall, JD, Kim, SY, Bechle, M, Gilliat, KS,
8 Vernon, SE, Robinson, AL and Burnett, RT (2019). Mortality Risk and Fine Particulate
9 Air Pollution in a Large, Representative Cohort of U.S. Adults. *Environmental Health*
10 *Perspectives* 127(7): 77007.
- 11 Pope, CA, Turner, MC, Burnett, R, Jerrett, M, Gapstur, SM, Diver, WR, Krewski, D and Brook,
12 RD (2015). Relationships between fine particulate air pollution, cardiometabolic
13 disorders and cardiovascular mortality. *Circulation Research* 116(1): 108-U258.
- 14 Puett, RC, Hart, JE, Suh, H, Mittleman, M and Laden, F (2011). Particulate matter exposures,
15 mortality and cardiovascular disease in the health professionals follow-up study.
16 *Environmental Health Perspectives* 119(8): 1130-1135.
- 17 Puett, RC, Hart, JE, Yanosky, JD, Paciorek, C, Schwartz, J, Suh, H, Speizer, FE and Laden, F
18 (2009). Chronic fine and coarse particulate exposure, mortality, and coronary heart
19 disease in the Nurses' Health Study. *Environmental Health Perspectives* 117(11): 1697-
20 1701.
- 21 Puett, RC, Hart, JE, Yanosky, JD, Spiegelman, D, Wang, M, Fisher, JA, Hong, B and Laden, F
22 (2014). Particulate matter air pollution exposure, distance to road, and incident lung
23 cancer in the Nurses' Health Study cohort. *Environmental Health Perspectives* 122(9):
24 926-932.
- 25 Pun, VC, Kazemiparkouhi, F, Manjourides, J and Suh, HH (2017). Long-term PM2.5 exposures
26 and respiratory, cancer and cardiovascular mortality in American older adults. *American*
27 *Journal of Epidemiology* 186(8): 961-969.
- 28 Qiu, X, Wei, Y, Wang, Y, Di, Q, Sofer, T, Awad, YA and Schwartz, J (2020). Inverse
29 probability weighted distributed lag effects of short-term exposure to PM2.5 and ozone
30 on CVD hospitalizations in New England Medicare participants - Exploring the causal
31 effects. *Environmental Research* 182: 109095.
- 32 Raaschou-Nielsen, O, Andersen, ZJ, Beelen, R, Samoli, E, Stafoggia, M, Weinmayr, G,
33 Hoffmann, B, Fischer, P, Nieuwenhuijsen, MJ, Brunekreef, B, Xun, WW, Katsouyanni,
34 K, Dimakopoulou, K, Sommar, J, Forsberg, B, Modig, L, Oudin, A, Oftedal, B,
35 Schwarze, PE, Nafstad, P, De Faire, U, Pedersen, NL, Östenson, CG, Fratiglioni, L,
36 Penell, J, Korek, M, Pershagen, G, Eriksen, KT, Sørensen, M, Tjønneland, A, Ellermann,
37 T, Eeftens, M, Peeters, PH, Meliefste, K, Wang, M, Bueno-De-mesquita, B, Key, TJ, De
38 Hoogh, K, Concin, H, Nagel, G, Vilier, A, Grioni, S, Krogh, V, Tsai, MY, Ricceri, F,
39 Sacerdote, C, Galassi, C, Migliore, E, Ranzi, A, Cesaroni, G, Badaloni, C, Forastiere, F,

- 1 Tamayo, I, Amiano, P, Dorronsoro, M, Trichopoulou, A, Bamia, C, Vineis, P and Hoek,
2 G (2013). Air pollution and lung cancer incidence in 17 European cohorts: Prospective
3 analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE). The
4 Lancet Oncology 14(9): 813-822.
- 5 Ramanathan, G, Yin, F, Speck, M, Tseng, CH, Brook, JR, Silverman, F, Urch, B, Brook, RD and
6 Araujo, JA (2016). Effects of urban fine particulate matter and ozone on HDL
7 functionality. Particle and Fibre Toxicology 13(1): 26.
- 8 Sacks, JD, Ito, K, Wilson, WE and Neas, LM (2012). Impact of covariate models on the
9 assessment of the air pollution-mortality association in a single- and multipollutant
10 context. American Journal of Epidemiology 176(7): 622-634.
- 11 Sanders, NJ, Barreca, AI and Neidell, MJ (2020). Estimating Causal Effects of Particulate Matter
12 Regulation on Mortality. Epidemiology 31(2): 160-167.
- 13 Schwartz, J, Austin, E, Bind, MA, Zanobetti, A and Koutrakis, P (2015). Estimating causal
14 associations of fine particles with daily deaths in Boston. American Journal of
15 Epidemiology 182(7): 644-650.
- 16 Schwartz, J, Bind, MA and Koutrakis, P (2017). Estimating causal effects of local air pollution
17 on daily deaths: Effect of low levels. Environmental Health Perspectives 125(1): 23-29.
- 18 Schwartz, J, Fong, K and Zanobetti, A (2018a). A National Multicity Analysis of the Causal
19 Effect of Local Pollution, [Formula: see text], and [Formula: see text] on Mortality.
20 Environmental Health Perspectives 126(8): 87004.
- 21 Schwartz, J, Wei, Y, Yitshak-Sade, M, Di, Q, Dominici, F and Zanobetti, A (2021). A national
22 difference in differences analysis of the effect of PM_{2.5} on annual death rates.
23 Environmental Research 194: 110649.
- 24 Schwartz, JD, Wang, Y, Kloog, I, Yitshak-Sade, M, Dominici, F and Zanobetti, A (2018b).
25 Estimating the Effects of PM_{2.5} on Life Expectancy Using Causal Modeling Methods.
26 Environmental Health Perspectives 126(12): 127002.
- 27 Shi, L, Zanobetti, A, Kloog, I, Coull, BA, Koutrakis, P, Melly, SJ and Schwartz, JD (2016).
28 Low-concentration PM_{2.5} and mortality: estimating acute and chronic effects in a
29 population-based study. Environmental Health Perspectives 124(1): 46-52.
- 30 Shin, HH, Gogna, P, Maquiling, A, Parajuli, RP, Haque, L and Burr, B (2021). Comparison of
31 hospitalization and mortality associated with short-term exposure to ambient ozone and
32 PM_{2.5} in Canada. Chemosphere 265: 128683.
- 33 Shin, S, Burnett, RT, Kwong, JC, Hystad, P, van Donkelaar, A, Brook, JR, Goldberg, MS, Tu, K,
34 Copes, R, Martin, R, Lia, Y, Kopp, A and Chen, H (2019). Ambient Air Pollution and the
35 Risk of Atrial Fibrillation and Stroke: A Population-Based Cohort Study. Environmental
36 Health Perspectives 127(8): 87009.

- 1 Sivagangabalan, G, Spears, D, Masse, S, Urch, B, Brook, RD, Silverman, F, Gold, DR, Lukic,
2 KZ, Speck, M, Kusha, M, Farid, T, Poku, K, Shi, E, Floras, J and Nanthakumar, K
3 (2011). The effect of air pollution on spatial dispersion of myocardial repolarization in
4 healthy human volunteers. *Journal of the American College of Cardiology* 57(2): 198-
5 206.
- 6 Stieb, DM, Szyszkowicz, M, Rowe, BH and Leech, JA (2009). Air pollution and emergency
7 department visits for cardiac and respiratory conditions: A multi-city time-series analysis.
8 *Environmental Health: A Global Access Science Source* 8(25): 25.
- 9 Sun, S, Stewart, JD, Eliot, MN, Yanosky, JD, Liao, D, Tinker, LF, Eaton, CB, Whitsel, EA and
10 Wellenius, GA (2019). Short-term exposure to air pollution and incidence of stroke in the
11 Women's Health Initiative. *Environment International* 132: 105065.
- 12 Szyszkowicz, M (2009). Air pollution and ED visits for chest pain. *American Journal of*
13 *Emergency Medicine* 27(2): 165-168.
- 14 Tétreault, LF, Doucet, M, Gamache, P, Fournier, M, Brand, A, Kosatsky, T and Smargiassi, A
15 (2016). Childhood exposure to ambient air pollutants and the onset of asthma: an
16 administrative cohort study in Québec. *Environmental Health Perspectives* 124(8): 1276-
17 1282.
- 18 Thurston, GD, Ahn, J, Cromar, KR, Shao, Y, Reynolds, HR, Jerrett, M, Lim, CC, Shanley, R,
19 Park, Y and Hayes, RB (2016). Ambient particulate matter air pollution exposure and
20 mortality in the NIH-AARP Diet and Health Cohort. *Environmental Health Perspectives*
21 124(4): 484-490.
- 22 Thurston, GD, Ito, K, Lall, R, Burnett, RT, Turner, MC, Krewski, D, Shi, Y, Jerrett, M, Gapstur,
23 SM, Diver, WR and Pope, CA, III (2013). NPACT Study 4. Mortality and long-term
24 exposure to PM_{2.5} and its components in the American Cancer Society's Cancer
25 Prevention Study II Cohort. Research Report 177. Health Effects Institute. Boston, MA.
- 26 Thurston, GD, Kipen, H, Annesi-Maesano, I, Balmes, J, Brook, RD, Cromar, K, De Matteis, S,
27 Forastiere, F, Forsberg, B, Frampton, MW, Grigg, J, Heederik, D, Kelly, FJ, Kuenzli, N,
28 Laumbach, R, Peters, A, Rajagopalan, ST, Rich, D, Ritz, B, Samet, JM, Sandstrom, T,
29 Sigsgaard, T, Sunyer, J and Brunekreef, B (2017). A joint ERS/ATS policy statement:
30 what constitutes an adverse health effect of air pollution? An analytical framework.
31 *European Respiratory Journal* 49(1): 1600419.
- 32 Tomczak, A, Miller, AB, Weichenthal, SA, To, T, Wall, C, van Donkelaar, A, Martin, RV,
33 Crouse, DL and Villeneuve, PJ (2016). Long-term exposure to fine particulate matter air
34 pollution and the risk of lung cancer among participants of the Canadian National Breast
35 Screening Study. *International Journal of Cancer* 139(9): 1958-1966.
- 36 Tong, H, Rappold, AG, Caughey, M, Hinderliter, AL, Bassett, M, Montilla, T, Case, MW,
37 Berntsen, J, Bromberg, PA, Cascio, WE, Diaz-Sanchez, D, Devlin, RB and Samet, JM
38 (2015). Dietary supplementation with olive oil or fish oil and vascular effects of

1 concentrated ambient particulate matter exposure in human volunteers. Environmental
2 Health Perspectives 123(11): 1173-1179.

3 Turner, MC, Jerrett, M, Pope, A, III, Krewski, D, Gapstur, SM, Diver, WR, Beckerman, BS,
4 Marshall, JD, Su, J, Crouse, DL and Burnett, RT (2016). Long-term ozone exposure and
5 mortality in a large prospective study. American Journal of Respiratory and Critical Care
6 Medicine 193(10): 1134-1142.

7 U.S. EPA (2001). Risk Assessment Guidance for Superfund: Volume III - Part A, Process for
8 Conducting Probabilistic Risk Assessment. Office of Emergency and Remedial
9 Response. Washington, D.C. U.S. EPA. EPA 540-R-02-002. Available at :
10 https://www.epa.gov/sites/production/files/2015-09/documents/rags3adt_complete.pdf.

11 U.S. EPA (2004). An Examination of EPA Risk Assessment Principles and Practices. Office of
12 Research and Development. Washington, D.C. U.S. EPA. EPA/100/B-04/001. Available
13 at: <https://semsub.epa.gov/work/10/500006305.pdf>.

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

18 U.S. EPA (2011). Policy Assessment for the Review of the Particulate Matter National Ambient
19 Air Quality Standards Office of Air Quality Planning and Standards, Health and
20 Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-
21 11-003 April 2011. Available at:
22 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100AUMY.txt>.

23 U.S. EPA (2013). Regulatory Impact Analysis for the Final Revisions to the National Ambient
24 Air Quality Standards for Particulate Matter. U.S. Environmental Protection Agency,
25 Office of Air Quality Planning and Standards. Research Triangle Park, NC. U.S. EPA.
26 EPA-452/R-12-005. February 2013. Available at:
27 [https://www.epa.gov/sites/default/files/2020-07/documents/naaqs-pm_ria_final_2012-
28 12.pdf](https://www.epa.gov/sites/default/files/2020-07/documents/naaqs-pm_ria_final_2012-12.pdf).

29 U.S. EPA (2015a). Regulatory Impact Analysis of the Final Revisions to the National Ambient
30 Air Quality Standards for Ground-Level Ozone. U.S. Environmental Protection Agency,
31 Office of Air Quality Planning and Standards. Research Triangle Park, NC. U.S. EPA.
32 EPA-452/R-15-007 September 2015. Available at:
33 <https://www.epa.gov/sites/default/files/2016-02/documents/20151001ria.pdf>.

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

- 1 U.S. EPA (2016). Integrated review plan for the national ambient air quality standards for
2 particulate matter. Office of Air Quality Planning and Standards. Research Triangle Park,
3 NC. U.S. EPA. EPA-452/R-16-005. December 2016. Available at:
4 [https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-](https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf)
5 [plan.pdf](https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf).
- 6 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
7 U.S. Environmental Protection Agency, Office of Research and Development, National
8 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
9 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
10 [standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 11 U.S. EPA (2020). Policy Assessment for the Review of the National Ambient Air Quality
12 Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health
13 and Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-
14 452/R-20-002. January 2020. Available at: [https://www.epa.gov/sites/default/files/2020-](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf)
15 [01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf).
- 16 U.S. EPA (2021a). Supplement to the 2019 Integrated Science Assessment for Particulate Matter
17 (External Review Draft). U.S. Environmental Protection Agency, Office of Research and
18 Development, Center for Public Health and Environmental Assessment. Washington,
19 DC. U.S. EPA. EPA/600/R-21/198. December 2019. Available at:
20 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
21 [assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 22 U.S. EPA (2021b). Technical Support Document (TSD) for the Final Revised Cross-State Air
23 Pollution Rule Update for the 2008 Ozone Season NAAQS Estimating PM_{2.5}- and
24 Ozone-Attributable Health Benefits. U.S. Environmental Protection Agency, Office of
25 Air Quality Planning and Standards. Research Triangle Park, NC. U.S. EPA. EPA-HQ-
26 OAR-2020-0272. March 2021. Available at:
27 [https://www.epa.gov/sites/default/files/2021-03/documents/estimating_pm2.5-](https://www.epa.gov/sites/default/files/2021-03/documents/estimating_pm2.5-and_ozone-attributable_health_benefits_tsd_march_2021.pdf)
28 [and_ozone-attributable_health_benefits_tsd_march_2021.pdf](https://www.epa.gov/sites/default/files/2021-03/documents/estimating_pm2.5-and_ozone-attributable_health_benefits_tsd_march_2021.pdf).
- 29 Urch, B, Speck, M, Corey, P, Wasserstein, D, Manno, M, Lukic, KZ, Brook, JR, Liu, L, Coull,
30 B, Schwartz, J, Gold, DR and Silverman, F (2010). Concentrated ambient fine particles
31 and not ozone induce a systemic interleukin-6 response in humans. *Inhalation Toxicology*
32 22(3): 210-218.
- 33 Urman, R, McConnell, R, Islam, T, Avol, EL, Lurmann, FW, Vora, H, Linn, WS, Rappaport,
34 EB, Gilliland, FD and Gauderman, WJ (2014). Associations of children's lung function
35 with ambient air pollution: Joint effects of regional and near-roadway pollutants. *Thorax*
36 69(6): 540-547.
- 37 USDA (2018). Rural America at a Glance, 2018 Edition. U.S. Department of Agriculture,
38 Economic Research Service Washington, DC. USDA. Economic Information Bulletin
39 200. November 2018. Available at:
40 <https://www.ers.usda.gov/webdocs/publications/90556/eib-200.pdf>.

- 1 van Donkelaar, A, Martin, RV, Li, C and Burnett, RT (2019). Regional estimates of chemical
2 composition of fine particulate matter using a combined geoscience-statistical method
3 with information from satellites, models, and monitors. *Environmental Science &*
4 *Technology* 53(5).
- 5 Vieira, JL, Guimaraes, GV, de Andre, PA, Cruz, FD, Nascimento Saldiva, PH and Bocchi, EA
6 (2016a). Respiratory filter reduces the cardiovascular effects associated with diesel
7 exhaust exposure a randomized, prospective, double-blind, controlled study of heart
8 failure: the FILTER-HF trial. *JACC: Heart Failure* 4(1): 55-64.
- 9 Vieira, JL, Guimaraes, GV, de Andre, PA, Nascimento Saldiva, PH and Bocchi, EA (2016b).
10 Effects of reducing exposure to air pollution on submaximal cardiopulmonary test in
11 patients with heart failure: Analysis of the randomized, double-blind and controlled
12 FILTER-HF trial. *International Journal of Cardiology* 215: 92-97.
- 13 Villeneuve, PJ, Weichenthal, SA, Crouse, D, Miller, AB, To, T, Martin, RV, van Donkelaar, A,
14 Wall, C and Burnett, RT (2015). Long-term exposure to fine particulate matter air
15 pollution and mortality among Canadian women. *Epidemiology* 26(4): 536-545.
- 16 Wang, B, Eum, KD, Kazemiparkouhi, F, Li, C, Manjourides, J, Pavlu, V and Suh, H (2020). The
17 impact of long-term PM_{2.5} exposure on specific causes of death: exposure-response
18 curves and effect modification among 53 million U.S. Medicare beneficiaries.
19 *Environmental Health: A Global Access Science Source* 19(1): 20.
- 20 Wang, Y, Shi, L, Lee, M, Liu, P, Di, Q, Zanobetti, A and Schwartz, JD (2017). Long-term
21 exposure to PM_{2.5} and mortality among older adults in the Southeastern US.
22 *Epidemiology* 28(2): 207-214.
- 23 Ward-Caviness, CK, Weaver, AM, Buranosky, M, Pfaff, ER, Neas, LM, Devlin, RB, Schwartz,
24 J, Di, Q, Cascio, WE and Diaz-Sanchez, D (2020). Associations between long-term fine
25 particulate matter exposure and mortality in heart failure patients. *Journal of the*
26 *American Heart Association* 9(6): e012517.
- 27 Wei, Y, Wang, Y, Di, Q, Choirat, C, Wang, Y, Koutrakis, P, Zanobetti, A, Dominici, F and
28 Schwartz, JD (2019). Short term exposure to fine particulate matter and hospital
29 admission risks and costs in the Medicare population: time stratified, case crossover
30 study. *BMJ (Clinical Research Edition)* 367: l6258.
- 31 Weichenthal, S, Crouse, DL, Pinault, L, Godri-Pollitt, K, Lavigne, E, Evans, G, van Donkelaar,
32 A, Martin, RV and Burnett, RT (2016a). Oxidative burden of fine particulate air pollution
33 and risk of cause-specific mortality in the Canadian Census Health and Environment
34 Cohort (CanCHEC). *Environmental Research* 146: 92-99.
- 35 Weichenthal, S, Hatzopoulou, M and Goldberg, MS (2014). Exposure to traffic-related air
36 pollution during physical activity and acute changes in blood pressure, autonomic and
37 micro-vascular function in women: A cross-over study. *Particle and Fibre Toxicology*
38 11(1): 70.

- 1 Weichenthal, S, Lavigne, E, Evans, G, Pollitt, K and Burnett, RT (2016b). Ambient PM_{2.5} and
2 risk of emergency room visits for myocardial infarction: Impact of regional PM_{2.5}
3 oxidative potential: A case-crossover study. *Environmental Health* 15: 46.
- 4 Weichenthal, S, Lavigne, E, Evans, GJ, Godri Pollitt, KJ and Burnett, RT (2016c). PM_{2.5} and
5 emergency room visits for respiratory illness: effect modification by oxidative potential.
6 *American Journal of Respiratory and Critical Care Medicine* 194(5): 577-586.
- 7 WHO (2008). Uncertainty and Data Quality in Exposure Assessment. The International
8 Programme on Chemical Safety. Geneva. WHO.
9 https://www.who.int/ipcs/publications/methods/harmonization/exposure_assessment.pdf.
- 10 Willis, A, Jerrett, M, Burnett, RT and Krewski, D (2003). The association between sulfate air
11 pollution and mortality at the county scale: an exploration of the impact of scale on a
12 long-term exposure study. *Journal of Toxicology and Environmental Health, Part A:*
13 *Current Issues* 66(16-19): 1605-1624.
- 14 Wu, X, Braun, D, Kioumourtzoglou, MA, Choirat, C, Di, Q and Dominici, F (2019). Causal
15 inference in the context of an error prone exposure: Air pollution and mortality. *Annals*
16 *of Applied Statistics* 13(1): 520-547.
- 17 Wu, X, Braun, D, Schwartz, J, Kioumourtzoglou, MA and Dominici, F (2020a). Evaluating the
18 impact of long-term exposure to fine particulate matter on mortality among the elderly.
19 *Science Advances* 6(29): eaba5692.
- 20 Wu, X, Netherly, RC, Sabath, MB, Braun, D and Dominici, F (2020b). Air pollution and
21 COVID-19 mortality in the United States: Strengths and limitations of an ecological
22 regression analysis. *Science Advances* 6(45): eabd4049.
- 23 Wyatt, LH, Devlin, RB, Rappold, AG, Case, MW and Diaz-Sanchez, D (2020a). Low levels of
24 fine particulate matter increase vascular damage and reduce pulmonary function in young
25 healthy adults. *Particle and fibre toxicology* 17(1): 1-12.
- 26 Wyatt, LH, Peterson, GCL, Wade, TJ, Neas, LM and Rappold, AG (2020b). The contribution of
27 improved air quality to reduced cardiovascular mortality: Declines in socioeconomic
28 differences over time. *Environment International* 136: 105430.
- 29 Wyatt, LH, Xi, YZ, Kshirsagar, A, Di, Q, Ward-Caviness, C, Wade, TJ, Cascio, WE and
30 Rappold, AG (2020c). Association of short-term exposure to ambient PM_{2.5} with
31 hospital admissions and 30-day readmissions in end-stage renal disease patients:
32 population-based retrospective cohort study. *British Medical Journal Open* 10(12):
33 e041177.
- 34 Yap, P, Gilbreath, S, Garcia, C, Jareen, N and Goodrich, B (2013). The influence of
35 socioeconomic markers on the association between fine particulate matter and hospital
36 admissions for respiratory conditions among children. *American Journal of Public Health*
37 103(4): 695-702.

- 1 Yazdi, D, Wang, Y, Di, Q, Zanobetti, A and Schwartz, J (2019). Long-term exposure to PM2.5
2 and ozone and hospital admissions of Medicare participants in the Southeast USA.
3 Environment International 130: 104879.
- 4 Yazdi, DM, Wang, Y, Di, Q, Wei, YG, Requia, WJ, Shi, LH, Sabath, MB, Dominici, F, Coull,
5 BA, Evans, JS, Koutrakis, P and Schwartz, JD (2021). Long-term association of air
6 pollution and hospital admissions among Medicare participants using a doubly robust
7 additive model. Circulation 143(16): 1584-1596.
- 8 Yitshak-Sade, M, Kloog, I, Zanobetti, A and Schwartz, JD (2019). Estimating the causal effect
9 of annual PM2.5 exposure on mortality rates in the Northeastern and mid-Atlantic states.
10 Environmental Epidemiology 3(4): e052.
- 11 Yorifuji, T, Kashima, S and Doi, H (2016). Fine-particulate air pollution from diesel emission
12 control and mortality rates in Tokyo: a quasi-experimental study. Epidemiology 27(6):
13 769-778.
- 14 Zanobetti, A, Dominici, F, Wang, Y and Schwartz, JD (2014). A national case-crossover analysis
15 of the short-term effect of PM_{2.5} on hospitalizations and mortality in subjects with
16 diabetes and neurological disorders. Environmental Health: A Global Access Science
17 Source 13(1): 38.
- 18 Zanobetti, A, Franklin, M, Koutrakis, P and Schwartz, J (2009). Fine particulate air pollution and
19 its components in association with cause-specific emergency admissions. Environmental
20 Health: A Global Access Science Source 8: 58.
- 21 Zanobetti, A and Schwartz, J (2009). The effect of fine and coarse particulate air pollution on
22 mortality: A national analysis. Environmental Health Perspectives 117(6): 1-40.
- 23 Zeger, S, Dominici, F, McDermott, A and Samet, J (2008). Mortality in the Medicare population
24 and chronic exposure to fine particulate air pollution in urban centers (2000-2005).
25 Environmental Health Perspectives 116(12): 1614-1619.
- 26 Zhang, Z, Wang, J, Kwong, JC, Burnett, RT, van Donkelaar, A, Hystad, P, Martin, RV, Bai, L,
27 McLaughlin, J and Chen, H (2021). Long-term exposure to air pollution and mortality in
28 a prospective cohort: The Ontario Health Study. Environment International 154: 106570.
- 29
30

4 RECONSIDERATION OF THE PRIMARY STANDARD FOR PM₁₀

This chapter presents and evaluates the policy implications of the scientific and technical information pertaining to reconsideration of the 2020 final decision on the primary PM₁₀ standard. In so doing, the chapter presents key aspects of the health effects evidence of PM_{10-2.5}, as documented in the 2019 ISA, with support from the prior ISA and AQCDs, and associated public health implications. This information provides the basis for our evaluation of the scientific information regarding health effects of PM₁₀ in ambient air and the potential for effects to occur under air quality conditions associated with the existing standard, as well as the associated implications for public health. Our evaluation is framed around key policy-relevant questions derived from the IRP (U.S. EPA, 2016, section 2.1) for the review completed in 2020, and the scientific conclusions regarding the relationship between short- and long-term PM_{10-2.5} exposure and health effects detailed in the 2019 ISA, while also taking into account conclusions reached in previous reviews. In this way, we identify key policy-relevant issues and summary conclusions regarding the public health protection provided by the current standard as the Administrator reconsiders the final 2020 decision on the primary PM₁₀ standard.

As described in Chapter 1, the scope of the updated scientific evaluation of the health effects evidence for PM₁₀ is based on those health effects categories where the 2019 ISA concluded a causal relationship exists. Therefore, the draft ISA Supplement does not include an evaluation of additional studies for PM_{10-2.5} and the 2019 ISA continues to serve as the scientific foundation for assessing the adequacy of the primary PM₁₀ standard in this reconsideration of the 2020 final decision (U.S. EPA, 2019, section 1.7; U.S. EPA, 2021). As such, this chapter draws heavily from the 2020 PA in identifying and summarizing key issues related to this reconsideration of the primary PM₁₀ standard.

Within this chapter, background information on the current standard is summarized in section 4.1. The general approach for evaluating the available information in this reconsideration, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 4.2. Key aspects of the available health effects evidence presented in the 2019 ISA and considered in the 2020 PA are addressed in section 4.3. Section 4.4 summarizes the key evidence-based considerations identified in our evaluation and presents associated preliminary conclusions on the adequacy of the current standard. Key remaining uncertainties and areas for future research are identified in section 4.5.

4.1 BACKGROUND ON THE CURRENT STANDARD

With the 2020 final decision on the PM NAAQS, the EPA retained the existing 24-hour primary PM₁₀ 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 short-term exposures to PM_{10-2.5} (85 FR 82725, December 18, 2020). This decision was based on the scientific information available at that time, as well as the Administrator’s judgments regarding the health effects evidence and the appropriate degree of public health protection for the existing standard.

The health effects evidence assessed in the 2019 ISA included an expanded body of scientific evidence linking short-term PM_{10-2.5} to health outcomes such as premature death and hospital visits (U.S. EPA, 2009, U.S. EPA, 2019). This evidence base assessed the causal nature of relationships between short-term exposure to PM_{10-2.5} and a broad range of health effects (U.S. EPA, 2020, section 1.4.2). These effects associated with short-term exposure ranged from hospital admissions and emergency department visits for cardiovascular effects (documented in epidemiologic studies that reported PM_{10-2.5} associations with cardiovascular hospital admissions and emergency department visits in study locations with mean 24-hour average PM_{10-2.5} concentrations ranging from 7.4 to 13 µg/m³) and respiratory effects (documented in epidemiologic studies that reported PM_{10-2.5} associations with respiratory hospital admissions and emergency department visits in study locations with mean 24-hour average concentrations ranging from 5.6 to 16.2 µg/m³) to mortality (documented in epidemiologic studies that reported PM_{10-2.5} associations with mortality in study areas with mean 24-hour average concentrations ranging from 6.1 µg/m³ to 16.4 µg/m³). In addition to the epidemiologic studies, the evidence base included few controlled human exposure studies and animal toxicologic studies that provided insight into the biological plausibility of these effects. Collectively, the epidemiologic studies, controlled human exposure, and animal toxicological studies, with their inherent uncertainties, contributed to the causality determinations of “suggestive of, but not sufficient to infer, a causal relationship” between short-term exposures to PM_{10-2.5} and cardiovascular effects, respiratory effects, and mortality (U.S. EPA, 2009, U.S. EPA, 2019, section 1.4.2).

Building on the evidence considered in the 2012 review, the primary focus in the 2020 review was on multi-city and single-city epidemiologic studies that evaluated associations between short-term PM_{10-2.5} and mortality, cardiovascular effects (hospital admissions and emergency department visits), and respiratory effects. Despite differences in the approaches used to estimate ambient PM_{10-2.5} concentrations, the majority of the studies reported positive, though often not statistically significant, associations with short-term PM_{10-2.5} exposures. Most PM_{10-2.5} effect estimates remained positive in copollutant models that included either gaseous pollutants or other particulate matter size fractions (e.g., PM_{2.5}). In U.S. study locations likely to have met

1 the PM₁₀ standard during the study period, a few studies reported positive associations between
2 PM_{10-2.5} and mortality that were statistically significant and remained so in copollutant models
3 (U.S. EPA, 2009, U.S. EPA, 2019).

4 In addition to the epidemiologic studies, there were a small number of controlled human
5 exposure studies assessed in the 2019 ISA that reported alterations in heart rate variability or
6 increased pulmonary inflammation following short-term exposure to PM_{10-2.5}, providing some
7 support for the associations in the epidemiologic studies. Toxicological studies that examined the
8 effects of PM_{10-2.5} used intratracheal instillation as opposed to inhalation. Therefore, these studies
9 provided limited evidence for the biological plausibility of PM_{10-2.5}-induced effects (U.S. EPA,
10 2009, U.S. EPA, 2019).

11 Although the scientific evidence available in the 2019 ISA expanded the understanding of
12 health effects associated with PM_{10-2.5} exposures, a number of important uncertainties remained.
13 These uncertainties, and their implications for interpreting the scientific evidence, include the
14 following:

- 15 • The potential for confounding by copollutants, notably PM_{2.5}, was addressed with
16 copollutant models in a relatively small number of PM_{10-2.5} epidemiologic studies (U.S.
17 EPA, 2009, U.S. EPA, 2019). This was particularly important given the relatively small
18 body of experimental evidence (i.e., controlled human exposure and animal toxicological
19 studies) available to support the independent effect of PM_{10-2.5} on human health. This
20 increases the uncertainty regarding the extent to which PM_{10-2.5} itself, rather than one or
21 more cooccurring pollutants, is responsible for the mortality and morbidity effects
22 reported in epidemiologic studies.
- 23 • There was greater spatial variability in PM_{10-2.5} concentrations than PM_{2.5} concentrations,
24 resulting in increased exposure error for PM_{10-2.5} (U.S. EPA, 2009, U.S. EPA, 2019).
25 Available measurements did not provide sufficient information to adequately characterize
26 the spatial distribution of PM_{10-2.5} concentrations (U.S. EPA, 2009, U.S. EPA, 2019). The
27 limitations in estimates of ambient PM_{10-2.5} concentrations “would tend to increase
28 uncertainty and make it more difficult to detect effects of PM_{10-2.5} in epidemiologic
29 studies” (U.S. EPA, 2009, U.S. EPA, 2019).
- 30 • The distributions of PM_{10-2.5} concentrations over which reported health outcomes occur
31 remain highly uncertain. Only a relatively small number of PM_{10-2.5} monitoring sites were
32 operating at the time of the 2012 review and such sites had only been in operation for a
33 relatively short period of time, limiting the spatial and temporal coverage for routine
34 measurement of PM_{10-2.5} concentrations. Given these limitations in routine monitoring,
35 epidemiologic studies employed a number of different approaches for estimating PM_{10-2.5}
36 concentrations. Given the relatively small number of PM_{10-2.5} monitoring sites, the
37 relatively large spatial variability in ambient PM_{10-2.5} concentrations, the use of different
38 approaches to estimating ambient PM_{10-2.5} concentrations across epidemiologic studies,
39 and the limitations inherent in such estimates, the distributions of PM_{10-2.5} concentrations
40 over which reported health outcomes occur remain highly uncertain (U.S. EPA, 2009,
41 U.S. EPA, 2019).

- 1 • There was relatively little information on the chemical and biological composition of
2 PM_{10-2.5} and the effects associated with the various components (U.S. EPA, 2019).
3 Without more information on the chemical speciation of PM_{10-2.5}, the apparent variability
4 in associations with health effects across locations was difficult to characterize (U.S.
5 EPA, 2009, U.S. EPA, 2019).

6 Consistent with the general approach routinely employed in NAAQS reviews, the initial
7 consideration in the 2020 review of the primary PM₁₀ standard was with regard to the adequacy
8 of protection provided by the then-existing standard. Key aspects of that consideration are
9 summarized in section 4.1.1 below.

10 **4.1.1 Considerations Regarding the Adequacy of the Existing Standards in the 2020** 11 **Review**

12 In the 2020 final decision, the EPA retained the existing 24-hour primary PM₁₀ standard
13 with its level of 150 µg/m³ and its one-expected-exceedance form on average over three years to
14 continue to provide public health protection against exposures to PM_{10-2.5} (85 FR 82727,
15 December 18, 2020). In reaching his decision, the Administrator specifically noted that, while
16 the health effects evidence was somewhat expanded since the prior reviews, the overall
17 conclusions in the 2019 ISA, including uncertainties and limitations, were generally consistent
18 with what was considered in the 2012 review (85 FR 82725, December 18, 2020). In addition,
19 the Administrator recognized that there were still a number of uncertainties and limitations
20 associated with the available evidence.

21 With regard to the evidence on PM_{10-2.5}-related health effects, the Administrator noted
22 that epidemiologic studies continued to report positive associations with mortality and morbidity
23 in cities across North America, Europe, and Asia, where PM_{10-2.5} sources and composition were
24 expected to vary widely. While significant uncertainties remained in the 2020 review, the
25 Administrator recognized that this expanded body of evidence had broadened the range of effects
26 that have been linked with PM_{10-2.5} exposures. The studies evaluated in the 2019 ISA expanded
27 the scientific foundation presented in the 2009 ISA and led to revised causality determinations
28 (and new determinations) for long-term PM_{10-2.5} exposures and mortality, cardiovascular effects,
29 metabolic effects, nervous system effects, and cancer (85 FR 82726, December 18, 2020).
30 Drawing from his consideration of this evidence, the Administrator concluded that the scientific
31 information available since the time of the last review supported a decision to maintain a primary
32 PM₁₀ standard to provide public health protection against PM_{10-2.5} exposures, regardless of
33 location, source of origin, or particle composition (85 FR 82726, December 18, 2020).

34 With regard to uncertainties in the available evidence, the Administrator first noted that a
35 number of limitations were identified in the 2012 review related to: (1) estimates of ambient
36 PM_{10-2.5} concentrations used in epidemiologic studies; (2) limited evaluation of copollutant

1 models to address the potential for confounding; and (3) limited experimental studies supporting
2 biological plausibility for PM_{10-2.5}-related effects. Despite the expanded body of evidence for
3 PM_{10-2.5} exposures and health effects, the Administrator recognized that uncertainties in the 2020
4 review continued to include those associated with the exposure estimates used in epidemiologic
5 studies, the independence of the PM_{10-2.5} health effect associations, and the biologically plausible
6 pathways for PM_{10-2.5} health effects (85 FR 82726, December 18, 2020). These uncertainties
7 contributed to the 2019 ISA determinations that the evidence is “suggestive of, but not sufficient
8 to infer” causal relationships (85 FR 82726, December 18, 2020).

9 Further, consistent with the approach in reaching the 2012 decision, the approach for the
10 2020 PM NAAQS review did not include quantitative assessments of estimated exposures or
11 risks allowed by the existing standard or potential alternative standards. Further, the available
12 evidence in the 2019 ISA did not provide support for evaluating air quality distributions in
13 locations of individual epidemiologic studies as was done in the 2012 review (78 FR 3176,
14 January 15, 2013). The substantial uncertainty in such analyses, if conducted based on the
15 available PM_{10-2.5} health studies, would have been of limited utility for informing conclusions on
16 the primary PM₁₀ standard.

17 In the 2020 decision, for all of the reasons discussed above and recognizing the CASAC
18 conclusion that the evidence provided support for retaining the current standard, the
19 Administrator concluded that it was appropriate to retain the existing primary PM₁₀ standard,
20 without revision. His decision was consistent with the CASAC advice related to the primary
21 PM₁₀ standard. Specifically, the CASAC agreed with the 2020 PA conclusions that, while these
22 effects are important, the “evidence does not call into question the adequacy of the public health
23 protection afforded by the current primary PM₁₀ standard” and “supports consideration of
24 retaining the current standard in this review” (Cox, 2019a, p. 3 of letter). Thus, the Administrator
25 concluded that the primary PM₁₀ standard (in all of its elements) was requisite to protect public
26 health with an adequate margin of safety against effects that have been associated with PM_{10-2.5}.
27 In light of this conclusion, the EPA retained the existing PM₁₀ standard.

28 **4.2 GENERAL APPROACH AND KEY ISSUES IN THIS** 29 **RECONSIDERATION OF THE 2020 FINAL DECISION**

30 As is the case for all such reviews, this reconsideration of the 2020 final decision on the
31 primary PM₁₀ standard is most fundamentally based on using the Agency’s assessment of the
32 scientific evidence and quantitative information, if available, to inform the Administrator’s
33 judgments regarding a primary standard that is requisite to protect public health with an adequate
34 margin of safety. The approach for this reconsideration builds on the substantial assessments and
35 evaluations performed over previous reviews (U.S. EPA, 2011, U.S. EPA, 2020). As noted

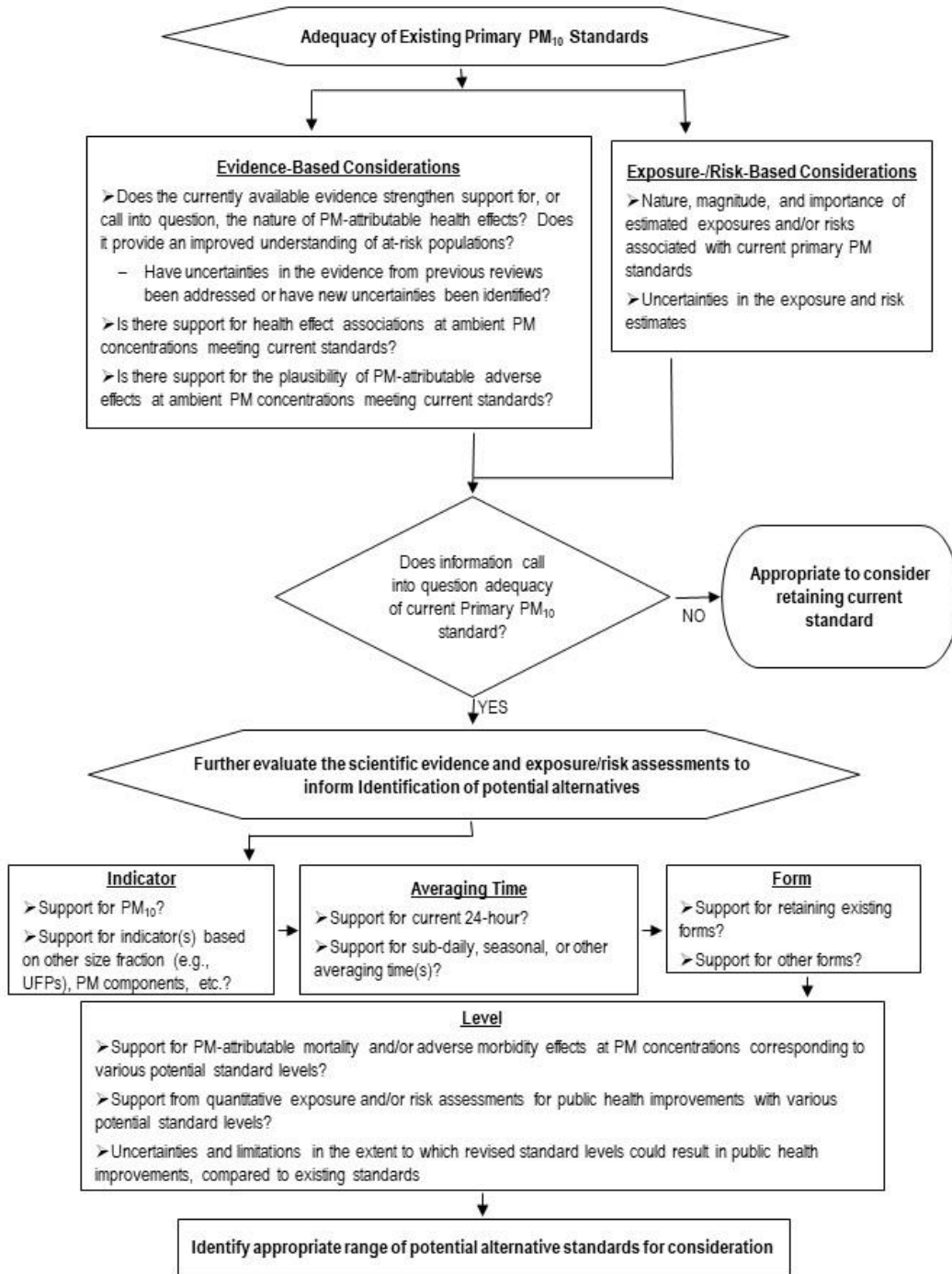
1 above, the draft ISA Supplement does not include an evaluation of studies for PM_{10-2.5} and the
2 2019 ISA continues to serve as the scientific foundation for this reconsideration. Given that there
3 is no new evidence for PM_{10-2.5}-related health effects assessed in the draft ISA Supplement that
4 would inform quantitative assessments or preliminary conclusions on the current primary PM₁₀
5 standard since the completion of the 2020 review, this draft PA draws from the evaluation of the
6 health effects evidence for PM_{10-2.5}-related effects in the 2019 ISA and considerations of such
7 effects in the 2020 PA (U.S. EPA, 2020).

8 The evaluations in this draft PA of the health effects evidence assessed in the 2019 ISA
9 are intended to inform the Administrator’s public health policy judgments and conclusions as a
10 part of this reconsideration of the 2020 final decision, including his decision as to whether to
11 retain or revise the primary PM₁₀ standard. The draft PA evaluations consider the potential
12 implications of various aspects of the scientific evidence and the associated uncertainties and
13 limitations. In so doing, the approach for this draft PA involves evaluating the available scientific
14 and technical information to address a series of key policy-relevant questions using evidence-
15 based considerations. Consideration of the full set of evidence in this reconsideration will inform
16 the answer to the following initial overarching question:

- 17 • **Does the scientific evidence support or call into question the adequacy of the**
18 **protection afforded by the current 24-hour primary PM₁₀ standard against health**
19 **effects associated with exposures to PM_{10-2.5}?**

20 In reflecting on this question, we consider the body of scientific evidence, assessed in the
21 2019 ISA, including whether it supports or calls into question the scientific conclusions reached
22 in previous reviews regarding health effects related to exposure to PM_{10-2.5} in ambient air.
23 Information available in the 2019 ISA that may be informative to public health judgments
24 regarding significance or adversity of key effects will also be considered. Further, in considering
25 this question with regard to the primary PM₁₀ standard, as in all NAAQS reviews, we give
26 particular attention to exposures and health risks to at-risk populations (including at-risk
27 lifestages). Evaluation of the scientific information with regard to this consideration of the
28 current standard will focus on key policy-relevant issues by addressing a series of questions
29 including the extent to which the available scientific evidence supports retaining or altering the
30 conclusions in the prior reviews regarding health effects attributed to PM_{10-2.5} exposures.
31 Furthermore, this draft PA will examine whether the previously identified uncertainties have
32 been reduced and if new uncertainties have been identified.

33 The general approach to reaching preliminary conclusions on the current primary PM₁₀
34 standard is summarized in Figure 4-1:



1
 2 **Figure 4-1. Overview of general approach for the reconsideration of the 2020 final decision**
 3 **on the primary PM₁₀ standard.**
 4

1 The Agency’s approach to reviewing the primary standards is consistent with the
2 requirements of the provisions of the CAA related to the review of the NAAQS and with how the
3 EPA and the courts have historically interpreted the CAA. As discussed in section 1.1 above,
4 these provisions require the Administrator to establish primary standards that, in the
5 Administrator’s judgment, are requisite (i.e., neither more nor less stringent than necessary) to
6 protect public health with an adequate margin of safety. Consistent with the Agency’s approach
7 across all NAAQS reviews, the approach of this draft PA to informing these judgments is based
8 on a recognition that the available health effects evidence generally reflects continuums that
9 include ambient air exposures for which scientists generally agree health effects are likely to
10 occur through lower levels at which the likelihood and magnitude of response become
11 increasingly uncertain. The CAA does not require the Administrator to establish a primary
12 standard at a zero-risk level or at background concentration levels, but rather at a level that
13 reduces risk sufficiently so as to protect public health, including the health of sensitive groups,¹
14 with an adequate margin of safety.

15 The decisions on the adequacy of the current primary PM₁₀ standard and on any
16 alternative standards considered in a review are largely public health policy judgments made by
17 the Administrator. The four basic elements of the NAAQS (i.e., indicator, averaging time, form,
18 and level) are generally considered collectively in evaluating the health protection afforded by
19 the current standard, and by any alternatives considered. The Administrator’s final decisions in a
20 review draw upon the scientific evidence for health effects, quantitative analyses of population
21 exposures and/or health risks, as available, and judgments about how to consider the
22 uncertainties and limitations that are inherent in the scientific evidence and quantitative analyses.

23 **4.3 HEALTH EFFECTS EVIDENCE**

24 This section draws from the EPA’s synthesis and assessment of the scientific evidence
25 presented in the 2019 ISA (U.S. EPA, 2019) to consider the following policy-relevant questions:

- 26 • **To what extent does the available scientific evidence strengthen, or otherwise alter, our**
27 **conclusions from previous reviews regarding health effects attributable to long- or**
28 **short-term PM_{10-2.5} exposures? Have previously identified uncertainties been reduced?**
29 **What important uncertainties remain and have new uncertainties been identified?**

30 Answers to these questions will inform our response to the overarching question on the adequacy
31 of the current primary PM₁₀ standard, posed at the beginning of this chapter. In section 4.3.1

¹ More than one population group may be identified as sensitive or at-risk in a NAAQS review. Decisions on NAAQS reflect consideration of the degree to which protection is provided for these sensitive population groups. To the extent that any particular population group is not among the identified sensitive groups, a decision that provides protection for the sensitive groups would be expected to also provide protection for other population groups.

1 below, we consider the nature of the effects attributable to long-term and short-term PM_{10-2.5}
 2 exposures.

3 **4.3.1 Nature of Effects**

4 As noted above, for the health effect categories and exposure duration combinations
 5 evaluated, the 2019 ISA concludes that the evidence supports causality determinations for
 6 PM_{10-2.5} that are “suggestive of, but not sufficient to infer, a causal relationship.” These health
 7 effect categories, along with their corresponding causality determinations from the 2009 ISA, are
 8 highlighted below in Table 4-1 (adapted from U.S. EPA, 2019, Table 1-4).

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

Health Outcome	Exposure Duration	2009 PM ISA	2019 PM ISA
Mortality	Long-term	Inadequate	Suggestive of, but not sufficient to infer
	Short-term	Suggestive of, but not sufficient to infer	
Cardiovascular effects	Long-term	Inadequate	
	Short-term	Suggestive of, but not sufficient to infer	
Respiratory effects	Short-term	Suggestive of, but not sufficient to infer	
Cancer	Long-term	Inadequate	
Nervous System effects	Long-term	---	
Metabolic effects	Long-term	---	

10
 11 While the evidence supporting the causal nature of relationships between exposure to
 12 PM_{10-2.5} has been strengthened for some of the health effect categories listed in Table 4-1 since
 13 the 2009 ISA, the 2019 ISA concludes that overall “the uncertainties in the evidence identified in
 14 the 2009 PM ISA have, to date, still not been addressed” (U.S. EPA, 2019, section 1.4.2, p. 1-
 15 41). Specifically, epidemiologic studies available in the 2012 review relied on various methods
 16 to estimate PM_{10-2.5} concentrations, and these methods had not been systematically compared to
 17 evaluate spatial and temporal correlations in PM_{10-2.5} concentrations. Methods included (1)
 18 calculating the difference between PM₁₀ and PM_{2.5} concentrations at co-located monitors, (2)
 19 calculating the difference between county-wide averages of monitored PM₁₀- and PM_{2.5}-based on
 20 monitors that are not necessarily co-located, and (3) direct measurement of PM_{10-2.5} using a
 21 dichotomous sampler (U.S. EPA, 2019, section 1.4.2). As described in the 2019 ISA, there
 22 continues to be variability across epidemiologic studies in the approaches used to estimate PM₁₀-

1 2.5 concentrations. Additionally, some studies estimate long-term PM_{10-2.5} exposures as the
2 difference between PM₁₀ and PM_{2.5} concentrations based on information from spatiotemporal or
3 land use regression (LUR) models, in addition to monitors. The various methods used to estimate
4 PM_{10-2.5} concentrations have not been systematically evaluated (U.S. EPA, 2019, section
5 3.3.1.1), contributing to uncertainty regarding the spatial and temporal correlations in PM_{10-2.5}
6 concentrations across methods and in the PM_{10-2.5} exposure estimates used in epidemiologic
7 studies (U.S. EPA, 2019, section 2.5.1.2.3). Given the greater spatial and temporal variability of
8 PM_{10-2.5} and the lower number of PM_{10-2.5} monitoring sites, compared to PM_{2.5}, this uncertainty is
9 particularly important for the coarse size fraction.

10 Beyond the uncertainty associated with PM_{10-2.5} exposure estimates in epidemiologic
11 studies, the limited information on the potential for confounding by copollutants and the limited
12 support available for the biological plausibility of health effects following PM_{10-2.5} exposures
13 also continue to contribute to uncertainty in the PM_{10-2.5} health evidence. Uncertainty related to
14 potential confounding stems from the relatively small number of epidemiologic studies that have
15 evaluated PM_{10-2.5} health effect associations in copollutants models with both gaseous pollutants
16 and other PM size fractions. On the other hand, uncertainty related to the biological plausibility
17 of effects attributed to PM_{10-2.5} exposures results from the small number of controlled human
18 exposure and animal toxicology² studies that have evaluated the health effects of experimental
19 PM_{10-2.5} inhalation exposures. The evidence supporting the 2019 ISA’s “suggestive of, but not
20 sufficient to infer, a causal relationship” causality determinations for PM_{10-2.5}, including
21 uncertainties in this evidence, is summarized in sections 4.3.1.1 to 4.3.1.6 below.

22 **4.3.1.1 Mortality**

23 Long-term exposures

24 Due to the dearth of studies examining the association between long-term PM_{10-2.5}
25 exposure and mortality, the 2009 ISA concluded that the evidence was “inadequate to determine
26 if a causal relationship exists” (U.S. EPA, 2009, U.S. EPA, 2019). As reported in the 2019 ISA,
27 some recent cohort studies conducted in the U.S. and Europe report positive associations
28 between long-term PM_{10-2.5} exposure and total (nonaccidental) mortality, though results are
29 inconsistent across studies (U.S. EPA, 2019, Table 11-11). The examination of copollutant
30 models in these studies remains limited and, when included, PM_{10-2.5} effect estimates were often
31 attenuated after adjusting for PM_{2.5} (U.S. EPA, 2019, Table 11-11). Across studies, PM_{10-2.5}
32 exposure concentrations were estimated using a variety of approaches, including direct

² Compared to humans, rats and mice have small nasal passages, allowing smaller fractions of inhaled PM_{10-2.5} to penetrate into the thoracic regions of the lungs of rats and mice (U.S. EPA, 2019, section 4.1.6), contributing to the relatively limited evaluation of PM_{10-2.5} exposures in animal studies.

1 measurements from dichotomous samplers, calculating the difference between PM₁₀ and PM_{2.5}
2 concentrations measured at collocated monitors, and calculating the difference between area-
3 wide concentrations of PM₁₀ and PM_{2.5}. As discussed above, temporal and spatial correlations
4 between these approaches have not been evaluated, contributing to uncertainty regarding the
5 potential for exposure measurement error (U.S. EPA, 2019, section 3.3.1.1 and Table 11-11).
6 The 2019 ISA concludes that this uncertainty “reduces the confidence in the associations
7 observed across studies” (U.S. EPA, 2019, p. 11-125). The 2019 ISA additionally concludes that
8 the evidence for long-term PM_{10-2.5} exposures and cardiovascular effects, respiratory morbidity,
9 and metabolic disease evidence provides limited biological plausibility for PM_{10-2.5}-related
10 mortality (U.S. EPA, 2019, sections 11.4.1 and 11.4). Taken together, the 2019 ISA concludes
11 that, “this body of evidence is suggestive of, but not sufficient to infer, a causal relationship
12 between long-term PM_{10-2.5} exposure and total mortality” (U.S. EPA, 2019, p. 11-125).

13 Short-term exposures

14 The 2009 ISA concluded that the evidence is "suggestive of a causal relationship between
15 short-term exposure to PM_{10-2.5} and mortality” (U.S. EPA, 2009). The 2019 ISA included
16 multicity epidemiologic studies conducted primarily in Europe and Asia which continue to
17 provide consistent evidence of positive associations between short-term PM_{10-2.5} exposure and
18 total (nonaccidental) mortality (U.S. EPA, 2019, Table 11-9). Although these studies contribute
19 to increasing confidence in the PM_{10-2.5}-mortality relationship, the use of a variety of approaches
20 to estimate PM_{10-2.5} exposures continues to contribute uncertainty to the associations observed.
21 Studies considered in the 2019 ISA continue to expand the assessment of potential copollutant
22 confounding of the PM_{10-2.5}-mortality relationship and provide evidence that PM_{10-2.5}
23 associations generally remain positive in copollutant models, though associations are attenuated
24 in some instances (U.S. EPA, 2019, section 11.3.4.1, Figure 11-28, Table 11-10). The 2019 ISA
25 concludes that, overall, the assessment of potential copollutant confounding is limited due to the
26 lack of information on the correlation between PM_{10-2.5} and gaseous pollutants and the small
27 number of locations in which copollutant analyses have been conducted. Associations with
28 cause-specific mortality provide some support for associations with total (nonaccidental)
29 mortality, though associations with cause-specific mortality, particularly respiratory mortality,
30 are more uncertain (i.e., wider confidence intervals) and less consistent (U.S. EPA, 2019, section
31 11.3.7). The 2019 ISA concludes that the evidence for PM_{10-2.5}-related cardiovascular and
32 respiratory effects provides only limited support for the biological plausibility of a relationship
33 between short-term PM_{10-2.5} exposure and cardiovascular mortality (U.S. EPA, 2019, section
34 11.3.7). Based on the overall evidence, the 2019 ISA concludes that, “this body of evidence is
35 suggestive of, but not sufficient to infer, a causal relationship between short-term PM_{10-2.5}
36 exposure and total mortality” (U.S. EPA, 2019, p. 11-120).

1 **4.3.1.2 Cardiovascular Effects**

2 Long-term exposures

3 In the 2009 ISA, the evidence describing the relationship between long-term exposure to
4 PM_{10-2.5} and cardiovascular effects was characterized as “inadequate to infer the presence or
5 absence of a causal relationship.” The limited number of epidemiologic studies reported
6 contradictory results and experimental evidence demonstrating an effect of PM_{10-2.5} on the
7 cardiovascular system was lacking (U.S. EPA, 2019, section 6.4).

8 The evidence relating long-term PM_{10-2.5} exposures to cardiovascular mortality remains
9 limited, with no consistent pattern of associations across studies and, as discussed above,
10 uncertainty stemming from the use of various approaches to estimate PM_{10-2.5} concentrations
11 (U.S. EPA, 2019, Table 6-70). The evidence for associations with cardiovascular morbidity has
12 grown since the 2009 ISA and, while results across studies are not entirely consistent, some
13 epidemiologic studies report positive associations with ischemic heart disease (IHD) and
14 myocardial infarction (MI) (U.S. EPA, 2019, Figure 6-34); stroke (U.S. EPA, 2019, Figure 6-
15 35); atherosclerosis; venous thromboembolism (VTE); and blood pressure and hypertension
16 (U.S. EPA, 2019, Section 6.4.6). PM_{10-2.5} cardiovascular mortality effect estimates are often
17 attenuated, but remain positive, in models that adjust for PM_{2.5}. For morbidity outcomes,
18 associations are inconsistent in models that adjust for PM_{2.5}, NO₂, and chronic noise pollution
19 (U.S. EPA, 2019, p. 6-276). The lack of toxicological evidence for long-term PM_{10-2.5} exposures
20 represents a substantial data gap (U.S. EPA, 2019, section 6.4.10), resulting in the 2019 ISA
21 conclusion that “evidence from experimental animal studies is of insufficient quantity to
22 establish biological plausibility” (U.S. EPA, 2019, p. 6-277). Based largely on the observation of
23 positive associations in some epidemiologic studies, the 2019 ISA concludes that “evidence is
24 suggestive of, but not sufficient to infer, a causal relationship between long-term PM_{10-2.5}
25 exposure and cardiovascular effects” (U.S. EPA, 2019, p. 6-277).

26 Short-term exposures

27 The 2009 ISA concluded that the available evidence for short-term PM_{10-2.5} exposure and
28 cardiovascular effects was “suggestive of a causal relationship.” This conclusion was based on
29 several epidemiologic studies reporting associations between short-term PM_{10-2.5} exposure and
30 cardiovascular effects, including IHD hospitalizations, supraventricular ectopy, and changes in
31 heart rate variability (HRV). In addition, dust storm events resulting in high concentrations of
32 crustal material were linked to increases in total cardiovascular disease emergency department
33 visits and hospital admissions. However, the prior reviews noted the potential for exposure
34 measurement error and copollutant confounding in these epidemiologic studies. In addition, there
35 was only limited evidence of cardiovascular effects from a small number of experimental studies
36 (e.g., animal toxicological studies and controlled human exposure studies) that examined short-

1 term PM_{10-2.5} exposures (U.S. EPA, 2009, U.S. EPA, 2019). In the 2019 ISA, key uncertainties
2 include the potential for exposure measurement error, copollutant confounding, and limited
3 evidence of biological plausibility for cardiovascular effects following inhalation exposure (U.S.
4 EPA, 2019, section 6.3.13).

5 The evidence for short-term PM_{10-2.5} exposure and cardiovascular outcomes has expanded
6 since the 2009 ISA, though important uncertainties remain. The 2019 ISA notes that there are a
7 small number of epidemiologic studies reporting positive associations between short-term
8 exposure to PM_{10-2.5} and cardiovascular-related morbidity outcomes. However, the evidence is
9 limited to suggest that these associations were biologically plausible, or independent of
10 copollutant confounding. The 2019 ISA also concludes that it remains unclear how the
11 approaches used to estimate PM_{10-2.5} concentrations in epidemiologic studies may impact
12 exposure measurement error. Taken together, the 2019 ISA concludes that “the evidence is
13 suggestive of, but not sufficient to infer, a causal relationship between short-term PM_{10-2.5}
14 exposures and cardiovascular effects” (U.S. EPA, 2019, p.6-254).

15 **4.3.1.3 Respiratory Effects**

16 Short-term exposures

17 Based on a small number of epidemiologic studies observing associations with some
18 respiratory effects and limited evidence from experimental studies to support biological
19 plausibility, the 2009 ISA concluded that the relationship between short-term exposure to PM₁₀₋
20 _{2.5} and respiratory effects is “suggestive of a causal relationship” (U.S. EPA, 2009).
21 Epidemiologic findings were consistent for respiratory infection and combined respiratory-
22 related diseases, but not for COPD. Studies were characterized by overall uncertainty in the
23 exposure assignment approach and limited information regarding potential copollutant
24 confounding. Controlled human exposure studies of short-term PM_{10-2.5} exposures found no lung
25 function decrements and inconsistent evidence for pulmonary inflammation. Animal
26 toxicological studies were limited to those using non-inhalation (e.g., intra-tracheal instillation)
27 routes of PM_{10-2.5} exposure.

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

1 sections 2.5.1.2.3 and 3.3.1.1). Taken together, the 2019 ISA concludes that “the collective
2 evidence is suggestive of, but not sufficient to infer, a causal relationship between short-term
3 PM_{10-2.5} exposure and respiratory effects” (U.S. EPA, 2019, p. 5-270).

4 **4.3.1.4 Cancer**

5 Long-term exposures

6 In the 2012 review, few studies examined cancer following inhalation exposures to PM₁₀₋
7 _{2.5}. Thus, the 2009 ISA determined the evidence was “inadequate to assess the relationship
8 between long-term PM_{10-2.5} exposures and cancer” (U.S. EPA, 2009). The scientific information
9 assessed in the 2019 ISA of long-term PM_{10-2.5} exposure and cancer remains limited, with a few
10 recent epidemiologic studies reporting positive, but imprecise, associations with lung cancer
11 incidence (U.S. EPA, 2019). Additionally, uncertainty remains in these studies with respect to
12 exposure measurement error due to the use of PM_{10-2.5} predictions that have not been validated
13 by monitored PM_{10-2.5} concentrations (U.S. EPA, 2019, sections 3.3.2.3 and 10.3.4). Relatively
14 few experimental studies of PM_{10-2.5} have been conducted, though available studies indicate that
15 PM_{10-2.5} exhibits two key characteristics of carcinogens: genotoxicity and oxidative stress. While
16 limited, such experimental studies provide some evidence of biological plausibility for the
17 findings in a small number of epidemiologic studies (U.S. EPA, 2019, section 10.3.4). Taken
18 together, the small number of epidemiologic and experimental studies, along with uncertainty
19 with respect to exposure measurement error, contribute to the determination in the 2019 ISA that,
20 “the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term
21 PM_{10-2.5} exposure and cancer” (U.S. EPA, 2019, p. 10-87).

22 **4.3.1.5 Metabolic Effects**

23 Long-term exposures

24 The 2009 ISA did not make a causality determination for PM_{10-2.5}-related metabolic
25 effects. One epidemiologic study is assessed in the 2019 ISA that reports an association between
26 long-term PM_{10-2.5} exposure and diabetes incidence, while additional cross-sectional studies
27 report associations with effects on glucose or insulin homeostasis (U.S. EPA, 2019, section 7.4).
28 As discussed above for other outcomes, uncertainties with the epidemiologic evidence include
29 the potential for copollutant confounding and exposure measurement error (U.S. EPA, 2019,
30 Tables 7-14 and 7-15). The evidence base to support the biological plausibility of metabolic
31 effects following PM_{10-2.5} exposures is limited, but a cross-sectional study that investigated
32 biomarkers of insulin resistance and systemic and peripheral inflammation may support a
33 pathway leading to type 2 diabetes (U.S. EPA, 2019, sections 7.4.1 and 7.4.3). Based on the
34 expanded, though still limited evidence base, the 2019 ISA concludes that, “[o]verall, the

1 evidence is suggestive of, but not sufficient to infer, a causal relationship between [long]-term
2 PM_{10-2.5} exposure and metabolic effects” (U.S. EPA, 2019, p. 7-56).

3 **4.3.1.6 Nervous system effects**

4 Long-term exposures

5 The 2009 ISA did not make a causality determination for PM_{10-2.5}-related nervous system
6 effects. In the 2019 ISA, available epidemiologic studies report associations between PM_{10-2.5}
7 and impaired cognition and anxiety in adults in longitudinal analyses (U.S. EPA, 2019, Table 8-
8 25, section 8.4.5). Associations of long-term exposure with neurodevelopmental effects are not
9 consistently reported in children (U.S. EPA, 2019, sections 8.4.4 and 8.4.5). Uncertainties in
10 these studies include the potential for copollutant confounding, as no studies examined
11 copollutants models (U.S. EPA, 2019, section 8.4.5), and for exposure measurement error, given
12 the use of various model-based subtraction methods to estimate PM_{10-2.5} concentrations (U.S.
13 EPA, 2019, Table 8-25). In addition, there is only limited animal toxicological evidence
14 supporting the biological plausibility of nervous system effects (U.S. EPA, 2019, sections 8.4.1
15 and 8.4.5). Overall, the 2019 ISA concludes that, “the evidence is suggestive of, but not
16 sufficient to infer, a causal relationship between long-term PM_{10-2.5} exposure and nervous system
17 effects (U.S. EPA, 2019, p. 8-75).

18 **4.3.1.7 Preliminary Conclusions Drawn from the Evidence**

19 With the evidence available in this reconsideration, as assessed in the 2019 ISA (U.S.
20 EPA, 2019) and summarized in subsections 4.3.1.1 to 4.3.1.6 above, we revisit the policy-
21 relevant questions posed at the beginning of this section:

- 22 • **To what extent does the available scientific evidence strengthen, or otherwise alter, our**
23 **conclusions from previous reviews regarding health effects attributable to long- or**
24 **short-term PM_{10-2.5} exposures? Have previously identified uncertainties been reduced?**
25 **What important uncertainties remain and have new uncertainties been identified?**

26 For each of these categories of effects listed above, the 2019 ISA concludes that the
27 evidence is “suggestive of, but not sufficient to infer, a causal relationship” (U.S. EPA, 2019).
28 As summarized in the sections above, key uncertainties in the evidence result from limitations in
29 the approaches used to estimate ambient PM_{10-2.5} concentrations in epidemiologic studies, limited
30 examination of the potential for confounding by co-occurring pollutants, and limited support for
31 the biological plausibility of the serious effects reported in many epidemiologic studies. The
32 evidence base for several PM_{10-2.5}-related health effects has expanded over time, broadening our
33 understanding of the range of health effects linked to PM_{10-2.5} exposures. This includes additional
34 evidence for the relationships between long-term exposures and cardiovascular effects, metabolic

1 effects, nervous system effects, cancer, and mortality. However, the 2019 ISA identifies a
2 number of key limitations in the evidence, including the following:

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

14 These uncertainties contribute to the conclusions in the 2019 ISA that the evidence for the PM₁₀-
15 2.5-related health effects discussed in this section for both short- and long-term exposures is
16 “suggestive of, but not sufficient to infer, a causal relationship.”

17 **4.4 PRELIMINARY CONCLUSIONS ON THE ADEQUACY OF THE** 18 **CURRENT PRIMARY PM₁₀ STANDARD**

19 This section describes our preliminary conclusions regarding the adequacy of the current
20 primary PM₁₀ standard. Our approach to reaching preliminary conclusions considers the EPA’s
21 assessment of the scientific evidence for PM_{10-2.5}-related health effects in the 2019 ISA. We
22 revisit the overarching question for this chapter:

- 23 • **Does the available scientific evidence support or call into question the adequacy of**
24 **the protection afforded by the current primary PM₁₀ standard against health effects**
25 **associated with exposures to PM_{10-2.5}?**

26 As an initial matter, we note that the scope of the updated scientific evaluation of the
27 health effects evidence for PM₁₀ is based on those health effects categories where the 2019 ISA
28 concludes a causal relationship exists. Therefore, the draft ISA Supplement does not include an
29 evaluation of additional studies for PM_{10-2.5} and the 2019 ISA continues to serve as the scientific
30 foundation for assessing the adequacy of the primary PM₁₀ standard in this reconsideration of the
31 2020 final decision (U.S. EPA, 2019, section 1.7; U.S. EPA, 2021). As such, this section
32 describing our preliminary conclusions regarding the adequacy of the current primary PM₁₀
33 standard draws heavily from the conclusions in the 2020 PA related to the primary PM₁₀
34 standard (U.S. EPA, 2020, section 4.4). Lastly, we recognize that a final decision on the primary
35 PM₁₀ standard in this reconsideration will be largely a public health policy judgement in which
36 the Administrator weighs the evidence, including its associated uncertainties.

1 With respect to the indicator, we note that the evidence continues to support retaining the
2 PM₁₀ indicator given that the varying concentrations of PM_{10-2.5} permitted in urban versus non-
3 urban areas under a PM₁₀ standard, based on the varying levels of PM_{2.5} present (i.e., lower
4 PM_{10-2.5} concentrations allowed in urban areas, where PM_{2.5} concentrations tend to be higher),
5 appropriately reflect differences in the strength of PM_{10-2.5} health effects evidence.

6 Regarding evidence for PM_{10-2.5}-related health effects, we note that the evidence for
7 several PM_{10-2.5}-related health effects has expanded, particularly for long-term exposures,
8 broadening our understanding of the range of effects linked to PM_{10-2.5} exposures. The
9 epidemiologic studies considered in the 2019 ISA continue to report positive associations with
10 mortality or morbidity in cities across North America, Europe, and Asia, where PM_{10-2.5} sources
11 and composition are expected to vary widely. Such studies provide an important part of the body
12 of evidence supporting the strengthened causality determinations (and new determinations) for
13 long-term PM_{10-2.5} exposures and mortality, cardiovascular effects, metabolic effects, nervous
14 system effects and cancer (U.S. EPA, 2019). Although most of these studies examined PM_{10-2.5}
15 health effect associations in urban areas, some studies have also linked mortality and morbidity
16 with relatively high ambient concentrations of particles of non-urban crustal origin from dust
17 storm events (U.S. EPA, 2019). Drawing from this evidence, we note continued support for
18 maintaining a standard that provides some measure of protection against exposures to PM_{10-2.5},
19 regardless of location, source of origin, or particle composition (78 FR 3176, January 15, 2013).
20 Thus, the scientific evidence evaluated for this reconsideration does not call into question the
21 decision in the 2020 review to maintain a primary standard that provides some measure of public
22 health protection against PM_{10-2.5} exposures, regardless of location, source of origin, or particle
23 composition.

24 With regard to uncertainties, the 2019 ISA notes that important uncertainties remain in the
25 evidence base for PM_{10-2.5}-related health effects. As summarized in section 4.3.1 above, these
26 include uncertainties in the PM_{10-2.5} exposure estimates used in epidemiologic studies, in the
27 independence of PM_{10-2.5} health effect associations, and in the biological plausibility of the
28 PM_{10-2.5}-related effects. Thus, the evidence available in the 2019 ISA for consideration in
29 reaching preliminary conclusions in this reconsideration is subject to the same broad
30 uncertainties present in the 2012 review (U.S. EPA, 2019). Consistent with the assessment of the
31 evidence in the 2009 ISA, these uncertainties contribute to the determinations in the 2019 ISA
32 that the evidence for key PM_{10-2.5}-related health effects is “suggestive of, but not sufficient to
33 infer” causal relationships (U.S. EPA, 2019). Drawing from this information, we reach the
34 preliminary conclusion that, as in previous reviews, such uncertainties raise questions regarding
35 the degree to which additional public health improvements would be achieved by revising the
36 existing PM₁₀ standard.

1 When the above information is taken together, we reach the preliminary conclusion that
2 the available evidence does not call into question the scientific judgments that informed the
3 decision in the 2020 review to retain the current primary PM₁₀ standard in order to protect
4 against PM_{10-2.5} exposures. Specifically, while the evidence supports maintaining a PM₁₀
5 standard to provide some measure of protection against PM_{10-2.5} exposures, uncertainties in the
6 evidence lead to questions regarding the potential public health implications of revising the
7 existing PM₁₀ standard. Thus, consistent with the approach taken in the previous reviews, we
8 reach the preliminary conclusion that the evidence does not call into question the adequacy of the
9 public health protection afforded by the current primary PM₁₀ standard. Furthermore, the
10 available evidence in this reconsideration of the 2020 final decision supports retaining the
11 current standard. As such, we have not evaluated alternative standards in this updated PA.

12 **4.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

13 As discussed above, a number of key uncertainties and limitations in the health evidence
14 have been considered, consistent with those identified in the 2009 ISA and 2019 ISA. In this
15 section, we highlight areas for future health-related research and data collection activities from
16 the 2020 PA to address these uncertainties and limitations in the evidence (U.S. EPA, 2020,
17 section 4.5). These efforts, if undertaken, could provide important evidence for informing future
18 reviews of the PM NAAQS. Key areas for future research efforts are summarized below.

- 19 • The body of experimental inhalation studies of exposure to PM_{10-2.5} (e.g., controlled
20 human exposure and animal toxicology studies) is relatively sparse. While coarse PM
21 inhalation studies in rats and mice are complicated by substantial differences in dosimetry
22 (i.e., compared to humans), additional experimental studies of short- or long-term PM₁₀₋
23 _{2.5} exposures could play an important role in weight of evidence judgments in future
24 ISAs. Experimental evaluation of effects that are plausibly related to the serious health
25 outcomes documented in epidemiologic studies could be particularly informative. Such
26 effects could include changes in markers of cardiovascular or respiratory function, similar
27 to the effects that have been evaluated following PM_{2.5} exposures (e.g., vascular function,
28 blood pressure, heart rate and heart rate variability, markers of potential for coagulation,
29 systemic and respiratory inflammation, respiratory function, etc.).
- 30 • The potential for exposure error is of particular concern for PM_{10-2.5}, given its less
31 homogeneous atmospheric distribution compared to fine particles (U.S. EPA, 2019, U.S.
32 EPA, 2009 section 1.2.1.5) and the relatively sparse PM_{10-2.5} monitoring network.
33 Therefore, efforts to develop and validate new exposure estimation approaches, or to
34 further validate existing approaches, would be informative.
- 35 • Existing epidemiologic studies have rarely examined associations with PM_{10-2.5} in
36 copollutant models, contributing to uncertainty in the degree to which reported health
37 effect associations are independent of potential confounding variables. Additional
38 epidemiologic studies that evaluate copollutants models would be informative.

- 1 • Epidemiologic studies use a variety of approaches to measure/estimate PM_{10-2.5}
2 concentrations, including: (1) difference method with co-located monitors, (2) difference
3 method with area-wide averages of monitored PM₁₀ and PM_{2.5}, (3) difference method
4 with area-wide averages of modeled PM₁₀ and PM_{2.5} or (4) direct measurement of
5 PM_{10-2.5} using a dichotomous sampler. It is important that we better understand how these
6 methods compare to one another, both in terms of absolute estimated concentrations and
7 in terms of the spatial and temporal correlations in those estimated concentrations
8 between methods.
- 9 • Measurement capabilities and the availability of PM_{10-2.5} ambient concentration data have
10 greatly increased since the 2009 ISA (U.S. EPA, 2019, U.S. EPA, 2009, section
11 2.5.1.1.3). Starting in 2011, PM_{10-2.5} has been monitored at NCore stations, IMPROVE
12 stations, and several sites run by State and local agencies. Furthermore, there has been an
13 increase in the deployment of PM_{2.5} FEM monitors that also measure PM_{10-2.5}. To date,
14 epidemiologic studies have used a variety of approaches to measure/estimate PM_{10-2.5}
15 concentrations but have not used direct measurements from NCore or IMPROVE stations
16 to evaluate health effects associations with PM_{10-2.5} exposure. A body of epidemiologic
17 studies that evaluate health effect associations using monitoring data from these stations
18 could allow more direct comparisons of results across studies.
- 19 • Evaluate and expand the PM_{10-2.5} network, along with speciation of PM_{10-2.5} including
20 multi-elements, major ions, carbon (including carbonate carbon), and bioaerosols.
- 21 • Characterize PM_{10-2.5} in different health-relevant exposure environments (e.g., city center,
22 suburban, roadside, agricultural, and rural areas) for mass, elements (including potential
23 toxic species), carbonaceous materials (including selected organic compounds and
24 carbonate), water-soluble ions, and bioaerosols (including endotoxins, 1,3 beta glucans,
25 and total protein).
- 26 • Additional areas of interest for future research include:
- 27 ○ Further evaluation of the potential for particular PM_{10-2.5} components, groups of
28 components, or other particle characteristics to contribute to exposure-related
29 health effects.
- 30 ○ Research to improve our understanding of concentration-response relationships
31 and the confidence bounds around these relationships, especially at lower ambient
32 PM_{10-2.5} concentrations.
- 33 ○ Identifying novel populations that could be at-risk of PM_{10-2.5}-related health
34 effects.
- 35 ○ Modeling to estimate PM_{10-2.5} mass and composition in areas with sparse or less-
36 than-daily monitoring.

1 **REFERENCES**

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

6 U.S. EPA (2011). Policy Assessment for the Review of the Particulate Matter National Ambient
7 Air Quality Standards. Office of Air Quality Planning and Standards, Health and
8 Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-
9 11-003. April 2011. Available at:
10 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100AUMY.txt>.

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

16 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
17 U.S. Environmental Protection Agency, Office of Research and Development, National
18 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
19 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-
20 standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).

21 U.S. EPA (2020). Policy Assessment for the Review of the National Ambient Air Quality
22 Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health
23 and Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-
24 452/R-20-002. January 2020. Available at: [https://www.epa.gov/sites/default/files/2020-
25 01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf).

26 U.S. EPA (2021). Supplement to the 2019 Integrated Science Assessment for Particulate Matter
27 (External Review Draft). U.S. Environmental Protection Agency, Office of Research and
28 Development, Center for Public Health and Environmental Assessment. Washington,
29 DC. U.S. EPA. EPA/600/R-21/198. December 2019. Available at:
30 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-
31 assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).

32

5 RECONSIDERATION OF THE SECONDARY STANDARDS FOR PM

This chapter presents and evaluates the policy implications of the scientific and technical information pertaining to this reconsideration of the 2020 final decision on the secondary PM standards. In so doing, the chapter presents key aspects of the evidence for the welfare effects of PM documented in the 2019 ISA and draft ISA Supplement, with support from the prior ISA and AQCDs, and associated public welfare implications, as well as key aspects of quantitative analyses of recent air quality that is presented in the appendix associated with this chapter. As described in detail in section 1.4.2, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. The selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for welfare effects, the focus within the draft ISA Supplement is on visibility effects. The draft ISA Supplement does not include an evaluation of studies on climate or materials effects. Together, the scientific evidence and quantitative information provides the foundation for our evaluation of the scientific information regarding welfare effects of PM in ambient air and the potential for welfare effects to occur under air quality conditions associated with the current standards, as well as the associated public welfare implications. Our evaluation is framed around key policy-relevant questions derived from the questions included in the IRP (U.S. EPA, 2016) for the review completed in 2020 and also takes into account the conclusions reached in the review. In this way we identify key policy-relevant considerations and summary conclusions regarding the public welfare protection provided by the current standards for the Administrator's consideration in this reconsideration of the 2020 final decision on the secondary PM standards.

Within this chapter, background information on the current standards, including key considerations in reaching the final decision in the 2020 review, is summarized in section 5.1. The general approach for considering the information in this reconsideration of the 2020 final decision, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 5.2. Key aspects of the welfare effects evidence, quantitative information, and associated public welfare implications and uncertainties are addressed in section 5.3. Section 5.3.1 presents our consideration of the available scientific evidence and quantitative information for visibility effects, while section 5.3.2 considers the scientific evidence for each of the non-

1 visibility welfare effects (climate effects and materials effects) separately.¹ Section 5.4
2 summarizes the key evidence- and quantitative-based considerations identified in our evaluation
3 and presents associated summary conclusions of this analysis. Key remaining uncertainties and
4 areas for future research are identified in section 5.5.

5 **5.1 BACKGROUND ON THE CURRENT STANDARDS**

6 The current secondary PM standards were affirmed in 2020 based on the scientific and
7 technical information available at that time, as well as the Administrator’s judgments regarding
8 the available welfare effects evidence, the appropriate degree of public welfare protection for the
9 existing standards, and available air quality information on visibility impairment that may be
10 allowed by such a standard (85 FR 82684, December 18, 2020). The welfare effects evidence
11 base available in the 2020 review included several decades of extensive research on the visibility
12 and non-visibility effects (climate effects, materials effects, and ecological effects) of PM,
13 conducted both in and outside of the U.S., that documents the impacts of PM (U.S. EPA, 2019;
14 U.S. EPA, 2009; U.S. EPA, 2004b; U.S. EPA, 2004a). With the 2020 decision, the EPA retained
15 the secondary 24-hour PM_{2.5} standard, with its level of 35 µg/m³, the annual PM_{2.5} standard, with
16 its level of 15.0 µg/m³, and the 24-hour PM₁₀ standard, with its level of 150 µg/m³. The sections
17 below focus on the key considerations, and the Administrator’s conclusions, for climate and
18 materials effects (section 5.1.1) and visibility effects (section 5.1.2) in the 2020 review.

19 **5.1.1 Non-Visibility Effects**

20 In light of the robust evidence base, the 2019 ISA concluded there to be causal
21 relationships between PM and climate effects and material effects (U.S. EPA, 2019, sections
22 13.3.9 and 13.4.2). For climate effects, the 2019 ISA concluded that aerosols² alter climate
23 processes directly through radiative forcing and by indirect effects on cloud brightness, changes

¹ Other welfare effects of PM, such as ecological effects, are being considered in the separate, on-going review of the secondary NAAQS for oxides of nitrogen, oxides of sulfur and PM. Accordingly, the public welfare protection provided by the secondary PM standards against ecological effects such as those related to deposition of nitrogen- and sulfur-containing compounds in vulnerable ecosystems is being considered in that separate review. Thus, the Administrator’s conclusion in this reconsideration of the 2020 final decision will be focused only and specifically on the adequacy of public welfare protection provided by the secondary PM standards from effects related to visibility, climate, and materials.

² In the climate sciences research community, PM is encompassed by what is typically referred to as aerosol. An aerosol is defined as a solid or liquid suspended in a gas, but PM refers to the solid or liquid phase of an aerosol. In this reconsideration of the 2020 final decision on the secondary PM NAAQS the discussion on climate effects of PM uses the term PM throughout for consistency with the 2019 ISA (U.S. EPA, 2019) as well as to emphasize that the climate processes altered by aerosols are generally altered by the PM portion of the aerosol. Exceptions to this practice include the discussion of climate effects in the 2012 review, when aerosol was used when discussing suspending aerosol particles, and for certain acronyms that are widely used by the climate community that include the term aerosol (e.g., aerosol optical depth, or AOD).

1 in precipitation, and possible changes in cloud lifetimes (U.S. EPA, 2019, section 13.3.9).
2 Additionally, the major aerosol components with the potential to affect climate processes (i.e.,
3 black carbon (BC), organic carbon (OC), sulfates, nitrates and mineral dusts) vary in their
4 reflectivity, forcing efficiencies, and direction of climate forcing (U.S. EPA, 2019, section
5 13.3.5). For materials effects, the 2019 ISA considered effects associated with the deposition of
6 PM (i.e., dry and wet deposition), including both physical damage (materials effects) and
7 aesthetic qualities (soiling effects). The deposition of PM can physically affect materials, adding
8 to the effects of natural weathering processes, by promoting or accelerating the corrosion of
9 metals; by degrading paints; and by deteriorating building materials such as stone, concrete, and
10 marble (U.S. EPA, 2019, section 13.4.2). Additionally, the deposition of PM from ambient air
11 can reduce the aesthetic appeal of buildings and objects through soiling.

12 The 2020 decision on the adequacy of the secondary standards for climate and materials
13 effects was a public welfare policy judgment made by the Administrator, which drew upon the
14 available scientific evidence for PM-attributable climate and materials effects and recognized
15 that the evidence did not support a quantitative assessment of exposures and public welfare risks
16 based on impacts to climate and materials. Noting the strong evidence indicating that aerosols
17 affect climate, the Administrator further considered what the available information indicated
18 regarding the adequacy of protection provided by the secondary PM standards. He noted that a
19 number of uncertainties in the scientific information affected our ability to quantitatively
20 evaluate the standards in this regard. For example, the 2019 ISA and 2020 PA noted the spatial
21 and temporal heterogeneity of PM components that contribute to climate forcing, uncertainties in
22 the measurement of aerosol components, inadequate consideration of aerosol impacts in climate
23 modeling, insufficient data on local and regional microclimate variations and heterogeneity of
24 cloud formations (U.S. EPA, 2019, section 13.3.9). In light of these uncertainties and the lack of
25 sufficient data, the 2020 PA concluded that “the data remain insufficient to conduct quantitative
26 analyses for PM effects on climate in the current review” (U.S. EPA, 2020, pp. 5-34 to 5-35) and
27 that there was insufficient information available to base a national ambient air quality standard
28 on climate impacts associated with ambient air concentrations of PM or its constituents (U.S.
29 EPA, 2020, section 5.4).

30 With regard to materials effects, the Administrator noted that the 2020 PA noted that
31 quantitative relationships were lacking between characteristics of PM and frequency of
32 repainting and repair of surfaces and that considerable uncertainty exists in the contributions of
33 co-occurring pollutants to materials damage and soiling processes (U.S. EPA, 2020, p. 5-35).
34 The 2020 PA concluded that none of the evidence available called into question the adequacy of
35 the existing secondary PM standards to protect against material effects (U.S. EPA, 2020, section
36 5.4).

1 The 2020 final decision was based on a thorough review in the 2019 ISA of the scientific
2 information on PM-induced climate and materials effects. The decision also took into account:
3 (1) assessments in the 2020 PA of the most policy-relevant information in the 2019 ISA
4 regarding evidence of adverse effects of PM to climate and materials, (2) uncertainties in the
5 available evidence to inform a quantitative assessment of PM-related climate and materials
6 effects, (3) CASAC advice and recommendations, and (4) public comments received during the
7 development of these documents and on the proposal notice.

8 Consistent with the general approach routinely employed in NAAQS reviews, the initial
9 consideration in the 2020 review of the secondary standards was with regard to the adequacy of
10 protection provided by the then-existing standards. Key aspects of that consideration are
11 summarized in section 5.1.1.1 below.

12 **5.1.1.1 Considerations Regarding Adequacy of the Existing Standards for Non-** 13 **Visibility Effects in the 2020 Review**

14 In considering non-visibility welfare effects in the 2020 review, as discussed above, the
15 Administrator concluded that, while it is important to maintain an appropriate degree of control
16 of fine and coarse particles to address non-visibility welfare effects, “it is generally appropriate
17 to retain the existing standards and that there is insufficient information to establish any distinct
18 secondary PM standards to address climate and materials effects of PM” (85 FR 82744,
19 December 18, 2020).

20 With regard to climate, the Administrator recognized that there were a number of
21 improvements and refinements to climate models since the 2012 review. However, while the
22 evidence continued to support a causal relationship between PM and climate effects, the
23 Administrator noted that significant limitations continued to exist related to quantifying the
24 contributions of direct and indirect effects of PM and PM components on climate forcing (U.S.
25 EPA, 2020, sections 5.2.2.1.1 and 5.4). He also recognized that the models continued to exhibit
26 considerable variability in estimates of PM-related climate impacts as regional scales (e.g., ~100
27 km) as compared to simulations at global scales. Therefore, the resulting uncertainty led the
28 Administrator to conclude that the available scientific information in the 2020 review remained
29 insufficient to quantify climate impacts associated with particular concentrations of PM in
30 ambient air (U.S. EPA, 2020, section 5.2.2.2.1) or to evaluate or consider a level of PM air
31 quality in the U.S. to protect against climate effects and that there was insufficient information
32 available to base a national ambient standard on climate impacts (85 FR 82744, December 18,
33 2020).

34 With regard to materials effects, the Administrator noted that the evidence available in
35 the 2019 ISA continued to support a causal relationship between materials effects and PM
36 deposition (U.S. EPA, 2019, section 13.4). He recognized that the deposition of fine and coarse

1 particles to materials can lead to physical damage and/or impaired aesthetic qualities. Particles
2 can contribute to materials damage by adding to the natural weathering processes and by
3 promoting the corrosion of metals, the degradation of building materials, and the weakening of
4 material components. While some new information was available in the 2019 ISA, the
5 information was from studies primarily conducted outside of the U.S. in areas where PM
6 concentrations in ambient air are typically higher than those observed in the U.S. (U.S. EPA,
7 2020, section 13.4). Additionally, the information assessed in the 2019 ISA did not support
8 quantitative analyses of PM-related materials effects in the 2020 review (U.S. EPA, section
9 5.2.2.2.2). Given the limited amount of information available and its inherent uncertainties and
10 limitations, the Administrator concluded that he was unable to relate soiling or damage to
11 specific levels of PM in ambient air or to evaluate or consider a level of air quality to protect
12 against such materials effects, and that there was insufficient information available to support a
13 distinct national ambient standard based on materials effects (85 FR 82744, December 18, 2020).

14 In the 2020 decision, for all of the reasons discussed above and recognizing the CASAC
15 conclusion that the evidence provided support for retaining the current secondary PM standards,
16 the Administrator concluded that it was appropriate to retain the existing secondary PM
17 standards, without revision. His decision was consistent with the CASAC advice related to non-
18 visibility effects. Specifically, the CASAC agreed with the 2020 PA conclusions that, while these
19 effects are important, “the available evidence does not call into question the protection afforded
20 by the current secondary PM standards” and recommended that the secondary standards “should
21 be retained” (Cox, 2019a, p. 3 of letter). For climate and materials effects, this conclusion
22 reflected his judgment that, although it remains important to maintain secondary PM_{2.5} and PM₁₀
23 standards to provide some degree of control over long- and short-term concentrations of both
24 fine and coarse particles, there was insufficient information to establish distinct secondary PM
25 standards to address non-visibility PM-related welfare effects (85 FR 82744, December 18,
26 2020). Thus, the Administrator concluded that it was appropriate to retain all aspects of the
27 existing 24-hour PM_{2.5}, annual PM_{2.5}, and 24-hour PM₁₀ secondary standards. With regard to the
28 secondary annual PM_{2.5} standard, the Administrator concluded that it was appropriate to retain a
29 level of 15.0 µg/m³ while revising only the form of the standard to remove the option for spatial
30 averaging (85 FR 82744, December 18, 2020).

31 **5.1.2 Visibility Effects**

32 Visibility refers to the visual quality of a human’s view with respect to color rendition
33 and contrast definition. It is the ability to perceive landscape form, colors, and textures. Visibility
34 involves optical and psychophysical properties involving human perception, judgment, and
35 interpretation. Light between the observer and the object can be scattered into or out of the sight

1 path and absorbed by PM or gases in the sight path. Given the strength of the evidence base, the
2 2019 ISA concluded that, “the evidence is sufficient to conclude that a causal relationship exists
3 between PM and visibility impairment” (U.S. EPA, 2019, section 13.2.6). Visibility impairment
4 is caused by light scattering and absorption by suspended particles and gases, including water
5 content of aerosols.³ The available evidence in the 2012 review indicated that specific
6 components of PM have been shown to contribute to visibility impairment. For example, at
7 sufficiently high relative humidity values, sulfate and nitrate are the PM components that scatter
8 more light and thus contribute most efficiently to visibility impairment. Elemental carbon (EC)
9 and OC are also important contributors, especially in the northwestern U.S. where their
10 contribution to PM_{2.5} mass is higher. Crustal materials can be significant contributors to visibility
11 impairment, particularly for remote areas in the arid southwestern U.S. (U.S. EPA, 2009, section
12 2.5.1; 2019 ISA, section 13.2.4.1).

13 Visibility impairment can have implications for people’s enjoyment of daily activities
14 and for their overall sense of well-being (U.S. EPA, 2009, section 9.2). Consistent with the
15 evidence available in the 2012, the 2019 ISA evaluated available visibility preference studies that
16 were part of the overall body of evidence, and these preference studies were considered in the
17 2020 PA (U.S. EPA, 2020, pp. 5-15 to 5-17). These preference studies provided information
18 about the potential public welfare implications of visibility impairment from surveys in which
19 participants were asked questions about their preferences or the values they placed on various
20 visibility conditions, as displayed to them in scenic photographs or in images with a range of
21 known light extinction levels.⁴

22 The 2020 decision on the adequacy of the secondary standards with regard to visibility
23 effects was a public welfare policy judgment made by the Administrator, which drew upon the
24 available scientific evidence for PM-related visibility effects and on analyses of visibility
25 impairment, as well as judgments about the appropriate weight to place on the range of
26 uncertainties inherent in the evidence and analyses. Consistent with the approach in the 2012
27 review, the analyses utilized a PM_{2.5} visibility index based on an algorithm, known as the

³ All particles scatter light and, although a larger particle scatters more light than a similarly shaped smaller particle of the same composition, the light scattered per unit of mass is greatest for particles with diameters from ~0.3-1.0 μm (U.S. EPA, 2009, section 2.5.1; 2019 ISA, section 13.2.1). Particles with hygroscopic components (e.g., particulate sulfate and nitrate) contribute more to light extinction at higher relative humidity than at lower relative humidity because they change size in the atmosphere in response to relative humidity.

⁴ Preference studies were available in four urban areas. Three western preference studies were available, including one in Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting, 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates, 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith and Howell, 2009). More details about these studies are available in Appendix D.

1 IMPROVE algorithm,⁵ that provides for the estimation of light extinction (b_{ext}), in units of Mm^{-1} ,
2 using routinely monitored components of fine ($PM_{2.5}$) and coarse ($PM_{10-2.5}$) PM. The quantitative
3 analyses focused on $PM_{2.5}$ based on conclusions in the 2019 ISA that fine particles scatter more
4 light than coarse particles on a per unit mass basis and include sulfates, nitrates, organics, light-
5 absorbing carbon, and soil (Malm et al., 1994). The 2019 ISA also concluded that hygroscopic
6 particles like ammonium sulfate, ammonium nitrate, and sea salt increase in size as relative
7 humidity increases, leading to increased light scattering (U.S. EPA, 2019, section 13.2.3).
8 Included in this decision were judgments on the weight to place on the visibility preference
9 studies; on the weight to give associated uncertainties, including those related to variability in
10 visibility preferences across the studies in different areas of the U.S.; variability in occurrence
11 of visibility impairment in areas of the U.S., especially in urban areas; and on the extent to which
12 such effects in such areas may be considered adverse to public welfare.

13 The 2020 final decision was based on a thorough review in the 2019 ISA of the scientific
14 information on PM-related visibility effects. The decision also took into account: (1) assessments
15 in the 2020 PA of the most policy-relevant information in the 2019 ISA regarding evidence of
16 adverse effects of PM on visibility; (2) air quality analyses of the $PM_{2.5}$ visibility index and
17 design values based on the form and averaging time of the existing standard; (3) CASAC advice
18 and recommendations; and (4) public comments received during the development of these
19 documents and on the 2020 proposal notice.

20 Consistent with the general approach routinely employed in NAAQS reviews, the initial
21 consideration in the 2020 review of the secondary PM standards was with regard to the adequacy
22 of the protection provided by the then-existing standards. Key aspects of that consideration are
23 summarized in section 5.1.2.1 below.

24 **5.1.2.1 Consideration Regarding the Adequacy of the Existing Standards for** 25 **Visibility Effects in the 2020 Review**

26 In considering the visibility effects in the 2020 review, the Administrator noted the long-
27 standing body of evidence for PM-related visibility impairment. This evidence, which is based
28 on the fundamental relationship between light extinction and PM mass, demonstrated that
29 ambient PM can impair visibility in both urban and remote areas, and had changed very little
30 since the 2012 review (U.S. EPA, 2019, section 13.1; U.S. EPA, 2009a, section 9.2.5). The
31 evidence related to public perception of visibility impairment was from studies from four areas in
32 North America. These studies provided information to inform our understanding of levels of

⁵ The algorithm is referred to as the IMPROVE algorithm as it was developed specifically to use monitoring data generated at IMPROVE network sites and with equipment specifically designed 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).

1 visibility impairment that the public judged to be “acceptable” (U.S. EPA, 2010b; 85 FR 24131,
2 April 30, 2020). In considering these public preference studies, the Administrator noted that, as
3 described in the 2019 ISA, no new visibility studies had been conducted in the U.S. and there
4 was little newly available information with regard to acceptable levels of visibility impairment in
5 the U.S. The Administrator recognized that visibility impairment can have implications for
6 people’s enjoyment of daily activities and their overall well-being, and therefore, considered the
7 degree to which the current secondary standards protect against PM-related visibility
8 impairment.

9 Consistent with the 2012 review, in the 2020 review, the Administrator first concluded
10 that a target level of protection for a secondary PM standard is most appropriately defined in
11 terms of a visibility index that directly takes into account the factors (i.e., species composition
12 and relative humidity) that influence the relationship between PM_{2.5} in ambient air and PM-
13 related visibility impairment. In defining a target level of protection, the Administrator
14 considered the specific aspects of such an index, including the appropriate indicator, averaging
15 time, form and level (78 FR 82742-82744, December 18, 2020).

16 First, with regard to indicator, the Administrator noted that in the 2012 review, the EPA
17 used an index based on estimates of light extinction by PM_{2.5} components calculated using an
18 adjusted version of the IMPROVE algorithm, which allows the estimation of light extinction
19 using routinely monitored components of PM_{2.5} and PM_{10-2.5}, along with estimates of relative
20 humidity. The Administrator recognized that, while there have been some revisions to the
21 IMPROVE algorithm since the time of the 2012 review, our fundamental understanding of the
22 relationship between PM in ambient air and light extinction had changed little and the various
23 IMPROVE algorithms appropriately reflected this relationship across the U.S. In the absence of
24 a monitoring network for direct measurement of light extinction, he concluded that calculated
25 light extinction indicator that utilizes the IMPROVE algorithms continued to provide a
26 reasonable basis for defining a target level of protection against PM-related visibility impairment
27 (78 FR 82742-82744, December 18, 2020).

28 In further defining the characteristics of a visibility index, the Administrator next
29 considered the appropriate averaging time, form, and level of the index. Given the available
30 scientific information in the review, and in considering the CASAC’s advice and public
31 comments, the Administrator concluded that, consistent with the decision in the 2012 review, a
32 visibility index with a 24-hour averaging time and a form based on the 3-year average of annual
33 90th percentile values remained reasonable. With regard to the averaging time and form of such
34 an index, the Administrator noted analyses conducted in the last review that demonstrated
35 relatively strong correlations between 24-hour and subdaily (i.e., 4-hour average) PM_{2.5} light
36 extinction (78 FR 3226, January 15, 2013), indicating that a 24-hour averaging time is an

1 appropriate surrogate for the sub-daily time periods of the perception of PM-related visibility
2 impairment and the relevant exposure periods for segments of the viewing public. This decision
3 in the 2020 review also recognized that a 24-hour averaging time may be less influenced by
4 atypical conditions and/or atypical instrument performance (78 FR 3226, January 15, 2013). The
5 Administrator recognized that there was no new information to support updated analyses of this
6 nature, and therefore, he believed these analyses continued to provide support for consideration
7 of a 24-hour averaging time for a visibility index in this review. With regard to the statistical
8 form of the index, the Administrator noted that, consistent with the 2012 review: (1) A multi-
9 year percentile form offers greater stability from the occasional effect of interannual
10 meteorological variability (78 FR 3198, January 15, 2013; U.S. EPA, 2011, p. 4–58); (2) a 90th
11 percentile represents the median of the distribution of the 20 percent worst visibility days, which
12 are targeted in Federal Class I areas by the Regional Haze Program; and (3) public preference
13 studies did not provide information to identify a different target than that identified for Federal
14 Class I areas (U.S. EPA, 2011, p. 4–59). Therefore, the Administrator judged that a visibility
15 index based on estimates of light extinction, with a 24-hour averaging time and a 90th percentile
16 form, averaged over three years, remained appropriate (78 FR 82742-82744, December 18,
17 2020).

18 With regard to the level of a visibility index, consistent with the 2012 review, the
19 Administrator judged that it was appropriate to establish a target level of protection of 30
20 deciviews (dv),^{6 7} reflecting the upper end of the range of visibility impairment judged to be
21 acceptable by at least 50% of study participants in the available public preference studies (78 FR
22 3226, January 15, 2013). As described above, the 2011 PA identified a range of levels from 20 to
23 30 dv based on the responses in the public preference studies available at that time. At the time
24 of the 2012 review, the Administrator noted a number of uncertainties and limitations in public
25 preference studies, including the small number of stated preference studies available, the
26 relatively small number of study participants, the extent to which the study participants may not
27 be representative of the broader study area population in some of the studies, and the variations
28 in the specific materials and methods used in each study. In considering the available preference
29 studies, with their inherent uncertainties and limitations, the prior Administrator concluded that
30 the substantial degree of variability and uncertainty in the public preference studies should be
31 reflected in a target level of protection based on the upper end of the range of candidate
32 protection levels (CPLs).

⁶ Deciview (dv) refers to a scale for characterizing visibility that is defined directly in terms of light extinction. The deciview scale is frequently used in the scientific and regulatory literature on visibility.

⁷ For comparison, 20 dv, 25 dv, and 30 dv are equivalent to 64, 112, and 191 megameters (Mm⁻¹), respectively.

1 Given that there were no new preference studies available in 2020 review, the
2 Administrator’s judgments were based on the same studies, with the same range of levels,
3 available in the 2012 review. As identified in the 2020 PA (U.S. EPA, 2020, section 5.5), there
4 were a number of limitations and uncertainties associated with these studies, including the
5 following:

- 6 • Available studies may not represent the full range of preferences for visibility in the U.S.
7 population, particularly given the potential variability in preferences based on the
8 conditions commonly encountered and the scenes being viewed.
- 9 • Available preference studies were conducted 15 to 30 years ago and may not accurately
10 represent the current day preferences of people in the U.S.
- 11 • The variety of methods used in the preference studies may potentially influence the
12 responses as to what level of impairment is deemed acceptable.
- 13 • Factors that are not captured in the methods of the preference studies, such as the time of
14 day when light extinction is the greatest or the frequency of impairment episodes, may
15 influence people’s judgment on acceptable visibility (U.S. EPA, 2020, section 5.2.1.1).

16 Therefore, in considering the scientific information, with its uncertainties and limitations,
17 as well as public comments on the level of the target level of protection against visibility
18 impairment, the Administrator concluded that it is appropriate to again use a level of 30 dv for
19 the visibility index (78 FR 82742-82744, December 18, 2020).

20 Having concluded that the protection provided by a standard defined in terms of a PM_{2.5}
21 visibility index, with a 24-hour averaging time, and a 90th percentile form, averaged over 3 years,
22 set at a level of 30 dv, was requisite to protect public welfare with regard to visual air quality, the
23 Administrator next considered the degree of protection from visibility impairment afforded by
24 the existing suite of secondary PM standards.

25 In this context, the Administrator considered the updated analyses of visibility
26 impairment presented in the 2020 PA (U.S. EPA, 2020, section 5.2.1.2), which reflected a
27 number of improvements since the 2012 review. Specifically, the updated analyses examined
28 multiple versions of the IMPROVE equation, including the version incorporating revisions since
29 the time of the 2012 review. These updated analyses provided a further understanding of how
30 variation in the inputs to the algorithms affect the estimates of light extinction (U.S. EPA, 2020,
31 Appendix D). Additionally, for a subset of monitoring sites with available PM_{10-2.5} data, the
32 updated analyses better characterized the influence of coarse PM on light extinction than in the
33 2012 review (U.S. EPA, 2020, section 5.2.1.2).

34 The results of the updated analyses in the 2020 PA were consistent with those from the
35 2012 review. Regardless of which version of the IMPROVE equation was used, the analyses
36 demonstrated that, based on 2015–2017 data, the 3-year visibility metric was at or below about
37 30 dv in all areas meeting the current 24-hour PM_{2.5} standard, and below 25 dv in most of those

1 areas. In locations with available PM_{10-2.5} monitoring, which met both the current 24-hour
2 secondary PM_{2.5} and PM₁₀ standards, 3-year visibility index metrics were at or below 30 dv
3 regardless of whether the coarse fraction was included as an input to the algorithm for estimating
4 light extinction (U.S. EPA, 2020, section 5.2.1.2). While the inclusion of the coarse fraction had
5 a relatively modest impact on the estimates of light extinction, the Administrator recognized the
6 continued importance of the PM₁₀ standard given the potential for larger impacts on light
7 extinction in areas with higher coarse particle concentrations, which were not included in the
8 analyses in the 2020 PA due to a lack of available data (U.S. EPA, 2019, section 13.2.4.1; U.S.
9 EPA, 2020, section 5.2.1.2). He noted that the air quality analyses showed that all areas meeting
10 the existing 24-hour PM_{2.5} standard, with its level of 35 µg/m³, had visual air quality at least as
11 good as 30 dv, based on the visibility index. Thus, the secondary 24-hour PM_{2.5} standard would
12 likely be controlling relative to a 24-hour visibility index set at a level of 30 dv. Additionally,
13 areas would be unlikely to exceed the target level of protection for visibility of 30 dv without
14 also exceeding the existing secondary 24-hour standard. Thus, the Administrator judged that the
15 24-hour PM_{2.5} standard provided sufficient protection in all areas against the effects of visibility
16 impairment, i.e., that the existing 24-hour PM_{2.5} standard would provide at least the target level
17 of protection for visual air quality of 30 dv which he judged appropriate (78 FR 82742-82744,
18 December 18, 2020).

19 **5.2 GENERAL APPROACH AND KEY ISSUES IN THIS** 20 **RECONSIDERATION OF THE 2020 FINAL DECISION**

21 This reconsideration of the 2020 final decision on the secondary PM standards is most
22 fundamentally based on using the Agency’s assessment of the scientific evidence and associated
23 quantitative analyses to inform the Administrator’s judgments regarding secondary standards that
24 are requisite to protect public welfare from known or anticipated adverse effects. This draft PA is
25 intended to help bridge the gap between the scientific evidence and information assessed in the
26 2019 ISA and draft ISA Supplement and the judgments required of the Administrator in
27 determining whether it is appropriate to retain or revised the secondary PM NAAQS. The
28 approach planned for this reconsideration of the 2020 final decision on the secondary PM
29 standards will build on previous reviews, including the substantial assessments and evaluations
30 performed in those reviews, and taking into account scientific information and air quality data to
31 inform our understanding of the key policy-relevant issues in this reconsideration.

1 The evaluations in this draft PA, of the scientific assessments in the 2019 ISA and draft
2 ISA Supplement⁸ augmented by quantitative air quality analyses, are intended to inform the
3 Administrator’s public welfare policy judgments and conclusions, including his decisions as to
4 whether to retain or revise these standards. The draft PA considers the potential implications of
5 various aspects of the scientific evidence, the air quality information, and the associated
6 uncertainties and limitations. In so doing, the approach for this draft PA involves evaluating the
7 scientific and technical information to address a series of key policy-relevant questions using
8 both evidence- and quantitative-based considerations. Together, consideration of the full set of
9 evidence and information in this reconsideration will inform the answer to the following initial
10 overarching question for the reconsideration:

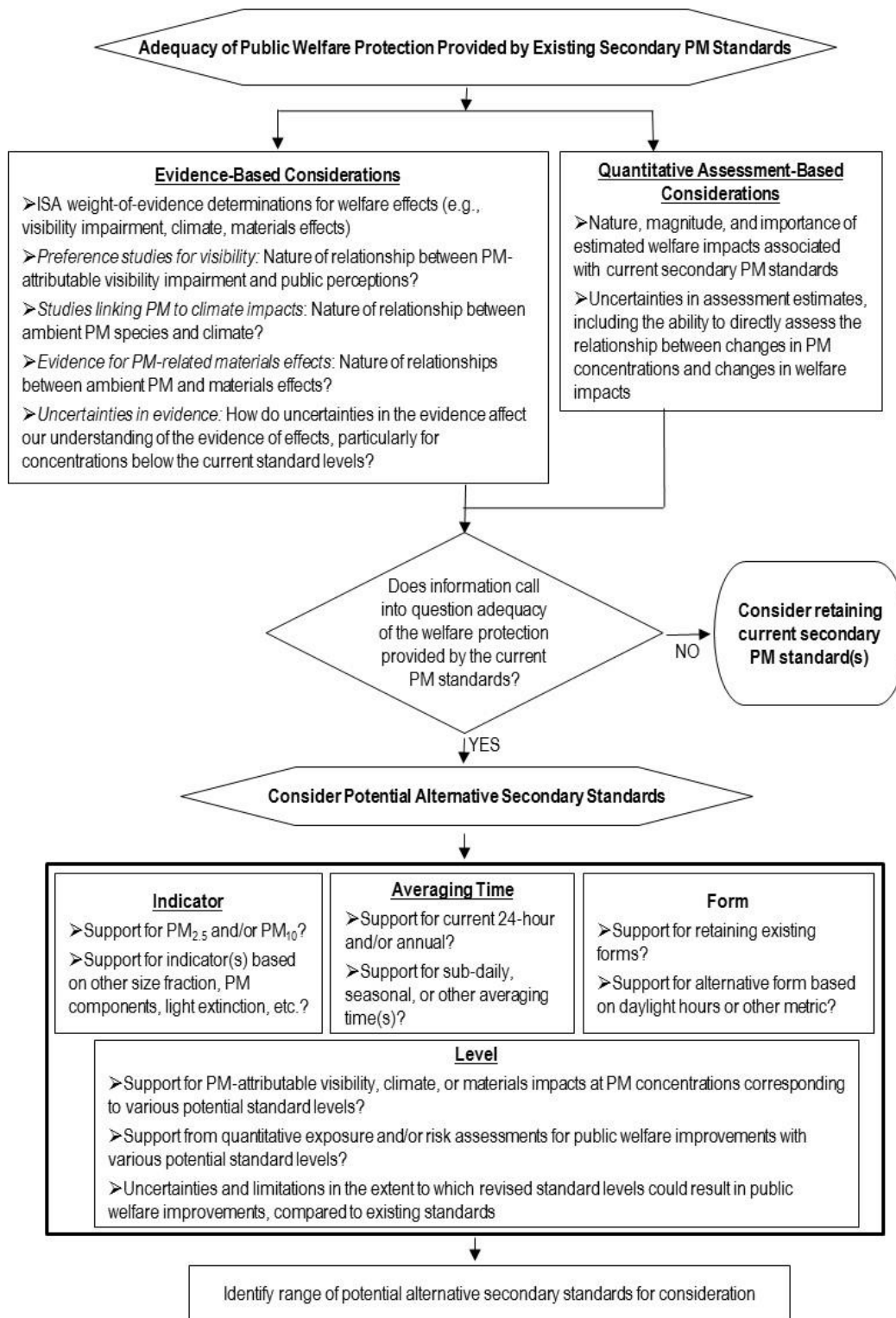
- 11 • **Do the scientific evidence and quantitative information support or call into question**
12 **the adequacy of the protection afforded by the current secondary PM standards?**

13 In reflecting on this question in the remaining sections of this chapter, we consider the
14 body of scientific evidence assessed in the 2019 ISA and draft ISA Supplement and considered
15 as basis for developing or interpreting air quality analyses, including whether it supports or calls
16 into question the scientific conclusions reached in the 2020 review regarding welfare effects
17 related to exposure to PM in ambient air. Information in this reconsideration of the 2020 final
18 decision that may be informative to public policy judgments on the significance or adversity of
19 key effects on the public welfare is also considered. Additionally, the quantitative information,
20 whether newly developed in this reconsideration or predominantly developed in the past and
21 interpreted in light of current information, is considered, including with regard to the extent to
22 which it may continue to support judgments made in previous reviews.

23 The approach to reaching conclusions on the current secondary PM standards and, as
24 appropriate, on potential alternative standards, including consideration of policy-relevant
25 questions that frame the current reconsideration, is illustrated in Figure 5-1.

26

⁸ As noted above and described in detail in section 1.4.2, the draft ISA Supplement focuses on a thorough evaluation of some studies that became available after the literature cutoff date of the 2019 ISA that could either further inform the adequacy of the current PM NAAQS or address key scientific topics that have evolved since the literature cutoff date for the 2019 ISA. The selection of the welfare effects to evaluate within the draft ISA Supplement were based on the causality determinations reported in the 2019 ISA and the subsequent use of scientific evidence in the 2020 PA. Specifically, for welfare effects, the focus within the draft ISA Supplement is on visibility effects. The draft ISA Supplement does not include an evaluation of studies on climate or materials effects.



1
2
3
4

Figure 5-1. Overview of general approach for the reconsideration of the 2020 final decision on the secondary PM standards.

1 The Agency’s approach in its reconsideration of the 2020 final decision on the secondary
2 standards is consistent with the requirements of the provisions of the CAA related to the review
3 of NAAQS and with how the EPA and the courts have historically interpreted the CAA. As
4 discussed in section 2.1 above, these provisions require the Administrator to establish secondary
5 standards that, in the Administrator’s judgment, are requisite (i.e., neither more nor less stringent
6 than necessary) to protect the public welfare from known or anticipated adverse effects
7 associated with the presence of the pollutant in ambient air. In so doing, the Administrator
8 considers advice from the CASAC and public comment.

9 Consistent with the Agency’s approach across all NAAQS reviews, the approach of this
10 draft PA to informing the Administrator’s judgments in this reconsideration of the 2020 final
11 decision on the secondary PM standards is based on a recognition that the evidence generally
12 reflects continuums that include ambient air exposures for which scientists generally agree that
13 effects are likely to occur through lower levels at which the likelihood and magnitude of
14 response become increasingly uncertain. The CAA does not require that standards be set at a
15 zero-risk level, but rather at a level that reduces risk sufficiently so as to protect the public
16 welfare from known or anticipated adverse effects. The Agency’s decisions on the adequacy of
17 the current secondary standards and, as appropriate, on any potential alternative standards
18 considered in a review, are largely public welfare policy judgments made by the Administrator.
19 The four basic elements of the NAAQS (i.e., indicator, averaging time, form, and level) are
20 considered collectively in evaluating the protection afforded by the current standard, or any
21 alternative standards considered. Thus, the Administrator’s final decisions in such reviews draw
22 upon the scientific information and analyses about welfare effects, environmental exposures and
23 risks, and associated welfare significance, as well as judgments about how to consider the range
24 and magnitude of uncertainties that are inherent in the scientific evidence and analyses.

25 **5.3 WELFARE EFFECTS AND QUANTITATIVE INFORMATION**

26 In considering the evidence for welfare effects attributable to PM presented in the 2019
27 ISA and the draft ISA Supplement, this section poses the following policy-relevant questions:

- 28 • **Does the scientific evidence and quantitative information support or call into**
29 **question the adequacy of the welfare protection afforded by the current secondary**
30 **PM standards?**

31 In answering this question, we have posed a series of more specific questions to aid in
32 considering the scientific evidence and quantitative information, as discussed below. In
33 considering the scientific and technical information, we reflect upon both the information in
34 previous reviews and information that is assessed and presented in the 2019 ISA (U.S. EPA,
35 2019) and in the draft ISA Supplement (U.S. EPA, 2021), focusing on welfare effects for which

1 the evidence supports either a “causal” or a “likely to be causal” relationship as described in the
2 Preamble to the ISA (U.S. EPA, 2015). Table 5-1 lists such causality determinations from the
3 2019 ISA for welfare effects. As in previous reviews, the evidence is sufficient to support a
4 causal relationship between PM and visibility effects (section 5.3.1), climate effects (section
5 5.3.2) and materials effects (section 5.3.2).

6 While the 2019 ISA provides the broad scientific foundation for this reconsideration, we
7 recognized that additional literature has become available since the cutoff date of the 2019 ISA
8 that expands the body of evidence related to visibility effects that can inform the Administrator’s
9 judgments on the adequacy of the current secondary PM standards. As such, the draft ISA
10 Supplement builds on the information in the 2019 ISA with a target identification and evaluation
11 of new scientific information regarding visibility effects (U.S. EPA, 2021, section 1.2). As
12 described in chapter 1, the selection of the welfare effects to evaluate within the draft ISA
13 Supplement were based on the causality determinations reported in the 2019 ISA and the
14 subsequent use of scientific evidence in the 2020 PA. The draft ISA Supplement focuses on U.S.
15 and Canadian studies that provide new information on public preference for visibility impairment
16 and/or developed new methodologies or conducted quantitative analyses of light extinction (U.S.
17 EPA, 2021, section 1.2). Such studies of visibility effects and quantitative relationships between
18 visibility impairment and PM in ambient air were considered to be of greatest utility in informing
19 the Administrator’s conclusions on the adequacy of the current secondary PM standards. The
20 visibility effects evidence presented within the 2019 ISA, along with the targeted identification
21 and evaluation of new scientific information in the draft ISA Supplement, provides the scientific
22 basis for the reconsideration of the 2020 final decision on the primary PM_{2.5} standards. For
23 climate and materials effects, the 2020 PA concluded that there were substantial uncertainties
24 associated with the quantitative relationships with PM concentrations and the concentration
25 patterns that limited the ability quantitatively assess the public welfare protection provided by
26 the standards from these effects. Therefore, for climate and materials effects, we draw heavily
27 from the 2020 PA in our evaluation of the information related to these effects and in reaching
28 preliminary conclusions in this draft PA.

29 **Table 5-1. Key causality determinations for PM-related welfare effects.**

Effect	2009 PM ISA	2019 PM ISA
Visibility effects	Causal	Causal
Climate effects	Causal	Causal
Materials effects	Causal	Causal

30

1 **5.3.1 Visibility Effects**

2 In the sections below, we consider the nature of visibility-related effects attributable to
3 PM (section 5.3.1.1) and the quantitative information (section 5.3.1.2).

4 **5.3.1.1 Nature of Effects**

5 In considering the evidence of visibility welfare effects attributable to PM as presented in
6 the 2019 ISA and the draft ISA Supplement, this section addresses the following policy-relevant
7 question:

- 8 • **Does the available scientific evidence alter our conclusions from the 2020 review**
9 **regarding the nature of visibility effects attributable to PM in ambient air?**

10 Visibility refers to the visual quality of a human’s view with respect to color rendition
11 and contrast definition. It is the ability to perceive landscape form, colors, and textures. Visibility
12 involves optical and psychophysical properties involving human perception, judgment, and
13 interpretation. Light between the observer and the object can be scattered into or out of the sight
14 path and absorbed by PM or gases in the sight path. As recognized above, the conclusion of the
15 2019 ISA that “the evidence is sufficient to conclude that a causal relationship exists between
16 PM and visibility impairment” is consistent with conclusions of causality in the 2012 review
17 (U.S. EPA, 2019, section 13.2.6). These conclusions are based on strong and consistent evidence
18 that ambient PM can impair visibility in both urban and remote areas (U.S. EPA, 2009, section
19 9.2.5).

20 These subsequent questions consider the characterization and quantification of light
21 extinction and preferences associated with varying degrees of visibility impairment.

- 22 • **To what extent is information available that changes or enhances our understanding**
23 **of the physics of light extinction and/or its quantification (e.g., through light**
24 **extinction or other monitoring methods or through algorithms such as IMPROVE)?**

25 Our understanding of the relationship between light extinction and PM mass has changed
26 little since the 2009 ISA (U.S. EPA, 2009). The combined effect of light scattering and
27 absorption by particles and gases is characterized as light extinction, i.e., the fraction of light that
28 is scattered or absorbed per unit of distance in the atmosphere. Light extinction is measured in
29 units of 1/distance, which is often expressed in the technical literature as visibility per
30 megameter (abbreviated Mm^{-1}). Higher values of light extinction (usually given in terms of Mm^{-1}
31 or dv) correspond to lower visibility. When PM is present in the air, its contribution to light
32 extinction is typically much greater than that of gases (U.S. EPA, 2019, section 13.2.1). The
33 impact of PM on light scattering depends on particle size and composition, as well as relative
34 humidity. All particles scatter light, as described by the Mie theory, which relates light scattering
35 to particle size, shape and index of refraction (U.S. EPA, 2019, section 13.2.3; Van de Hulst,

1 1981; Mie, 1908). Fine particles scatter more light than coarse particles on a per unit mass basis
2 and include sulfates, nitrates, organics, light-absorbing carbon, and soil (Malm et al., 1994).
3 Hygroscopic particles like ammonium sulfate, ammonium nitrate, and sea salt increase in size as
4 relative humidity increases, leading to increased light scattering (U.S. EPA, 2019, section
5 13.2.3).

6 Direct measurements of PM light extinction, scattering, and absorption are considered
7 more accurate for quantifying visibility impairment than PM mass-based estimates because they
8 do not depend on assumptions about particle characteristics (e.g., size, shape, density, component
9 mixture, etc.). Measurements of light extinction can be made with high time resolution, allowing
10 for characterization of subdaily temporal patterns of visibility impairment. Measurement
11 methods include transmissometers for measurement of light extinction and the determination of
12 visual range and integrating nephelometers for measurement of light scattering, as well as
13 teleradiometers and telephotometers, and photography and photographic modeling (U.S. EPA,
14 2009; U.S. EPA, 2004b). While some recent research confirms and adds to the body of
15 knowledge regarding direct measurements as is described in the 2019 ISA and draft ISA
16 Supplement, no major new developments have been made with these measurement methods
17 since prior reviews (U.S. EPA, 2019, section 13.2.2.2; U.S. EPA, 2021, section 4.2).

18 A theoretical relationship between light extinction and PM characteristics has been
19 derived from Mie theory (U.S. EPA, 2019, Equation 13-5) and can be used to estimate light
20 extinction by combining mass scattering efficiencies of particles with particle concentrations
21 (U.S. EPA, 2019, section 13.2.3; U.S. EPA, 2009, sections 9.2.2.2 and 9.2.3.1). However,
22 routine ambient air monitoring rarely includes measurements of particle size and composition
23 information with sufficient detail for these calculations. Accordingly, a much simpler algorithm
24 has been developed to make estimating light extinction more practical.

25 The algorithm, known as the IMPROVE algorithm,⁹ estimates light extinction (b_{ext} ,
26 measured in units of Mm^{-1}), using routinely monitored components of fine ($PM_{2.5}$) and coarse
27 ($PM_{10-2.5}$) PM. Relative humidity data are also needed to estimate the contribution by liquid
28 water that is in solution with the hygroscopic components of PM. To estimate each component's
29 contribution to light extinction, their concentrations are multiplied by extinction coefficients and
30 are additionally multiplied by a water growth factor that accounts for their expansion with
31 moisture. Both the extinction efficiency coefficients and water growth factors of the IMPROVE
32 algorithm have been developed by a combination of empirical assessment and theoretical

⁹ The algorithm is referred to as the IMPROVE algorithm as it was developed specifically to use monitoring data generated at IMPROVE network sites and with equipment specifically designed 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).

1 calculation using particle size distributions associated with each of the major aerosol components
2 (U.S. EPA, 2019, section 13.2.3.1, section 13.2.3.3).

3 The *original IMPROVE algorithm* (Equation D-1 in Appendix D), so referenced here to
4 distinguish it from subsequent variations developed later, was found to underestimate the highest
5 light scattering values and overestimate the lowest values at IMPROVE monitors throughout the
6 U.S. (Malm and Hand, 2007; Ryan et al., 2005; Lowenthal and Kumar, 2004) and at sites in
7 China (U.S. EPA, 2019, section 13.2.3.3). To resolve these biases, a *revised IMPROVE equation*,
8 shown in Equation D-2 in Appendix D, was developed (Pitchford et al., 2007) that divides PM
9 components into smaller and larger sizes of particles in PM_{2.5}, with separate mass scattering
10 efficiencies and hygroscopic growth functions for each size category. The revised IMPROVE
11 equation was described in detail in the 2009 ISA (U.S. EPA, 2009) and at that time, it both
12 reduced bias at the lowest and highest scattering values and improved the accuracy of the
13 calculated light b_{ext} . However, poorer precision was observed with the revised IMPROVE
14 equation compared to the original IMPROVE equation (U.S. EPA, 2009).¹⁰ Recent research
15 suggests that changes in PM composition in ambient air can impact the accuracy of estimating
16 light extinction using the IMPROVE algorithms (U.S. EPA, 2021, section 4.2.2). As an example,
17 a study by Prenni et al. (2019) found that the relationship between directly measured light
18 scattering and estimated light scattering using the revised IMPROVE equation has changed over
19 time in recent years. In particular, Prenni et al. (2019) compared estimated light extinction using
20 the revised IMPROVE equation with measured light extinction using nephelometers from 2001-
21 2016 and found that the revised IMPROVE equation underestimated light extinction at many
22 sites, especially for locations that experienced large decreases in sulfate and organic mass
23 concentrations. They further found that the underestimation results from splitting the components
24 into smaller and larger sizes of particles, with too much of the mass being allocated to the
25 smaller size fraction which has a lower dry mass scattering efficiency (U.S. EPA, 2021, section
26 4.2.2; Prenni et al., 2019).

27 Since the 2012 review, Lowenthal and Kumar (2016) have tested and evaluated a number
28 of modifications to the revised IMPROVE equation based on evaluations of monitoring data
29 from remote IMPROVE sites. In these locations, they observed that the multiplier to estimate the
30 concentration of organic matter, [OM], from the concentration of organic carbon, [OC], was

¹⁰ In the most recent IMPROVE report, a combination of the original and revised IMPROVE equations (the *modified original IMPROVE equation*) was used (Hand et al., 2011). This equation uses the sea salt term of the revised equation but does not subdivide the components into two size classes. Further, it uses a factor of 1.8 to estimate organic matter from organic carbon concentrations and also replaces the constant value of 10 Mm⁻¹ used for Rayleigh scattering in the original and revised equations with a site-specific term based on elevation and mean temperature.

1 closer to 2.1 than the value of 1.8 used in the revised IMPROVE equation.¹¹ They also observed
2 that water soluble organic matter absorbs water as a function of relative humidity, which is not
3 accounted for in either the original or revised IMPROVE equations and was therefore
4 underestimated in these equations. They further suggested that light scattering by sulfate was
5 overestimated because the assumption that all sulfate is fully neutralized ammonium sulfate is
6 not always true (U.S. EPA, 2019, section 13.2.3.3). Modifications based on these points are
7 reflected in Equation D-3 in Appendix D.

8 In summary, rather than altering our understanding from previous reviews, we continue
9 to recognize that direct measurements are better at characterizing light extinction than estimating
10 light extinction with an algorithm. However, in the absence of advances in the monitoring
11 methods and/or network for directly measuring light extinction, the use of the IMPROVE
12 equation for estimating light extinction continues to be supported by the evidence, with some
13 refinements to the inputs of the IMPROVE equation. Accordingly, as in previous reviews, this
14 reconsideration focuses on calculated light extinction when quantifying visibility impairment
15 resulting from recent concentrations of PM in ambient air.

- 16 • **What does the information indicate with regard to factors that influence light**
17 **extinction and visibility, as well as variation in these factors and resulting light**
18 **extinction across the U.S.?**

19 The 2019 ISA provides a comprehensive discussion of the spatial and temporal patterns
20 of PM_{2.5} composition and its contribution to light extinction from IMPROVE and CSN
21 monitoring sites, which are mostly rural and urban, respectively.¹² The data from these sites for
22 the periods of 2005-2008 and 2011-2014 were used in the 2019 ISA to identify differences in
23 species contributing to light extinction in urban and rural areas by region and season. This is an
24 expansion over the analysis in the 2009 ISA, in that the measurements at that time were
25 primarily based measurements from monitors located in rural areas and at remote sites (U.S.
26 EPA, 2019, section 13.2.4.1, Figures 13-1 through 13-14).

27 Focusing on the more recent time period of 2011-2014, some major differences in
28 estimated light extinction are apparent among regions of the U.S. Annual average calculated b_{ext}
29 was considerably greater in the East and Midwest than in the Southwest. Based on IMPROVE

¹¹ In areas near sources, PM is often less oxygenated, and therefore, in these locations, much of the organic PM mass is present as OC (Jimenez et al., 2009). In areas further away from PM sources, organic PM mass is often more oxygenated as a result of photochemical activity and interactions with other PM and gaseous components in the atmosphere (Jimenez et al., 2009). Under these conditions, the multiplier to convert OC to OM may be higher than in locations with less aged organic PM.

¹² Monitors were grouped into 28 IMPROVE regions and 31 CSN regions based on site location and PM concentrations for major species. For comparison purposes, and where possible, CSN regions were defined similarly to those for the IMPROVE network (Hand et al., 2011; U.S. EPA, 2019, section 13.2.4.1).

1 data, annual average b_{ext} was greater than 40 Mm^{-1} in the Southeast, East Coast, Mid-South,
2 Central Great Plains, and Appalachian regions, with the highest annual average b_{ext} (greater than
3 50 Mm^{-1}) in the Ohio River Valley,¹³ while annual average b_{ext} was below 40 Mm^{-1} for all
4 Western IMPROVE regions. Annual average b_{ext} values were also generally higher in the East
5 than the West based on CSN data, although the highest annual average b_{ext} was in the
6 Sacramento/San Joaquin Valley and Los Angeles areas (U.S. EPA, 2019, section 13.2.4.1, Figure
7 13-1, Figure 13-3, Figure 13-5).

8 Consistent with the analysis in the 2019 ISA, a recent study analyzed national and
9 regional trends in light extinction based on reconstructed total light extinction estimated from
10 IMPROVE data using 5-year aggregates of annual mean b_{ext} (Mm^{-1}) for 2000-2004 and 2014-
11 2018 (U.S. EPA, 2021, section 4.2.2). Hand et al. (2020) found that, for 2000-2004, the highest
12 levels of b_{ext} occurred in the Appalachian Mountains and Ohio River valley ($\sim 100 \text{ Mm}^{-1}$ or
13 greater), with decreasing values in the central U.S. ($\sim 70 \text{ Mm}^{-1}$). Values of b_{ext} in the East
14 significantly decreased over time, reduced to $\sim 50 \text{ Mm}^{-1}$ in the 2014-2018 time period, likely
15 corresponding to decreases in sulfate concentrations over time. However, for 2014-2018, the
16 highest values of b_{ext} were in the central U.S. ($50\text{-}60 \text{ Mm}^{-1}$), which is an area with high
17 agricultural activity and nitrate and ammonium concentrations. During both time periods, lower
18 b_{ext} occurred in the western U.S. ($20\text{-}30 \text{ Mm}^{-1}$), with improvements in b_{ext} closer to the West
19 Coast in 2014-2018 compared to 2004-2008.

20 Moreover, Hand et al. (2020) also explored changes in b_{ext} over time as relative trends ($\%$
21 yr^{-1}) and found spatial variability in long-term and short-term trends. Generally, similar
22 magnitudes and spatial variability were found for both long-term and short-term trends, with the
23 strongest reductions in b_{ext} across the eastern U.S. ($-4\% \text{ yr}^{-1}$ or greater) and along the West Coast,
24 particularly in Southern California. There was less improvement in the Intermountain West¹⁴ ($-$
25 $2\% \text{ yr}^{-1}$), although air quality in these areas have been increasingly impacted by wildfire activity
26 and biomass smoke in recent years (Hand et al., 2020). Decreased trends also occurred across the
27 Southwest, but at a lower rate than in the Eastern U.S. Over the entire continental U.S., on
28 average, b_{ext} decreased at a rate of $-2.8\% \text{ yr}^{-1}$ from 2002 to 2018 and $-18\% \text{ yr}^{-1}$ from 1992 to
29 2018, with much of the improvement occurring in the eastern U.S. (U.S. EPA, 2021, section
30 4.2.2; Hand et al., 2020).

31 Components of $\text{PM}_{2.5}$ contributing to light extinction vary regionally. For example, in the
32 analysis completed in the 2019 ISA, in the Eastern regions, ammonium sulfate accounted for
33 approximately 35 to 60% of the annual average b_{ext} , with the greatest contributions typically

¹³ A b_{ext} value of 40 Mm^{-1} corresponds to a visual range of about 100 km.

¹⁴ The Intermountain West area includes Idaho, Montana, northern Wyoming, and portions of northern California.

1 occurring in the summer (U.S. EPA, 2019, section 13.2.4.1). The second greatest contribution to
2 light extinction came from particulate organic matter (POM), ranging from about 20 to 30% of
3 annual average b_{ext} with less seasonal variation on average than ammonium sulfate. Ammonium
4 nitrate also contributed approximately 10% to 35% of annual average b_{ext} , with much higher
5 concentrations in the winter than in the summer (U.S. EPA, 2019, section 13.2.4.1). In the
6 Northwest, POM was the largest contributor to annual average b_{ext} , up to 70%, in most urban and
7 rural regions with the greatest contributions in the fall. This seasonal contribution of POM may
8 be related to wildfires. A few exceptions included Boise and sites in North Dakota, where
9 ammonium nitrate was the greatest contributor, and sites in the Alaska IMPROVE region, where
10 ammonium sulfate was the greatest contributor (U.S. EPA, 2019, section 13.2.4.1). In the
11 Southwest, based on IMPROVE data, ammonium sulfate or POM were generally the greatest
12 contributors to annual average b_{ext} , with nearly equivalent contributions in several regions. Based
13 on CSN data, ammonium nitrate was often the greatest contributor, with especially high b_{ext}
14 contributions in the winter. While $PM_{10-2.5}$ mass scattering was relatively small in the eastern and
15 northwestern U.S., in the Southwest, $PM_{10-2.5}$ mass scattering contributed to more than 20% of
16 light extinction (U.S. EPA, 2019, section 13.2.4.1).

17 Differences also exist between the urban CSN and the mainly rural IMPROVE data.
18 Light extinction is generally higher in CSN regions than the geographically corresponding
19 IMPROVE regions. Annual average b_{ext} was greater than 50 Mm^{-1} in 11 CSN regions, compared
20 to only one IMPROVE region, and was greater than 20 Mm^{-1} in all CSN regions, compared to
21 just over half of the IMPROVE regions. Light absorbing carbon was the greatest contributor to
22 light extinction in several Western CSN regions but was not a large contributor in any of the
23 IMPROVE regions (U.S. EPA, 2019, Figure 13-11). Ammonium nitrate also accounted for more
24 light extinction in the CSN regions, while it was only a top contributor to b_{ext} in one IMPROVE
25 region (U.S. EPA, 2019, section 13.2.4.1).

26 From the 2005-2008 time period to the 2011-2014 time period, the annual average b_{ext} in
27 most CSN regions in the Eastern U.S. decreased by more than 20 Mm^{-1} . This corresponds to an
28 improvement in average visual range in most Eastern U.S. regions of more than 6 Mm^{-1} (or 15
29 km) from 2005-2008 to 2011-2014. Additionally, the contribution of ammonium sulfate to light
30 extinction has also changed over this period. Due to decreased atmospheric sulfate
31 concentrations, the impact on visibility impairment is evident with a smaller fraction of the total
32 b_{ext} accounted for by ammonium sulfate in 2011-2014 compared to 2005-2008 (U.S. EPA, 2019,
33 section 13.2.4.1).

34 Additionally, Hand et al. (2020) observed that changes in PM composition in ambient air
35 also affect trends for annual, regional mean speciated b_{ext} at IMPROVE monitoring locations
36 across the U.S. In the East, annual mean total b_{ext} decreased by $-4.3\% \text{ yr}^{-1}$ during from 2002 to

1 2018, much of which is attributable to reductions of light extinction from ammonium sulfate.
2 Light extinction was also decreased for ammonium nitrate, although at a lower rate and a lower
3 magnitude than ammonium sulfate. Light extinction by POM, EC, and fine dust also decreased
4 over time, while light extinction by coarse PM increased slightly. In the Intermountain West and
5 Southwest, annual mean total b_{ext} decreased by $-0.9\% \text{ yr}^{-1}$ from 2002 to 2018. The composition
6 of PM in these regions are different than in the East, and while light extinction from ammonium
7 sulfate and ammonium nitrate generally decreased over these time periods, their contribution to
8 light extinction in the Intermountain West and Southwest is less than in the East. Light extinction
9 by POM, EC, and fine dust decreased over time, while the trend for coarse PM remained
10 relatively the same, although the composition of the particles responsible for light extinction in
11 these areas shifted towards a more carbon-dominated composition over time. It is also important
12 to note that the trends observed in the Intermountain West and Southwest regions are likely
13 influenced by biomass smoke, as wildfire smoke emissions are the largest contributor to light
14 extinction by POM and the impacts of wildfires on air quality in these regions has increased in
15 recent years (Hand et al., 2020). Light extinction levels in the West Coast region were higher
16 than in the Intermountain West and Southwest regions, but generally decreased over time (-1.5%
17 yr^{-1}). Light extinction by ammonium nitrate decreased at the highest rate in the West Coast
18 region, and was the only area where the rate decreased at a greater rate than ammonium sulfate.
19 Light extinction by EC and fine dust also decreased, while the trend for POM generally remained
20 flat and light extinction by coarse mass increased slightly. The mix of positive and negative
21 trends in the West Coast region are likely due to the influence of biomass smoke in northern
22 California and Oregon, in particular during 2017 and 2018, as well as reductions in NO_x
23 emissions in Southern California and reductions in light extinction by ammonium sulfate across
24 the region (U.S. EPA, 2021, section 4.2.2; Hand et al., 2020).

25 Since the completion of the 2019 ISA, additional research has emerged that explores the
26 impact of wildfire smoke and biomass smoke on PM composition in the U.S. The increases in
27 PM emissions from these sources coincides with decreases in SO_2 and NO_x emissions, which
28 influences the contribution of different PM species to light extinction. The evidence suggests that
29 PM emissions from wildfire and biomass smoke can impact visibility impairment due to general
30 changes in the dominant PM species in the ambient air during these events, as well as the
31 influence of particle size and aging of the PM over time (U.S. EPA, 2021, section 4.2.2; Laing et
32 al., 2016; Kleinman et al., 2020).

33 In summary, the spatial and temporal analysis of PM monitoring network data in the
34 2019 ISA and recent evidence presented in the draft ISA Supplement emphasize that the extent
35 of light extinction by $\text{PM}_{2.5}$ depends on $\text{PM}_{2.5}$ composition and relative humidity. Regional
36 differences in $\text{PM}_{2.5}$ composition greatly influence light extinction spatially and temporally.

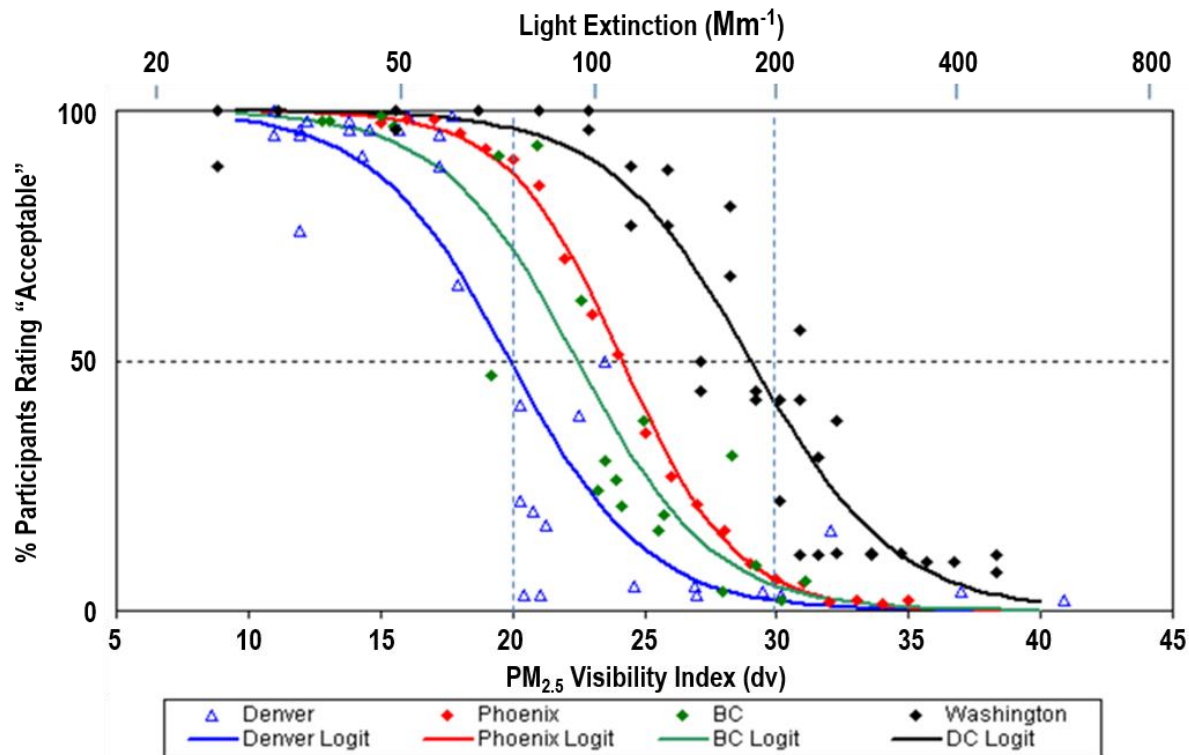
1 Changes in PM_{2.5} composition over time can also affect light extinction based on concentrations
2 of specific PM components in ambient air.

- 3 • **To what extent are recent studies available that might inform judgments about the**
4 **potential adversity to public welfare of PM-attributable visibility impairment and**
5 **the nature of the relationship between PM-attributable visibility impairment and**
6 **public perceptions of such impairment?**

7 In the 2012 review, visibility preference studies were available from four areas in North
8 America,¹⁵ as described in section 5.1.2 above. Study participants were queried regarding
9 multiple images that, depending on the study, were either photographs of the same location and
10 scenery that had been taken on different days on which measured extinction data were available
11 or digitized photographs onto which a uniform “haze” had been superimposed. Results of these
12 studies indicated a wide range of judgments on what study participants considered to be
13 acceptable visibility across the different study areas, depending on the setting depicted in each
14 photograph. As a part of the 2010 UFVA, each study was evaluated separately, and figures were
15 developed to display the percentage of participants that rated the visual air quality depicted as
16 “acceptable” (U.S. EPA, 2010). Figure 5-2 represents a graphical summary of the results of the
17 studies in the four cities and identifies a range encompassing the PM_{2.5} visibility index values
18 from images that were judged to be acceptable by at least 50% of study participants across all
19 four of the urban preference studies (U.S. EPA, 2010, p. 4-24).¹⁶ As shown in Figure 5-2, much
20 lower visibility (considerably more haze resulting in higher values of light extinction) was
21 considered acceptable in Washington, D.C. than was in Denver. The median judgment for the
22 study groups in the two areas differed by 9.2 dv (which roughly corresponds to about 30 μg/m³
23 of PM) (U.S. EPA, 2010).

¹⁵ As noted above, preference studies were available in four urban areas in the last review: Denver, Colorado (Ely et al., 1991, Pryor, 1996), Vancouver, British Columbia, Canada (Pryor, 1996), Phoenix, Arizona (BBC Research & Consulting, 2003), and Washington, DC (Abt Associates, 2001; Smith and Howell, 2009). More details about these studies are available in Appendix D.

¹⁶ Figure 5-2 shows the results of a logistical regression analysis using a logit model of the acceptable or unacceptable ratings from participants of the studies. The logit model is a generalized linear model used for binomial regression analysis which fits explanatory data about binary outcomes (in this case, a person rating an image as acceptable or unacceptable) to a logistic function curve. A detailed description is available in Appendix J of the 2010 UFVA (U.S. EPA, 2010).



1
2 **Figure 5-2. Relationship of viewer acceptability ratings to light extinction.** (Source: U.S.
3 EPA, 2011, Figure 4-2; U.S. EPA, 2010, Figure 2-16)

4
5 Since the completion of the 2012 review, there has been very little research on visibility
6 preferences, with one visibility preference study conducted in the Grand Canyon, AZ (Malm et
7 al., 2019) and one in Beijing, China (Fajardo et al., 2013). The Grand Canyon study reported a
8 lower range of acceptable visibility impairment among participants than was found in preference
9 studies previously conducted in the U.S. (Malm et al., 2019). The Malm et al. (2019) study
10 design is similar to that used in the public preference studies discussed above, but differs from
11 those studies in that this study was conducted in a Federal Class I area, as opposed to in an urban
12 area, with a scene depicted in the photographs that did not include urban features.¹⁷ The Malm et
13 al. (2019) study also used a much lower range of superimposed “haze” than the preference
14 studies discussed above, which may bias the participant responses given the generally lower
15 visibility range presented compared to the other studies.¹⁸

¹⁷ The Grand Canyon study used a single scene looking west down the canyon with a small landscape feature of a 100-km-distant mountain (Mount Trumbull), along with other closer landscape features. The scenes presented in the previously available visibility preference studies are presented in more detail in Table D-9 in Appendix D.

¹⁸ The Grand Canyon study superimposed light extinction ranging from 3 dv to 20 dv on the image slides shown to participants compared to the previously available preference studies. In those studies, the visibility ranges

1 The study conducted in Beijing found a higher range of acceptable visibility impairment
2 among participants than was found in preference studies previously conducted in the U.S. This
3 finding may be related to the common occurrence of higher PM_{2.5} concentrations in Beijing (with
4 associated visibility impairment) than is typical in the U.S. (U.S. EPA, 2019, section 13.2.5).

5 Similarly, there is little recent information regarding acceptable levels of visibility
6 impairment in the U.S. One study explored alternate methods for evaluating “acceptable” levels
7 of visual air quality from the preference studies, including the use of scene-specific visibility
8 indices as potential indicators of visibility levels as perceived by the observer (Malm et al.,
9 2019). In addition to measures of atmospheric haze, such as atmospheric extinction, used in
10 previously available preference studies, other indices for visual air quality include color and
11 achromatic contrast of single landscape figures, average and equivalent contrast of an entire
12 scene, edge detection algorithms such as the Sobel index, and just-noticeable difference or
13 change indexes. The results reported by Malm et al. (2019) suggest that scene-dependent metrics,
14 such as contrast, may be useful alternate predictors of preference levels compared to universal
15 metrics like light extinction (U.S. EPA, 2021, section 4.2.1). This is because extinction alone is
16 not a measure of “haze,” but of light attenuation per unit distance, and visible “haze” is
17 dependent on both light extinction and distance to a landscape feature (U.S. EPA, 2021, section
18 4.2.1).

19 • **To what extent have important uncertainties in the evidence from the last review
20 been addressed, and have new uncertainties emerged?**

21 Since the 2012 review, some refinements have been made to the IMPROVE equation to
22 better estimate light extinction, but there has been no expansion of monitoring efforts for direct
23 measurement of light extinction. At the time of the 2012 review, it was noted that a PM_{2.5} light
24 extinction monitoring program could help with characterizing visibility conditions and the
25 relationships between PM component concentrations and light extinction.

26 Little new research is available that helps to expand our understanding of visibility
27 preferences or our characterization of visibility conditions. Uncertainties and limitations
28 consistent with those identified in the past reviews persist in this reconsideration.

- 29 • Given the potential for people to have different preferences based on the visibility they are
30 used to based on conditions that they commonly encounter, and the potential for them to
31 also have different preferences for different types of scenes, the preference studies may
32 not capture the range of preferences of people in the U.S.
- 33 • Most of the preference studies were conducted 15 to 30 years ago and may not reflect the
34 visibility preferences of the U.S. population today. Given that air quality has improved

presented were as low as 9 dv and as high as 45 dv. The visibility ranges presented in the previously available
visibility preference studies are described in more detail in Table D-9 in Appendix D.

1 over the last several decades, the older studies may not reflect current preferences of
2 people in the U.S. Newer studies may not capture the extent to which preferences may be
3 changing over time.

- 4 • The preference studies have used different methods to evaluate what level of visibility
5 impairment is acceptable. Variability in study methodology may influence an individual's
6 response as to what level of visibility impairment is deemed acceptable, and thereby
7 influence the results of the study.
- 8 • Many factors that are not captured by the methods used in the preference studies may
9 influence people's judgments on acceptable visibility. For example, an individual's
10 perception of an acceptable level of visibility impairment could be influenced by the
11 duration of visibility impairment experienced, the time of day during which light
12 extinction is greatest, and the frequency of episodes of visibility impairment, as well as
13 the intensity of the visibility impairment (i.e., the focus of the studies).
- 14 • Methods for quantitatively evaluating people's judgments on acceptability are evolving
15 but are still inconsistent in their application across studies. Variability in quantitative
16 methods for comparing visual air quality in public preference studies may influence the
17 consistency and comparability of results and the interpretation of these results in the
18 context of regional or national preferences for visibility impairment in urban, non-urban,
19 and Federal Class I areas.

20 Overall, the body of evidence regarding visibility effects remains largely unchanged since
21 the time of the 2012 review. While one new study provides refinements to the methods for
22 estimating light extinction, uncertainties and limitations in the scientific evidence during the
23 previous reviews remain.

24 **5.3.1.2 Quantitative and Air Quality Information**

25 Beyond our consideration of the scientific evidence, discussed in section 5.3.1.1 above,
26 we have also considered quantitative analyses of PM air quality and visibility impairment with
27 regard to the extent they could inform conclusions on the adequacy of the public welfare
28 protection provided by the current secondary PM standards. In the 2012 review, quantitative
29 analyses focused on daily visibility impairment, given the short-term nature of PM-related
30 visibility effects. Such quantitative analyses conducted as part of the 2012 review informed the
31 decision on the secondary standards in that review (U.S. EPA, 2010, U.S. EPA, 2011; 78 FR
32 3189-3192, January 15, 2013). The information available since the 2012 review includes an
33 updated equation for estimating light extinction, summarized in section 5.3.1.1 above and
34 described in the 2019 ISA, as well as more recent air monitoring data, that together allow for
35 development of an updated assessment with the potential to substantially add to our
36 understanding of PM-related visibility impairment. Thus, we have conducted updated analyses
37 for this reconsideration based on the technical information, tools, and methods.

- 1 • **How much visibility impairment is estimated to occur in areas that meet the current**
2 **secondary PM standards? What are the factors contributing to the estimates in areas**
3 **with higher values?**

4 Consistent with the analyses conducted in the 2012 and 2020 reviews, we have conducted
5 analyses examining the relationship between PM mass concentrations and calculated light
6 extinction using the 3-year design values¹⁹ for the current secondary standards and a 3-year
7 average visibility metric based on light extinction estimated using IMPROVE equations using air
8 quality data for 2017 to 2019.²⁰ These analyses are intended to inform our understanding of
9 visibility impairment in the U.S. under recent air quality conditions, particularly those conditions
10 that meet the current standards, and the relative influence of various factors on light extinction.
11 Given the relationship of visibility with short-term PM, we focus particularly on the short-term
12 PM standards.

13 Given that visibility-related effects are often associated with short-term PM
14 concentrations, and recognizing the relatively larger role of PM_{2.5} and its components in light
15 extinction and as inputs to the IMPROVE equation, we have given somewhat more attention to
16 consideration of the 24-hour PM_{2.5} standard. Analyses were conducted using three versions of
17 the IMPROVE equation (Equations D-1 through D-3 in Appendix D) to estimate light extinction
18 to better understand the influence of variability in inputs across the three equations. This analysis
19 included 60 monitoring sites that are geographically distributed across the U.S. in both urban and
20 rural areas (see Figure D-1 in Appendix D). These sites are those that have a valid 24-hour PM_{2.5}
21 design value for the 2017-2019 period and met strict criteria for PM species for this analysis.²¹
22 We present results for these 60 sites using the original IMPROVE equation, with modifications
23 to the equation consistent with those made in evaluating light extinction in the 2012 review
24 (described in detail in section D.1 of Appendix D). We then present results for these 60 sites with
25 light extinction calculated using the Lowenthal and Kumar (2016) IMPROVE equation described
26 in section 5.3.1.1 above.

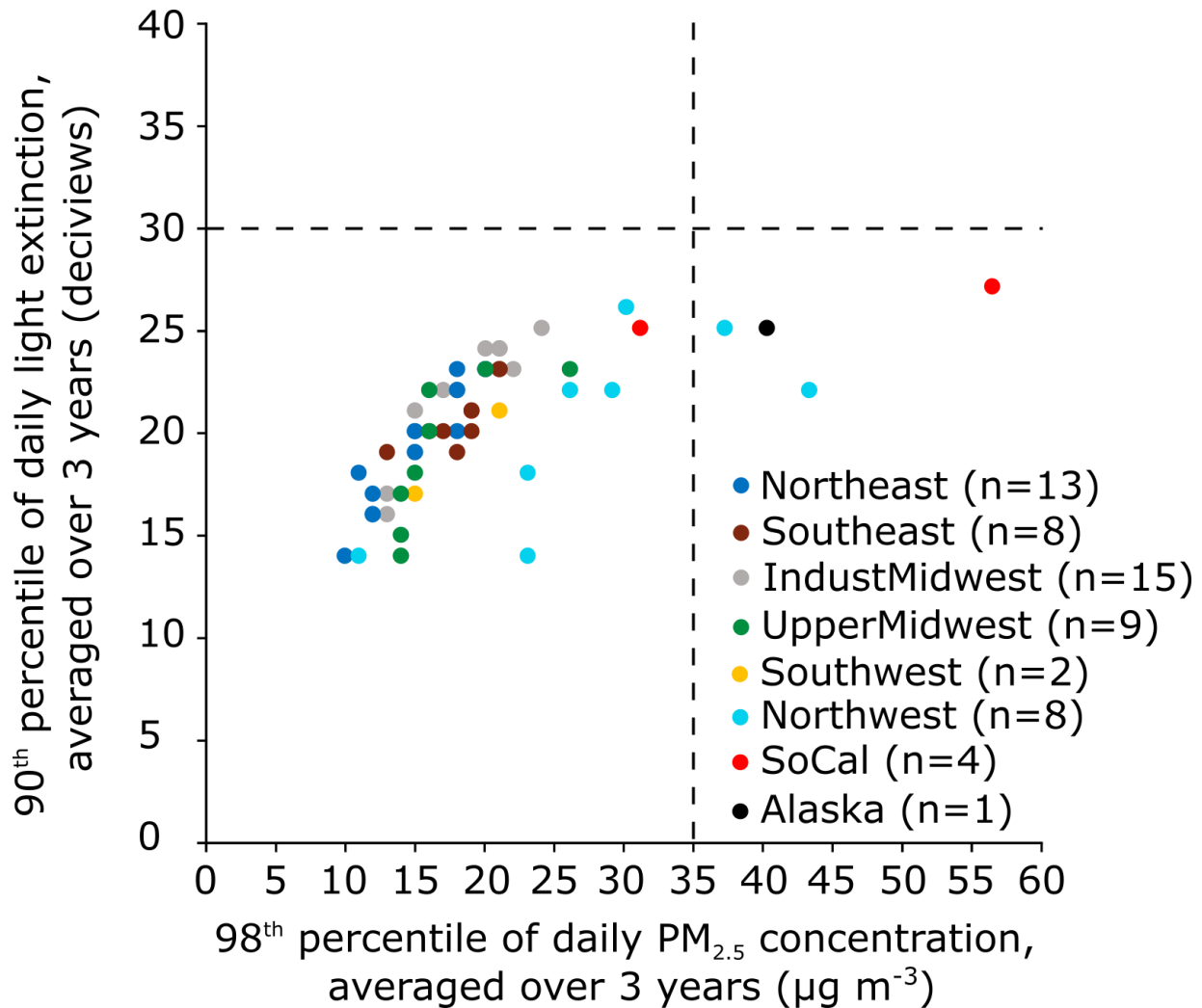
¹⁹ A design value is a statistic that summarizes the air quality data for a given area in terms of the indicator, averaging time, and form of the standard. Design values can be compared to the level of the standard and are typically used to designate areas as meeting or not meeting the standard and assess progress towards meeting the NAAQS.

²⁰ This is the 3-year visibility metric that was used to evaluate visibility impairment in the 2012 and 2020 reviews. Given that there has been little new research since the time of the 2012 review to better inform our understanding of visibility preferences in the U.S., there is no new information available to inform selection of a visibility metric for evaluating visibility impairment in the current review different from the one identified in the 2012 review.

²¹ For this analysis, completeness criteria for speciated PM data at these sites included having all 12 quarters in the 2017-2019 period with at least 11 days in each quarter with a valid PM_{2.5} and PM_{10-2.5} mass, sulfate, nitrate, organic carbon, elemental carbon, sea salt (chlorine or chloride), and fine soil (aluminum, silica, calcium, iron, and titanium) measurement.

1 In considering the relationship between the 24-hour PM_{2.5} mass-based design value and
2 the 3-year visibility metric using recent air quality data, we first examine the relationship using
3 the original IMPROVE equation, consistent with the methods used in the 2012 review (Kelly et
4 al., 2012; 78 FR 3201, January 15, 2013; Appendix D). In those areas that meet the current 24-
5 hour PM_{2.5} standard, all sites have light extinction estimates at or below 26 dv (Figure 5-3; 78 FR
6 3218, January 15, 2013). For the four locations that exceed the current 24-hour PM_{2.5} standard,
7 light extinction estimates range from 22 dv to 29 dv (Figure 5-3). These findings are consistent
8 with the findings of the analysis using the same IMPROVE equation in the 2012 review with
9 data from 102 sites with data from 2008-2010 and in the 2020 review with data from 67 sites
10 with data from 2015-2017. This indicates similar findings from this analysis as was the case with
11 the similar analysis in the 2012 and 2020 reviews, i.e., the updated quantitative analysis shows
12 that the 3-year visibility metric was no higher than 30 dv²² at sites meeting the current secondary
13 PM standards, and at most such sites the 3-year visibility index values are much lower (e.g., an
14 average of 20 dv across the 60 sites).
15

²² For comparison purposes in these air quality analyses, we use a 3-year visibility metric with a level of 30 dv, which is the highest level of visibility impairment judged to be acceptable by at least 50 percent of the participants in the preference studies that were available at the time of the 2012 review (78 FR 3191, January 15, 2013).



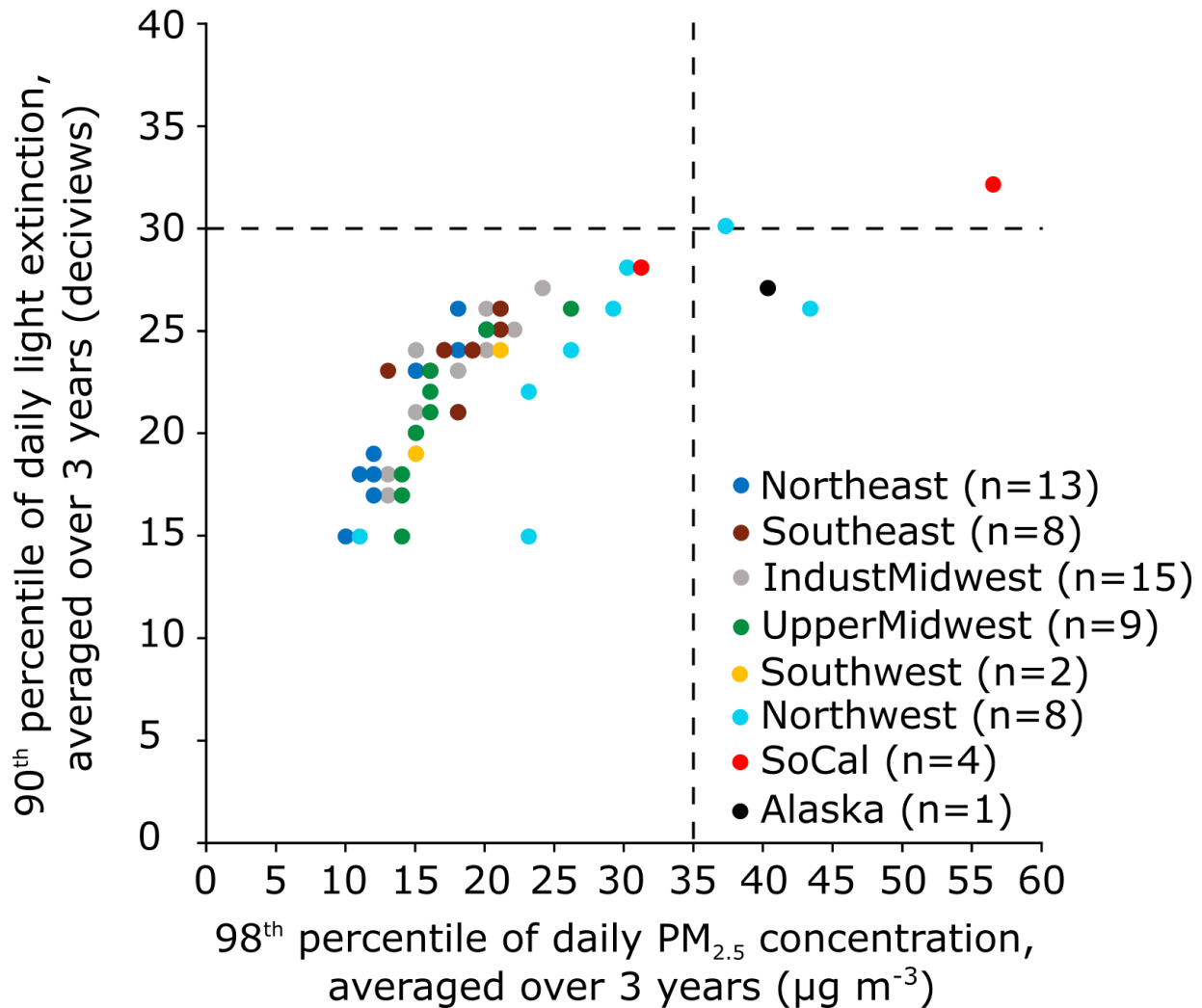
1
 2 **Figure 5-3. Comparison of 90th percentile of daily light extinction, averaged over three**
 3 **years, and 98th percentile of daily PM_{2.5} concentrations, averaged over three years, for**
 4 **2017-2019 using the original IMPROVE equation.** (Note: Dashed lines indicate the level
 5 of current 24-hour PM_{2.5} standard (35 µg/m³) and the target level of protection identified for
 6 the 3-year visibility metric (30 dv).)
 7

8 When light extinction was calculated using the refined equation from Lowenthal and
 9 Kumar (2016), the resulting 3-year visibility metrics are slightly higher at all sites compared to
 10 light extinction estimates calculated using the original IMPROVE equation (Figure 5-4). As
 11 noted in section 5.3.1.1, this version of the IMPROVE equation uses a multiplier of 2.1 to
 12 convert the measured OC to OM for input into the equation and also accounts for water
 13 absorption by water soluble organic matter as a function of relative humidity, likely contributing
 14 to the slightly higher estimates of light extinction. As noted in section 5.3.1.1, the Lowenthal and
 15 Kumar (2016) refinements to the IMPROVE equation are based on evaluations of monitoring
 16 data from remote IMPROVE sites. More remote areas tend to have more aged organic particles

1 than urban areas, and these adjustments to the IMPROVE equation account for the higher
2 concentration of organic matter as a result of more aged organic particles at these sites. It is
3 important to note that, since the Lowenthal and Kumar (2016) refinements to the IMPROVE
4 equation likely result in one of the higher estimates of light extinction, this equation may
5 overestimate light extinction in non-remote areas, including those urban areas in our analyses.

6 Using the Lowenthal and Kumar (2016) equation, for those sites that meet the current 24-
7 hour PM_{2.5} standard, the 3-year visibility metric is at or below 28 dv when light extinction is
8 calculated. For those sites that exceed the current 24-hour PM_{2.5} standard, three of these sites
9 have a 3-year visibility metric ranging between 26 dv and 30 dv, while one site in Fresno,
10 California that exceeds the current 24-hour PM_{2.5} standard and has a 3-year visibility index value
11 of 32 dv (compared to 29 dv when light extinction is calculated with the original IMPROVE
12 equation) (see Table D-3 in Appendix D). At this site, it is likely that the 3-year visibility metric
13 using the Lowenthal and Kumar (2016) equation would be below 30 dv if PM_{2.5} concentrations
14 were reduced such that the 24-hour PM_{2.5} level of 35 µg/m³ was attained.

15



1
2 **Figure 5-4. Comparison of 90th percentile of daily light extinction, averaged over three**
3 **years, and 98th percentile of daily PM_{2.5} concentrations, averaged over three years, for**
4 **2015-2017 using the Lowenthal and Kumar equation.** (Note: Dashed lines indicate the
5 level of current 24-hour PM_{2.5} standard (35 µg/m³) and the target level of protection
6 identified for the 3-year visibility metric (30 dv).)
7

8 In considering visibility impairment under recent air quality conditions, we recognize that
9 the differences in the inputs to equations estimating light extinction can influence the resulting
10 values. For example, given the varying chemical composition of emissions from different
11 sources, the 2.1 multiplier in the Lowenthal and Kumar (2016) equation may not be appropriate
12 for all source types. At the time of the 2012 review, the EPA judged that a 1.6 multiplier for
13 converting OC to OM was more appropriate, for the purposes of estimating visibility index at
14 sites across the U.S., than the 1.4 or 1.8 multipliers used in the original and revised IMPROVE
15 equations, respectively. A multiplier of 1.8 or 2.1 would account for the more aged and
16 oxygenated organic PM that tends to be found in more remote regions than in urban regions,

1 whereas a multiplier of 1.4 may underestimate the contribution of organic PM found in remote
2 regions when estimating light extinction (78 FR 3206, January 15, 2013; U.S. EPA, 2012a, p.
3 IV-5). The information and analyses indicate that it may be appropriate to select inputs to the
4 IMPROVE equation (e.g., the multiplier for OC to OM) on a regional basis rather than a national
5 basis when calculating light extinction. This is especially true when comparing sites with
6 localized PM sources (such as sites in urban or industrial areas) to sites with PM derived largely
7 from biogenic precursor emissions (that contribute to widespread secondary organic aerosol
8 formation), such as those in the southeastern U.S. We note, however, that conditions involving
9 PM from such different sources have not been well studied in the context of applying a multiplier
10 to estimate light extinction, contributing uncertainty to estimates of light extinction for such
11 conditions.

12 At the time of the 2012 review, the EPA noted that PM_{2.5} is the size fraction of PM
13 responsible for most of the visibility impairment in urban areas (77 FR 38980, June 29, 2012).
14 Data available at the time of the 2012 review suggested that, generally, PM_{10-2.5} was a minor
15 contributor to visibility impairment most of the time (U.S. EPA, 2010) although the coarse
16 fraction may be a major contributor in some areas in the desert southwestern region of the U.S.
17 Moreover, at the time of the 2012 review, there were few data available from PM_{10-2.5} monitors
18 to quantify the contribution of coarse PM to calculated light extinction. Since that time, an
19 expansion in PM_{10-2.5} monitoring efforts has increased the availability of data for use in
20 estimating light extinction with both PM_{2.5} and PM_{10-2.5} concentrations included as inputs in the
21 equations. The analysis in the 2020 review addressed light extinction at 20 of the 67 PM_{2.5} sites
22 where collocated PM_{10-2.5} monitoring data were available. Since the 2020 review, PM_{10-2.5}
23 monitoring data are available at more locations and the analyses presented in this draft PA
24 include those for light extinction estimated with coarse and fine PM at all 60 sites. Generally, the
25 contribution of the coarse fraction to light extinction at these sites is minimal, contributing less
26 than 1 dv to the 3-year visibility metric (U.S. EPA, 2020, section 5.2.1.2). However, we note that
27 in our analysis, only a few sites were in locations that would be expected to have high
28 concentrations of coarse PM, such as the Southwest. These results are consistent with those in
29 the analyses in the 2019 ISA, which found that mass scattering from PM_{10-2.5} was relatively
30 small (less than 10%) in the eastern and northwestern U.S., whereas mass scattering was much
31 larger in the Southwest (more than 20%) particularly in southern Arizona and New Mexico (U.S.
32 EPA, 2019, section 13.2.4.1, p. 13-36).

33 In summary, the findings of these updated quantitative analyses are generally consistent
34 with those in the 2012 and 2020 reviews. The 3-year visibility metric was generally below 26 dv
35 in most areas that meet the current 24-hour PM_{2.5} standard. Small differences in the 3-year
36 visibility metric were observed between the variations of the IMPROVE equation, which may

1 suggest that it may be more appropriate to use one version over another in different regions of
2 the U.S. based on PM characteristics such as particle size and composition to more accurately
3 estimate light extinction.

4 **5.3.2 Non-Visibility Effects**

5 **5.3.2.1 Nature of Effects**

6 In considering the evidence for non-visibility welfare effects attributable to PM as
7 presented in the 2019 ISA, this section poses the following policy-relevant questions:

- 8 • **To what extent has the scientific evidence improved our understanding of the nature
9 and magnitude of non-visibility welfare effects of PM in ambient air, including the
10 variability associated with such effects? To what extent have important uncertainties
11 in the evidence from the last review been addressed, and have new uncertainties
12 emerged?**

13 As an initial matter, we note that the draft ISA Supplement does not include an evaluation
14 of additional studies for climate and materials effects and the causality determinations from PM-
15 related climate and materials effects presented in the 2019 ISA continue to serve as the scientific
16 foundation for these effects. As such, the sections below that address these questions for PM and
17 climate effects (section 5.3.2.1.1) and materials effects (section 5.3.2.1.2) draw from the
18 evaluation of the welfare effects evidence for PM-related climate and materials effects in the
19 2019 ISA and considerations of such effects in the 2020 PA (U.S. EPA, 2020).

20 **5.3.2.1.1 Climate Effects**

21 In considering the evidence of climate effects attributable to PM, this section poses the
22 following policy-relevant question:

- 23 • **To what extent is information available that changes or enhances our understanding
24 of the climate impacts of PM-related aerosols, particularly regarding a quantitative
25 relationship between PM concentrations and effects on climate (e.g., through
26 radiative forcing)?**

27 In the 2012 review, the 2009 PM ISA concluded that there was “sufficient evidence to
28 determine a causal relationship between PM and climate effects – specifically on the radiative
29 forcing of the climate system, including both direct effects of PM on radiative forcing and
30 indirect effects that involve cloud feedbacks that influence precipitation formation and cloud
31 lifetimes” (U.S. EPA, 2009, section 9.3.10).²³ Since the 2012 review, climate impacts have been

²³ Radiative forcing (RF) for a given atmospheric constituent is defined as the perturbation in net radiative flux, at the tropopause (or the top of the atmosphere) caused by that constituent, in watts per square meter (Wm^{-2}), after allowing for temperatures in the stratosphere to adjust to the perturbation but holding all other climate responses constant, including surface and tropospheric temperatures (Fiore et al., 2015, Myhre et al., 2013). A positive

1 extensively studied and the 2019 ISA concludes that “overall the evidence is sufficient to
2 conclude that a causal relationship exists between PM and climate effects” (U.S. EPA, 2019,
3 section 13.3.9). Recent research reinforces and strengthens the evidence evaluated in the 2009
4 ISA. Recent evidence provides greater specificity about the details of these radiative forcing
5 effects and increased understanding of additional climate impacts driven by PM radiative effects.
6 The Intergovernmental Panel on Climate Change (IPCC) assesses the role of anthropogenic
7 activity in past and future climate change. In the 2012 review, the 2009 ISA relied heavily on the
8 Fourth IPCC Assessment Report (AR4); since the 2012 review, the IPCC issued an updated
9 report, as described in the 2019 ISA. The Fifth IPCC Assessment Report (AR5; IPCC, 2013)
10 reports on the key scientific advances in understanding the climate effects of PM since AR4. The
11 2019 ISA draws substantially upon AR5 in summarizing these effects.

12 Atmospheric PM has the potential to affect climate in multiple ways, including absorbing
13 and scattering of incoming solar radiation, alterations in terrestrial radiation, effects on the
14 hydrological cycle, and changes in cloud properties (U.S. EPA, 2019, section 13.3.1).

15 Atmospheric PM interacts with incoming solar radiation. Many species of PM (e.g., sulfate and
16 nitrate) efficiently scatter solar energy. By enhancing reflection of solar energy back to space,
17 scattering PM exerts a cooling effect on the surface below. Certain species of PM such as black
18 carbon (BC), brown carbon (BrC), or dust can also absorb incoming sunlight. A recent study
19 found that whether absorbing PM warms or cools the underlying surface depends on several
20 factors, including the altitude of the PM layer relative to cloud cover and the albedo of the
21 surface (Ban-Weiss et al., 2014). PM also perturbs incoming solar energy by influencing cloud
22 cover and cloud lifetime. For example, PM provides nuclei upon which water vapor condenses,
23 forming cloud droplets. Finally, absorbing PM deposited on snow and ice can diminish surface
24 albedo and lead to regional warming (U.S. EPA, 2019, section 13.3.2).

25 PM has direct and indirect effects on climate processes. PM interactions with solar
26 radiation through scattering and absorption, collectively referred to as aerosol-radiation
27 interactions (ARI), are also known as the direct effects of PM on climate, as opposed to the
28 indirect effects that involve aerosol-cloud interactions (ACI). The direct effects of PM on climate
29 result primarily from particles scattering light away from Earth and sending a fraction of solar
30 energy back into space, decreasing the transmission of visible radiation to the surface of the
31 Earth and resulting in a decrease in the heating rate of the surface and the lower atmosphere. The
32 IPCC AR5, taking into account both model simulations and satellite observations, reports a
33 radiative forcing from aerosol-radiation interactions (RFari) from anthropogenic PM of $-0.35 \pm$

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).

1 0.5 watts per square meter (Wm^{-2}) (Boucher, 2013), which is slightly reduced compared to AR4.
2 Estimates of effective radiative forcing²⁴ from aerosol-radiation interactions (ERFari), which
3 include the rapid feedback effects of temperature and cloud cover, rely mainly on model
4 simulations, as this forcing is complex and difficult to observe (U.S. EPA, 2019, section
5 13.3.4.1). The IPCC AR5 best estimate for ERFari is $-0.45 \pm 0.5 \text{ Wm}^{-2}$, which reflects this
6 uncertainty (Boucher, 2013).

7 By providing cloud condensation nuclei, PM increases cloud droplet number, thereby
8 increasing cloud droplet surface area and albedo (Twomey, 1977). The climate effects of these
9 perturbations are more difficult to quantify than the direct effects of aerosols with RF but likely
10 enhance the cooling influence of clouds by increasing cloud reflectivity (traditionally referred to
11 as the first indirect effect) and lengthening cloud lifetime (the second indirect effect). These
12 effects are reported as the radiative forcing from aerosol-cloud interactions (RFaci) and the
13 effective radiative forcing from aerosol-cloud interactions (ERFaci) (U.S. EPA, 2019, section
14 13.3.3.2). IPCC AR5 estimates ERFaci at -0.45 Wm^{-2} , with a 90% confidence interval of -1.2 to
15 0 Wm^{-2} (U.S. EPA, 2019, section 13.3.4.2).²⁵ Studies have also calculated the combined
16 effective radiative forcing from aerosol-radiation and aerosol-cloud interactions (ERFari+aci)
17 (U.S. EPA, 2019, section 13.3.4.3). IPCC AR5 reports a best estimate of ERFari+aci of -0.90 (-
18 1.9 to -0.1) Wm^{-2} , consistent with these estimates (Boucher, 2013).

19 PM can also strongly reflect incoming solar radiation in areas of high albedo, such as
20 snow- and ice-covered surfaces. The transport and subsequent deposition of absorbing PM such
21 as BC to snow- and ice-covered regions can decrease the local surface albedo, leading to surface
22 heating. The absorbed energy can then melt the snow and ice cover and further depress the
23 albedo, resulting in a positive feedback loop (U.S. EPA, 2019, section 13.3.3.3; Bond et al.,
24 2013; U.S. EPA, 2012b). Deposition of absorbing PM, such as BC, may also affect surface
25 temperatures over glacial regions (U.S. EPA, 2019, section 13.3.3.3). The IPCC AR5 best
26 estimate of RF from the albedo effect is $+0.04 \text{ Wm}^{-2}$, with an uncertainty range of $+0.02$ to $+0.09$
27 Wm^{-2} (Boucher, 2013).

28 While research on PM-related effects on climate has expanded since the 2012 review,
29 there are still significant uncertainties associated with the accurate measurement of PM
30 contributions to the direct and indirect effects of PM on climate.

²⁴ Effective radiative forcing (ERF), new in the IPCC AR5, takes into account not just the instantaneous forcing but also a set of climate feedbacks, involving atmospheric temperature, cloud cover, and water vapor, that occur naturally in response to the initial radiative perturbation (U.S. EPA, 2019, section 13.3.2.2).

²⁵ While the 2019 ISA includes estimates of RFaci and ERFaci from a number of studies (U.S. EPA, 2019, sections 13.3.4.2, 13.3.4.3, 13.3.3.3), this draft PA focuses on the single best estimate with a range of uncertainty, as reported in IPCC AR5 (Boucher, 2013).

- 1 • **To what extent does the information provide evidence of a quantitative relationship**
2 **between specific PM constituents (i.e., BC, OC, sulfate) and climate-related effects?**

3 Since the 2012 review, a number of studies have examined the individual climate effects
4 associated with key PM components, including sulfate, nitrate, OC, BC, and dust, along with
5 updated quantitative estimates of the radiative forcing associated with the individual species.

6 Sulfate particles form through oxidation of SO₂ by OH in the gas phase and in the
7 aqueous phase by a number of pathways, including in particular those involving ozone and H₂O₂
8 (U.S. EPA, 2019, section 13.3.5.1). The main source of anthropogenic sulfate is from coal-fired
9 power plants, and global trends in the anthropogenic SO₂ emissions are estimated to have
10 increased dramatically during the 20th and early 21st centuries, although the recent
11 implementation of more stringent air pollution controls on sources has led to a reversal in such
12 trends in many places (U.S. EPA, 2019, section 13.3.5.1). Sulfate particles are highly reflective.
13 Consistent with other recent estimates, on a global scale, the IPCC AR5 estimates that sulfate
14 contributes more than other PM types to RF, with RF_{ari} of -0.4 (-0.6 to -0.2) Wm⁻², where the
15 5% and 95% uncertainty range is represented by the numbers in the parentheses (Myhre et al.,
16 2013). This uncertainty range indicates the challenges associated with estimating SO₂ from
17 sources in developing regions and estimating the lifetime of sulfate against wet deposition.
18 Sulfate is also a major contributor to the influence of PM on clouds (Takemura, 2012). A total
19 effective radiative forcing (ERF_{ari+aci}) for anthropogenic sulfate has been estimated to be nearly
20 -1.0 Wm⁻² (Adams et al., 2001, Zelinka et al., 2014).

21 Nitrate particles form through the oxidation of nitrogen oxides and occur mainly in the
22 form of ammonium nitrate. Ammonium preferentially associates with sulfate rather than nitrate,
23 leading to formation of ammonium sulfate at the expense of ammonium nitrate (Adams et al.,
24 2001). As anthropogenic emissions of SO₂ decline, more ammonium will be available to react
25 with nitrate, potentially leading to future increases in ammonium nitrate particles in the
26 atmosphere (U.S. EPA, 2019, section 13.3.5.2; Hauglustaine et al., 2014; Lee et al., 2013;
27 Shindell et al., 2013). Warmer global temperatures, however, may decrease nitrate abundance
28 given that it is highly volatile at higher temperatures (Tai et al., 2010). The IPCC AR5 estimates
29 RF_{ari} of nitrate of -0.11 (-0.3 to -0.03) Wm⁻² (Boucher, 2013), which is one-fourth of the RF_{ari}
30 of sulfate.

31 Primary organic carbonaceous PM, including BrC, are emitted from wildfires,
32 agricultural fires, and fossil fuel and biofuel combustion. Secondary organic aerosols (SOA)
33 form when anthropogenic or biogenic nonmethane hydrocarbons are oxidized in the atmosphere,
34 leading to less volatile products that may partition into PM (U.S. EPA, 2019, section 13.3.5.3).
35 Organic particles are generally reflective, but in the case of BrC, a portion is significantly
36 absorbing at shorter wavelengths (<400 nm). The IPCC AR5 estimates an RF_{ari} for primary

1 organic PM from fossil fuel combustion and biofuel use of -0.09 (-0.16 to -0.03) Wm^{-2} and an
2 RFari estimate for SOA from these sources of -0.03 (-0.27 to $+0.20$) Wm^{-2} (Myhre et al., 2013).
3 The wide range in these estimates, including inconsistent signs for forcing, reflect uncertainties
4 in the optical properties of organic PM and its atmospheric budgets, including the production
5 pathways of anthropogenic SOA (Scott et al., 2014; Myhre et al., 2013; McNeill et al., 2012;
6 Heald et al., 2010). The IPCC AR5 also estimates an RFari of -0.2 Wm^{-2} for primary organic PM
7 arising from biomass burning (Boucher, 2013).

8 Black carbon (BC) particles occur as a result of inefficient combustion of carbon-
9 containing fuels. Like directly emitted organic PM, BC is emitted from biofuel and fossil fuel
10 combustion and by biomass burning. BC is absorbing at all wavelengths and likely has a large
11 impact on the Earth's energy budget (Bond et al., 2013). The IPCC AR5 estimates a RFari from
12 anthropogenic fossil fuel and biofuel use of $+0.4$ ($+0.5$ to $+0.8$) Wm^{-2} (Myhre et al., 2013).
13 Biomass burning contributes an additional $+0.2$ ($+0.03$ to $+0.4$) Wm^{-2} to BC RFari, while the
14 albedo effect of BC on snow and ice adds another $+0.04$ ($+0.02$ to $+0.09$) Wm^{-2} (Myhre et al.,
15 2013; U.S. EPA, 2019, section 13.3.5.4, section 13.3.4.4).

16 Dust, or mineral dust, is mobilized from dry or disturbed soils as a result of both
17 meteorological and anthropogenic activities. Dust has traditionally been classified as scattering,
18 but a recent study found that dust may be substantially coarser than currently represented in
19 climate models, and thus more light-absorbing (Kok et al., 2017). The IPCC AR5 estimates
20 RFari as -0.1 ± 0.2 Wm^{-2} (Boucher, 2013), although the results of the study by Kok et al. (2017)
21 would suggest that in some regions dust may have led to warming, not cooling (U.S. EPA, 2019,
22 section 13.3.5.5).

23 Recent research expands upon the evidence from the 2012 review. Consistent with the
24 evidence in the 2012 review, the key PM components, including sulfate, nitrate, OC, BC, and
25 dust, that contribute to climate processes vary in their reflectivity, forcing efficiencies, and
26 direction of forcing.

27 • **To what extent does the evidence change or improve our understanding of the spatial
28 and temporal variation in climate responses to PM?**

29 Radiative forcing due to PM elicits a number of responses in the climate system that can
30 lead to significant effects on weather and climate over a range of spatial and temporal scales,
31 mediated by a number of feedbacks that link PM and climate. Since the 2012 review, the
32 evidence base has expanded with respect to the mechanisms of climate responses and feedbacks
33 to PM radiative forcing, described below, although considerable uncertainties continue to exist.
34 We focus our discussion primarily on the climate impacts in the U.S.

35 Unlike well-mixed, long-lived greenhouse gases in the atmosphere, PM has a very
36 heterogenous distribution across the Earth. As such, patterns of RFari and RFaci tend to correlate

1 with PM loading, with the greatest forcings centralized over continental regions. The climate
2 response is more complicated since the perturbation to one climate variable (e.g., temperature,
3 cloud cover, precipitation) can lead to a cascade of effects on other variables. While the initial
4 PM radiative forcing may be concentrated regionally, the eventual climate response can be much
5 broader spatially or be concentrated in remote regions (U.S. EPA, 2019, section 13.3.6). The
6 complex climate system interactions lead to variation among climate models, with some studies
7 showing relatively close correlation between forcing and surface response temperatures (e.g.,
8 Leibensperger et al., 2012), while other studies show much less correlation (e.g., Levy et al.,
9 2013). Many studies have examined observed trends in PM and temperature in the U.S. Climate
10 models have suggested a range of factors which can influence large-scale meteorological
11 processes and may affect temperature, including local feedback effects involving soil moisture
12 and cloud cover, changes in the hygroscopicity of the PM, and interactions with clouds alone
13 (U.S. EPA, 2019, section 13.3.7). While evidence described in the 2019 ISA suggests that PM
14 influenced temperature trends across the southern and eastern U.S. in the 20th century,
15 uncertainties continue to exist and further research is needed to better characterize the effects of
16 PM on regional climate in the U.S.

17 • **To what extent have important uncertainties identified in prior reviews been**
18 **reduced and/or have new uncertainties emerged?**

19 Since 2009, significant progress has been made in evaluating PM-related climate effects
20 and uncertainties. The IPCC AR5 states that “climate-relevant aerosol processes are better
21 understood, and climate-relevant aerosol properties are better observed, than at the time of the
22 AR4” (Boucher, 2013). However, significant uncertainties remain that make it difficult to
23 quantify the climate effects of PM. Such uncertainties include those related to our understanding
24 of:

- 25 • The magnitude of PM radiative forcing and the portion of that associated with
26 anthropogenic emissions;
- 27 • The contribution of regional differences in PM concentrations, and of individual
28 components, to radiative forcing;
- 29 • The mechanisms of climate responses and feedbacks resulting from PM-related radiative
30 forcing; and,
- 31 • The process by which PM interacts with clouds and how to represent such interactions in
32 climate models.

33 While research has progressed significantly since the 2012 review, substantial
34 uncertainties still remain with respect to key processes linking PM and climate, because of the
35 small scale of PM-relevant atmospheric processes compared to the resolution of state-of-the-art
36 models, and because of the complex cascade of indirect impacts and feedbacks in the climate

1 system that result from an initial PM-related radiative perturbation (U.S. EPA, 2019, section
2 13.3.9).

3 **5.3.2.1.2 Materials Effects**

4 In considering the evidence on materials effects attributable to PM, this section poses the
5 following policy-relevant question:

- 6 • **To what extent is information available to link PM to materials effects, including**
7 **degradation of surfaces, and deterioration of materials such as metal, stone, concrete**
8 **and marble?**

9 In the 2012 review, the 2009 ISA concluded that there was “a causal relationship between
10 PM and effects on materials” (U.S. EPA, 2009, sections 2.5.4 and 9.5.4). Rather than altering our
11 conclusions from the 2012 review, the evidence in the 2019 ISA continues to support prior
12 conclusions regarding materials effects associated with PM deposition. Effects of deposited PM,
13 particularly sulfates and nitrates,²⁶ to materials include both physical damage and impaired
14 aesthetic qualities. Because of their electrolytic, hygroscopic, and acidic properties and their
15 ability to sorb corrosive gases, particles contribute to materials damage by adding to the effects
16 of natural weathering processes, by potentially promoting or accelerating the corrosion of metals,
17 degradation of painted surfaces, deterioration of building materials, and weakening of material
18 components. The majority of the evidence on materials effects of PM are from outside the U.S.
19 on buildings and other items of cultural heritage; however, they provide limited new data for
20 consideration. (U.S. EPA, 2019, section 13.4).

21 Materials damage from PM generally involves one or both of two processes: soiling and
22 corrosion (U.S. EPA, 2019, section 13.4.2). Soiling and corrosion are complex, interdependent
23 processes, typically beginning with deposition of atmospheric PM or SO₂ to exposed surfaces.
24 Constituents of deposited PM can interact directly with materials or undergo further chemical
25 and/or physical transformation to cause soiling, corrosion, and physical damage. Weathering,
26 including exposure to moisture, ultraviolet (UV) radiation and temperature fluctuations, affects
27 the rate and degree of damage (U.S. EPA, 2019, section 13.4.2).

28 Soiling is the result of PM accumulation on an object that alters its optical characteristics
29 or appearance. These soiling effects can affect the aesthetic value of a structure or result in
30 reversible or irreversible damage to the surface. The presence of air pollution can increase the
31 frequency and duration of cleaning and can enhance biodeterioration processes on the surface of
32 materials. For example, deposition of carbonaceous components of PM can lead to the formation

²⁶ In the case of materials effects, it is difficult to isolate the effects of gaseous and particulate nitrogen and sulfur wet deposition so both will be considered along with other PM-related deposition effects on materials.

1 of black crusts on surfaces, and the buildup of microbial biofilms²⁷ can discolor surfaces by
2 trapping PM more efficiently (U.S. EPA, 2009, p. 9-195; U.S. EPA, 2019, section 13.4.2). The
3 presence of PM may alter light transmission or change the reflectivity of a surface. Additionally,
4 the organic or nutrient content of deposited PM may enhance microbial growth on surfaces.

5 Since the 2012 review, very little evidence has become available related to deposition of
6 SO₂ to materials such as limestone, granite, and metal. Deposition of SO₂ onto limestone can
7 transform the limestone into gypsum, resulting in a rougher surface, which allows for increased
8 surface area for accumulation of deposited PM (Camuffo and Bernardi, 1993; U.S. EPA, 2019,
9 section 13.4.2). Oxidation of deposited SO₂ that contributes to the transformation of limestone to
10 gypsum can be enhanced by the formation of surface coatings from deposited carbonaceous PM
11 (both elemental and organic carbon) (Grossi et al., 2007, McAlister et al., 2008). Ozga et al.
12 (2011) characterized damage to two concrete buildings in Poland and Italy. Gypsum was the
13 main damage product on surfaces of these buildings that were sheltered from rain runoff, while
14 PM embedded in the concrete, particularly carbonaceous particles, were responsible for
15 darkening of the building walls (Ozga et al., 2011).

16 Building on the evidence in the 2009 ISA, research has progressed on the theoretical
17 understanding of soiling of cultural heritage in a number of studies. Barca et al. (2010)
18 developed and tested a new methodological approach for characterizing trace elements and
19 heavy metals in black crusts on stone monuments to identify the origin of the chemicals and the
20 relationship between the concentrations of elements in the black crusts and local environmental
21 conditions. Recent research has also used isotope tracers to distinguish between contributions
22 from local sources versus atmospheric pollution to black crusts on historical monuments in
23 France (Kloppmann et al., 2011). A study in Portugal found that biological activity played a
24 major role in soiling, specifically in the development of colored layers and in the detachment
25 process (de Oliveira et al., 2011). Another study found damage to cement renders, often used for
26 restoration, consolidation, and decorative purposes on buildings, following exposure to sulfuric
27 acid, resulting in the formation of gypsum (Lanzon and Garcia-Ruiz, 2010).

28 Corrosion of stone and the decay of stone building materials by acid deposition and
29 sulfate salts were described in the 2009 ISA (U.S. EPA, 2009, section 9.5.3). Since that time,
30 advances have been made on the quantification of degradation rates and further characterization
31 of the factors that influence damage of stone materials (U.S. EPA, 2019, section 13.4.2). Decay
32 rates of marble grave stones were found to be greater in heavily polluted areas compared to a
33 relatively pristine area (Mooers et al., 2016). The time of wetness and the number of

²⁷ Microbial biofilms are communities of microorganisms, which may include bacteria, algae, fungi and lichens, that colonize an inert surface. Microbial biofilms can contribute to biodeterioration of materials via modification of the chemical environment.

1 dissolution/crystallization cycles were identified as hazard indicators for stone materials, with
2 greater hazard during the spring and fall when these indicators are relatively high (Casati et al.,
3 2015).

4 A study examining the corrosion of steel as a function of PM composition and particle
5 size found that changes in the composition of resulting rust gradually changed with particle size
6 (Lau et al., 2008). In a study of damage to metal materials under in Hong Kong, which generally
7 has much higher PM concentrations than those observed in the U.S., Liu et al. (2015) found that
8 iron and steel were corroded by both PM and gaseous pollutants (SO₂ and NO₂), while copper
9 and copper alloys were mainly corroded by gaseous pollutants (SO₂ and O₃) and aluminum and
10 aluminum alloy corrosion was mainly attributed to PM and NO₂.

11 A number of studies have also found materials damage from PM components besides
12 sulfate and black carbon and atmospheric gases besides SO₂. Studies have characterized impacts
13 of nitrates, NO_x, and organic compounds on direct materials damage or on chemical reactions
14 that enhance materials damage (U.S. EPA, 2019, section 13.4.2). Other studies have found that
15 soiling of building materials can be attributed to enhanced biological processes and colonization,
16 including the development and thickening of biofilms, resulting from the deposition of PM
17 components and atmospheric gases (U.S. EPA, 2019, section 13.4.2).

18 Since the 2012 review, other materials have been studied for damage attributable to PM,
19 including glass and photovoltaic panels. Soiling of glass can affect its optical and thermal
20 properties, and can lead to increased cleaning costs and frequency. The development of haze²⁸ on
21 modern glass has been measured and modeled, with a strong correlation between the size
22 distribution of particles and the evolution of the mass deposited on the surface of the glass.
23 Measurements showed that, under sheltered conditions, mass deposition accelerated regularly
24 with time in areas closest to sources of PM (i.e., near roadways) and coarse mineral particles
25 were more prevalent compared to other sites (Alfaro et al., 2012). Model predictions were found
26 to correctly simulate the development of haze at site locations when compared with
27 measurements (Alfaro et al., 2012).

28 Soiling of photovoltaic panels can lead to decreased energy efficiency. For example,
29 soiling by carbonaceous PM decreased solar efficiency by nearly 38%, while soil particles
30 reduced efficiency by almost 70% (Radonjic et al., 2017). The rate of photovoltaic power output
31 can also be degraded by soiling and has been found to be related to the rate of dust accumulation.

²⁸ In this discussion of non-visibility welfare effects (section 5.3.2), haze is used as it has been defined in the scientific literature on soiling of glass, i.e., the ratio of diffuse transmitted light to direct transmitted light (Lombardo et al., 2010). This differs from the definition of haze as used in the discussion of visibility welfare effects in section 5.3.1, where it is used as a qualitative description of the blockage of sunlight by dust, smoke, and pollution.

1 In five sites in the U.S. representing different meteorological and climatological conditions,²⁹
2 photovoltaic module power transmission was reduced by approximately 3% for every g/m² of
3 PM deposited on the cover plate of the photovoltaic panel, independent of geographical location
4 (Boyle et al., 2017). Another study found that photovoltaic module power output was reduced by
5 40% after 10 months of exposure without cleaning, although a number of anti-reflective coatings
6 can generally mitigate power reduction resulting from dust deposition (Walwil et al., 2017).
7 Energy efficiency can also be impacted by the soiling of building materials, such as light-colored
8 marble panels on building exteriors, that are used to reflect a large portion of solar radiation for
9 passive cooling and to counter the urban heat island effect. Exposure to acidic pollutants in urban
10 environments have been found to reduce the solar reflectance of marble, decreasing the cooling
11 effect (Rosso et al., 2016). Highly reflective roofs, or cool roofs, have been designed and
12 constructed to increase reflectance from buildings in urban areas, to both decrease air
13 conditioning needs and urban heat island effects, but these efforts can be impeded by soiling of
14 materials used for constructing cool roofs. Methods have been developed for accelerating the
15 aging process of roofing materials to better characterize the impact of soiling and natural weather
16 on materials used in constructing cool roofs (Sleiman et al., 2014).

- 17 • **To what extent has information emerged for quantifying material damage**
18 **attributable to PM through dose-response relationships or damage functions? Are**
19 **there studies linking perceptions of reduced aesthetic appeal of buildings and other**
20 **objects to PM or wet deposition of nitrogen and sulfur species?**

21 Some progress has been made since the 2012 review in the development of dose-response
22 relationships for soiling of building materials, although some key relationships remain poorly
23 characterized. The first general dose-response relationships for soiling of materials were
24 generated by measuring contrast reflectance of a soiled surface to the reflectance of the unsoiled
25 substrate for different materials, including acrylic house paint, cedar siding, concrete, brick,
26 limestone, asphalt shingles, and window glass with varying total suspended particulate (TSP)
27 concentrations (Beloin and Haynie, 1975; U.S. EPA, 2019, section 13.4.3). Continued efforts to
28 develop dose-response curves for soiling have led to some advancements for modern materials,
29 but these relationships remain poorly characterized for limestone. One study quantified the dose-
30 response relationships between PM₁₀ and soiling for painted steel, white plastic, and
31 polycarbonate filter material, but there was too much scatter in the data to produce a dose-
32 response relationship for limestone (Watt et al., 2008). A dose-response relationship for silica-

²⁹ Of the five sites studied, three were in rural, suburban, and urban areas representing a semi-arid environment (Front Range of Colorado), one site represented a hot and humid environment (Cocoa, Florida), and one represented a hot and arid environment (Albuquerque, New Mexico) (U.S. EPA, 2019, section 13.4.2; Boyle et al., 2017).

1 soda-lime window glass soiling by PM₁₀, NO₂, and SO₂ was quantified based on 31 different
2 locations (Lombardo et al., 2010; U.S. EPA, 2019, section 13.4.3, Figure 13-32, Equation 13-8).
3 The development of this dose-response relationship required several years of observation time
4 and had inconsistent data reporting across the locations.

5 Since the 2012 review, there has also been progress in developing methods to more
6 rapidly evaluate soiling of different materials by PM mixtures. Modern buildings typically have
7 simpler lines, less detailed surfaces, and a greater use of glass, tile, and metal, which are easier to
8 clean than stone. There have also been major changes in the types of materials used for
9 buildings, including a variety of polymers available for use as coatings and sealants. New
10 economic and environmental considerations beyond aesthetic appeal and structural damage are
11 emerging (U.S. EPA, 2019, section 13.4.3). Changes in building materials and design, coupled
12 with new approaches in quantifying the dose-response relationship between PM and materials
13 effects, may reduce the amount of time needed for observations to support the development of
14 material-specific dose-response relationships.

15 In addition to dose-response functions, damage functions have also been used to quantify
16 material decay as a function of pollutant type and load. Damage can be determined from sample
17 surveys or inspection of actual damage and a damage function can be developed to link the rate
18 of material damage to time of replacement or maintenance. A cost function can then link the time
19 for replacement and maintenance to a monetary cost, and an economic function links cost to the
20 dose of pollution based on the dose-response relationship (U.S. EPA, 2019, section 13.4.3).
21 Damage functions are difficult to assess because it depends on human perception of the level of
22 soiling deemed to be acceptable and evidence in this area remains limited. As described in the
23 2019 ISA, damage functions for a wide range of building materials (i.e., stone, aluminum, zinc,
24 copper, plastic, paint, rubber, stone) have been developed and reviewed (Brimblecombe and
25 Grossi, 2010). One study estimated long-term deterioration of building materials and found that
26 damage to durable building material (such as limestone, iron, copper, and discoloration of stone)
27 is no longer controlled by pollution as was historically documented but rather that natural
28 weathering is a more important influence on these materials in modern times (Brimblecombe and
29 Grossi, 2009). Even as PM-attributable damage to stone and metals has decreased over time, it
30 has been predicted that there will be potentially higher degradation rates for polymeric materials,
31 plastic, paint, and rubber due to increased oxidant concentrations and solar radiation
32 (Brimblecombe and Grossi, 2009).

- 33 • **To what extent have important uncertainties identified in prior reviews been**
34 **reduced and/or have new uncertainties emerged?**

35 While there are a number of studies in the 2019 ISA that investigate the effect of PM on
36 newly studied materials and further characterize the effects of PM on previously studied

1 materials, there remains insufficient evidence to relate soiling or damage to specific PM levels or
2 to establish a quantitative relationship between PM in ambient air and materials degradation.
3 Uncertainties that were identified in the 2012 review still largely remain with respect to
4 quantitative relationships between particle size, concentration, chemical concentrations, and
5 frequency of repainting and repair. No new studies are assessed in the 2019 ISA that link
6 perceptions of reduced aesthetic appeal of buildings and other objects to PM-related materials
7 effects. Moreover, uncertainties about the deposition rates of airborne PM to surfaces and the
8 interaction of co-pollutants still remain.

9 **5.3.2.2 Quantitative Information**

10 Beyond our consideration of the scientific evidence, discussed above in section 5.3.2.1
11 **Error! Reference source not found.** above, we also consider the extent to which quantitative
12 analyses of PM air quality and quantitative assessments for climate and materials effects could
13 inform conclusions on the adequacy of the public welfare protection provided by the current
14 secondary PM standards. We have evaluated the potential support for conducting new analyses
15 of PM air quality concentrations and non-visibility welfare effects.

16 **5.3.2.2.1 Climate Effects**

17 While expanded since the 2012 review, our current understanding of PM-related climate
18 effects is still limited by significant uncertainties. Large spatial and temporal heterogeneities in
19 direct and indirect PM climate forcing can occur for a number of reasons, including the
20 frequency and distribution of emissions of key PM components contributing to climate forcing,
21 the chemical and microphysical processing that occurs in the atmosphere, and the atmospheric
22 lifetime of PM relative to other pollutants contributing to climate forcing (U.S. EPA, 2019,
23 section 13.3). These issues particularly introduce uncertainty at the local and regional scales in
24 the U.S. that would likely be most relevant to a quantitative assessment of the potential effects of
25 a national PM standard on climate in this review. Limitations and uncertainties in the evidence
26 make it difficult to quantify the impact of PM on climate and in particular how changes in the
27 level of PM mass in ambient air would result in changes to climate in the U.S. Thus, as in the
28 2012 review, the data remain insufficient to conduct quantitative analyses for PM effects on
29 climate.

30 **5.3.2.2.2 Materials Effects**

31 As at the time of the 2012 review, sufficient evidence is not available to conduct a
32 quantitative assessment of PM-related soiling and corrosion effects. While soiling associated
33 with PM can lead to increased cleaning frequency and repainting of surfaces, no quantitative
34 relationships have been established between characteristics of PM or the frequency of cleaning
35 or repainting that would help inform our understanding of the public welfare implications of

1 soiling (U.S. EPA, 2019, section 13.4). Similarly, while some information is available with
2 regard to microbial deterioration of surfaces and the contribution of carbonaceous PM to the
3 formation of black crusts that contribute to soiling, the available evidence does not support
4 quantitative analyses (U.S. EPA, 2019, section 13.4). While some evidence is available with
5 respect to PM-attributable materials effects, the data are insufficient to conduct quantitative
6 analyses for PM effects on materials.

7 **5.4 PRELIMINARY CONCLUSIONS REGARDING THE ADEQUACY OF** 8 **THE SECONDARY PM STANDARDS**

9 This section discusses preliminary staff conclusions for the Administrator’s consideration
10 in judging the adequacy of the current secondary PM standards. These preliminary conclusions
11 are based on consideration of the assessment and integrative synthesis of evidence presented in
12 the 2019 ISA and draft ISA Supplement, as well as analyses of recent air quality. Taking into
13 consideration the responses to specific questions discussed above, we revisit the overarching
14 policy question for this chapter:

- 15 • **Does the scientific evidence and quantitative information support or call into**
16 **question the adequacy of the protection afforded by the current secondary PM**
17 **standards?**

18 As provided in section 109(b)(2) of the CAA, the secondary standard is to “specify a
19 level of air quality the attainment and maintenance of which in the judgment of the
20 Administrator...is requisite to protect public welfare from any known or anticipated adverse
21 effects associated with the presence of such air pollutant in the ambient air.” Effects on welfare
22 include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials,
23 animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and
24 hazards to transportation, as well as effects on economic values and on personal comfort and
25 well-being” (CAA section 302(h)). The secondary standards are not meant to protect against all
26 known or anticipated PM-related effects, but rather those that are judged to be adverse to the
27 public welfare (78 FR 3212, January 15, 2013). Similarly, the extent to which secondary
28 standards are concluded to provide adequate protection from such effects also depends on
29 judgments by the Administrator.

30 Therefore, we recognize that, as is the case in NAAQS reviews in general, the extent to
31 which the current secondary PM standards are judged to be adequate will depend on a variety of
32 factors and judgments to be made by the Administrator. Such judgments include those
33 concerning the extent or severity of welfare effects that may be considered adverse to the public
34 welfare, and accordingly, what level of protection from such known or anticipated effects may be
35 judged requisite. In general, the public welfare significance of PM-related effects for different air

1 quality conditions and in different locations depend upon the type and severity of the effects, as
2 well as the strength of the underlying information and associated uncertainties. Thus, in the
3 discussion below, our intention is to focus on such aspects of the evidence and quantitative
4 analyses.

5 With regard to visibility, climate, and materials effects of PM, our response to the
6 question above takes into consideration the discussions that address the specific policy-relevant
7 questions in prior sections of this chapter (see sections 5.3.1 and 5.3.2) and the approach
8 described in section 5.2 that builds on the approach from previous reviews. With respect to the
9 evidence-based considerations, we note that the evidence, while somewhat expanded since
10 previous reviews, does not include evidence of effects at lower concentrations or other welfare
11 effects of PM than those identified at the time of prior reviews. There continue to be significant
12 uncertainties related to quantifying the relationships between PM mass concentrations in ambient
13 air and welfare effects, including visibility impairment, climate effects, and materials effects.

14 With respect to the visibility effects of PM, the evidence continues to support a causal
15 relationship. With respect to evidence for visibility effects of PM, we note that the evidence,
16 while somewhat expanded since the 2012 review, does not include evidence of effects at lower
17 concentrations than those identified at the time of the 2012 review. Consistent with the evidence
18 available at the time of the 2012 review, significant limitations remain in directly measuring light
19 extinction. However, a number of small refinements have been made to the algorithm commonly
20 used to estimate light extinction (U.S. EPA, 2019, section 13.2.3.3; section 5.3.1.1 above). Light
21 extinction by PM_{2.5} is dependent on PM_{2.5} composition and relative humidity, which varies
22 regionally, with component contributions to light extinction also changing over time with
23 changes in emissions, as can be seen in analyses of recent air quality. We also note that limited
24 new research is available on methods of characterizing visibility or on how visibility is valued by
25 the public, such as visibility preference studies. Thus, while limited new research has further
26 informed our understanding of the influence of atmospheric components of PM_{2.5} on light
27 extinction, the available evidence to inform consideration of the public welfare implications of
28 PM-related visibility impairment remains relatively unchanged.

29 With respect to quantitative-based considerations, analyses using recent air quality and
30 considering updated and alternative methods for estimating visibility impairment provide results
31 generally similar to those given a focus in the decision for the 2012 and 2020 reviews. We
32 recognize that conclusions reached regarding visibility in previous reviews were based primarily
33 on the quantitative analyses that considered the relationship of estimated visibility impairment
34 (light extinction) with design values for the secondary 24-hour PM_{2.5} standard. These analyses
35 demonstrated that visibility index values were below 30 dv – the value identified as the target
36 level of protection for visibility-related welfare effects – at all locations that met the daily

1 standard. In our evaluation in this chapter, we have considered the information regarding the
2 equations to estimate light extinction and the inputs to the equations and regarding identification
3 of the target level of protection. With regard to the equations, consistent with the approach in the
4 2020 review, we have utilized both the most recently published equations as well as alternatives
5 considered in the 2012 review in recognition of the uncertainties inherent in the quantitative
6 relationship between PM and light extinction and the variability in applicability to different
7 locations. Further, we have considered key coefficients in estimating and adjusting
8 concentrations of specific PM_{2.5} components, a key example of which is the multiplier used to
9 estimate the concentration of organic matter from the concentration of organic carbon. For
10 consistency with the analyses on which the decisions were based in the 2012 and 2020 reviews,
11 we have focused on a 3-year average of the 90th percentile of daily light extinction (calculated
12 using old and new algorithms) in considering visibility impairment at the analyzed locations.

13 In reaching a conclusion in the 2012 and 2020 reviews with regard to the adequacy of
14 visibility protection provided by the secondary PM standards, both Administrators identified 30
15 μv as an appropriate target level of protection. We have not identified new information available
16 since the completion of the 2020 review in this reconsideration of the 2020 final decision that
17 would challenge this public policy. Thus, in our consideration of the current information and
18 analyses in this document, we have compared the results of the updated analyses to the value of
19 30 μv , finding that all sites meet this target level of protection while also meeting the current
20 daily standards. In so finding, we additionally note the uncertainties recognized above regarding
21 estimation of OM for use in the IMPROVE equations, and also the variability across sites in
22 characteristics that affect the relationship between PM in ambient air and light extinction, and in
23 characteristics that affect human visibility and preferences in that regard. Based on the findings
24 of this comparison, in light of all of these considerations, we find it reasonable to conclude that
25 the quantitative information available in this reconsideration of the 2020 final decision does not
26 call into question the adequacy of visibility-related public welfare protection provided by the
27 current secondary PM standards. As a result, we have not conducted additional analyses to
28 evaluate the level of visibility protection that might be afforded by potential alternative
29 standards.

30 With respect to the non-visibility welfare effects of PM, the available evidence continues
31 to support causal relationships between climate effects and PM and materials effects and PM.
32 The evidence related to climate effects and PM, while expanded since previous reviews, has not
33 appreciably improved our understanding of the spatial and temporal heterogeneity of PM
34 components that contribute to climate forcing. We note that, as at the time of the 2012 review,
35 the evidence describes differences among individual PM components in their reflective
36 properties and direction of climate forcing. We also note that, while climate research has

1 continued, there are still significant limitations in our ability to quantify contributions of PM, and
2 of individual PM components, to the direct and indirect effects of PM on climate (e.g. changes to
3 the pattern of rainfall, changes to wind patterns, effects on vertical mixing in the atmosphere).
4 While climate models have been improved and refined since the 2012 review, climate models
5 simulating aerosol-climate interactions on regional scales (e.g., ~100 km) tend to have more
6 variability in estimates of the PM-related climate effects than simulations at the global scale, and
7 fewer studies are available that simulate specific regions (e.g., the U.S.) than that provide global-
8 scale simulations. While recent research has added to the understanding of climate forcing on a
9 global scale, there remain significant limitations to quantifying potential adverse effects from
10 PM on climate in the U.S. and how they would vary in response to changes in PM concentrations
11 in the U.S. That is, the information with regard to climate does not provide a clear understanding
12 of a quantitative relationship between concentrations of PM mass in ambient air and associated
13 climate-related effects, and consequently, precludes a quantitative evaluation of the level of
14 protection provided by a PM concentration-based secondary standard from adverse climate-
15 related effects on the public welfare in the U.S. Thus, on the whole, we do not find the
16 information to provide support for different conclusions than were reached in the 2012 and 2020
17 reviews with regard to climate-related effects of PM in ambient air.

18 In considering the evidence related to materials effects and PM, we note that there is
19 some evidence that informs our understanding on the soiling process and types of materials
20 affected, and provides limited information on dose-response relationships and damage functions,
21 although most of the recent evidence comes from studies outside of the U.S. In particular, there
22 is a growing body of research on PM and energy efficiency-related materials, such as solar
23 panels and passive cooling building materials, affecting the optical and thermal properties,
24 thereby impacting the intended energy efficiency of these materials. While recent research has
25 added to the understanding of PM-related materials effects, there remains a lack of research
26 related to quantifying materials effects and understanding the public welfare implications of such
27 effects.

28 In summary, with regard to the two main non-visibility effects – climate effects and
29 materials effects – the available evidence, as in previous reviews, documents a causal role for
30 PM in ambient air. This evidence, however, as in the 2012 and 2020 reviews, also includes
31 substantial uncertainties with regard to quantitative relationships with PM concentrations and
32 concentration patterns that limit our ability to quantitatively assess the public welfare protection
33 provided by the standards from these effects. Thus, as a whole, the available information does
34 not call into question the adequacy of protection provided by the current standards for these
35 effects.

1 Based on all of the above considerations, we find that the available evidence does not call
2 into question the protection afforded by the current secondary PM standards against PM-related
3 welfare effects. Thus, our preliminary conclusion for the Administrator’s consideration is that it
4 is appropriate to consider retaining the current secondary PM standards, without revision. In so
5 concluding, we recognize, as noted above, that the final decision on this reconsideration of the
6 secondary PM standards to be made by the Administrator is largely a public welfare judgment,
7 based on his judgment as to the requisite protection of the public welfare from any known or
8 anticipated adverse effects. This final decision will draw upon the available scientific evidence
9 and quantitative analyses on PM-attributable welfare effects, and on judgments about the
10 appropriate weight to place on the range of uncertainties inherent in the evidence and analyses.

11 **5.5 AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

12 In this section, we highlight key uncertainties in the available information related to the
13 effects of PM on public welfare. Such key uncertainties and areas for future research, model
14 development, and data gathering are outlined below. We note, however, that a full set of research
15 recommendations is beyond the scope of this discussion. Rather, listed below are key
16 uncertainties, research questions and data gaps that have been thus far highlighted in this review
17 of the secondary PM standards.

- 18 • A critical aspect of our consideration of the evidence and quantitative information for
19 visibility impairment is our understanding of human perception of visibility impairment
20 in the preference studies. This is essential to the Administrator’s consideration of the
21 public welfare implications of visibility effects and to decisions on the adequacy of
22 protection provided by the secondary PM standards from them. Additional information
23 related to several areas would reduce uncertainty in in our interpretation of the available
24 information for purposes of characterizing visibility impairment. These areas include the
25 following:
 - 26 – Expanding the number and geographic coverage of preference studies in urban,
27 rural and Class I areas to account for the potential for people to have different
28 preferences based on the conditions that they commonly encounter and potential
29 differences in preferences based on the scene types;
 - 30 – Evaluating visibility preferences of the U.S. population today, given that the
31 preference studies were conducted more than 15 years ago, during which time air
32 quality in the U.S. has improved;
 - 33 – Accounting for the influence that varying study methods may have on an
34 individual’s response as to what level of visibility impairment is acceptable; and
 - 35 – Providing insights regarding people’s judgments on acceptable visibility based on
36 those factors that can influence an individual’s perception of visibility
37 impairment, including the duration of visibility impairment experiences, the time
38 of day during which light extinction is greatest, and the frequency of episodes of
39 visibility impairment, as well as the intensity of the visibility impairment.

- 1 • The development and implementation of direct monitoring of PM_{2.5} light extinction would
2 help to characterize visibility and the relationships between PM component
3 concentrations and light extinction and to evaluate and refine light extinction calculation
4 algorithms for use in areas near anthropogenic sources, and would provide measurements
5 for future visibility effects assessments.
- 6 • Substantial uncertainties still remain with respect to key processes linking PM and
7 climate, because of the small scale of PM-relevant atmospheric processes compared to
8 the resolution of state-of-the-art models, and because of the complex cascade of indirect
9 impacts and feedbacks in the climate system that result from an initial PM-related
10 radiative perturbation. Such uncertainties include those related to our understanding of:
- 11 – The magnitude of PM radiative forcing and the portion of that associated with
12 anthropogenic emissions;
 - 13 – The contribution of regional differences in PM concentrations, and of individual
14 components, to radiative forcing; and,
 - 15 – The process by which PM interacts with clouds and how to represent such
16 interactions in climate models.
- 17 • Research on more accurate U.S. and global emission inventories would provide source-
18 specific data on PM and PM component contributions to climate effects, particularly
19 those effects resulting from climate forcing.
- 20 • Insufficient evidence is available to relate soiling or damage to specific PM
21 concentrations or to establish a quantitative relationship between PM concentrations in
22 ambient air and materials degradation. Additional information would reduce uncertainty
23 in our interpretation of the available information, including in the following areas:
- 24 – Identifying quantitative relationships between particle size, PM concentration,
25 chemical concentrations, and frequency of repainting and repair;
 - 26 – Understanding human perceptions of reduced aesthetic appeal of buildings, and
27 other objects to PM-related materials effects; and
 - 28 – Characterizing deposition rates of airborne PM to surfaces and the interaction of
29 co-pollutants.

1 **REFERENCES**

- 2 Abt Associates, Inc. (2001). Assessing public opinions on visibility impairment due to air
3 pollution: Summary report. U.S. Environmental Protection Agency. Research Triangle
4 Park, NC.
- 5 Adams, PJ, Seinfeld, JH, Koch, D, Mickley, L and Jacob, D (2001). General circulation model
6 assessment of direct radiative forcing by the sulfate-nitrate-ammonium-water inorganic
7 aerosol system. *Journal of Geophysical Research* 106(D1): 1097-1111.
- 8 Alfaro, SC, Chabas, A, Lombardo, T, Verney-Carron, A and Auset, P (2012). Predicting the
9 soiling of modern glass in urban environments: A new physically-based model.
10 *Atmospheric Environment* 60: 348-357.
- 11 Ban-Weiss, GA, Jin, L, Bauer, SE, Bennartz, R, Liu, X, Zhang, K, Ming, Yi, Guo, H and Jiang,
12 JH (2014). Evaluating clouds, aerosols, and their interactions in three global climate
13 models using satellite simulators and observations. *Journal of Geophysical Research:*
14 *Atmospheres* 119(18): 10876-10901.
- 15 Barca, D, Belfiore, CM, Crisci, GM, La Russa, MF, Pezzino, A and Ruffolo, SA (2010).
16 Application of laser ablation ICP-MS and traditional techniques to the study of black
17 crusts on building stones: a new methodological approach. *Environmental Science and*
18 *Pollution Research* 17(8): 1433-1447.
- 19 BBC Research & Consulting (2003). Phoenix area visibility survey. Denver, CO.
20 http://www.azdeq.gov/environ/air/download/vis_021903f.pdf.
- 21 Beloin, NJ and Haynie, FH (1975). Soiling of building materials. *Journal of the Air and Waste*
22 *Management Association* 25(4): 399-403.
- 23 Bond, TC, Doherty, SJ, Fahey, DW, Forster, PM, Berntsen, T, Deangelo, BJ, Flanner, MG,
24 Ghan, S, Kärcher, B, Koch, D, Kinne, S, Kondo, Y, Quinn, PK, Sarofim, MC, Schultz,
25 MG, Schulz, M, Venkataraman, C, Zhang, H, Zhang, S, Bellouin, N, Guttikunda, SK,
26 Hopke, PK, Jacobson, MZ, Kaiser, JW, Klimont, Z, Lohmann, U, Schwarz, JP, Shindell,
27 D, Storelvmo, T, Warren, SG and Zender, CS (2013). Bounding the role of black carbon
28 in the climate system: A scientific assessment. *Journal of Geophysical Research:*
29 *Atmospheres* 118(11): 5380-5552.
- 30 Boucher, O (2013). *Clouds and Aerosols. In Climate Change 2013: The Physical Science Basis.*
31 *Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental*
32 *Panel on Climate Change.* Cambridge University Press. Cambridge, United Kingdom
33 and New York, NY, USA.
- 34 Boyle, L, Burton, PD, Danner, V, Hannigan, MP and King, B (2017). Regional and national
35 scale spatial variability of photovoltaic cover plate soiling and subsequent solar
36 transmission losses. *7(5): 1354-1361.*

- 1 Brimblecombe, P and Grossi, CM (2009). Millennium-long damage to building materials in
2 London. *Science of the Total Environment* 407(4): 1354-1361.
- 3 Brimblecombe, P and Grossi, CM (2010). Potential damage to modern building materials from
4 21st century air pollution. *The Scientific World Journal* 10: 116-125.
- 5 Camuffo, D and Bernardi, A (1993). Microclimatic factors affecting the trajan column. *Science*
6 *of the Total Environment* 128(2-3): 227-255.
- 7 Casati, M, Rovelli, G, D'Angelo, L, Perrone, MG, Sangiorgi, G, Bolzacchini, E and Ferrero, L
8 (2015). Experimental measurements of particulate matter deliquescence and
9 crystallization relative humidity: Application in heritage climatology. *Aerosol and Air*
10 *Quality Research* 15(2): 399-409.
- 11 de Oliveira, BP, de la Rosa, JM, Miller, AZ, Saiz-Jimenez, C, Gomez-Bolea, A, Braga, MAS and
12 Dionisio, A (2011). An integrated approach to assess the origins of black films on a
13 granite monument. *Environmental Earth Sciences* 63(6-7 SI): 1677-1690.
- 14 Ely, DW, Leary, JT, Stewart, TR and Ross, DM (1991). *The establishment of the Denver*
15 *Visibility Standard*. Colorado Department of Health. Denver, Colorado.
- 16 Fajardo, OA, Jiang, J and Hao, J (2013). Assessing young people's preferences in urban visibility
17 in beijing. *Aerosol and Air Quality Research* 13(5): 1536-1543.
- 18 Fiore, AM, Naik, V and Leibensperger, EM (2015). Air quality and climate connections. *Journal*
19 *of the Air and Waste Management Association* 65(6): 645-685.
- 20 Grossi, CM, Brimblecombe, P, Esbert, RM and Javier Alonso, F (2007). Color changes in
21 architectural limestones from pollution and cleaning. *Color Research and Application*
22 32(4): 320-331.
- 23 Hand, JL, Copeland, SA, Day, DA, Dillner, AM, Indresand, H, Malm, WC, McDade, CE,
24 Moore, CT, Jr., Pitchford, ML, Schichtel, BA and Watson, JG (2011). Spatial and
25 seasonal patterns and temporal variability of haze and its constituents in the United
26 States, IMPROVE Report V. Colorado State University. Fort Collins, CO.
- 27 Hand, JL, Prenni, AJ, Copeland, S, Schichtel, BA and Malm, WC (2020). Thirty years of the
28 Clean Air Act Amendments: Impacts on haze in remote regions of the United States
29 (1990–2018). *Atmospheric Environment* 243: 117865.
- 30 Hauglustaine, DA, Balkanski, Y and Schulz, M (2014). A global model simulation of present
31 and future nitrate aerosols and their direct radiative forcing of climate. *Atmospheric*
32 *Chemistry and Physics* 14(20): 11031-11063.
- 33 Heald, CL, Ridley, DA, Kreidenweis, SM and Drury, EE (2010). Satellite observations cap the
34 atmospheric organic aerosol budget. *Geophysical Research Letters* 37.

- 1 IPCC (2013). Climate change 2013: The physical science basis. Contribution of working group I
2 to the fifth assessment report of the Intergovernmental Panel on Climate Change. T. F.
3 Stocker, D. Qin, G. K. Plattner et al. Cambridge University Press. Cambridge, UK.
- 4 Jimenez, JL, Canagaratna, MR, Donahue, NM, Prevot, AS, Zhang, Q, Kroll, JH, Decarlo, PF,
5 Allan, JD, Coe, H, Ng, NL, Aiken, AC, Docherty, KS, Ulbrich, IM, Grieshop, AP,
6 Robinson, AL, Duplissy, J, Smith, JD, Wilson, KR, Lanz, VA, Hueglin, C, Sun, YL,
7 Tian, J, Laaksonen, A, Raatikainen, T, Rautiainen, J, Vaattovaara, P, Ehn, M, Kulmala,
8 M, Tomlinson, JM, Collins, DR, Cubison, MJ, Dunlea, EJ, Huffman, JA, Onasch, TB,
9 Alfarra, MR, Williams, PI, Bower, K, Kondo, Y, Schneider, J, Drewnick, F, Borrmann,
10 S, Weimer, S, Demerjian, K, Salcedo, D, Cottrell, L, Griffin, R, Takami, A, Miyoshi, T,
11 Hatakeyama, S, Shimojo, A, Sun, JY, Zhang, YM, Dzepina, K, Kimmel, JR, Sueper, D,
12 Jayne, JT, Herndon, SC, Trimborn, AM, Williams, LR, Wood, EC, Middlebrook, AM,
13 Kolb, CE, Baltensperger, U and Worsnop, DR (2009). Evolution of organic aerosols in
14 the atmosphere. *Science* 326(5959): 1525-1529.
- 15 Kelly, J, Schmidt, M, Frank, N, Timin, B, Solomon, D and Venkatesh, R. (2012). Memorandum
16 to PM NAAQS Review Docket (EPA-HQ-OAR-2007-0492). Technical Analyses to
17 Support Surrogacy Policy for Proposed Secondary PM_{2.5} NAAQS under NSR/PSD
18 Programs. June 14, 2012. Docket ID No. EPA-HQ-OAR-2007-0492. Office of Air
19 Quality Planning and Standards Research Triangle Park, NC. Available at:
20 <https://www3.epa.gov/ttn/naaqs/standards/pm/data/20120614Kelly.pdf>.
- 21 Kleinman, LI, Sedlacek III, AJ, Adachi, K, Buseck, PR, Collier, S, Dubey, MK, Hodshire, AL,
22 Lewis, E, Onasch, TB, Pierce, JR, Shilling, J, Springston, SR, Wang, J, Zhang, Q, Zhou,
23 S and Yokelson, RJ (2020). Rapid evolution of aerosol particles and their optical
24 properties downwind of wildfires in the western US. *Atmospheric Chemistry and Physics*
25 20(21): 13319-13341.
- 26 Klopman, W, Bromblet, P, Vallet, JM, Verges-Belmin, V, Rolland, O, Guerrot, C and
27 Gosselin, C (2011). Building materials as intrinsic sources of sulphate: A hidden face of
28 salt weathering of historical monuments investigated through multi-isotope tracing (B, O,
29 S). *Science of the Total Environment* 409(9): 1658-1669.
- 30 Kok, JF, Ridley, DA, Zhou, Q, Miller, RL, Zhao, C, Heald, CL, Ward, DS, Albani, S and
31 Haustein, K (2017). Smaller desert dust cooling effect estimated from analysis of dust
32 size and abundance. *Nature Geoscience* 10(4): 274-278.
- 33 Laing, JR, Jaffe, DA and Hee, JR (2016). Physical and optical properties of aged biomass
34 burning aerosol from wildfires in Siberia and the Western USA at the Mt. Bachelor
35 Observatory. *Atmospheric Chemistry and Physics* 16(23): 15185-15197.
- 36 Lanzon, M and Garcia-Ruiz, PA (2010). Deterioration and damage evaluation of rendering
37 mortars exposed to sulphuric acid. *Materials and Structures* 43(3): 417-427.
- 38 Lau, NT, Chan, CK, Chan, L and Fang, M (2008). A microscopic study of the effects of particle
39 size and composition of atmospheric aerosols on the corrosion of mild steel. *Corrosion*
40 *Science* 50(10): 2927-2933.

- 1 Lee, YH, Lamarque, JF, Flanner, MG, Jiao, C, Shindell, DT, Berntsen, T, Bisiaux, MM, Cao, J,
2 Collins, WJ, Curran, M, Edwards, R, Faluvegi, G, Ghan, S, Horowitz, LW, McConnell,
3 JR, Ming, J, Myhre, G, Nagashima, T, Naik, V, Rumbold, ST, Skeie, RB, Sudo, K,
4 Takemura, T, Thevenon, F, Xu, B and Yoon, JH (2013). Evaluation of preindustrial to
5 present-day black carbon and its albedo forcing from Atmospheric Chemistry and
6 Climate Model Intercomparison Project (ACCMIP). *Atmospheric Chemistry and Physics*
7 13(5): 2607-2634.
- 8 Leibensperger, EM, Mickley, LJ, Jacob, DJ, Chen, WT, Seinfeld, JH, Nenes, A, Adams, PJ,
9 Streets, DG, Kumar, N and Rind, D (2012). Climatic effects of 1950-2050 changes in US
10 anthropogenic aerosols - Part 2: Climate response. *Atmospheric Chemistry and Physics*
11 12(7): 3349-3362.
- 12 Levy, H, Horowitz, LW, Schwarzkopf, MD, Ming, Yi, Golaz, JC, Naik, V and Ramaswamy, V
13 (2013). The roles of aerosol direct and indirect effects in past and future climate change.
14 *Journal of Geophysical Research: Atmospheres* 118(10): 4521-4532.
- 15 Liu, B, Wang, DW, Guo, H, Ling, ZH and Cheung, K (2015). Metallic corrosion in the polluted
16 urban atmosphere of Hong Kong. *Environmental Monitoring and Assessment* 187(1):
17 4112.
- 18 Lombardo, T, Ionescu, A, Chabas, A, Lefevre, RA, Ausset, P and Candau, Y (2010). Dose-
19 response function for the soiling of silica-soda-lime glass due to dry deposition. *Science*
20 *of the Total Environment* 408(4): 976-984.
- 21 Lowenthal, DH and Kumar, N (2004). Variation of mass scattering efficiencies in IMPROVE.
22 *Journal of the Air and Waste Management Association* (1990-1992) 54(8): 926-934.
- 23 Lowenthal, DH and Kumar, N (2016). Evaluation of the IMPROVE Equation for estimating
24 aerosol light extinction. *Journal of the Air and Waste Management Association* 66(7):
25 726-737.
- 26 Malm, WC and Hand, JL (2007). An examination of the physical and optical properties of
27 aerosols collected in the IMPROVE program. *Atmospheric Environment* 41(16): 3407-
28 3427.
- 29 Malm, WC, Schichtel, B, Molenaar, J, Prenni, A and Peters, M (2019). Which visibility indicators
30 best represent a population's preference for a level of visual air quality? *Journal of the*
31 *Air & Waste Management Association* 69(2): 145-161.
- 32 Malm, WC, Sisler, JF, Huffman, D, Eldred, RA and Cahill, TA (1994). Spatial and seasonal
33 trends in particle concentration and optical extinction in the United States. *Journal of*
34 *Geophysical Research* 99(D1): 1347-1370.
- 35 McAlister, J, Smith, BJ and Torok, A (2008). Transition metals and water-soluble ions in
36 deposits on a building and their potential catalysis of stone decay. *Atmospheric*
37 *Environment* 42(33): 7657-7668.

- 1 McNeill, VF, Woo, JL, Kim, DD, Schwier, AN, Wannell, NJ, Sumner, AJ and Barakat, JM
2 (2012). Aqueous-phase secondary organic aerosol and organosulfate formation in
3 atmospheric aerosols: a modeling study. *Environmental Science and Technology* 46(15):
4 8075-8081.
- 5 Mie, G (1908). Beitrage zur Optik truber Medien, speziell kolloidaler Metallosungen [Optics of
6 cloudy media, especially colloidal metal solutions]. *Annalen der Physik* 25(3): 377-445.
- 7 Mooers, HD, Cota-Guertin, AR, Regal, RR, Sames, AR, Dekan, AJ and Henkels, LM (2016). A
8 120-year record of the spatial and temporal distribution of gravestone decay and acid
9 deposition. *Atmospheric Environment* 127: 139-154.
- 10 Myhre, G, Shindell, D, Bréon, FM, Collins, W, Fuglestvedt, J, Huang, J, Koch, D, Lamarque, JF,
11 Lee, D, Mendoza, B, Nakajima, T, Robock, A, Stephens, G, Takemura, T and Zhang, H,
12 Eds. (2013). *Anthropogenic and natural radiative forcing*. Cambridge University Press
13 Cambridge, UK.
- 14 Ozga, I, Bonazza, A, Bernardi, E, Tittarelli, F, Favoni, O, Ghedini, N, Morselli, L and Sabbioni,
15 C (2011). Diagnosis of surface damage induced by air pollution on 20th-century concrete
16 buildings. *Atmospheric Environment* 45(28): 4986-4995.
- 17 Pitchford, M, Maim, W, Schichtel, B, Kumar, N, Lowenthal, D and Hand, J (2007). Revised
18 algorithm for estimating light extinction from IMPROVE particle speciation data. *Journal*
19 *of the Air and Waste Management Association* 57(11): 1326-1336.
- 20 Prenni, AJ, Hand, JL, Malm, WC, Copeland, S, Luo, G, Yu, F, Taylor, N, Russell, LM and
21 Schichtel, BA (2019). An examination of the algorithm for estimating light extinction
22 from IMPROVE particle speciation data. *Atmospheric Environment* 214: 116880.
- 23 Pryor, SC (1996). Assessing public perception of visibility for standard setting exercises.
24 *Atmospheric Environment* 30(15): 2705-2716.
- 25 Radonjic, IS, Pavlovic, TM, Mirjanic, DLj, Radovic, MK, Milosavljevic, DD and Pantic, LS
26 (2017). Investigation of the impact of atmospheric pollutants on solar module energy
27 efficiency. *Thermal Science* 21(5): 2021-2030.
- 28 Rosso, F, Pisello, AL, Jin, WH, Ghandehari, M, Cotana, F and Ferrero, M (2016). Cool marble
29 building envelopes: The effect of aging on energy performance and aesthetics.
30 *Sustainability* 8(8): Article #753.
- 31 Ryan, PA, Lowenthal, D and Kumar, N (2005). Improved light extinction reconstruction in
32 interagency monitoring of protected visual environments. *Journal of the Air and Waste*
33 *Management Association* 55(11): 1751-1759.
- 34 Scott, CE, Rap, A, Spracklen, DV, Forster, PM, Carslaw, KS, Mann, GW, Pringle, KJ, Kivekas,
35 N, Kulmala, M, Lihavainen, H and Tunved, P (2014). The direct and indirect radiative
36 effects of biogenic secondary organic aerosol. *Atmospheric Chemistry and Physics* 14(1):
37 447-470.

- 1 Shindell, DT, Lamarque, JF, Schulz, M, Flanner, M, Jiao, C, Chin, M, Young, PJ, Lee, YH,
2 Rotstayn, L, Mahowald, N, Milly, G, Faluvegi, G, Balkanski, Y, Collins, WJ, Conley,
3 AJ, Dalsoren, S, Easter, R, Ghan, S, Horowitz, L, Liu, X, Myhre, G, Nagashima, T, Naik,
4 V, Rumbold, ST, Skeie, R, Sudo, K, Szopa, S, Takemura, T, Voulgarakis, A, Yoon, JH
5 and Lo, F (2013). Radiative forcing in the ACCMIP historical and future climate
6 simulations. *Atmospheric Chemistry and Physics* 13(6): 2939-2974.
- 7 Sleiman, M, Kirchstetter, TW, Berdahl, P, Gilbert, HE, Quelen, S, Marlot, L, Preble, CV, Chen,
8 S, Montalbano, A, Rosseler, O, Akbari, H, Levinson, R and Destailats, H (2014). Soiling
9 of building envelope surfaces and its effect on solar reflectance - Part II: Development of
10 an accelerated aging method for roofing materials. *Solar Energy Materials and Solar*
11 *Cells* 122: 271-281.
- 12 Smith, AE and Howell, S (2009). An assessment of the robustness of visual air quality
13 preference study results. CRA International. Washington, DC.
14 [http://yosemite.epa.gov/sab/sabproduct.nsf/B55911DF9796E5E385257592006FB737/\\$File/CRA+VAQ+Pref+Robustness+Study+3+30+09+final.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/B55911DF9796E5E385257592006FB737/$File/CRA+VAQ+Pref+Robustness+Study+3+30+09+final.pdf).
15
- 16 Tai, APK, Mickley, LJ and Jacob, DJ (2010). Correlations between fine particulate matter
17 (PM_{2.5}) and meteorological variables in the United States: Implications for the sensitivity
18 of PM_{2.5} to climate change. *Atmospheric Environment* 44(32): 3976-3984.
- 19 Takemura, T (2012). Distributions and climate effects of atmospheric aerosols from the
20 preindustrial era to 2100 along Representative Concentration Pathways (RCPs) simulated
21 using the global aerosol model SPRINTARS. *Atmospheric Chemistry and Physics*
22 12(23): 11555-11572.
- 23 Twomey, S (1977). The influence of pollution on the shortwave albedo of clouds. *Journal of the*
24 *Atmospheric Sciences* 34(7): 1149-1152.
- 25 U.S. EPA (2004a). Air Quality Criteria for Particulate Matter. (Vol II of II). Office of Research
26 and Development. Research Triangle Park, NC. U.S. EPA. EPA-600/P-99-002bF.
27 October 2004. Available at:
28 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100LG7Q.txt>.
- 29 U.S. EPA (2004b). Air Quality Criteria for Particulate Matter. (Vol I of II). Office of Research
30 and Development. Research Triangle Park, NC. U.S. EPA. EPA-600/P-99-002aF.
31 October 2004. Available at:
32 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100LFIQ.txt>.
- 33 U.S. EPA (2009). Integrated Science Assessment for Particulate Matter (Final Report). Office of
34 Research and Development, National Center for Environmental Assessment. Research
35 Triangle Park, NC. U.S. EPA. EPA-600/R-08-139F. December 2009. Available at:
36 <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546>.
- 37 U.S. EPA (2010). Particulate Matter Urban-Focused Visibility Assessment (Final Document).
38 Office of Air Quality Planning and Standards, Health and Environmental Impacts

- 1 Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-10-004. July 2010.
2 Available at: <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FO5D.txt>.
- 3 U.S. EPA (2011). Policy Assessment for the Review of the Particulate Matter National Ambient
4 Air Quality Standards. Office of Air Quality Planning and Standards, Health and
5 Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-
6 11-003. April 2011. Available at:
7 <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100AUMY.txt>.
- 8 U.S. EPA (2012a). Responses to Significant Comments on the 2012 Proposed Rule on the
9 National Ambient Air Quality Standards for Particulate Matter (June 29, 2012; 77 FR
10 38890). Research Triangle Park, NC. U.S. EPA. Docket ID No. EPA-HQ-OAR-2007-
11 0492. Available at: <https://www3.epa.gov/ttn/naaqs/standards/pm/data/20121214rtc.pdf>.
- 12 U.S. EPA (2012b). Report to Congress on Black Carbon. U.S. Environmental Protection
13 Agency, Office of Air and Radition. Washington, DC. U.S. EPA. EPA-450/R-12-001.
14 March 2012. Availble at: <http://www.epa.gov/blackcarbon/2012report/fullreport.pdf>.
- 15 U.S. EPA (2015). Preamble to the integrated science assessments. U.S. Environmental Protection
16 Agency, Office of Research and Development, National Center for Environmental
17 Assessment, RTP Division. Research Triangle Park, NC. U.S. EPA. EPA/600/R-15/067.
18 November 2015. Available at:
19 <https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=310244>.
- 20 U.S. EPA (2016). Integrated review plan for the national ambient air quality standards for
21 particulate matter. Office of Air Quality Planning and Standards. Research Triangle Park,
22 NC. U.S. EPA. EPA-452/R-16-005. December 2016. Available at:
23 [https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-](https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf)
24 [plan.pdf](https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review-plan.pdf).
- 25 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
26 U.S. Environmental Protection Agency, Office of Research and Development, National
27 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
28 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
29 [standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 30 U.S. EPA (2020). Policy Assessment for the Review of the National Ambient Air Quality
31 Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health
32 and Environmental Impacts Division. Research Triangle Park, NC. U.S. EPA. EPA-
33 452/R-20-002. January 2020. Available at: [https://www.epa.gov/sites/default/files/2020-](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf)
34 [01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf](https://www.epa.gov/sites/default/files/2020-01/documents/final_policy_assessment_for_the_review_of_the_pm_naaqs_01-2020.pdf).
- 35 U.S. EPA (2021). Supplement to the 2019 Integrated Science Assessment for Particulate Matter
36 (External Review Draft). U.S. Environmental Protection Agency, Office of Research and
37 Development, Center for Public Health and Environmental Assessment. Washington,
38 DC. U.S. EPA. EPA/600/R-21/198. December 2019. Available at:
39 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
40 [assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).

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

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

5 Watt, J, Jarrett, D and Hamilton, R (2008). Dose-response functions for the soiling of heritage
6 materials due to air pollution exposure. *Science of the Total Environment* 400(1-3): 415-
7 424.

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

11

APPENDIX A. SUPPLEMENTAL INFORMATION ON PM AIR QUALITY ANALYSES

This appendix provides supplemental information on the data sources and methods used to generate the figures and table presented in Chapter 2 of this draft PA. Sections A.1 to A.4 describe the data sources and methods used to generate figures and tables in section 2.3.2. Section A.5 describes the data sources and methods used to generate figures and tables in section 2.3.3. Section A.6 describes the data sources and methods used to generate figures and tables in section 2.4. Section A.7 described the methods used for the comparison on PM_{2.5} fields in estimating exposure and relative to design values.

A.1 DATA SOURCES AND METHODS FOR GENERATING NATIONAL PM_{2.5}, PM₁₀, PM_{10-2.5}, AND PM_{2.5} SPECIATION FIGURES

- PM_{2.5} annual average and 98th percentile mass concentrations: calculated from regulatory-quality (Federal Reference Method or Federal Equivalent Method) 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98th percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2019 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM₁₀ annual average and 98th percentile mass concentrations: calculated from regulatory-quality (Federal Reference Method or Federal Equivalent Method) 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98th percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2019 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM_{10-2.5} annual average and 98th percentile mass concentrations: calculated from both regulatory and non-regulatory methods using 24-hour average values from monitors with at least 75% completeness for each year. When a single site has multiple monitors, the figure shows the average of the annual averages and 98th percentiles from each monitor at the site. We downloaded the monitor-level concentrations for all sites in the United States for all available days (including potential exceptional events) for 2000-2019 from the EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)
- PM_{2.5} speciated annual average mass concentrations: calculated from filter-based, 24-hour averages from monitors with at least 75% completeness for each year. We downloaded data from monitors that are part of the Interagency Monitoring of Protected Visual

1 Environments (IMPROVE) network, Chemical Speciation Network (CSN), and the
2 NCore Multipollutant Monitoring Network for 2017-2019.

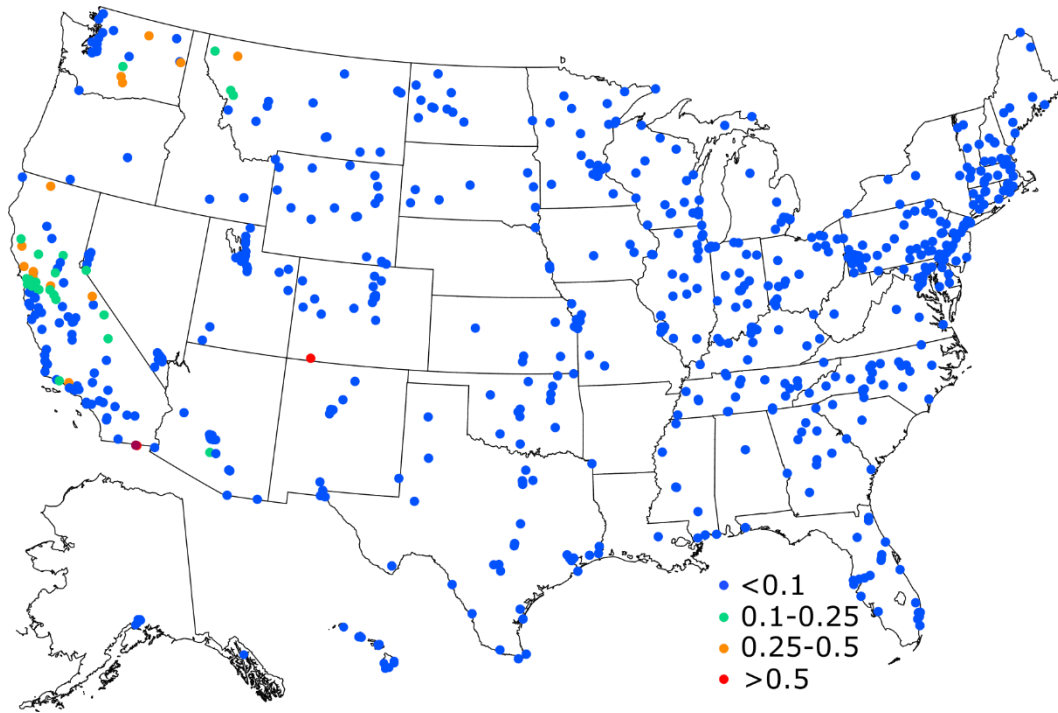
- 3 • The 2000-2019 trends are calculated from the Pearson correlation coefficient for monitors
4 having at least 75% of the available years with 75% completeness within each year.
5 When a single site has multiple monitors, the average of the annual averages and 98th
6 percentiles from each monitor at the site is taken prior to calculation of the Pearson
7 correlation coefficient.

8 **A.2 DATA SOURCES AND METHODS FOR GENERATING NEAR- 9 ROAD PM_{2.5} DESIGN VALUE TABLE AND INCREMENT FIGURES**

- 10 • PM_{2.5} design values: calculated using the data handling described by 40 CFR Appendix N
11 to Part 50 - Interpretation of the National Ambient Air Quality Standards for PM_{2.5}. We
12 downloaded the design values for all sites in the United States for all available days
13 (including potential exceptional events) for 2017-2019 from the EPA's Air Quality
14 System (AQS, <https://www.epa.gov/aqs>)
- 15 • PM_{2.5} hourly, daily, and annual average mass concentrations: calculated from regulatory-
16 quality (Federal Reference Method or Federal Equivalent Method) monitors. When a
17 single site has multiple monitors, the figures show the average from all monitors at the
18 site. We downloaded the monitor-level concentrations for all sites in the United States
19 for all available days (including potential exceptional events) for 2000-2019 from the
20 EPA's Air Quality System (AQS, <https://www.epa.gov/aqs>)

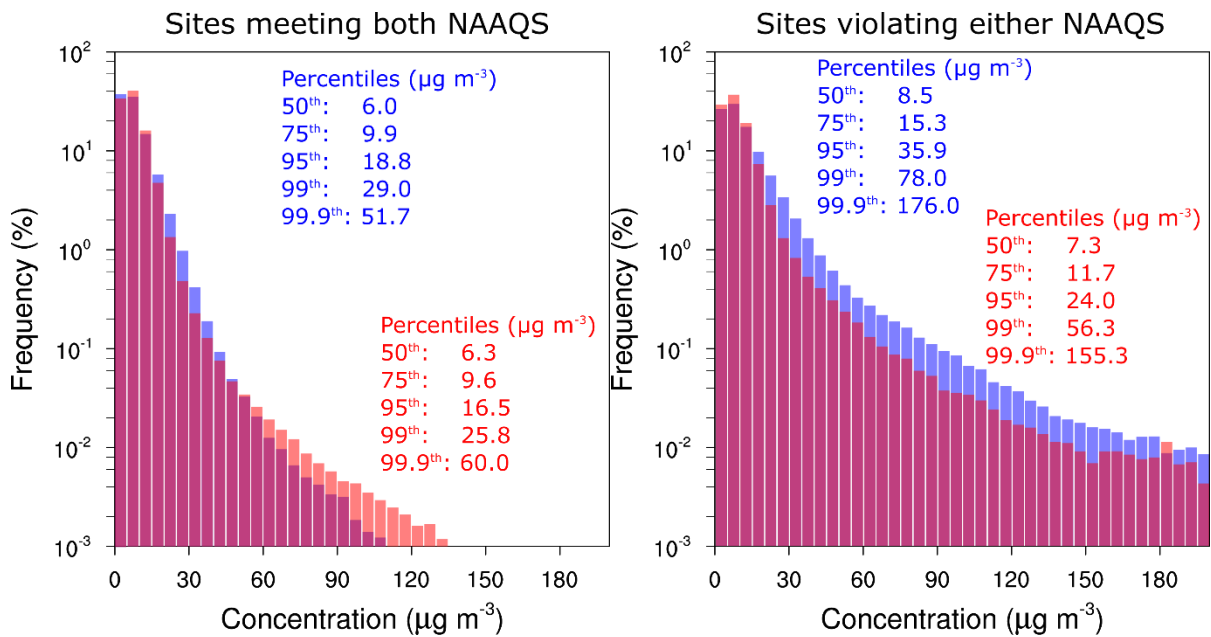
21 **A.3 DATA SOURCES FOR SUB-DAILY PM_{2.5} CONCENTRATION 22 FIGURE**

- 23 • PM_{2.5} hourly average mass concentrations: calculated from regulatory-quality Federal
24 Equivalent Method monitors. The 2-hour and 5-hour averages were calculated for periods
25 with each hourly average available. Only sites with a valid annual or 24-hour design
26 value for 2017-2019 are shown in the figure. The percentages of 2-hour average PM_{2.5}
27 mass concentrations above 140 µg/m³ at individual sites are illustrated in Figure A-1.
28 Frequency distributions of 5-hour averages are presented in Figure A-2.



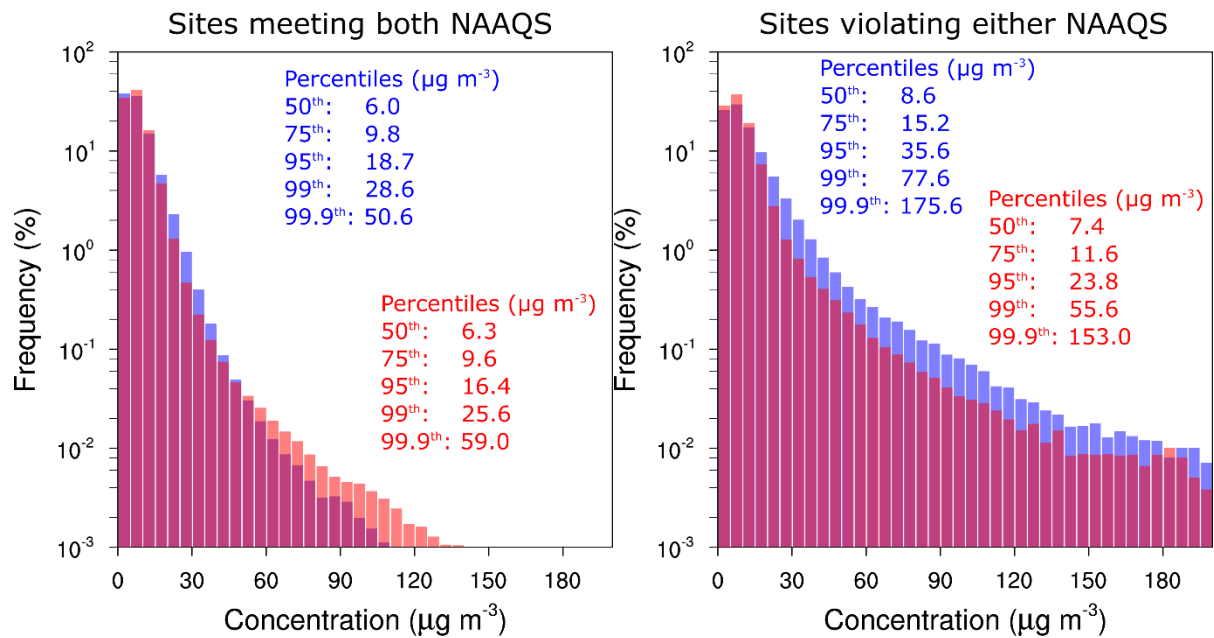
1
2
3
4

Figure A-1. Percentages of 2017-2019 2-hour average PM_{2.5} mass concentrations above 140 µg/m³.



5
6
7
8
9

Figure A-2. Frequency distribution of 2017-2019 4-hour averages for sites meeting both or violating either PM_{2.5} NAAQS for October to March (blue) and April to September (red).



1
2 **Figure A-3. Frequency distribution of 2017-2019 5-hour averages for sites meeting both or**
3 **violating either PM_{2.5} NAAQS for October to March (blue) and April to September**
4 **(red).**

5
6 **A.4 DATA SOURCES FOR ULTRAFINE FRACTION OF PM_{2.5} MASS**
7 **FIGURE**

- 8 • Annual average particle number and mass concentrations for Bondville, IL: calculated
9 from 24-hour average values for years with 66% data completion in 75% of the months
10 of the year from 2000-2019. We downloaded the mass concentrations from the EPA's
11 Air Quality System (AQS, <https://www.epa.gov/aqs>) and particle number concentrations
12 from NOAA's Earth System Research Laboratory's Global Monitoring Division
13 (<https://www.esrl.noaa.gov/gmd>).

14 **A.5 METHODS FOR PREDICTING AMBIENT PM_{2.5} BASED ON HYBRID**
15 **MODELING APPROACHES**

16 **A.5.1 Data Sources for 2011 PM_{2.5} Spatial Fields**

- 17 • The "HU2017" fields were provided by Professor Yang Liu of Emory University in the
18 form of comma-separated-values files (*.csv) of daily average PM_{2.5} on a national grid.
- 19 • The "DI2016" fields were provided by Dr. Qian Di of Harvard in the form of MATLAB
20 files (*.mat) of daily average PM_{2.5} on a national grid.
- 21 • The "VD2019" fields were provided by Dr. Aaron van Donkelaar in the form of netCDF
22 files (*.nc) of annual average concentration. These files are also available at:
23 http://fizz.phys.dal.ca/~atmos/martin/?page_id=140.

- 1 • The “downscaler” files were developed in terms of daily average Downscaler predictions
2 on a national grid following methods described in the risk assessment appendix.

3 **A.5.2 Data Averaging and Coefficient of Variation**

- 4 • PM_{2.5} concentration fields were loaded into R version 3.4.4, and daily fields were
5 averaged to the annual period. Concentrations for each method at prediction points were
6 then averaged to the corresponding CMAQ grid cells to enable consistent comparisons
7 for Figure 2-28, Figure 2-29, and Table 2-2.
- 8 • The coefficient of variation (CoV) was calculated for each grid cell using the following
9 formula

$$10 \quad CoV(\%) = \frac{100}{\bar{P}} \sqrt{\frac{\sum_{i=1}^N (P_i - \bar{P})^2}{N}}$$

11 where P is the prediction for each of the four methods (i.e., N=4).

12 **A.6 ANALYSES OF BACKGROUND PM**

- 13 • Data sources for Figure 2-38: Smoke and fire detections observed by MODIS in August
14 2017
 - 15 – Image was produced using the NASA Worldview platform
16 (<https://worldview.earthdata.nasa.gov/>). Layers selected were 1) Corrected
17 Reflectance and 2) Fires and Thermal Anomalies, both from Aqua/MODIS. Day
18 selected was August 4, 2017.
- 19 • Data sources for Figure 2-39: Fine PM mass time series during 2017 from North Cascades
20 IMPROVE site
 - 21 – Image was archived from the IMPROVE website
22 (http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_Pm
23 [HazeComp](http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_Pm)); hosted by CIRA/CSU and sponsored by NPS and USFS) for the
24 North Cascades (NOCA1) site in 2017.
- 25 • Data sources for Figure 2-40: Speciated annual average fine PM mass from IMPROVE at
26 select remote monitors in 2004 and 2016
 - 27 – Speciated IMPROVE data from 2004 and 2016
28 (http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_Pm
29 [HazeComp](http://views.cira.colostate.edu/fed/SiteBrowser/Default.aspx?appkey=SBCF_Pm)) were averaged annually for each monitor. Corresponding monitor
30 locations are shown in Figure 2-41.

31
32
33

1 **A.7 COMPARISON OF PM_{2.5} FIELDS IN ESTIMATING EXPOSURE AND** 2 **RELATIVE TO DESIGN VALUES: METHODS**

3 Section 2.3.3.2.4 outlines analyses comparing the PM_{2.5} concentrations in estimating
4 exposure relative to design values. Below details the data sources and methods used.

5 To calculate annual average concentrations over the U.S. for 2000-2016, gridded
6 concentration fields were obtained based on the DI2019 (Di et al., 2019) and the HA2020
7 (Hammer et al., 2020) and (Van Donkelaar et al., 2019) methods. The DI2019 concentrations
8 were acquired from a [Google Drive](#) and the HA2020 concentrations (version V4.NA.03) were
9 acquired from a [web link](#). To identify grid cells that fall within the contiguous U.S. and Core
10 Based Statistical Areas (CBSAs) boundaries, cartographic boundary shapefiles
11 (“cb_2017_us_state_5m” and “cb_2017_us_cbsa_5m”) were downloaded from the census.gov
12 [website](#). The concentration data and shapefiles were read into R version 3.62 (R Core Team,
13 2019), and grid cells within the contiguous U.S. and CBSAs were identified using the Simple
14 Features package version 0.8-0 (Pebesma, 2018) in R. Average concentrations were then
15 calculated for each year and for each region (i.e., contiguous U.S. and CBSAs within the
16 contiguous U.S.) using the dplyr package version 0.8.3 (Wickham et al., 2019) in R.

17 To generate the population-weighting for the DI2019 and HA2020 PM_{2.5} concentrations,
18 2015 gridded population counts at 0.05×0.05° from the fourth version of the Gridded Population
19 of the World (GPWv4; <https://sedac.ciesin.columbia.edu/data/collection/gpw-v4>) were spatially-
20 collocated with the PM_{2.5} concentrations surfaces after conversion to latitude-longitude
21 coordinates. A similar CBSA filtering was performed for the gridded population and spatially-
22 collocated PM_{2.5} surfaces from DI2019 and HA2020 and the fractional population for each grid
23 was multiplied by the PM_{2.5} concentrations within each CBSA.

24 Regulatory design values were calculated using the data handling described by 40 CFR
25 Appendix N to Part 50 - Interpretation of the National Ambient Air Quality Standards for PM_{2.5},
26 by CBSA, for each 3-year period of available hybrid modeling surface data from the EPA’s Air
27 Quality System (AQS, <https://www.epa.gov/aqs>). Within each CBSA, by each 3-year period, the
28 ratio of design values to estimated PM_{2.5} concentrations was calculated.

1 **REFERENCES**

2 Di, Q, Amini, H, Shi, L, Kloog, I, Silvern, R, Kelly, J, Sabath, MB, Choirat, C, Koutrakis, P and
3 Lyapustin, A (2019). An ensemble-based model of PM2.5 concentration across the
4 contiguous United States with high spatiotemporal resolution. *Environment International*
5 130: 104909.

6 Hammer, MS, van Donkelaar, A, Li, C, Lyapustin, A, Sayer, AM, Hsu, NC, Levy, RC, Garay,
7 MJ, Kalashnikova, OV and Kahn, RA (2020). Global estimates and long-term trends of
8 fine particulate matter concentrations (1998–2018). *Environmental Science &*
9 *Technology* 54(13): 7879-7890.

10 Pebesma, EJ (2018). Simple features for R: standardized support for spatial vector data. *R J.*
11 10(1): 439.

12 R Core Team (2019). R: A language and environment for statistical computing R Foundation for
13 Statistical Computing Vienna, Austria.

14 Van Donkelaar, A, Martin, RV, Li, C and Burnett, RT (2019). Regional estimates of chemical
15 composition of fine particulate matter using a combined geoscience-statistical method
16 with information from satellites, models, and monitors. *Environmental science &*
17 *technology* 53(5): 2595-2611.

18 Wickham, H, François, R, Henry, L and Müller, K (2019). *dplyr: A Grammar of Data*
19 *Manipulation*. R package version 0.8.3.

20

1
2
3

**APPENDIX B. SUPPLEMENTAL STUDY
INFORMATION: SELECTION CRITERIA, STUDY
METHODS AND DETAILS**

TABLE OF CONTENTS

1

2 B.1 Forest Plots..... B-1

3 B.2 Monitored PM_{2.5} Concentrations in Key Epidemiologic Studies B-1

4 B.3 Hybrid Model Predicted PM_{2.5} Concentrations in Key Epidemiologic Studies B-3

5 B.4 Details of Key Epidemiologic Studies, Including Study Design, Exposure Metric, and

6 Statistical Analysis..... B-8

7 References B-85

8

1 This appendix presents supplemental information on the key epidemiologic studies
2 evaluated in section 3.3.3 of this draft PA. Section B.1 provides supplemental information on the
3 forest plots presented in Figures 3-3 to 3-6. Section B.2 provide supplemental information on the
4 study-reported PM_{2.5} concentrations presented in Figure 3-8, Figure 3-9, while section B.3
5 provides supplemental information on studies presented Figure 3-10 and Figure 3-11. Section
6 B.4 provides details on key elements of epidemiologic studies, including the study design and
7 details on the statistical analyses employed, including control for confounding effects.

8 **B.1 FOREST PLOTS**

9 Figure 3-3 through 3-6 in Chapter 3 present forest plots that include the effect estimates
10 and 95% confidence intervals from 92 epidemiologic studies that were assessed in the 2019 ISA
11 and draft ISA Supplement that have the potential to be most informative in reaching conclusions
12 on the adequacy of the current primary PM_{2.5} standards. Epidemiologic studies included in these
13 figures support “causal” or “likely to be causal” relationships with PM exposures in the 2019
14 ISA and include mortality (all-cause mortality, cardiovascular (CVD) mortality, respiratory
15 mortality, lung cancer mortality), and morbidity (asthma incidence, lung cancer incidence, lung
16 function and lung development, CVD and respiratory emergency room visit or hospital
17 admission) health endpoints. Further, studies included in Figure 3-3 to Figure 3-6 were restricted
18 to multi-city studies in the United States or Canada. Multi-city studies within a single State were
19 not included, with the exception of respiratory morbidity endpoints, where multi-city studies
20 were limited (U.S. EPA, 2019). For some of the major cohort studies included in the 2009 ISA,
21 like the American Cancer Society (ACS) cohort, we included more recent studies that reanalyze
22 epidemiologic associations for multiple mortality endpoints (e.g. lung cancer mortality and IHD
23 mortality) and an extension of follow-up periods (e.g., Pope et al., 2015a, Turner et al. (2016),
24 Jerrett et al. (2016), and Thurston et al. (2016b)), as well as a reanalysis (Krewski et al. (2009) of
25 the original ACS dataset, including an extended follow-up period, that was evaluated in the 2009
26 ISA (U.S. EPA, 2009)).

27 **B.2 MONITORED PM_{2.5} CONCENTRATIONS IN KEY EPIDEMIOLOGIC** 28 **STUDIES**

29 Based on the 92 key studies identified in Figure 3-3 to Figure 3-6, a subset of studies are
30 depicted in Figure 3-8 and Figure 3-9 and includes key epidemiologic studies that report an
31 overall study mean or median concentration of PM_{2.5} (as opposed to a study mean/median range
32 across study area locations) and based on ambient PM_{2.5} monitored data. The plots include
33 studies that report significant effect estimates (29 studies) and studies that report non-significant
34 effect estimates (4 studies). Further, to be included, only key studies for which the years of air

1 quality data used to estimate exposures overlap entirely with the years during which health
 2 events are reported were included. The PM_{2.5} concentrations reported by studies that estimate
 3 exposures from air quality corresponding to only part of the study period, often including only
 4 the later years of the health¹ are not likely to reflect the full ranges of ambient PM_{2.5}
 5 concentrations that contributed to reported associations.²

6 Some of the key epidemiologic studies assessed in the 2019 ISA also provide city-
 7 specific study mean concentrations and city-specific health events, but this information was not
 8 available in studies evaluated in the draft ISA Supplement. PM_{2.5} exposure estimates
 9 corresponding to the 10th and 25th percentiles of those events were calculated in the following
 10 manner. City-specific cases and PM_{2.5} concentrations were input in ascending order by PM_{2.5}
 11 concentration. The city-specific percent of cases was calculated as a proportion of the total study
 12 cases and the cumulative percent of cases was determined. The PM_{2.5} concentration associated
 13 with the cumulative percent closest to the 10th and 25th percentiles are presented in Figure 3-8
 14 and Figure 3-9 and the cumulative percent values closest to the associated 10th and 25th percentile
 15 values are shown in Table B-1.³ Data for Bell et al. (2008) and Zanobetti and Schwartz (2009)
 16 were previously provided by the study authors, as described in Rajan (2011).

17

18 **Table B-1. PM_{2.5} concentrations corresponding to the 25th and 10th percentiles of estimated**
 19 **health events.**

Citation	10 th Percentile PM _{2.5} (µg/m ³) (Cumulative percent value closest)	25 th Percentile PM _{2.5} (µg/m ³) (Cumulative percent value closest)
Bell et al. (2008)	9.8	11.5
Franklin et al. (2007)	10.4 (11.1%)	12.9 (25.3%)
Stieb et al. (2009)	6.7 (16.5%)	6.8 (20.5%)
Szyszkowicz (2009)	6.4 (4.1%)	6.5 (18.6%)
Zanobetti and Schwartz (2009)	10.3	12.5

20

¹ The following studies do not have an overlap between the years of PM_{2.5} air quality data and the years during which health effects are reported: Miller et al., 2007 ; Hart et al., 2011 ; Thurston et al., 2013; Weichenthal et al., 2014; Pope et al., 2015b ; Villeneuve et al., 2015; Turner et al., 2016; Weichenthal et al., 2016a; Pinault et al., 2017; Parker et al., 2018; and Pope et al., 2019.

² This is an issue only for some studies of long-term PM_{2.5} exposures. While this approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term PM_{2.5} exposures, under the assumption that spatial patterns in PM_{2.5} concentrations are not appreciably different during time periods for which air quality information is not available (e.g., Chen et al., 2016), our interest is in understanding the distribution of ambient PM_{2.5} concentrations that could have contributed to reported health outcomes.

³ That is, 25% of the total health events occurred in study locations with mean PM_{2.5} concentrations (i.e., averaged over the study period) below the 25th percentiles identified in Figure 3-8 and Figure 3-9 and 10% of the total health events occurred in study locations with mean PM_{2.5} concentrations below the 10th percentiles identified.

1 **B.3 HYBRID MODEL PREDICTED PM_{2.5} CONCENTRATIONS IN KEY** 2 **EPIDEMIOLOGIC STUDIES**

3 Figure 3-10 and Figure 3-11 focus on multicity/multistate studies in the U.S. and Canada,
4 that are part of the evidence supporting “causal” or “likely to be causal” determinations in the
5 2019 ISA and that use hybrid modeling methods to estimate PM_{2.5} exposures, as well as studies
6 assessed in the draft ISA Supplement. In addition, as detailed in section 3.2.3.2.1, for studies
7 included in Figure 3-10 and Figure 3-11 we also consider the approach used to estimate PM_{2.5}
8 concentrations and the approach used to validate hybrid model predictions when determining
9 those studies that we identify as key epidemiologic studies. Such studies are identified as those
10 that use hybrid modeling approaches for which recent methods and models were used (e.g.,
11 recent versions and configurations of the air quality models); studies that are fused with PM_{2.5}
12 data from national monitoring networks (i.e., FRM/FEM data); and studies that reported a
13 thorough model performance evaluation for core years of the study.⁴

14 Figure 3-10 and Figure 3-11 present overall means of hybrid model-predicted PM_{2.5}
15 concentrations for key studies, and the concentrations corresponding to the 25th and 10th
16 percentiles of estimated exposures or health events, when available. For Di et al. (2017b), we
17 present 25th and 10th percentiles of annual PM_{2.5} concentrations by zip code corresponding to
18 long-term exposure estimates, while for Di et al. (2017a), we present daily air pollution
19 concentrations (short-term exposure estimates) corresponding to the 25th and 10th percentiles of
20 deaths at the zip-code level. These values, along with other percentiles, are illustrated in Figure
21 B-1 and Figure B-2 (Jenkins, 2019a, Jenkins, 2019b). The study authors for Di et al. (2017b)
22 additionally provided information on population weighted percentile values corresponding to
23 long-term PM_{2.5} exposure (Chan, 2019). These are presented in Table B-2. For other studies
24 included in Figure 3-10 and 3-11 [Bai et al., 2019, Erickson et al., 2019, Kloog et al. (2012),
25 Kloog et al. (2014), Shi et al. (2016), U.S. EPA, 2021, and Wang et al. (2017)], 25th percentiles
26 of exposure estimates were derived from study manuscripts of air quality descriptive statistics
27 and can be found in Table B-3.

⁴ The following studies do not meet these criteria: Bravo et al., 2017, Crouse et al., 2015; Puett et al., 2009, Puett et al., 2011, Hystad et al., 2012; Hystad et al., 2013, Hayes et al., 2020; Elliott et al., 2020; Lefler et al., 2019;; Pappin et al., 2019; Cakmak et al., 2018; Fisher et al., 2019; Sun et al., 2019; McClure et al., 2017; Loop et al., 2018 ; and Honda et al., 2017.

Percentiles of PM_{2.5} By Zip Code

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

Percentile of PM _{2.5} , Based on ZIP code	PM _{2.5} Value
0%	0.0209025
5%	6.1962803
10%	7.2742546
15%	8.0043245
20%	8.5892973
25%	9.0612931
30%	9.4644903
35%	9.8273901
40%	10.1797192
45%	10.5371831
50%	10.9015790
55%	11.2791073
60%	11.6666804
65%	12.0707952
70%	12.4916270
75%	12.9386305
80%	13.4294338
85%	13.9765291
90%	14.6375324
95%	15.6106067
100%	32.5759482

1

2 **Figure B-1. Percentiles of annual PM_{2.5} concentrations by zip code corresponding to long-**
3 **term exposure estimates in Di et al., 2017b.**

4

1 **Table B-2. Population weighted percentiles of annual PM_{2.5} concentrations by zip code**
2 **corresponding to long-term exposure estimates in Di et al., 2017b.**

3

Percentile	Population Weighted PM _{2.5} (µg/m ³)
0.0	0.0
5.0	7.1
10.0	7.9
15.0	8.6
20.0	9.1
25.0	9.5
30.0	9.9
35.0	10.3
40.0	10.6
45.0	11.0
50.0	11.4
55.0	11.7
60.0	12.1
65.0	12.5
70.0	12.9
75.0	13.4
80.0	13.9
85.0	14.4
90.0	15.1
95.0	16.1
100.0	32.6

4

Percentiles of PM_{2.5} By Zip Code

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

Percentile of Daily PM _{2.5} , Based on ZIP code	PM _{2.5} Value
0%	0.0006378
5%	3.8286960
10%	4.7224770
15%	5.4309290
20%	6.0727840
25%	6.6863868
30%	7.2922285
35%	7.9031599
40%	8.5292050
45%	9.1836408
50%	9.8740436
55%	10.6124979
60%	11.4111824
65%	12.2910351
70%	13.2835707
75%	14.4301324
80%	15.8159815
85%	17.5894591
90%	20.0959732
95%	24.4759063
100%	201.3071287

1
2 **Figure B-2. Daily air pollution concentrations (short-term exposure estimates)**
3 **corresponding to various percentiles of deaths at the zip-county level in Di et al., 2017a.**

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

Citation	10 th Percentile PM _{2.5} (µg/m ³)	25 th Percentile PM _{2.5} (µg/m ³)
Di et al. (2017a)	4.7	6.7
Di et al. (2017b)	7.3	9.1
Kloog et al. (2012)		6.4
Kloog et al. (2014)		7.9
Shi et al. (2016)		4.6
Shi et al. (2016)		6.2
Wang et al. (2017)		9.1
Bai et al. (2019)		7.9
Christidis et al. (2019)		4.3
Shin et al. (2019)		8

3

1 **B.4 DETAILS OF KEY EPIDEMIOLOGIC STUDIES, INCLUDING STUDY DESIGN, EXPOSURE**
 2 **METRIC, AND STATISTICAL ANALYSIS**

3 Table B-4 below summarizes additional details related to the designs of the U.S. and Canadian epidemiologic studies included
 4 in Figure 3-3 to 3-6, and Figure 3-8 to Figure 3-11, as well as studies included in the risk assessment (Table 3-13).

5 **Table B-4. Study characteristics from key studies.**
 6
 7

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Baxter et al., 2017	ST	All-cause mortality	77 US Cities	Time Series study (NCHS)	<p>EPA's National and State Local Ambient Monitoring Stations providing integrated daily measurements and operated more than 6 months or had more than 30 observations (2001-2005) considered. Monitors representing the general population exposure in the cities were selected. For this correlation was assessed between each pair of monitors within the county and the ones uncorrelated (coefficient<0.8 with majority of other monitors) were excluded. Once appropriate valid monitors were identified the summary measure of PM_{2.5} concentration over the county was calculated.</p> <p>2-day moving average (lag 0-1 days) of PM_{2.5} conc included in the model.</p>	<p>Poisson regression model and meta-regression</p> <p>In stage 1, ran single city Poisson time-series models; adjusted for temperature and dew point temperature, including variables for previous day temperature, temporal trends, and trends by age. In stage 2, meta-regression with cluster analysis (5 clusters) based on characteristics of residential infiltration.</p>	<p>Average daily PM_{2.5} values were calculated for each city. First, a global mean and variance were created within each city for the entire time period. Using the valid monitor measurements. Next, all values were standardized and average PM_{2.5} within a given day in each city was calculated. Finally, the standardized daily value was reversed to calculate average daily PM_{2.5} for each city.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Bell et al., 2008	ST	CVD HA Age 65+	202 US Counties with populations ≥ 200,000	Time Series study (MEDICARE enrollees)	<p>PM_{2.5} concentrations obtained from EPA monitors providing data daily or every 3 days for the period 1999-2005. Used 10% trimmed mean to calculate daily average across monitors after correction for yearly monitor averages (to protect against outliers as applied in Dominici et al. 2006).</p> <p>Used lag0 PM_{2.5} in the model.</p>	<p>2-stage Bayesian hierarchical model</p> <p>In stage 1, adjusted for temperature and dew point temperature, including variables for previous day's conditions, day-of-the-week, temporal trends, and differential temporal trends by age. In stage 2, county-specific estimates were combined, accounting for their statistical uncertainty.</p>	Average daily PM _{2.5} concentrations for each county used to calculate overall mean for the study area and duration.

Bell et al., 2014	ST	CVD, Asthma, and COPD HA Age 65+	4 Counties in MA and CT	Time-series study (MEDICARE enrollees)	<p>PM_{2.5} Teflon filter samples (measuring PM_{2.5} total mass) obtained from CT and MA DEP for the period of 2000-2004. Used data from five monitoring locations (providing daily or every third day data) within four county regions. Assigned daily PM_{2.5} concentration from a single monitor to three counties. For Fairfield County with two monitors: daily PM_{2.5} concentration was calculated by using population-weighted averaging of census tract PM_{2.5} concentrations. First, each census tract in the Fairfield county (209 tracts in total) was assigned the PM_{2.5} exposure of the nearest monitor. Then, PM_{2.5} exposures for all tracts were averaged and weighted by each tract's 2000 U.S. census population to calculate a county-level exposure for the Fairfield county.</p>	<p>Log-linear Poisson regression analysis</p> <p>Adjusted for temperature and dew point temperature, including previous day's temperature and dew point temperature, day-of-the-week temporal trends, and region.</p>	<p>Daily PM_{2.5} concentrations for all four counties (three with single monitor and one with two monitors that used population weighted approach) over the period of 2000-2004 were used to calculate the overall mean PM_{2.5} for the study location and period.</p>
-------------------	----	----------------------------------	-------------------------	--	--	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					Explored various lags and presented lag0 PM _{2.5} model.		
Bell et al., 2015	ST	HF HA 65+	213 U.S. Counties	Time-series study (MEDICARE enrollees)	<p>Daily monitored PM_{2.5} data from the US EPA AQS monitors for the period of 1999-2010. On average, county-level PM_{2.5} data was available for 56.5% of study days (range: 7.8%-99.9%; no imputation done for missing data). For each county, daily PM_{2.5} measurement was calculated by averaging the PM_{2.5} values from all monitors within a county in a given day.</p> <p>Explored various lags and presented lag0 PM_{2.5} model.</p>	<p>2-stage Bayesian hierarchical model</p> <p>The stage 1 model included county-specific model adjusted for weather (temperature, dew point, previous days' temperature, and dew point), day-of-the-week, and temporal trends. In stage 2 county-specific effect estimates were pulled together to present overall association.</p>	<p>Daily PM_{2.5} concentrations for 213 counties over the period of 1999-2010 were used to calculate region-specific mean PM_{2.5}, and overall mean PM_{2.5} for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Bravo et al., 2017	ST	CVD HA Age 65+	418 U.S. Counties	Time-series study (MEDICARE enrollees)	<p>Daily (24-hr) monitored PM_{2.5} data from the US EPA AQS monitors (NAMS/SLAMS) obtained for the period of 2002-2006. Approximately 80% of PM_{2.5} monitors recorded observation once every 3 days. For each county (>=50K population), daily (24-hr) PM_{2.5} concentration was calculated by averaging multiple monitor measurements for the same day.</p> <p>Explored various lags and distributed lags of PM_{2.5} exposure.</p>	<p>2-stage Bayesian hierarchical model</p> <p>The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of PM_{2.5} conc simultaneously in the county-specific model.</p> <p>The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.</p>	<p>Daily PM_{2.5} concentrations for 418 counties over the period of 2002-2006 were used to calculate overall mean PM_{2.5} for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Bravo et al., 2017	ST	CVD HA Age 65+	708 U.S. Counties	Time-series study (MEDICARE enrollees)	<p>Daily PM_{2.5} concentrations were estimated at census tract centroids using the downscaler method (input from the US EPA AQS NAMS/SLAMS monitoring data, and gridded 12x12 km CMAQ) for the period of 2002-2006. County-level daily PM_{2.5} exposures were calculated from a population-weighted averages of PM_{2.5} concentrations predicted at census tract within each county using 2000 U.S. Census data. CMAQds was generated for all days in the study period 2002-2006. CMAQds-subset was calculated by taking population-weighted county level exposures only for counties and days with monitoring data (n=418 counties).</p> <p>Explored various lags and distributed lags of PM_{2.5} exposure.</p>	<p>2-stage Bayesian hierarchical model</p> <p>The stage 1 included log-linear Poisson regression models with over-dispersion fit at county-level. Model adjusted for same-day temperature and dew point temperature, 3-day moving average of temperature and dew point temperature, temporal trends in hospitalizations, day-of-the-week, and age. Fitted distributed lag model with multiple lags (0- to 7-day lags) of PM_{2.5} conc simultaneously in the county-specific model. The stage 2 estimated the association for the entire study area using two-level normal independent sampling estimation with priors thus allowing to combine risk estimates across counties while accounting for within county SE and between-county variability in the true RR.</p>	<p>24-hr average PM_{2.5} concentrations for 708 counties over the period of 2002-2006 were used to calculate overall mean PM_{2.5} for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Burnett and Goldberg, 2003	ST	All-cause mortality	8 Canadian Cities	Time-series study	<p>PM_{2.5} data obtained from dichotomous sampler with Teflon filters operating on 6-day schedule for the period of 1986-1996.</p> <p>Each city had one sampler and two cities have two samplers. If two samplers then data was averaged between the samplers and assigned to the city.</p> <p>Lag 1 explored.</p>	<p>Generalized additive model (GAM) analysis to generate pooled estimate of air pollution effect among the eight cities.</p> <p>The model adjusted for day-of-the-week, temporal trends, and weather variables (daily average temperature, daily average relative humidity, and barometric pressure lagged 0 and 1 days).</p>	<p>Daily PM_{2.5} concentrations (day before the death) for 8 Canadian cities over the period of 1986-1996 were averaged to get overall mean for the study area and period</p>
Burnett et al., 2004	ST	All-cause mortality	12 Canadian Cities	Time-series study (data from Statistics Canada)	<p>Monitoring data available for 12 cities from the Statistics Canada for the period of 1981-1999. PM_{2.5} data available every 6th-day sampling schedule. Daily PM_{2.5} concentrations were calculated for each city by averaging data over all monitors with each city.</p> <p>Explore various lags and moving average and presented data for lag 1 for PM_{2.5}.</p>	<p>Random-effects regression model.</p> <p>Adjusted for temporal trends in mortality and effects of weather using humidex index at lag 0 and lag 1 (a measure of combined effect of temperature and humidity)</p>	<p>Daily PM_{2.5} concentrations for all 12 cities over the period of 1981-1999 were used along with population information to calculate an overall population weighted PM_{2.5} concentration for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Cakmak et al., 2018	LT	Non-accidental, CVD, respiratory and lung cancer mortality	Canada Nationwide	Cohort study (CanCHE C)	PM _{2.5} estimates obtained from median satellite-derived concentrations for the period of 1998 – 2011. The concentration was determined at 10 km ² resolution as detailed in (van Donkelaar, 2010). Changes in PM _{2.5} between 1998 and 2006 was inferred using satellite instruments, MISR and SeaWiFS (Boys, 2014). Annual estimates of PM _{2.5} concentration was assigned to participants based on postal code of residence and was used to calculate 7-year moving average (at least 4 out of 7 years of data is available) PM _{2.5} concentration for each year of follow-up in the study.	Cox proportional hazards models to estimate the relationship between long-term exposure and date of death accounting for residential mobility. Model adjusted for individual-level covariates (aboriginal ancestry, minority status, marital status, education, immigrant status and income)	The 7-year moving averages for study participants were then used to calculate overall mean PM _{2.5} concentration (all and by geographic zones)

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Chen et al., 2020	LT	CVD mortality	Ontario, Canada	Cohort study (ONPHEC)	PM _{2.5} concentration estimated from multiple satellite retrievals of AOD combined with geophysical relationship between AOD and PM _{2.5} simulated by GEOS-Chem, which were then calibrated with surface measurements by GWR as detailed in (van Donkelaar, 2019). Annual estimates of exposure to PM _{2.5} and the composition for each participant was estimated by interpolating the annual mean concentrations of PM _{2.5} and the corresponding proportion of PM _{2.5} attributed to the seven major components to the centroid of their residential postal code for that year, thereby accounting for residential mobility.	Component-adjusted approach that jointly estimated the health impacts of PM _{2.5} and its major components while allowing for a potential nonlinear PM _{2.5} -outcome relationship. Compared this approach with three traditional approaches using Cox Hazard models. Adjusted for individual-level covariates, four time-varying variables for neighborhood-level SES, area-level indicators.	Annual PM _{2.5} concentrations in the Ontario region were then used to calculate overall mean PM _{2.5} concentration for the study location and period.

Christidis et al., 2019	LT	Non-accidental mortality	Canada Nationwide	Cohort study (mCHHS)	<p>PM_{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Spatial variation from modeled surface used with simulate PM_{2.5} and constrained with local ground-based monitors to estimate PM_{2.5} concentrations through 2015 (Meng, 2019). Linked postal codes to PM_{2.5} concentrations using points of latitude and longitude. When multiple points of latitude and longitude was available for a single urban postal code, equal weighting of the multiple air pollutant values was used to provide a singular value. In rural communities, population-weighted average of the values associated with duplicate postal codes was used. Used population-weighting to average multiple values to create inputs for partial postal codes (2 to 5 digit). For each individual and year of follow-up, PM_{2.5}</p>	<p>Cox proportional hazard models to assess the relationship between PM_{2.5} exposure and non-accidental death in low-exposure environment.</p> <p>C-R relationship observed using Shape constrained health impact function (SCHIF Model) Adjusted for socio-economic, behavioral, and time-varying contextual covariates</p>	<p>3-year moving average PM_{2.5} concentrations were for the participants used to calculate overall mean PM_{2.5} concentration for the study period-</p>
-------------------------	----	--------------------------	-------------------	----------------------	--	--	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					estimates was calculated as 3-year moving average with one-year lag.		

Crouse et al., 2012	LT	All-cause mortality	11 Canadian Cities	National Cohort study (Subset of Canadian census mortality follow-up study; 43% non-immigrant population)	<p>Monitor data from ground-based stations available for 11 cities for the 15-yr period including the 5-yr prior to baseline and 10-yr of follow-up (1987-2001) from Statistics Canada. PM_{2.5} data available every 6th-day sampling schedule. To address missing monthly PM_{2.5} data for some stations, data from all stations within 6-km of each other were pooled to calculate monthly, seasonal, annual and five-yr (1987-1991, 1992-1996, 1997-2001) means at each monitored location. Mean annual concentration (averaged over 1987-2001) from ground-based monitors was then assigned to the cohort member based on the 11 census divisions of their residence.</p> <p>A second set of exposure (10x10 km) was created using estimates of PM_{2.5} from remote sensing during period 2001-2006 to calculate 6-yr average. The mean concentration of PM_{2.5} within boundaries of each enumeration area</p>	<p>2 different modelling approach. Approach 1: Cox proportional hazards model, and Approach 2: nested, spatial random-effects Cox model with spatial clusters.</p> <p>Models adjusted for individual-level covariates, urban/rural indicator, and ecological covariates (% unemployed, % without high school diploma, lowest income quintile, and rural/urban indicator).</p>	Annual PM _{2.5} concentrations for the study participants were used to calculate overall mean PM _{2.5} for the study population and duration.
---------------------	----	---------------------	--------------------	--	--	---	---

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					<p>was calculated by overlaying PM surface over the surface of enumeration area across country. Satellite derived PM_{2.5} estimate was then assigned to participants based on their enumeration area of residence in 1991.</p>		

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Crouse et al., 2019	LT	Non-accidental, CVD, respiratory mortality, and lung cancer	Canada Nationwide	Cohort study (CanCHEC)	PM _{2.5} concentrations derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Spatial variation from modeled surface used with simulate PM _{2.5} and constrained with local ground-based monitors to estimate PM _{2.5} concentrations through 2015 as detailed in (Meng, 2019). Linked postal codes to PM _{2.5} concentrations from grid cells. Annual PM _{2.5} estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate moving average at various temporal and spatial scales based on the location and year of follow-up.	Cox Hazard model to assess the relationship between PM _{2.5} exposure at different temporal and spatial scales. Adjusted for individual-level variables (aboriginal identity, visible minority status, marital status, highest level of education, employment status, and household income adequacy quintiles)	The average annual PM _{2.5} concentrations were used to calculate overall mean PM _{2.5} concentration for the study period at various temporal and spatial scales.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Dai et al., 2014	ST	All-cause, CVD, and Respiratory mortality	75 U.S. Cities (with available daily mortality data and PM _{2.5} data for at least 400 days between 2000 and 2006)	Time-series study (NCHS)	Monitored data obtained from US EPA AQS for the period of 2000-2006. Daily PM _{2.5} concentrations from each monitor assigned to corresponding city. For cities with more than one sampling site, concentration data were averaged across all monitors within the city. Used average of 2-day lag (lag 01) PM _{2.5} .	Two stage: Stage 1. City-specific season-stratified time-series analysis using Poisson regression in GAM Model adjusted for 24-hr average temperature from closest weather station to the city center at lag0 and lag1, temporal trends, and day-of-the-week. Stage 2. Multivariate random effects meta-analysis to combined 300 (i.e. 75 cities * 4 seasons) effect estimates to obtain overall association.	Daily PM _{2.5} concentrations for all 75 cities over the period of 2000-2006 were used to calculate an overall mean PM _{2.5} concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
deSouza et al., 2021	ST	First CVD HA	US Nationwide	Time-stratified case-crossover design (MEDICAID)	PM _{2.5} concentration were derived for 1 km ² grid cells in the continental United States by integrating remote sensing, outputs from a chemical transport model, and other variables such as meteorological and land-use variables (Di et al. 2019); from an ensemble-based model that integrated multiple machine learning algorithms for the period of 2000-2012. Daily PM _{2.5} estimates of all grid cells averaged at-Zip code were assigned to study participants based on the zip code of residence. Used lag01 average exposure in the model.	Conditional logistic regression models to estimate the associations between short-term exposure to PM _{2.5} and CVD hospitalization rates. Adjusted for individual-level covariates, air and dew-point temperature.	Daily PM _{2.5} concentration from case days were then used to calculate overall case day mean PM _{2.5} concentration for the study location and period.

Di et al., 2017b	LT	All-cause mortality 65+	US Nationwide	Cohort (MEDICARE enrollees)	<p>Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of PM_{2.5}. The neural network was fit with monitored PM_{2.5} data and daily PM_{2.5} concentrations were predicted for nationwide grids that were 1x1 km. While not explicitly detailed in the study, it was assumed that the 1 km x 1 km grid cells were averaged up to the zip code spatial resolution. For each calendar year during which a person was at risk of death the annual average PM_{2.5} concentration was assigned according to the ZIP Code of the person's residence. As part of a sensitivity analysis, monitored PM_{2.5} data was matched with each person in the study within a distance of 50 km of the nearest monitoring site.</p>	<p>Two-pollutant Cox proportional hazards model with generalized estimating equation to account for correlation between ZIP codes.</p> <p>Accounted for individual variables, (sex, race, Medicaid eligibility, and average age at study entry), zip code-level variables (% Hispanic, % Black, median household income, median value of housing, % > 65 living below poverty level, % > 65 with less than high school education, % of owner-occupied housing units, and population density), county-level variables (county-level BMI and % ever smokers), hospital service area-level variables (% low-density lipoprotein level measured, % glycated hemoglobin level measured, and % >1 ambulatory visits), 32 km² gridded weather and 1 km² gridded pollution variables (annual average PM_{2.5} concentration, annual average temperature, and annual average humidity), monitor level air pollution variables (PM_{2.5} monitored data), and a regional dummy variable.</p>	<p>Average PM_{2.5} concentrations for all Zip Codes (entire US or ZIP codes with study participants only) from 2000 to 2012 were used to calculate overall mean PM_{2.5} for the study location and period.</p>
------------------	----	-------------------------	---------------	-----------------------------	---	--	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Di et al., 2017b (< 12 ug/m ³)					Analysis restricted to persons-years with PM _{2.5} exposures lower than 12 ug/m ³		

Di et al., 2017a	ST	All-cause mortality 65+	US Nationwide	Case-crossover study (MEDICARE enrollees)	<p>Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data to predict daily concentrations of PM_{2.5}. The neural network was fit with monitored PM_{2.5} data and daily PM_{2.5} concentrations were predicted for nationwide grids that were 1x1 km. For each case day (date of death) and its control days, the 24-hour PM_{2.5} concentrations were assigned based on zip code of residence of the individual.</p> <p>As part of a sensitivity analysis, monitored PM_{2.5} data was matched with each person in the study within a distance of 50 km of the nearest monitoring site, and cross-validation was performed between predicted and monitored concentrations.</p> <p>Used average of 2-day lag (lag 01) PM_{2.5}.</p>	<p>Conditional logistic regression.</p> <p>“Case Day” defined as death. For the same person, compared daily air pollution exposure on the case day vs. daily air pollution exposure on “control days.” Control days were chosen (1) on the same day of the week as the case day to control for potential confounding effect by day of week; (2) before and after the case day to control for time trend; and (3) only in the same month as the case day to control for seasonal and sub-seasonal patterns. Individual-level covariates and zip code-level covariates that did not vary day to day (e.g., age, sex, race/ethnicity, SES, smoking, and other behavioral risk factors) were not considered to be confounders as they remain constant when comparing case days vs control days.</p> <p>The regression model adjusted for air and dew point temperature.</p>	<p>The case and control days PM_{2.5} concentrations for study participants were averaged to get mean PM_{2.5} concentration for the study area and period.</p>
------------------	----	-------------------------	---------------	---	---	---	---

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Dominici et al., 2006	ST	HF and COPD HA 65+	204 Urban U.S. counties	Time-series study (MEDICARE enrollees)	<p>Monitored PM_{2.5} concentrations available from US EPA AQS for the period of 1999-2002. Of the 204 counties (>200,000 population), 90 counties had daily PM_{2.5} data across the study period and the remaining counties had PM_{2.5} data collected once every 3 days for at least 1 full year. To protect against consequences of outliers, used 10% trimmed mean to calculate daily average across monitors after correction for yearly averages for each monitor.</p> <p>Various lags (lag 0, 1, 2 days) and distributed lags assessed and presented.</p>	<p>2-stage Bayesian hierarchical models to estimate county-specific, region-specific, and national-average associations.</p> <p>Stage 1 model included single lag and distributed lag over-dispersed Poisson regression models to estimate county-specific risk. Models adjusted for temperature and dew point on the same day and the 3 previous days, calendar time to control for seasonality and other time-varying influences, daily numbers of individuals at risk, and day-of-the-week. In Stage 2, to produce a national average estimate, Bayesian hierarchical models were used to combine RRs across counties and accounting for within-county statistical error and for between-county variability or heterogeneity. To produce regional estimates. The Stage 2 hierarchical models described above was used for 7 regions separately.</p>	<p>Daily PM_{2.5} concentrations for all 204 US counties over the period of 1999-2002 were used to calculate an overall mean PM_{2.5} concentration for the study regions and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Dominici et al., 2019	LT	Non-accidental mortality	Nationwide	Cohort study (MEDICARE)	Artificial neural network that incorporated satellite-based measurements, simulation outputs from a chemical transport model, land-use terms, meteorological data, and other data was used to predict daily concentrations of PM _{2.5} (Di et al. 2017). Daily PM _{2.5} concentrations were predicted for nationwide grids at 1 km ² resolution for the period 2000–2012.	Survival analyses using the Andersen-Gill method, a variant of the traditional Cox proportional hazards model C-R relationship assessed fitting a log-linear model with thin-plate splines. Adjusted for individual-level covariates, county-and ZIP code-level variables, meteorological variables, and other area-level variables.	Daily PM _{2.5} concentrations over all ZIP codes were used to calculate overall mean PM _{2.5} for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Erickson et al., 2020	LT	Non-accidental, CVD, respiratory mortality, and lung cancer	Canada Nationwide	Cohort study (CanCHE C)	PM _{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). Linked postal codes to PM _{2.5} concentrations from grid cells. Annual PM _{2.5} estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year moving average based on the location and year of follow-up.	Cox proportional hazards models to examine the associations between ambient PM _{2.5} exposure and non-accidental and cause-specific mortality. Adjusted for individual-level and contextual-level covariates.	The average PM _{2.5} concentrations were used to calculate overall mean PM _{2.5} concentration for the study period by immigrant status and duration in Canada.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Eum et al., 2018	LT	All-cause mortality	Geographic regions: "East" of the Mississippi River, "Center" between the Mississippi River and the Sierra Nevada mountain range, and "West" of the Sierra Nevada mountain range	Cohort study (MEDICARE)	PM _{2.5} concentration obtained from US EPA's AQS for the period of 2000-2012. Monitoring sites with daily measurements for at least 8 calendar years with each year having 9+ months and with 4+ daily measurements included. 798 sites then were used to calculate long-term concentration (yearly moving average with 350+ days of valid data) using Greven et al. Annual average assigned to individuals that lived in ZIP codes with centroids within 6 miles of a valid monitor.	Age-stratified log-linear model including offset terms for the size of the population as a base model. Also included the temporal and spatiotemporal components. Ran base model using data for entire 13-year study period (2000-2012) and for shorter periods ranging between 3 and 12 years and compared MRRs to assess temporal confounding. In addition to base model, also assessed temporal confounding using three approaches (decomposition-based, residual-based, and spline models) Adjusted for individual-covariates, as well as county-level behavioral covariates, % of non-whites, smoking status, comorbidities, access to health care, income, and BMI.	Annual average PM _{2.5} concentrations were used to calculate overall mean concentration for the study location (all and by study region) and study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Fisher et al., 2019	ST	Stroke (Self-reported stroke adjudicated by physician medical record)	Nationwide	Time-stratified case-crossover study (HPFS)	<p>Validated national-scale, log-normal ordinary kriging model for PM_{2.5} were used to estimate daily PM_{2.5} concentration. US EPA's AQS data used to calculate monitor specific daily averages (monitors >=18 hours measures). These inputs were then used to produced kriged surfaces of daily mean PM_{2.5} concentrations at the geocoded residential addresses of all HPFS participants for the period 1999-2010.</p> <p>Lag periods up to 3 days prior to the stroke event and a 4-day average used in model.</p>	<p>Conditional logistic regression models</p> <p>Adjusted for mean daily temperature, and stratified models to examine effect modification by individual-level characteristics.</p>	<p>Daily PM_{2.5} concentration on the case day were used to calculate overall case day PM_{2.5} mean for the study period.</p>

Franklin et al., 2007	ST	All-cause, CVD, and Respiratory mortality	27 U.S. communities (with PM _{2.5} monitoring and daily mortality data for at least 2 years of 6-year study period 1997-2000)	Case-crossover study (NCHS)	<p>Monitored daily PM_{2.5} concentrations available from US EPA AQS (NAMS/SLAMS) for the period of 1997-2000. Data for Boston area available from Harvard University.</p> <p>To determine which monitors in the county are representative of exposure for a general population in the county, correlation was assessed between monitor pairs and excluded the monitors with $r < 0.8$ for 2 or more monitor pairs. Once appropriate monitors were identified then a summary measure of PM_{2.5} conc for the county was calculated using alternate averaging method described in Schwartz 2000 to account for data availability variation (daily vs 3-6 days for each monitors in the county) and calculate daily average PM_{2.5} conc for each of the 27 counties and corresponding communities.</p>	<p>2-stage time-stratified analysis:</p> <p>1) Conditional logistic regression analysis to generate community specific estimates;</p> <p>2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate.</p> <p>Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as “deaths” and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1, while effect modification of community-specific characteristics including geographic location, annual PM_{2.5} concentration > 15 ug/m³ and central AC prevalence was used in stage 2.</p>	Daily PM _{2.5} concentrations for all 27 US communities over the period of 1997-2000 were used to calculate an overall mean PM _{2.5} concentration for the study location and period.
-----------------------	----	---	--	-----------------------------	---	--	---

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					Calculated and presented various lags and averages for PM _{2.5} .		

Franklin et al., 2008	ST	All-cause, CVD, and Respiratory mortality	25 U.S. communities (with PM _{2.5} monitoring and daily mortality data for at least 4 years of 6-year period between 2000-2005)	Case-crossover study (NCHS)	<p>Monitored daily PM_{2.5} concentrations available from US EPA AQS (NAMS/SLAMS) for the period of 2000-2005. Data for Boston area available from Harvard University.</p> <p>To determine which monitors in the county are representative of exposure for a general population in the county, correlation was assessed between monitor pairs and excluded the monitors with $r < 0.8$ for 2 or more monitor pairs. Once appropriate monitors were identified then a summary measure of PM_{2.5} conc for the county was calculated using alternate averaging method described in Schwartz 2000 to account for data availability variation (daily vs 3-6 days for each monitors in the county) and calculate daily average PM_{2.5} conc for each of the 27 counties and corresponding communities.</p>	<p>2-stage time-stratified analysis:</p> <p>1) Conditional logistic regression analysis to generate community specific estimates;</p> <p>2) Meta-regression analysis to combined community specific estimates to generate overall pooled effect estimate.</p> <p>Stage 1 of the model adjusted for day-of-the-week, as well as apparent temperature at lag0 and lag1. Cases were defined as “deaths” and control days for a particular subject were chosen to be every third day within the same month and year that death occurred. Effect modification of age and gender was examined using interaction terms in stage 1.</p>	Daily PM _{2.5} concentrations for all 25 US communities over the period of 2000-2005 were used to calculate an overall mean PM _{2.5} concentration for the study location and period (overall and by seasons).
-----------------------	----	---	--	-----------------------------	---	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					Calculated and presented various lags and averages for PM _{2.5} .		
Gharibvand et al., 2016	LT	Lung cancer incidence	US Nationwide	Cohort study (AHSMOG-2 study)	Monitor data obtained from US EPA AQS for the period of 2000-2001 (2-year prior to start of the study). Using monitored PM _{2.5} data, inverse distance weighted interpolations methods, monthly pollution surfaces for PM _{2.5} were created for the US. Monthly exposure averages were based on daily PM _{2.5} measurements. Only months with at least 75% valid data were included in the exposure estimation. Participants were assigned monthly exposure based on their baseline residential address.	Cox proportional hazards model Covariates included sex, race, smoking status, years since participant quit smoking, average number of cigarettes per day during all smoking years, and education level. Additional covariates included calendar time, alcohol consumption, family income, BMI, physical activity, and marital status. 3 variables identified a priori as either as confounders or effect modifiers: hours/day spent outdoors, years of pre-study residence length at enrollment address, and moving distance from enrollment address during follow-up.	Monthly PM _{2.5} concentrations for study participants were used to calculate overall 2-yr mean PM _{2.5} for the study period 2000-2001.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Hart et al., 2015 (monitored)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	Monitored data obtained from US EPA AQS for the period 1999-2006. Monthly average PM _{2.5} concentration calculated from the nearest monitoring location for all addresses. The monthly data was again averaged to get the previous 12-month moving average at each residential address prior to mortality. Nearest monitor exposures were validated against personal exposures to PM _{2.5} of ambient origin.	Cox proportional hazards model. Information on potential confounders was available every two years (4 years for diet information) and each woman was assigned updated covariate values for each questionnaire cycle. Confounders examined include age, race, region, season, physical activity, BMI, hypercholesterolemia, family history of MI, smoking history, Current smoking status, diet, SES (education level, occupation of	Monthly PM _{2.5} concentrations at residence locations during the follow-up period of 2000 to 2012 were averaged to calculate overall mean PM _{2.5} exposure for the participants included in the study.

Hart et al., 2015 (modeled)	LT	All-cause mortality	US Nationwide	Cohort study (Nurses' Health study)	<p>Spatio-temporal models (developed using monitored data from US EPA AQS, the IMPROVE network, and also included meteorological and GIS-derived covariates, such as urban land use within 1 km, elevation, tract- and county-level population density, distance to the nearest road for road classes A1-A3 and point-source emission density within 7.5 km) was used to estimate monthly PM_{2.5} exposures at each geocoded address. The monthly data was again averaged to get the previous 12-month moving average prior to mortality for each residential address. Modeled exposures were validated against personal exposures to PM_{2.5} of ambient origin.</p> <p>Previous 12-month moving average of exposure either from nearest monitor or spatio-temporal models were</p>	<p>both of the nurses' parents when she was 16, marital status, and husband's education if applicable). Also adjusted for area-level SES (census tract level median income and house value), and long-term temporal trends. Risk set regression calibration for time-varying exposures was used to correct for bias due to exposure measurement error in the hazard ratios of all-cause mortality using the personal exposure validation data.</p>	
--------------------------------	----	---------------------	---------------	-------------------------------------	---	--	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					assigned to study participants.		
Hayes et al., 2020	LT	CVD mortality	6 US states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and 2 urban areas (Atlanta, GA, and Detroit, MI,)	Cohort study (NIH-AARP Diet and Health study)	Modelled (Hybrid land use regression geostatistical model developed by Kim et al. 2017) for the period of 1980-2010. Mean annual estimates of PM _{2.5} for each census tract in the US from spatio-temporal model were used till 1998. For period 1999-2010, monitored US EPA monitor and IMPROVE network was used to derive annual average estimates. Annual average PM _{2.5} concentrations assigned at census tract level lagged by 1 year in time-dependent manner. Annual PM _{2.5} exposure analyzed as continuous and categorical <8, 8-<12, 12-<20, and 20+ ug/m ³ variables.	Cox regression modelling with time-dependent covariates. Adjusted for individual-level variables (age, race/ethnicity, education, marital status, BMI, alcohol, and smoking status), as well as census tract variables.	Annual PM _{2.5} concentrations of the study participants for the year 2000 was used to calculate overall mean PM _{2.5} concentration for the period 2000.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Ito et al., 2013 ⁵	ST	All-cause mortality	150 U.S. cities	Time-series study	<p>24-hr average PM_{2.5} mass data in a given city, and when data from multiple monitors were available in a given city, computed the average of the daily values after standardizing each site's data using the mean and standard deviation of the sites data.</p> <p>Pollutant concentration is expressed in the model as a deviation from the monthly mean to reduce the influence of the seasonal cycles of the pollutants on the overall associations and help focus on the short-term associations.</p>	<p>Poisson regression analysis</p> <p>First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach</p> <p>Adjusted for temporal trends (annual cycles and influenza epidemics), immediate and delayed temperature, and day-of-week pattern, for entire years (2001-2006) and for warm (April-September) and cold (October-March) seasons.</p> <p>In second stage, assessed effect modification using land-use variables and average air pollution levels.</p>	

⁵ This study is not referenced individually in the ISA, but is study 3 of the National Particle Component Toxicity (NPACT) Initiative published in HEI (Lippmann et al., 2013).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Jerrett et al., 2016	LT	IHD mortality 30+	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Multiple exposure estimation approaches evaluated within the study – risk assessment uses results based on an ensemble approach that incorporates chemical transport modeling, land use data, satellite data, and data from ground-based monitors	Cox proportional hazards regression Covariates included current and former smoking status as well as smoking duration, amount, age started, second hand cigarette smoke (hours/day exposed), exposure to PM _{2.5} in the workplace for each of the subject's major lifetime occupation, self-reported exposure to dust/fumes at work, marital status, level of education, BMI, alcohol consumption, dietary vegetable/fruit/fiber index, dietary fat index, missing nutrition information. Ecologic characteristics included median household income, percentage of people with < 125% of poverty-level income, percentage of persons > 16 who are unemployed, percentage of adults with < 12 th grade education, and percentage of population who were Black or Hispanic.	

<p>Kioumourtzoglou et al., 2016</p>	<p>LT</p>	<p>All-cause mortality 65+</p>	<p>207 U.S. cities</p>	<p>Open Cohort study (MEDICARE enrollees)</p>	<p>Monitored data available from US EPA AQS for the period of 2000-2010. City-specific annual and 2-year PM_{2.5} averages was calculated using data from all available monitors in each city.</p>	<p>2-stage approach for modelling.</p> <p>In Stage 1, Cox proportional hazards model was fit for each city stratified by age, gender, race, and follow-up time in study. Control for slowly varying potential confounders (e.g., SES) and confounders that vary across subjects, city, and time.</p> <p>City-characteristics for: proportion of city population > 65, median household income, proportion in poverty, proportion of city families in poverty, proportion of white, black, and Asian residents, proportion of residents with/without high-school degrees and a college degree, and city-specific smoking and obesity rates.</p> <p>Population-weighted city averages were developed based on census data at the county level. Also included average annual temperature in the model.</p> <p>In stage 2, combined the city-specific estimates using a random effects meta-analysis to generate region-specific effects. Assessed effect modification by annual temperature levels, and population and city characteristics (greenness,</p>	<p>Annual PM_{2.5} concentrations for 207 cities during the period of 2000 to 2010 were averaged to calculate overall mean PM_{2.5} exposure for the study location (all and region specific) and study period.</p>
-------------------------------------	-----------	--------------------------------	------------------------	---	--	---	---

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
						poverty, racial composition, etc.).	
Klemm and Mason, 2003	ST	All-cause mortality	Harvard Six-City study reanalysis	Time-series study	24-hour PM _{2.5} concentration obtained from Dichotomous samplers placed at the central residential monitoring sites in each of the six cities. Integrated 24-hour samples were collected daily for part of the study periods but were collected at least every other day until the late 1980s.	Generalized additive and Generalized linear models Model adjusted for temporal trends, day-of-the-week, weather (average daily temperature and average daily dew point temperature).	Daily PM _{2.5} concentration of six cities over the period of 1979-1988 were used to calculate overall mean, median and percentiles of PM _{2.5} exposure for the study location (all and by study center) and period.

<p>Kloog et al., 2012</p>	<p>ST, LT</p>	<p>CVD HA Age 65+</p>	<p>New England Area with 6 U.S. States</p>	<p>Mixed study design (with time series and cohort components)</p>	<p>Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground PM_{2.5} measurements. Incorporated land use regressions and meteorological variables (temperature, wind speed, visibility, elevation, distance to major road, percent of open space, point emissions and area emissions) for the period of 2000-2006. Model predicted daily PM_{2.5} concentrations at a 10 x 10 km spatial resolution. The PM_{2.5} concentration then was matched to ZIP codes based on spatial location and date. Short-term exposure: used the mean of PM_{2.5} on the day of admission and day before admission. Long-term exposure: calculated as the mean exposure in each zip-code across the 7-year study period. Short term exposure was defined as the difference between the two-day average and the long-term average.</p>	<p>Equivalence between Poisson regression and the piecewise constant proportional hazard model to model the time to a hospital admission as a function of both long-term and short-term exposure simultaneously and enabling simultaneously examination of short term and long-term associations with hospital admissions (Hierarchical mixed Poisson regression model). The model adjusts for temperature, age, percent minorities, median income, and percent of people with no high school education.</p>	<p>Daily PM_{2.5} concentration of all grids within the NE area for the acute (0 day lag) and chronic (365 day moving average) were used to calculate overall mean short- and long-term PM_{2.5} exposure respectively, for the study location and period.</p>
---------------------------	---------------	---------------------------	--	--	--	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Kloog et al., 2014	ST	CVD and COPD HA Age 65+	7 U.S. Mid-Atlantic States and D.C.	Case-crossover design (MEDICARE enrollees)	<p>Spatiotemporal model: Used day-specific calibrations of aerosol optical depth (AOD) data, using ground PM_{2.5} measurements. Incorporated land use regression (elevation, distance to major roads, percent of open space, point emissions and area emissions) and meteorological variables (temperature, wind speed, relative humidity, and visibility) for the period of 2000-2006. Model used to predict daily PM_{2.5} concentrations at a 10 x 10 km spatial resolution. Daily predicted PM_{2.5} exposure estimates at grids were matched to zip codes.</p> <p>Average of 2-day lag (lag 0 and 1) PM_{2.5} used.</p>	<p>Conditional logistic regression analysis</p> <p>Temperature with the same moving average as PM_{2.5} was included in the model as a potential confounder. Study design samples only cases and compares each subject's exposure experience in a time period just before a case-defining event with the subject's exposure at other times, eliminating confounding (unmeasured or measured) that do not vary over time. Cases were matched on day of the week and defined the relevant exposure time window as the mean exposure of the day of and day before the patient's hospital admission. Effect modification: 1) assessed whether subject residence within 30 km of a monitor or farther modified the PM_{2.5} association; 2) examined interaction between exposure and income level and gender.</p>	<p>2-day moving average of PM_{2.5} concentration of all grids within the mid-Atlantic states were used to calculate overall mean (all area and rural/urban areas) PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Krall et al., 2013	ST	All-cause mortality	72 Urban U.S. Communities	Time-series study (NCHS)	<p>Monitored data available from US EPA AQS for the period of 2000-2005. Excluded data from source-oriented monitors that may not be representative of typical population exposures. Daily community-level pollutant exposure as the arithmetic mean of daily monitor observations within the community. For communities with single monitor pollutant concentration represented by that monitor.</p> <p>Used lag 1 PM_{2.5} in model.</p>	<p>Log-linear Poisson Regression Model</p> <p>Model adjusted for temperature and previous day's temperature, long-term and seasonal trends, age, and day-of-the-week. Also included interaction term for pollutant concentration and seasons.</p>	<p>Daily PM_{2.5} concentration of 72 US urban communities were used to calculate overall mean PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Krall et al., 2018	ST	ED Visits for CVD (CHF, Cardiac dysrhythmia, IHD, Stroke) or RD (asthma/wheeze, COPD, pneumonia, URI)	Multi-city (5 Metropolitan areas)	Time-series study (Electronic billing of ED visits)	PM _{2.5} concentrations obtained from ambient monitoring stations located within each of the metropolitan areas were fused with Community Multi-Scale Air Quality model estimates (Friberg et al, 2016, 2017) to obtain population-weighted average estimates of the 24-hour average PM _{2.5} concentrations.	Poisson time-series regression model accounting for overdispersion (Peng et al. 2009; Krall et al. 2013) to calculate city specific associations. To calculate overall and posterior city-specific associations, applied Bayesian hierarchical models (Everson and Morris 2000). Adjusted for weekday, season, holidays, metrology, temporal trends.	Daily (24-hr) PM concentrations for specific cities were used to calculate overall PM _{2.5} concentration by city for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Lavigne et al., 2018	ST	Non-accidental, CVD, and respiratory mortality	24 Canadian cities	Case-crossover study	Daily (24-hour) average PM _{2.5} concentrations obtained from monitors in Canada's NAPS network and were used to estimate PM _{2.5} concentrations for the period of 1998-2011. Exposure estimates were assigned to each study participant based on the monitoring station(s) located in participants' city of residence. If PM _{2.5} measurements were available from multiple monitors in a single city, daily concentrations were averaged across monitors.	Conditional logistic regression analysis. Performed stratified analyses examining the relationship between PM _{2.5} and mortality across tertiles of Oxidant capacity.	Daily PM _{2.5} concentrations in 24 Canadian cities were used to calculate overall mean PM _{2.5} concentration over the study location and period.

Lee et al., 2015	ST	All-cause, Cardiovascular, respiratory mortality	3 U.S. Southeast States	Case-crossover design (Dept. of Pub Health data)	<p>Spatio-temporal model that used satellite AOD data to predict daily PM_{2.5} at 1X1 km resolution for the period of 2007-2011. Daily PM_{2.5} concentration at 1km grids were aggregated into the zip code level. For this, 1 km grid cells were matched to zip code area by assigning the centroid of each 1 km grid cell to the centroid of the closest zip code. Zip code areas that contained one or more 1 km grid cells were given the averaged PM_{2.5} and zip codes that were smaller than 1 km² were given the predictions from the closest grid cell.</p> <p>Finally, PM_{2.5} concentrations from zip codes were assigned to the study participants based on their residence zip code and for specific days.</p> <p>For sensitivity: Daily monitored PM_{2.5} concentrations from the nearest EPA and IMPROVE monitors from</p>	<p>Conditional logistic regression</p> <p>Model adjusted for temperature and day of the week</p> <p>Also ran stratified analysis by age, sex, race, education, and primary cause of death.</p> <p>Analysis also restricted for zip codes where annual average of PM_{2.5} <12 or daily average <35 separately.</p> <p>Sensitivity analysis: potential non-linear relationship between temp and mortality modelled using natural spline to the temperature term.</p>	Daily PM _{2.5} concentrations for ZIP code from 2007-2011 were averaged to get overall mean PM _{2.5} (all states and by state)
------------------	----	--	-------------------------	--	---	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					<p>resident zip code (no distance limit) were identified and assigned to individuals.</p> <p>Used lag0 and lag1 in model.</p>		

Lefler et al., 2019	LT	All-cause mortality, Cardiopulmonary mortality	Nationwide	Cohort study (NHIS)	<p>Annual average PM_{2.5} was modeled using regulatory monitors and land use data as described in (Kim, 2018). PM_{2.5} exposure prior to 1999 were estimated using PM10 data. Estimates for each pollutant-year through 2015 were generated at the census-block level using year-2010 Census block centroids. Tract-level estimates for year 2000 Census tracts and year-2010 Census tracts were estimated by mapping year-2010 Census blocks to census tracts and then calculating a population-weighted average of the census blocks within a census tract. PM_{2.5} exposure estimates were assigned to home census tracts as either 2-year (i.e., cohort year and previous year) or 5-year (i.e., cohort year and previous 4 years) average PM_{2.5} concentrations, 17-year average PM_{2.5} concentrations (1999 – 2015), or 28-year average</p>	<p>Cox hazard model 2 versions: Basic PH model, and complex PH model using SURVEYPHREG.</p> <p>Basic model adjusted for age, sex, and race/ethnicity. Complex model adjusted for complex survey design. Both models controlled for marital status, household income, education, smoking status, BMI, urban/rural, census regions and survey year.</p>	<p>Annual PM_{2.5} concentration were for participants were used to calculate overall mean concentration for the 17-year study period 1999-2015.</p>
---------------------	----	--	------------	---------------------	--	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
					PM _{2.5} concentrations (1988 – 2015).		
Lepeule et al., 2012	LT	All-cause, Cardiovascular, lung cancer mortality	HARVARD 6 cities	Prospective Cohort/Longitudinal follow-up study (HARVARD 6 cities data)	PM _{2.5} data from monitors in the participant's city. PM _{2.5} data 1979-1986/1988 from monitors, end of monitoring to 1998 estimated from PM10 using US EPA monitors, 1999-2009 direct PM _{2.5} measurement from US EPA monitors. 1-yr or 1-3yr or 1-5 yr. moving PM _{2.5} averages were assigned to participants based on city of residence.	Cox proportional hazard models, Poisson survival analysis Stratified analysis by sex, age, and time in the study (1-yr interval). Confounders included: Baseline information on smoking status, smoking pack-years, education, linear and quadratic term for BMI. Also explored effect modification of PM _{2.5} on mortality by smoking status at enrollment, as well as time period in study.	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Liu et al., 2019	ST	All-cause and cause-specific mortality	107 U.S. Cities	Time-series study (MCC Collaborative Research Network)	Monitored PM _{2.5} concentration obtained from MCC database for the period of 1987-2006. Hourly data was used to calculate 24-hr daily average. Daily PM _{2.5} concentrations were averaged across stations within each city. Finally, 2-day moving average for the city was calculated. 2-day moving average (lag01) was used in model.	Used two-stage analytic protocol, which had been developed and widely applied in previous multicity time-series studies. First stage estimated city-specific association using quasi-Poisson generalized additive models. Second stage used random-effects models to pool the estimates of the city-specific associations. Two-stage regional analysis was also performed by WHO regions. Also explored the shape of the relationship using C-R curves with PM term appearing with a B-spline function with two knots at 25th and 75th percentiles.	Daily PM _{2.5} concentration were used to calculate overall mean concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Malig et al., 2013	ST	Respiratory morbidity (Asthma and COPD ED and HA)	35 CA counties (9 counties included for PM _{2.5} analysis)	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	PM _{2.5} data obtained from California Air Resources Board. Same day lag and various days lags average were calculated for PM _{2.5} . Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid. Only participants living in zip codes within 20 km of PM _{2.5} monitors were included to increase validity of pollution exposure metrics.	<p>County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis</p> <p>Time-invariant confounders and seasonal trends were controlled for given the study design.</p> <p>Other confounders included in the models were: other gaseous pollutants including ozone, linear and squared term for daily average temperature.</p> <p>Stratified analysis also by distance to monitor within 10 km vs. 10-20 km</p>	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
McConnell et al., 2010	LT	Asthma Incidence	13 CA communities	Cohort Study (CHS)	PM _{2.5} concentration data measured in central site monitors in each community (for 9 of 13 communities since 1994 and others different time period). This study considered 2003-2004 PM _{2.5} measurements at each community monitor. Average annual PM _{2.5} concentration from each community was assigned to study participants based on their community of residence.	Multi-level Cox proportional hazard model accounting for residual variation in time to asthma onset and clustering of children around schools and communities Models adjusted for: secondhand smoke, pets in home, race/ethnicity, age at study entry, sex, and random effects for community and school.	Average annual PM _{2.5} concentrations assigned to study participants were used to calculate overall mean PM _{2.5} exposure for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Ostro et al., 2016	ST	Asthma and COPD ED	8 metropolitan areas/counties in CA	Case-crossover design (CA Office of Statewide Health Planning and Development Data)	PM _{2.5} (24-hour average) data obtained from U.S. EPA provided by California Air Resources Board for the period of 2005-2009. Participants were assigned exposure from the closest monitor from the residential population-weighted zip code centroid. Only participants living in zip codes within 20 km of PM _{2.5} monitors were included to increase validity of pollution exposure metrics. Used lag0, lag1 and lag2 in model.	County-level conditional logistic regression analysis. Overall estimate was then calculated by combining county-level estimates using a random-effects meta-analysis Time-invariant confounders and seasonal trends were controlled for given the study design. Other confounders included in the models were: linear and squared term for lag0 temperature, day of the week.	Daily PM _{2.5} concentrations for all 8 metropolitan counties over the period of 2005-2009 were used to calculate an overall mean PM _{2.5} concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Pappin et al., 2019	LT	Non-accidental mortality	Canada Nationwide	Cohort study (CanCHEC)	PM _{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). For PM _{2.5} concentrations prior to 1998, back casting method employed that applied observed trends in ground monitoring data for PM _{2.5} to adjust pre-gridded PM _{2.5} estimates (Meng, 2019). Annual M2.5 estimates from the postal code and assigned to study participants based on the postal code for residence was used to calculate 3-year moving average based on the location and year of follow-up.	Cox Hazard model and DAG approach. Also performed C-R analysis using a 3 step approaches: (1) fit the data using restricted cubic splines (RCS) with a large number of knots; (2) smooth potential erratic predictions from the large number of knots using monotonically increasing smoothing splines (MISS); and (3) fit the shape constrained health impact function (SCHIF) to the MISS predictions. Cox model stratified by age, sex, and immigration status separately by CanCHEC cohorts. Two covariate adjustment models. First based on DAG and controlled for airshed, urban form, CMA/CA size. Second model "full" model adjusted for individual-level variables (income, education, occupation, marital status).	The annual average PM _{2.5} concentrations were used to calculate overall mean PM _{2.5} concentration for the study cohorts and periods.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Peng et al., 2009	ST	CVD HA Age 65+	119 U.S. Urban counties >150,000 populations	Time-series analysis (MEDICARE enrollees)	<p>PM_{2.5} data (daily or every 3 days) obtained from US EPA's AQS and STN for the period of 2000-2006. Countywide PM_{2.5} total mass concentration was calculated by averaging the daily PM_{2.5} values from all the monitors in a county.</p> <p>Used lag0, lag1 and lag2 in model.</p>	<p>Log-linear Poisson Regression analysis</p> <p>Adjusted for potential confounders including weather, day of the week, unobserved seasonal factors. In county-specific regression model, following indicators were included: indicator for the day of the weeks, a smooth function of time per calendar year to control for seasonality and long-term trends, a smooth function of current-day temperature, a smooth function of the 3-day running mean temperature, a smooth function of current-day dew-point temperature, and a smooth function of the 3-day running mean dew-point temperature. To model smooth functions, we used a natural spline basis.</p>	<p>Daily PM_{2.5} concentrations for all 119 counties over the period of 2000-2006 were used to calculate an overall median PM_{2.5} concentration for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Pinault et al., 2016	LT	All-cause, CVD and lung cancer mortality	Multicity Canada	Prospective Cohort Study (subset of participants of the Canadian Community Health Survey)	<p>PM_{2.5} concentration derived from MODIS. Geographically weighted regression including monitoring and land use data was applied to the estimates from MODIS to produce average PM_{2.5} concentration at 1 km² resolution. These model estimates extended to 1998-2003 using inter-annual variation of Boys et al.</p> <p>Participants were assigned exposure based on their postal code of residence. For each year in the cohort, respondents were assigned a PM_{2.5} concentration corresponding to the mean of the three previous years to the follow-up year.</p>	<p>Cox proportional hazards models</p> <p>Models were stratified by age (5-yr interval) and sex. Models adjusted for individual socioeconomic covariates and behavioral (BMI, smoking and alcohol consumption, fruit, and vegetable consumption) covariates, ecological variables including neighborhood socioeconomic status (both social and material deprivation).</p>	Annual 3-year PM _{2.5} average concentration for the study participants were used to calculate overall PM _{2.5} concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Pinault et al., 2017	LT	Non-accidental, CVD, respiratory and lung cancer mortality	Canada Nationwide	Cohort study (CanCHE C)	PM _{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). PM _{2.5} concentrations extended back to 1998 by applying interannual variation of a publishing PM _{2.5} dataset (Boys, 2014). Annual PM _{2.5} estimates from the postal code was assigned to study participants based on the postal code for residence and used to calculate 3-year moving average based on the location and year of follow-up for years 1998 – 2012.	Cox survival models. Also estimated Shape Constrained Health Impact Functions (a concentration-response function) for selected causes of death. Adjusted for individual demographic and socioeconomic variables at baseline (on Census day): Aboriginal identity, visible minority status, marital status, educational attainment, income adequacy quintile, and labor force status, and contextual variables at the census division scale.	The annual 3-year moving average PM _{2.5} concentrations for study participants were used to calculate overall mean PM _{2.5} concentration for the study period

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Pinault et al., 2018	LT	CVD mortality	Canada Nationwide	Cohort study (CanCHEC, mCHHS)	PM _{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). PM _{2.5} concentrations extended back to 1998 by applying interannual variation of a publishing PM _{2.5} dataset (Boys, 2014). Annual PM _{2.5} estimates from the postal code was assigned to study participants based on the postal code for residence and used to calculate 3-year moving average based on the location and year of follow-up for years 1998 – 2012.	Cox proportional hazard models. Considered co-occurring diabetes with and without other contributing causes of death: hypertension, dementia or Alzheimer's disease, and chronic kidney disease, as these comorbidities are medically related to diabetes. Also considered diabetes status at baseline as effect modifier using CCHS-mortality cohort. Adjusted model for individual-level variables (aboriginal identity, visible minority status, education, labor force status and income adequacy), and neighborhood-level variables.	The annual 3-year moving average PM _{2.5} concentrations for study participants were used to calculate overall mean PM _{2.5} concentration for the study period

Pope et al., 2015b	LT	All-cause, IHD mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	Monthly exposure to PM _{2.5} was estimated by linking geocoded home addresses of the study participants to ambient PM _{2.5} concentrations derived using a national-level hybrid land use regression (LUR) and Bayesian Maximum Entropy (BME) interpolation model (LUR-BME) that incorporated data from ground-based monitors for the study period of 1999-2004.	Cox proportional hazards models The individual-level covariates incorporated in the models included 13 variables that characterized current and former smoking habits (including smoking status of never, former, or current smoker, linear and squared terms for years smoked, and cigarettes smoked per day, indicator for starting smoking at aged <18 years, and pipe/cigar smoker). 1 continuous variable that assessed exposure to second-hand cigarette smoke (hours/d exposed); 7 variables that reflected workplace PM _{2.5} exposure in each subject's main lifetime occupation; a variable that indicated self-reported exposure to dust and fumes in the workplace; variables that represented marital status (separated/divorced/widowed or single versus married); variables that characterized the level	Monthly mean PM _{2.5} concentration for study participants were used to calculate overall mean concentration for the study period.
--------------------	----	--------------------------------	-----------------	---	--	--	---

						<p>of education (high school, more than high school versus less than high school); 2 body mass index variables (linear and squared terms for body mass index); variables that characterized the consumption of alcohol (beer, missing beer, wine, missing wine, liquor, and missing liquor); and variables that indicated quartile ranges of dietary fat index and quartile ranges of a dietary vegetable/fruit/fiber index.</p> <p>Ecological covariates included median household income; percentage of people with <125% of poverty-level income; percentage of unemployed individual aged ≥16 years; percentage of adults with <12th grade education; and percentage of the population who were black or Hispanic. These ecological covariates were included in the models using both zip code level data and zip code deviations from</p>	
--	--	--	--	--	--	--	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
						the county means.	
Pope et al., 2019	LT	All-cause and cause-specific mortality	Nationwide	Cohort study	PM _{2.5} concentration estimated for census block using regulatory monitoring data from 1999-2015 within a universal kriging framework employing land-use regression methods and other variables (Kim 2018). Pop-weighted annual averages were calculated for all 17 years for each 2000 and 2010 census tract. Individual were assigned air pollution conc based on their census tract of residency at the time of survey, e.g.: using year-2000 census tract for individuals surveyed 1986-2010 and using year-2010 census tract for individuals surveyed 2011-2014. For primary analysis: PM _{2.5} exposure is an average concentration over the 17 yrs.	<p>Cox Hazard models. Ran 2 models: one accounting for complex survey design and sampling strategy including sample weights (SURVEYPHREG) and another without accounting for complex survey design (PHREG). Ran model using full-cohort and sub-cohort with additional data on BMI and smoking. The shape of the PM_{2.5}-mortality relationship was also explored using an integrated modeling approach.</p> <p>Adjusted for age, sex, and race-ethnicity, income inflation-adjusted to 2015, education levels, marital status, urban vs rural, US regions, survey years, smoking status.</p>	Annual PM _{2.5} average concentrations over the 17-years (1999-2015) were used to calculate overall PM _{2.5} concentration.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Shi et al., 2016	ST and LT	Total mortality (65+)	New England Area with 6 U.S. States	Open Cohort study (MEDICARE enrollees)	<p>Daily PM_{2.5} for the New England area was predicted at 1-km² spatial resolution from novel 3-stage statistical models for the period of 2003-2008.</p> <p>365-day moving average (for long-term exposure) and average lag0-1 (for short-term exposure) were calculated for each grid cell. The long-term and short-term averages at grid-cells were matched to ZIP codes by linking the ZIP code centroid to the nearest PM_{2.5} grid. Participants were assigned PM_{2.5} concentrations based on the ZIP codes of residence.</p> <p>Used lag0-1 average for short-term exposure analysis in model.</p>	<p>Chronic effects of air pollution assessed using Cox proportional hazard models. Acute effects of air pollution assessed using Poisson log-linear models. Both acute and chronic effects were assessed using Poisson survival analysis. Analysis performed in full-cohort as well as low exposure cohorts.</p> <p>Poisson survival models were adjusted for smooth function of time, temporal covariates such as temperatures and day of the week, spatial covariates such as zip code-level socio-economic variables.</p>	<p>Long-term average: Average annual PM_{2.5} concentrations of all grid cells in the study area were used to calculate overall mean PM_{2.5} exposure for the study location and period.</p> <p>Short-term average: Lag01 PM_{2.5} concentrations of all grid cells in the study area were used to calculate overall mean PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Shin et al., 2019	LT	AF and Stroke (1 st HA)	Ontario, Canada	Cohort study (ONPHEC)	PM _{2.5} concentrations estimated using AOD and PM _{2.5} simulated by the GEOS-Chem chemical transport model (i.e. individual's exposure in 2001 was estimated as mean exposure from 1996-2000). Final surface with 1 × 1 km resolution was generated for Ontario. Annual PM _{2.5} estimates was calculated for the postal code and assigned to study participants based on the postal code for residence. PM _{2.5} concentration was used to calculate 5-year moving average based on the location and year of follow-up.	Cox proportional hazards models. Adjusted for individual-level variables (age and sex), neighborhood-level SES variables, and geographic indicators.	The 5-year moving average PM _{2.5} concentrations for study participants were used to calculate overall mean PM _{2.5} concentration for the study period

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Shin et al., 2021	ST	All-cause hospitalization and all-cause mortality	22 Canadian cities	Time series study (Statistics Canada)	Daily (24-hour) average PM _{2.5} concentrations were calculated for each study city using ambient monitoring data available from Canada's NAPS for the period 2001-2012. Daily PM _{2.5} concentrations were averaged across monitors within a city when multiple monitoring sites were present.	Generalized additive Poisson model and Bayesian hierarchical model. Static approach to estimate the nationwide overall associations between air pollution and health outcomes for all years combined. A two-stage hierarchical model was employed: firstly, a generalized additive Poisson model for city-specific associations between individual health outcomes and individual air pollutants, respectively, and secondly a Bayesian random effects model to combine the city-specific associations to obtain nationwide associations.	Daily PM _{2.5} concentrations of 22 Canadian cities were used to calculate overall mean PM _{2.5} concentration for the study location and period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Stieb et al., 2009	ST	Cardiac and Respiratory ED visits	Seven Canadian Cities	Time series study (Hospital cases)	PM data obtained from National Air Pollution Surveillance (NAPS) system for the period of the 1990s and early 2000s. City averages of the PM _{2.5} exposure were calculated by averaging all monitoring stations within the city. Used lag 0, 1 and 2, in model.	Generalized Linear Models with natural spline functions of time to adjust for seasonal cycles in air pollution and health Confounders included: Mean daily temperature and relative humidity at lag 0, 1, and 2 days, day of the week and holidays.	Daily PM _{2.5} concentrations of the cities were used to calculate the overall mean PM _{2.5} exposure for the study location (by site) and study period.
Sun et al., 2019	ST	Incident stroke: Total, HS and IS (self-reported)	Nationwide	Time-stratified case-crossover (WHI)	PM _{2.5} concentration obtained from log-normal ordinary kriging model as previously described (Liao et al., 2006). This model estimates daily air pollutants at each address based on weighted average of measurement from nearby monitors (Legendre and Fortin, 1989). Daily mean PM _{2.5} concentrations were estimated at geocoded participant address for the period of 1993-2012.	Conditional logistic regression. Adjusted for time-varying variables (daily mean ambient temperature, dew point temperature, and relative humidity) Various lags assessed (1-day moving average to 6-day moving average)	Daily PM _{2.5} concentrations on the case days for the participants were used to calculate the overall mean PM _{2.5} concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Szyszkowicz, 2009	ST	Angina ED	Seven Canadian Cities	Time series study (Hospital cases)	<p>PM data obtained from National Air Pollution Surveillance (NAPS) system. City averages of the exposure were calculated by averaging stations within the city.</p> <p>Used lag 0, 1 and 2, in model.</p>	<p>Generalized Linear Mixed models</p> <p>Models adjusted for meteorological variables such as relative humidity, temperature, and atmospheric pressure (a daily 24-hr average measurements were calculated). Temperature and relative humidity in models were represented by natural splines. Stratified analysis by season as well as combined for the whole period.</p>	<p>Daily PM_{2.5} concentrations of the cities were used to calculate the overall mean PM_{2.5} exposure for the study location (all and by cities) and study period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Thurston et al., 2016a	LT	All-cause, CVD and respiratory mortality	6 U.S. States and 2 MSAs	Cohort study (NIH_AARP cohort)	PM data obtained from US EPA AQS for the period of 2000-2008. Census-tract estimates were generated using hybrid LUR and BME models, which were combined to generate monthly estimates of PM _{2.5} . Participants exposure was estimated at census-tract of residence and included annual mean concentration in prior year of mortality.	Cox proportional hazard models Stratified analysis by age, sex, regions (6 states and 2 MSAs). Confounders adjusted included: race, education, marital status, BMI, alcohol consumption, smoking history, contextual variables such as median household income and % pop with less than high school education. Several interactions between PM _{2.5} and socio-demographics were also tested.	Average annual PM _{2.5} concentrations of census tract estimates were used to calculate overall mean PM _{2.5} exposure for the study location and period.

Turner et al., 2016	LT	Lung cancer mortality (30+)	U.S. Nationwide	Cohort study (ACS Cancer Prevention Study II)	<p>Estimated PM_{2.5} concentrations were obtained using a national-level hybrid land use regression (LUR) and Bayesian maximum entropy (BME) interpolation model. Monthly PM_{2.5} monitoring data were collected from 1,464 sites from 1999 through 2008, with 10% reserved for cross-validation. The base LUR model that predicted PM_{2.5} concentrations included traffic within 1 km and green space within 100 m³. Residual spatiotemporal variation in PM_{2.5} concentrations was interpolated with a BME interpolation model. The two estimates were then combined. The cross validation R² was approximately 0.79. Mean PM_{2.5} (1999–2004) concentrations were used here.</p>	<p>Cox proportional hazards model</p> <p>Models were adjusted for education; marital status; BMI and BMI squared; cigarette smoking status; cigarettes per day and cigarettes per day squared; years smoked, and years smoked squared; started smoking at younger than 18 years of age; passive smoking (hours); vegetable, fruit, fiber, and fat intake; beer, wine, and liquor consumption; occupational exposures; an occupational dirtiness index; and six sociodemographic ecological covariates at both the postal code and postal code minus county-level mean derived from the 1990 U.S. Census (median household income and percentage of African American residents, Hispanic residents, adults with postsecondary education, unemployment, and poverty).</p> <p>Potential confounding examined by elevation, MSA size, annual average daily maximum air temperature, mean county-level residential radon</p>	
---------------------	----	-----------------------------	-----------------	---	--	---	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
						concentrations, and 1980 percentage of air conditioning.	
Urman et al., 2014	LT	Lung-function decline	8 Southern CA communities/counties	Cohort study (CHS)	Central monitors in each community provided data on air pollutants. Each child was assigned exposure based on the child's resident community.	Linear Regression model Models were adjusted for demographic, socio-economic and anthropometric variables (BMI, height), study community.	
Wang et al., 2017	LT	Total mortality (65+)	7 U.S. Southeast States	Cohort study (MEDICARE enrollees)	Three stage Hybrid model to predict daily PM _{2.5} concentration at 1km X 1km resolution for the period 2000-2013. Annual average of PM _{2.5} for each grid cell calculated and took arithmetic mean of the annual average PM _{2.5} across all grids in each of the zip code tabulation area (ZCTA). Participants were assigned annual averages of PM _{2.5} based on their ZCTA of residence.	Cox Proportional hazard models Models were stratified by age groups, sex, race. Adjusted for variables: year of enrollment, previous admission due to CHF, COPD, MI and diabetes, numbers of days spent in ICU and CCU, state, ZCTA level socio-demographic variables such as % pop below poverty, urbanicity, lower education, median income and median home value, and behavioral variables such as % smokers and obesity at county level. Further model also included yearly mean summer temperature at ZCTA level.	Average annual PM _{2.5} concentrations of ZCTAs were used to calculate overall median PM _{2.5} exposure for the study location (overall and by state), and period (overall and by year).

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Wang et al., 2020	LT	Non-accidental cause-specific mortality (Resp, CVD, cancer)	Nationwide	Cohort study (MEDICARE)	Daily PM _{2.5} was estimated on a 6-km grid using a spatio-temporal model described in (Yanosky, 2014) for the period of 2000-2008. Model inputs included monitored PM _{2.5} , meteorological and geospatial covariates, and traffic-related PM estimated using a Gaussian line-source dispersion model. Medicare beneficiaries were matched to the grid point closest to their ZIP code centroid and PM _{2.5} concentrations were averaged for the 12-month period prior to death.	Cox hazard models. Also fit models using restricted cubic splines (RCS) with three knots to characterize non-linearity. Effect-modification assess for age, sex, race and urbanicity. Adjusted for SES variables.	Annual average PM _{2.5} concentration for participants were used to calculate overall annual mean PM _{2.5} exposure for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Weichenthal et al., 2016c	ST	Asthma and COPD ED	15 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	<p>Daily average concentration of PM_{2.5} collected from fixed-monitoring stations for the period of 2004-2011 in Ontario, which is part of Canada's National Air Pollution Data. PM data obtained from 19 sites located in 15 cities. 2 years of data available for 3 cities and remaining had 5-8 years of daily air pollution data. Case and control days of study participants were assigned PM_{2.5} concentration based on the city of residence and based on monitoring station closest to the population-weighted centroid of each subject's 3-digit postal code (if multiple monitors available in participants city such as Toronto and Hamilton).</p> <p>Various lags assessed: lag0, lag1, lag2 and mean of lag0-2.</p>	<p>Conditional logistic regression models</p> <p>Models adjusted for 3-day mean temperature and relative humidity using cubic splines.</p>	<p>Daily PM_{2.5} concentrations in Ontario over the period of 2004-2011 were used to calculate the overall mean PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Weichenthal et al., 2016b	ST	MI ED	16 cities in Ontario	Case-crossover Design (cases extracted from NACRS database)	<p>Daily average concentration of PM_{2.5} collected from fixed-monitoring stations for the period of 2004-2011 in Ontario, which is part of Canada's National Air Pollution Data. PM data obtained from 20 provincial monitoring sites located in 16 cities. Case and control days of study participants were assigned PM_{2.5} concentration based on the monitoring station closest to the population-weighted centroid of each subject's 3-digit postal code.</p> <p>Various lags assessed: lag0, lag1, lag2 and mean of lag0-2.</p>	<p>Conditional logistic regression models</p> <p>Models adjusted for 3-day mean temperature and relative humidity using cubic splines.</p>	<p>Daily PM_{2.5} concentrations in Ontario over the period of 2004-2011 were used to calculate the overall mean PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Wu et al., 2020	LT	All-cause mortality (65+)	US Nationwide	Cohort study (MEDICARE)	Annual PM _{2.5} exposure. Modeled PM _{2.5} exposure at 1km ² grid cells across the US using well-validated ensemble models (Di et al. 2019a, Di 2019b) for the period of 2000-2016. Daily concentration in grid cells were then averaged to estimate annual concentration at ZIP code and then assigned to individual based on ZIP code of residence.	Five statistical approaches: 2 regression approach (Cox Hazard, Poisson reg); 3 causal inference approach (GPSs) Stratified by individual-level characteristics. Further adjusted for community-level factors such as smoking and BMI, zip code-level census variables and meteorological variables, geographic regions, and calendar years (2000-2016).	Annual average PM _{2.5} concentration for participants were used to calculate overall mean PM _{2.5} concentration for the study period.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Wyatt et al., 2020	ST	All-cause, CVD, RD 30-day hospital readmissions	530 US counties	Case-crossover and Cohort study designs (USRDS hemodialysis patients)	<p>PM_{2.5} concentration estimates from AOD integrated with chemical transport model predictions, meteorology, land use variables for 1 km grid cells (Di,2016). Gridded PM_{2.5} estimates were subsequently converted to population-weighted county-level estimates using 2010 Census tract population values. Daily PM_{2.5} was linked to patient hospitalizations based on the county of their last dialysis visit.</p> <p>Examined Lag 0 and unconstrained distributive lag model.</p>	<p>The relative risks of hospital admissions associated with daily PM_{2.5} were estimated with conditional Poisson models for each of the three health outcomes separately. Cox proportional hazards models were used to assess the relative risk of early (1–7 days post discharge) and late (8–30 days post discharge) readmission associated with daily PM_{2.5} following all-cause and cause-specific index hospitalizations. Cox model adjusted for time-dependent (daily PM_{2.5}, daily temperature, daily RH, and day of the week) and time-independent (patient-specific hospitalization event and county SES) risk factors.</p>	<p>Daily estimates at county-level were used to calculate overall PM_{2.5} concentration for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Yap et al., 2013	ST	Asthma HA	12 CA counties	Time Series study (Hospital admissions)	<p>PM_{2.5} data for the period of 2000-2005 obtained from California Air Resources Board that maintains information from the National Air Monitoring Stations. PM_{2.5} reported was 24-hr average mass concentration based on measurements taken every 1, 3, or 6 days. For counties with more than 1 monitoring site, daily average PM_{2.5} was calculated by taking the average across monitors within the county. Missing values were computed based on data from other monitoring stations.</p> <p>PM at various lags lag0-lag6 were assessed.</p>	<p>Generalized Additive Poisson Regression analysis were run at county-level</p> <p>Models adjusted for: long-term time trends and seasonality, day of the week and smoothing splines within different lags for temperature. Effect modification by single or composite area-based SES assessed.</p>	<p>Daily PM_{2.5} concentrations in 12 CA counties over the period of 2000-2005 were used to calculate the overall mean PM_{2.5} exposure for the study location and period.</p>

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Yazdi et al., 2019	LT	First HA: Stroke, COPD, pneumonia, MI, lung cancer, and HF	7 Southeastern states: FL, AL, MS, GA, NC, SC, and TN	Cohort study (MEDICA RE)	PM _{2.5} concentration estimated from spatio-temporal prediction model at 1-km ² grid cell (Di et al. 2017) for the period of 2000-2012. Daily PM _{2.5} concentrations for grid cells were averaged to create annual PM _{2.5} concentration at zip code level and assigned to study participants based on the zip code of residence	Marginal structural Cox proportional hazards models which was weighted with stabilized IPWs (to approximate a causal model). Adjusted for individual-level variables (sex, race, year, state, Medicaid eligibility), as well as census SES.	NR

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Zanobetti et al., 2009	ST	Heart Failure and MI HA 65+	26 US communities	Time Series study (MEDICARE enrollees data)	<p>PM_{2.5} data obtained from US EPA AQS for the period of 2000-2003. For majority of cities, metropolitan counties encompassed the city and its suburbs but some cities like Boston, Minneapolis-St Paul included multiple counties. Daily PM_{2.5} data available for various monitors were averaged over the county and community (Monitors ranged from 1-4). Before averaging, however, monitors were tested for correlations and those with correlation <0.8 with 2 or more monitor pairs within a county were excluded considering it does not represent exposure for general population.</p> <p>Generated 2-day moving average (lag01) concentration</p>	<p>Poisson regression analysis</p> <p>Models stratified by season. Controlled for long-term trend with natural cubic spline for each season and year, day of the week, three-day average temperature and dew point temperature.</p>	

Zanobetti and Schwartz, 2009	ST	All-cause, CVD and respiratory mortality	112 US cities	Time Series study (NCHS data)	<p>PM_{2.5} data obtained from US EPA AQS (NAMS and SLAMS providing daily PM_{2.5} concentration) for the period of 1999-2005. For majority of cities, counties encompassed the city but some cities like Boston, Atlanta, Washington DC, the city included multiple counties. Daily PM_{2.5} (24-hr) data available for various monitors were averaged over the county and city. Before averaging, however, monitors were tested for correlations and those with correlation <0.8 with 2 or more monitor pairs within a county were excluded considering it does not represent exposure for general population. Used standardized method to fill in the missing data in some monitors with at least 265 days of data in at least one year.</p> <p>Generated 2-day moving average (lag 01) concentration</p>	<p>Poisson regression analysis</p> <p>First city- and season-specific Poisson regression was run, and then city-specific estimates were combined using random effects approach in total by season and region.</p> <p>Controlled for long-term trend with natural cubic spline for each season and year, day of the week, same day, and previous day temperature.</p>	Daily PM _{2.5} concentrations in 112 US cities over the period of 1999-2005 were used to calculate the overall mean PM _{2.5} exposure for the study location and period???
------------------------------	----	--	---------------	-------------------------------	---	--	--

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Zanobetti et al., 2014	ST	All-cause mortality 65+	121 US communities/cities	Case-Crossover Design (MEDICARE enrollees)	PM _{2.5} data obtained from US EPA AQS. Daily PM _{2.5} data available for various monitors were averaged over the communities. Participants were assigned 2-day moving average (lag 0 and 1) based on community of residence.	<p>Conditional logistic regression models at community level. In a second stage of analysis, the community specific results were combined using the multivariate meta-analysis techniques</p> <p>Conditional logistic regression controlled for confounders such as average temp for the same and previous day. Temperature was modelled using spline to account for nonlinear relationship. Effect modification tested for cause of prior admission due to neurological disorders or diabetes, primary or secondary hospitalization for other disease conditions. Stratified analysis by sex, age, or race.</p>	

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Zeger et al., 2008	LT	All-cause mortality 65+	668 U.S Urban counties	Retrospective Cohort Study of MEDICARE enrollees (MCAPS)	PM _{2.5} data (every 6 th day at many locations) available from US EPA's AirData Database for the period of 2000-2005. Calculated mean annual PM _{2.5} concentration for all 4,568 ZIP code centroids within 6 miles of a monitor with >10 months of data per year. Given the focus of study on long-term exposure, ZIP code 6-year average of PM _{2.5} was calculated and assigned to study participants living within a zip code both during the 6 years of follow-up and some time before cohort enrollment.	Log-linear Regression model ran for specific US regions separately Models adjusted for individual socio-demographic variables and ZIP code level SES variables (education, income, poverty etc.). Also included standardized mortality ratio for COPD as a surrogate indicator of long-term smoking pattern of its residents.	Average annual PM _{2.5} concentrations of ZIP codes were used to calculate overall mean PM _{2.5} exposure for the study location (all and by region) for the study period 2000-2005.

Citation	Long-term (LT)/Short-term (ST)	Health Endpoint	Geographic Area	Study Design	Exposure Metric	Statistical Analysis Including Confounding Variables Addressed	Calculation of study reported mean PM _{2.5} concentrations
Zhang et al., 2021	LT	Non-accidental, CVD and respiratory mortality	Ontario, Canada	Cohort study (Ontario Health Study)	PM _{2.5} exposures derived from AOD retrievals using GEOS-Chem calibrated to surface measurements by GWR (van Donkelaar, 2015). PM _{2.5} estimates at 1 km ² were used to estimate annual PM _{2.5} average and then 3-year and 5-year moving averages. These annual estimates were then assigned to participants based on postal code of residence (updated annually to account for residential mobility).	Cox proportional hazard models. Basic model stratified by age, sex, ethnicity, enrollment year to control for baseline risks. Models were adjusted for born in Canada, education, marital status, household income, BMI, fruits, and vegetable intake, smoking and drinking, physical activity, urban/rural, and various neighborhood level SES indicators.	The 5-year average PM _{2.5} concentrations were used to calculate overall mean PM _{2.5} concentration for the baseline year.

1

1 REFERENCES

- 2 Bai, L, Shin, S, Burnett, RT, Kwong, JC, Hystad, P, van Donkelaar, A, Goldberg, MS, Lavigne,
3 E, Copes, R, Martin, RV, Kopp, A and Chen, H (2019). Exposure to ambient air pollution
4 and the incidence of congestive heart failure and acute myocardial infarction: A
5 population-based study of 5.1 million Canadian adults living in Ontario. *Environment*
6 *International* 132: 105004.
- 7 Baxter, LK, Crooks, JL and Sacks, JD (2017). Influence of exposure differences on city-to-city
8 heterogeneity in PM_{2.5}-mortality associations in US cities. *Environmental Health: A*
9 *Global Access Science Source* 16(1): 1.
- 10 Bell, ML, Ebisu, K, Leaderer, BP, Gent, JF, Lee, HJ, Koutrakis, P, Wang, Y, Dominici, F and
11 Peng, RD (2014). Associations of PM_{2.5} constituents and sources with hospital
12 admissions: analysis of four counties in Connecticut and Massachusetts (USA) for
13 persons ≥ 65 years of age. *Environmental Health Perspectives* 122(2): 138-144.
- 14 Bell, ML, Ebisu, K, Peng, RD, Walker, J, Samet, JM, Zeger, SL and Dominic, F (2008).
15 Seasonal and regional short-term effects of fine particles on hospital admissions in 202
16 U.S. counties, 1999-2005. *American Journal of Epidemiology* 168(11): 1301-1310.
- 17 Bell, ML, Son, JY, Peng, RD, Wang, Y and Dominici, F (2015). Brief report: Ambient PM_{2.5} and
18 risk of hospital admissions: do risks differ for men and women? *Epidemiology* 26(4):
19 575-579.
- 20 Bravo, MA, Ebisu, K, Dominici, F, Wang, Y, Peng, RD and Bell, ML (2017). Airborne fine
21 particles and risk of hospital admissions for understudied populations: Effects by
22 urbanicity and short-term cumulative exposures in 708 U.S. counties. *Environmental*
23 *Health Perspectives* 125(4): 594-601.
- 24 Burnett, RT and Goldberg, MS (2003). Size-fractionated particulate mass and daily mortality in
25 eight Canadian cities. Health Effects Institute. Boston, MA.
- 26 Burnett, RT, Stieb, D, Brook, JR, Cakmak, S, Dales, R, Raizenne, M, Vincent, R and Dann, T
27 (2004). Associations between short-term changes in nitrogen dioxide and mortality in
28 Canadian cities. *Archives of Environmental and Occupational Health* 59(5): 228-236.
- 29 Cakmak, S, Hebbern, C, Pinault, L, Lavigne, E, Vanos, J, Crouse, DL and Tjepkema, M (2018).
30 Associations between long-term PM_{2.5} and ozone exposure and mortality in the
31 Canadian Census Health and Environment Cohort (CANHEC), by spatial synoptic
32 classification zone. *Environment International* 111: 200-211.
- 33 Chan, E. (2019). Email and attachment from Elizabeth Chan, EPA, Question re: Request for
34 additional PM_{2.5} information from Di et al 2017. Available in PM NAAQS Review
35 Docket (EPA-HQ-OAR-2015-0072). December 3, 2019. Office of Air Quality Planning
36 and Standards Research Triangle Park, NC.

- 1 Chen, H, Burnett, RT, Copes, R, Kwong, JC, Villeneuve, PJ, Goldberg, MS, Brook, RD, van
2 Donkelaar, A, Jerrett, M, Martin, RV, Brook, JR, Kopp, A and Tu, JV (2016). Ambient
3 fine particulate matter and mortality among survivors of myocardial infarction:
4 population-based cohort study. *Environmental Health Perspectives* 124(9): 1421-1428.
- 5 Chen, H, Zhang, ZL, van Donkelaar, A, Bai, L, Martin, RV, Lavigne, E, Kwong, JC and Burnett,
6 RT (2020). Understanding the Joint Impacts of Fine Particulate Matter Concentration and
7 Composition on the Incidence and Mortality of Cardiovascular Disease: A Component-
8 Adjusted Approach. *Environmental Science and Technology* 54(7): 4388-4399.
- 9 Christidis, T, Erickson, AC, Pappin, AJ, Crouse, DL, Pinault, LL, Weichenthal, SA, Brook, JR,
10 van Donkelaar, A, Hystad, P, Martin, RV, Tjepkema, M, Burnett, RT and Brauer, M
11 (2019). Low concentrations of fine particle air pollution and mortality in the Canadian
12 Community Health Survey cohort. *Environmental Health: A Global Access Science*
13 *Source* 18(1): 84.
- 14 Crouse, DL, Peters, PA, Hystad, P, Brook, JR, van Donkelaar, A, Martin, RV, Villeneuve, PJ,
15 Jerrett, M, Goldberg, MS, Pope, CA, Brauer, M, Brook, RD, Robichaud, A, Menard, R
16 and Burnett, RT (2015). Ambient PM 2.5, O₃, and NO₂ exposures and associations with
17 mortality over 16 years of follow-up in the Canadian Census Health and Environment
18 Cohort (CanCHEC). *Environmental Health Perspectives* 123(11): 1180-1186.
- 19 Crouse, DL, Peters, PA, van Donkelaar, A, Goldberg, MS, Villeneuve, PJ, Brion, O, Khan, S,
20 Atari, DO, Jerrett, M, Pope, CA, Brauer, M, Brook, JR, Martin, RV, Stieb, D and
21 Burnett, RT (2012). Risk of nonaccidental and cardiovascular mortality in relation to
22 long-term exposure to low concentrations of fine particulate matter: a Canadian national-
23 level cohort study. *Environmental Health Perspectives* 120(5): 708-714.
- 24 Crouse, DL, Pinault, L, Balram, A, Brauer, M, Burnett, RT, Martin, RV, van Donkelaar, A,
25 Villeneuve, PJ and Weichenthal, S (2019). Complex relationships between greenness, air
26 pollution, and mortality in a population-based Canadian cohort. *Environment*
27 *International* 128: 292-300.
- 28 Dai, L, Zanobetti, A, Koutrakis, P and Schwartz, JD (2014). Associations of fine particulate
29 matter species with mortality in the United States: a multicity time-series analysis.
30 *Environmental Health Perspectives* 122(8): 837-842.
- 31 deSouza, P, Braun, D, Parks, RM, Schwartz, J, Dominici, F and Kioumourtzoglou, MA (2021).
32 Nationwide Study of Short-term Exposure to Fine Particulate Matter and Cardiovascular
33 Hospitalizations Among Medicaid Enrollees. *Epidemiology* 32(1): 6-13.
- 34 Di, Q, Dai, L, Wang, Y, Zanobetti, A, Choirat, C, Schwartz, JD and Dominici, F (2017a).
35 Association of short-term exposure to air pollution with mortality in older adults. *JAMA:*
36 *Journal of the American Medical Association* 318(24): 2446-2456.
- 37 Di, Q, Wang, Y, Zanobetti, A, Wang, Y, Koutrakis, P, Choirat, C, Dominici, F and Schwartz, JD
38 (2017b). Air pollution and mortality in the Medicare population. *New England Journal of*
39 *Medicine* 376(26): 2513-2522.

- 1 Dominici, F, Peng, RD, Bell, ML, Pham, L, McDermott, A, Zeger, SL and Samet, JL (2006).
2 Fine particulate air pollution and hospital admission for cardiovascular and respiratory
3 diseases. *JAMA: Journal of the American Medical Association* 295(10): 1127-1134.
- 4 Dominici, F, Schwartz, J, Di, Q, Braun, D, Choirat, C and Zanobetti, A (2019). Assessing
5 adverse health effects of long-term exposure to low levels of ambient air pollution: Phase
6 1. Health Effects Institute. Boston, MA.
7 <https://www.healtheffects.org/system/files/dominici-rr-200-report.pdf>.
- 8 Elliott, EG, Laden, F, James, P, Rimm, EB, Rexrode, KM and Hart, JE (2020). Interaction
9 between Long-Term Exposure to Fine Particulate Matter and Physical Activity, and Risk
10 of Cardiovascular Disease and Overall Mortality in US Women. *Environmental Health
11 Perspectives* 128(12): 127012.
- 12 Erickson, AC, Brauer, M, Christidis, T, Pinault, L, Crouse, DL, van Donkelaar, A, Weichenthal,
13 S, Pappin, A, Tjepkema, M, Martin, RV, Brook, JR, Hystad, P and Burnett, RT (2019).
14 Evaluation of a method to indirectly adjust for unmeasured covariates in the association
15 between fine particulate matter and mortality. *Environmental Research* 175: 108-116.
- 16 Erickson, AC, Christidis, T, Pappin, A, Brook, JR, Crouse, DL, Hystad, P, Li, C, Martin, RV,
17 Meng, J, Pinault, L, von Donkelaar, A, Weichenthal, S, Tjepkema, M, Burnett, RT and
18 Brauer, M (2020). Disease assimilation: The mortality impacts of fine particulate matter
19 on immigrants to Canada. *Health Reports* 31(3): 14-26.
- 20 Eum, KD, Suh, HH, Pun, VC and Manjourides, J (2018). Impact of long-term temporal trends in
21 fine particulate matter (PM_{2.5}) on associations of annual PM_{2.5} exposure and mortality:
22 An analysis of over 20 million Medicare beneficiaries. *Environmental Epidemiology
23* 2(2): e009.
- 24 Fisher, JA, Puett, RC, Laden, F, Wellenius, GA, Sapkota, A, Liao, D, Yanosky, JD, Carter-
25 Pokras, O, He, X and Hart, JE (2019). Case-crossover analysis of short-term particulate
26 matter exposures and stroke in the health professionals follow-up study. *Environment
27 International* 124: 153-160.
- 28 Franklin, M, Koutrakis, P and Schwartz, J (2008). The role of particle composition on the
29 association between PM_{2.5} and mortality. *Epidemiology* 19(5): 680-689.
- 30 Franklin, M, Zeka, A and Schwartz, J (2007). Association between PM_{2.5} and all-cause and
31 specific-cause mortality in 27 US communities. *Journal of Exposure Science and
32 Environmental Epidemiology* 17(3): 279-287.
- 33 Gharibvand, L, Shavlik, D, Ghamsary, M, Beeson, WL, Soret, S, Knutsen, R and Knutsen, SF
34 (2016). The association between ambient fine particulate air pollution and lung cancer
35 incidence: Results from the AHSMOG-2 study. *Environmental Health Perspectives
36* 125(3): 378-384.

- 1 Hart, JE, Garshick, E, Dockery, DW, Smith, TJ, Ryan, L and Laden, F (2011). Long-term
2 ambient multi-pollutant exposures and mortality. *American Journal of Respiratory and*
3 *Critical Care Medicine* 183(1): 73-78.
- 4 Hart, JE, Liao, X, Hong, B, Puett, RC, Yanosky, JD, Suh, H, Kioumourtzoglou, MA,
5 Spiegelman, D and Laden, F (2015). The association of long-term exposure to PM_{2.5} on
6 all-cause mortality in the Nurses' Health Study and the impact of measurement-error
7 correction. *Environmental Health: A Global Access Science Source* 14: 38.
- 8 Hayes, RB, Lim, C, Zhang, YL, Cromar, K, Shao, YZ, Reynolds, HR, Silverman, DT, Jones,
9 RR, Park, Y, Jerrett, M, Ahn, J and Thurston, GD (2020). PM_{2.5} air pollution and cause-
10 specific cardiovascular disease mortality. *International Journal of Epidemiology* 49(1):
11 25-35.
- 12 Honda, T, Eliot, MN, Eaton, CB, Whitsel, E, Stewart, JD, Mu, L, Suh, H, Szpiro, A, Kaufman,
13 JD, Vedal, S and Wellenius, GA (2017). Long-term exposure to residential ambient fine
14 and coarse particulate matter and incident hypertension in post-menopausal women.
15 *Environment International* 105: 79-85.
- 16 Hystad, P, Demers, PA, Johnson, KC, Brook, J, van Donkelaar, A, Lamsal, L, Martin, R and
17 Brauer, M (2012). Spatiotemporal air pollution exposure assessment for a Canadian
18 population-based lung cancer case-control study. *Environmental Health: A Global Access*
19 *Science Source* 11: 22.
- 20 Hystad, P, Demers, PA, Johnson, KC, Carpiano, RM and Brauer, M (2013). Long-term
21 residential exposure to air pollution and lung cancer risk. *Epidemiology* 24(5): 762-772.
- 22 Ito, K, Ross, Z, Zhou, J, Nádas, A, Lippmann, M and Thurston, GD (2013). National Particle
23 Component Toxicity (NPACT) initiative: Study 3. Time-series analysis of mortality,
24 hospitalizations, and ambient PM_{2.5} and its components. 177. Health Effects Institute.
25 Boston, MA. [https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-](https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-AppendixG_0.pdf)
26 [AppendixG_0.pdf](https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-AppendixG_0.pdf).
- 27 Jenkins, S. (2019a). Email and attachment from Scott Jenkins, EPA, Question re: PM_{2.5} LT
28 estimates Di et al. (2017) Studies. Available in PM NAAQS Review Docket (EPA-HQ-
29 OAR-2015-0072-0022). May 8, 2019. Office of Air Quality Planning and Standards
30 Research Triangle Park, NC.
- 31 Jenkins, S. (2019b). Email and attachment from Scott Jenkins, EPA, Question re: PM_{2.5} ST
32 estimates Di et al. (2017) Studies. Available in PM NAAQS Review Docket (EPA-HQ-
33 OAR-2015-0072-0025). May 9, 2019. Office of Air Quality Planning and Standards
34 Research Triangle Park, NC.
- 35 Jerrett, M, Turner, MC, Beckerman, BS, Pope, CA, van Donkelaar, A, Martin, RV, Serre, M,
36 Crouse, D, Gapstur, SM, Krewski, D, Diver, WR, Coogan, PF, Thurston, GD and
37 Burnett, RT (2016). Comparing the health effects of ambient particulate matter estimated
38 using ground-based versus remote sensing exposure estimates. *Environmental Health*
39 *Perspectives* 125(4): 552-559.

- 1 Kioumourtzoglou, MA, Schwartz, J, James, P, Dominici, F and Zanobetti, A (2016). PM_{2.5} and
2 mortality in 207 us cities: Modification by temperature and city characteristics.
3 *Epidemiology* 27(2): 221-227.
- 4 Klemm, RJ and Mason, R (2003). *HEI Special Report: Revised Analyses of Time-Series Studies*
5 *of Air Pollution and Health, Part II Replication of reanalysis of Harvard Six-City*
6 *mortality study*. Health Effects Institute. Boston, MA.
- 7 Kloog, I, Coull, BA, Zanobetti, A, Koutrakis, P and Schwartz, JD (2012). Acute and chronic
8 effects of particles on hospital admissions in New-England. *PLoS ONE* 7(4): e34664.
- 9 Kloog, I, Nordio, F, Zanobetti, A, Coull, BA, Koutrakis, P and Schwartz, JD (2014). Short term
10 effects of particle exposure on hospital admissions in the Mid-Atlantic states: a
11 population estimate. *PLoS ONE* 9(2): e88578.
- 12 Krall, JR, Anderson, GB, Dominici, F, Bell, ML and Peng, RD (2013). Short-term exposure to
13 particulate matter constituents and mortality in a national study of U.S. urban
14 communities. *Environmental Health Perspectives* 121(10): 1148-1153.
- 15 Krall, JR, Chang, HH, Waller, LA, Mulholland, JA, Winquist, A, Talbott, EO, Rager, JR,
16 Tolbert, PE and Sarnat, SE (2018). A multicity study of air pollution and
17 cardiorespiratory emergency department visits: Comparing approaches for combining
18 estimates across cities. *Environment International* 120: 312-320.
- 19 Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, III,
20 Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, Deluca, P, Finkelstein, N, Ito, K,
21 Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H and Tempalski, B (2009).
22 Extended follow-up and spatial analysis of the American Cancer Society study linking
23 particulate air pollution and mortality. HEI Research Report 140. Health Effects Institute.
24 Boston, MA. <https://www.healtheffects.org/system/files/Krewski140Statement.pdf>.
- 25 Lavigne, E, Burnett, RT and Weichenthal, S (2018). Association of short-term exposure to fine
26 particulate air pollution and mortality: effect modification by oxidant gases. *Scientific*
27 *Reports* 8(1): 16097.
- 28 Lee, H, Honda, Y, Hashizume, M, Guo, YL, Wu, CF, Kan, H, Jung, K, Lim, YH, Yi, S and Kim,
29 H (2015). Short-term exposure to fine and coarse particles and mortality: A multicity
30 time-series study in East Asia. *Environ Pollut* 207: 43-51.
- 31 Lefler, JS, Higbee, JD, Burnett, RT, Ezzati, M, Coleman, NC, Mann, DD, Marshall, JD, Bechle,
32 M, Wang, Y, Robinson, AL and Arden Pope, C (2019). Air pollution and mortality in a
33 large, representative U.S. cohort: multiple-pollutant analyses, and spatial and temporal
34 decompositions. *Environmental Health: A Global Access Science Source* 18(1): 101.
- 35 Lepeule, J, Laden, F, Dockery, D and Schwartz, J (2012). Chronic exposure to fine particles and
36 mortality: an extended follow-up of the Harvard Six Cities study from 1974 to 2009.
37 *Environmental Health Perspectives* 120(7): 965-970.

- 1 Lippmann, M, Chen, LC, Gordon, T, Ito, K and Thurston, GD (2013). National Particulate
2 Component Toxicity (NPACT) Initiative: Integrated epidemiologic and toxicologic
3 studies of the health effects of particulate matter components. 177. Health Effects
4 Institute. Boston, MA. <https://www.healtheffects.org/system/files/RR177-Lippmann.pdf>.
- 5 Liu, C, Chen, R, Sera, F, Vicedo-Cabrera, AM, Guo, Y, Tong, S, Coelho, M, Saldiva, PHN,
6 Lavigne, E, Matus, P, Valdes Ortega, N, Osorio Garcia, S, Pascal, M, Stafoggia, M,
7 Scortichini, M, Hashizume, M, Honda, Y, Hurtado-Díaz, M, Cruz, J, Nunes, B, Teixeira,
8 JP, Kim, H, Tobias, A, Íñiguez, C, Forsberg, B, Åström, C, Ragettli, MS, Guo, YL, Chen,
9 BY, Bell, ML, Wright, CY, Scovronick, N, Garland, RM, Milojevic, A, Kysely, J, Urban,
10 A, Orru, H, Indermitte, E, Jaakkola, JJK, Rytí, NRI, Katsouyanni, K, Analitis, A,
11 Zanobetti, A, Schwartz, J, Chen, J, Wu, T, Cohen, A, Gasparrini, A and Kan, H (2019).
12 Ambient Particulate Air Pollution and Daily Mortality in 652 Cities. *New England
13 Journal of Medicine* 381(8): 705-715.
- 14 Loop, MS, McClure, LA, Levitan, EB, Al-Hamdan, MZ, Crosson, WL and Safford, MM (2018).
15 Fine particulate matter and incident coronary heart disease in the REGARDS cohort.
16 *American Heart Journal* 197: 94-102.
- 17 Malig, BJ, Green, S, Basu, R and Broadwin, R (2013). Coarse particles and respiratory
18 emergency department visits in California. *American Journal of Epidemiology* 178(1):
19 58-69.
- 20 McClure, LA, Loop, MS, Crosson, W, Kleindorfer, D, Kissela, B and Al-Hamdan, M (2017).
21 Fine Particulate Matter (PM_{2.5}) and the Risk of Stroke in the REGARDS Cohort. *Journal
22 of Stroke & Cerebrovascular Diseases* 26(8): 1739-1744.
- 23 McConnell, R, Islam, T, Shankardass, K, Jerrett, M, Lurmann, F, Gilliland, F, Gauderman, J,
24 Avol, E, Künzli, N, Yao, L, Peters, J and Berhane, K (2010). Childhood incident asthma
25 and traffic-related air pollution at home and school. *Environmental Health Perspectives*
26 118(7): 1021-1026.
- 27 Miller, KA, Siscovick, DS, Sheppard, L, Shepherd, K, Sullivan, JH, Anderson, GL and
28 Kaufman, JD (2007). Long-term exposure to air pollution and incidence of
29 cardiovascular events in women. *New England Journal of Medicine* 356(5): 447-458.
- 30 Ostro, B, Malig, B, Hasheminassab, S, Berger, K, Chang, E and Sioutas, C (2016). Associations
31 of source-specific fine particulate matter with emergency department visits in California.
32 *American Journal of Epidemiology* 184(6): 450-459.
- 33 Pappin, AJ, Christidis, T, Pinault, LL, Crouse, DL, Brook, JR, Erickson, A, Hystad, P, Li, C,
34 Martin, RV, Meng, J, Weichenthal, S, van Donkelaar, A, Tjepkema, M, Brauer, M and
35 Burnett, RT (2019). Examining the Shape of the Association between Low Levels of Fine
36 Particulate Matter and Mortality across Three Cycles of the Canadian Census Health and
37 Environment Cohort. *Environmental Health Perspectives* 127(10): 107008.

- 1 Parker, JD, Kravets, N and Vaidyanathan, A (2018). Particulate matter air pollution exposure and
2 heart disease mortality risks by race and ethnicity in the United States: 1997-2009 NHIS
3 with mortality followup through 2011. *Circulation* 137(16): 1688-1697.
- 4 Peng, RD, Dominici, F and Welty, LJ (2009). A Bayesian hierarchical distributed lag model for
5 estimating the time course of risk of hospitalization associated with particulate matter air
6 pollution. *J Roy Stat Soc Ser C (Appl Stat)* 58: 3-24.
- 7 Pinault, L, Brauer, M, Crouse, DL, Weichenthal, S, Erickson, A, van Donkelaar, A, Martin, RV,
8 Charbonneau, S, Hystad, P, Brook, JR, Tjepkema, M, Christidis, T, Ménard, R,
9 Robichaud, A and Burnett, RT (2018). Diabetes Status and Susceptibility to the Effects of
10 PM2.5 Exposure on Cardiovascular Mortality in a National Canadian Cohort.
11 *Epidemiology* 29(6): 784-794.
- 12 Pinault, L, Tjepkema, M, Crouse, DL, Weichenthal, S, van Donkelaar, A, Martin, RV, Brauer,
13 M, Chen, H and Burnett, RT (2016). Risk estimates of mortality attributed to low
14 concentrations of ambient fine particulate matter in the Canadian community health
15 survey cohort. *Environmental Health: A Global Access Science Source* 15(1): 18.
- 16 Pinault, LL, Weichenthal, S, Crouse, DL, Brauer, M, Erickson, A, Donkelaar, AV, Martin, RV,
17 Hystad, P, Chen, H, Finès, P, Brook, JR, Tjepkema, M and Burnett, RT (2017).
18 Associations between fine particulate matter and mortality in the 2001 Canadian Census
19 Health and Environment Cohort. *Environmental Research* 159: 406-415.
- 20 Pope, CA, III, Muhlestein, JB, Anderson, JL, Cannon, JB, Hales, NM, Meredith, KG, Viet, L
21 and Horne, BD (2015a). Short-Term Exposure to Fine Particulate Matter Air Pollution Is
22 Preferentially Associated With the Risk of ST-Segment Elevation Acute Coronary
23 Events. *Journal of the American Heart Association* 4(12).
- 24 Pope, CA, Lefler, JS, Ezzati, M, Higbee, JD, Marshall, JD, Kim, SY, Bechle, M, Gilliat, KS,
25 Vernon, SE, Robinson, AL and Burnett, RT (2019). Mortality Risk and Fine Particulate
26 Air Pollution in a Large, Representative Cohort of U.S. Adults. *Environmental Health
27 Perspectives* 127(7): 77007.
- 28 Pope, CA, Turner, MC, Burnett, R, Jerrett, M, Gapstur, SM, Diver, WR, Krewski, D and Brook,
29 RD (2015b). Relationships between fine particulate air pollution, cardiometabolic
30 disorders and cardiovascular mortality. *Circulation Research* 116(1): 108-U258.
- 31 Puett, RC, Hart, JE, Suh, H, Mittleman, M and Laden, F (2011). Particulate matter exposures,
32 mortality and cardiovascular disease in the health professionals follow-up study.
33 *Environmental Health Perspectives* 119(8): 1130-1135.
- 34 Puett, RC, Hart, JE, Yanosky, JD, Paciorek, C, Schwartz, J, Suh, H, Speizer, FE and Laden, F
35 (2009). Chronic fine and coarse particulate exposure, mortality, and coronary heart
36 disease in the Nurses' Health Study. *Environmental Health Perspectives* 117(11): 1697-
37 1701.

- 1 Rajan, P, Schmidt, M., Hassett-Sipple, B. (2011). Memorandum to PM NAAQS Review Docket
2 (EPA-HQ-OAR-2007-0492). PM_{2.5} Distributional Statistical Analyses. April 7, 2011.
3 Docket ID No. EPA-HQ-OAR-2007-0492. Office of Air Quality Planning and Standards
4 Research Triangle Park, NC. Available at:
5 <https://www3.epa.gov/ttn/naaqs/standards/pm/data/Rajan040711.pdf>.
- 6 Shi, L, Zanobetti, A, Kloog, I, Coull, BA, Koutrakis, P, Melly, SJ and Schwartz, JD (2016).
7 Low-concentration PM_{2.5} and mortality: estimating acute and chronic effects in a
8 population-based study. *Environmental Health Perspectives* 124(1): 46-52.
- 9 Shin, HH, Gogna, P, Maquiling, A, Parajuli, RP, Haque, L and Burr, B (2021). Comparison of
10 hospitalization and mortality associated with short-term exposure to ambient ozone and
11 PM_{2.5} in Canada. *Chemosphere* 265: 128683.
- 12 Shin, S, Burnett, RT, Kwong, JC, Hystad, P, van Donkelaar, A, Brook, JR, Goldberg, MS, Tu, K,
13 Copes, R, Martin, R, Lia, Y, Kopp, A and Chen, H (2019). Ambient Air Pollution and the
14 Risk of Atrial Fibrillation and Stroke: A Population-Based Cohort Study. *Environmental*
15 *Health Perspectives* 127(8): 87009.
- 16 Stieb, DM, Szyszkowicz, M, Rowe, BH and Leech, JA (2009). Air pollution and emergency
17 department visits for cardiac and respiratory conditions: A multi-city time-series analysis.
18 *Environmental Health: A Global Access Science Source* 8(25): 25.
- 19 Sun, S, Stewart, JD, Eliot, MN, Yanosky, JD, Liao, D, Tinker, LF, Eaton, CB, Whitsel, EA and
20 Wellenius, GA (2019). Short-term exposure to air pollution and incidence of stroke in the
21 Women's Health Initiative. *Environment International* 132: 105065.
- 22 Szyszkowicz, M (2009). Air pollution and ED visits for chest pain. *American Journal of*
23 *Emergency Medicine* 27(2): 165-168.
- 24 Thurston, GD, Ahn, J, Cromar, KR, Shao, Y, Reynolds, HR, Jerrett, M, Lim, CC, Shanley, R,
25 Park, Y and Hayes, RB (2016a). Ambient particulate matter air pollution exposure and
26 mortality in the NIH-AARP Diet and Health Cohort. *Environmental Health Perspectives*
27 124(4): 484-490.
- 28 Thurston, GD, Burnett, RT, Turner, MC, Shi, Y, Krewski, D, Lall, R, Ito, K, Jerrett, M, Gapstur,
29 SM, Diver, WR and Pope, CA, III (2016b). Ischemic heart disease mortality and long-
30 term exposure to source-related components of U.S. fine particle air pollution. *Environ*
31 *Health Perspect* 124(6): 785-794.
- 32 Thurston, GD, Ito, K, Lall, R, Burnett, RT, Turner, MC, Krewski, D, Shi, Y, Jerrett, M, Gapstur,
33 SM, Diver, WR and Pope, CA, III (2013). NPACT Study 4. Mortality and long-term
34 exposure to PM_{2.5} and its components in the American Cancer Society's Cancer
35 Prevention Study II Cohort. Research Report 177. Health Effects Institute. Boston, MA.
- 36 Turner, MC, Jerrett, M, Pope, A, III, Krewski, D, Gapstur, SM, Diver, WR, Beckerman, BS,
37 Marshall, JD, Su, J, Crouse, DL and Burnett, RT (2016). Long-term ozone exposure and

- 1 mortality in a large prospective study. American Journal of Respiratory and Critical Care
2 Medicine 193(10): 1134-1142.
- 3 U.S. EPA (2009). Integrated Science Assessment for Particulate Matter (Final Report). Office of
4 Research and Development, National Center for Environmental Assessment. Research
5 Triangle Park, NC. U.S. EPA. EPA-600/R-08-139F. December 2009. Available at:
6 <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546>.
- 7 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
8 U.S. Environmental Protection Agency, Office of Research and Development, National
9 Center for Environmental Assessment. Washington, DC. U.S. EPA. EPA/600/R-19/188.
10 December 2019. Available at: [https://www.epa.gov/naaqs/particulate-matter-pm-
11 standards-integrated-science-assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 12 U.S. EPA (2021). Supplement to the 2019 Integrated Science Assessment for Particulate Matter
13 (External Review Draft). U.S. Environmental Protection Agency, Office of Research and
14 Development, Center for Public Health and Environmental Assessment. Washington,
15 DC. U.S. EPA. EPA/600/R-21/198. December 2019. Available at:
16 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-
17 assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 18 Urman, R, McConnell, R, Islam, T, Avol, EL, Lurmann, FW, Vora, H, Linn, WS, Rappaport,
19 EB, Gilliland, FD and Gauderman, WJ (2014). Associations of children's lung function
20 with ambient air pollution: Joint effects of regional and near-roadway pollutants. Thorax
21 69(6): 540-547.
- 22 Villeneuve, PJ, Weichenthal, SA, Crouse, D, Miller, AB, To, T, Martin, RV, van Donkelaar, A,
23 Wall, C and Burnett, RT (2015). Long-term exposure to fine particulate matter air
24 pollution and mortality among Canadian women. Epidemiology 26(4): 536-545.
- 25 Wang, B, Eum, KD, Kazemiparkouhi, F, Li, C, Manjourides, J, Pavlu, V and Suh, H (2020). The
26 impact of long-term PM_{2.5} exposure on specific causes of death: exposure-response
27 curves and effect modification among 53 million U.S. Medicare beneficiaries.
28 Environmental Health: A Global Access Science Source 19(1): 20.
- 29 Wang, Y, Shi, L, Lee, M, Liu, P, Di, Q, Zanobetti, A and Schwartz, JD (2017). Long-term
30 exposure to PM_{2.5} and mortality among older adults in the Southeastern US.
31 Epidemiology 28(2): 207-214.
- 32 Weichenthal, S, Crouse, DL, Pinault, L, Godri-Pollitt, K, Lavigne, E, Evans, G, van Donkelaar,
33 A, Martin, RV and Burnett, RT (2016a). Oxidative burden of fine particulate air pollution
34 and risk of cause-specific mortality in the Canadian Census Health and Environment
35 Cohort (CanCHEC). Environmental Research 146: 92-99.
- 36 Weichenthal, S, Hatzopoulou, M and Goldberg, MS (2014). Exposure to traffic-related air
37 pollution during physical activity and acute changes in blood pressure, autonomic and
38 micro-vascular function in women: A cross-over study. Particle and Fibre Toxicology
39 11(1): 70.

- 1 Weichenthal, S, Lavigne, E, Evans, G, Pollitt, K and Burnett, RT (2016b). Ambient PM_{2.5} and
2 risk of emergency room visits for myocardial infarction: Impact of regional PM_{2.5}
3 oxidative potential: A case-crossover study. *Environmental Health* 15: 46.
- 4 Weichenthal, S, Lavigne, E, Evans, GJ, Godri Pollitt, KJ and Burnett, RT (2016c). PM_{2.5} and
5 emergency room visits for respiratory illness: effect modification by oxidative potential.
6 *American Journal of Respiratory and Critical Care Medicine* 194(5): 577-586.
- 7 Wu, X, Braun, D, Schwartz, J, Kioumourtzoglou, MA and Dominici, F (2020). Evaluating the
8 impact of long-term exposure to fine particulate matter on mortality among the elderly.
9 *Science Advances* 6(29): eaba5692.
- 10 Wyatt, LH, Xi, YZ, Kshirsagar, A, Di, Q, Ward-Caviness, C, Wade, TJ, Cascio, WE and
11 Rappold, AG (2020). Association of short-term exposure to ambient PM_{2.5} with hospital
12 admissions and 30-day readmissions in end-stage renal disease patients: population-based
13 retrospective cohort study. *British Medical Journal Open* 10(12): e041177.
- 14 Yap, P, Gilbreath, S, Garcia, C, Jareen, N and Goodrich, B (2013). The influence of
15 socioeconomic markers on the association between fine particulate matter and hospital
16 admissions for respiratory conditions among children. *American Journal of Public Health*
17 103(4): 695-702.
- 18 Yazdi, D, Wang, Y, Di, Q, Zanobetti, A and Schwartz, J (2019). Long-term exposure to PM_{2.5}
19 and ozone and hospital admissions of Medicare participants in the Southeast USA.
20 *Environment International* 130: 104879.
- 21 Zanobetti, A, Dominici, F, Wang, Y and Schwartz, JD (2014). A national case-crossover analysis
22 of the short-term effect of PM_{2.5} on hospitalizations and mortality in subjects with
23 diabetes and neurological disorders. *Environmental Health: A Global Access Science*
24 *Source* 13(1): 38.
- 25 Zanobetti, A, Franklin, M, Koutrakis, P and Schwartz, J (2009). Fine particulate air pollution and
26 its components in association with cause-specific emergency admissions. *Environmental*
27 *Health: A Global Access Science Source* 8: 58.
- 28 Zanobetti, A and Schwartz, J (2009). The effect of fine and coarse particulate air pollution on
29 mortality: A national analysis. *Environmental Health Perspectives* 117(6): 1-40.
- 30 Zeger, S, Dominici, F, McDermott, A and Samet, J (2008). Mortality in the Medicare population
31 and chronic exposure to fine particulate air pollution in urban centers (2000-2005).
32 *Environmental Health Perspectives* 116(12): 1614-1619.
- 33 Zhang, Z, Wang, J, Kwong, JC, Burnett, RT, van Donkelaar, A, Hystad, P, Martin, RV, Bai, L,
34 McLaughlin, J and Chen, H (2021). Long-term exposure to air pollution and mortality in
35 a prospective cohort: The Ontario Health Study. *Environment International* 154: 106570.

36

1
2
3
4

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

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28

TABLE OF CONTENTS

C.1	Additional Technical Detail on the Risk Assessment Approach	C-1
C.1.1	Selection of Key Health Endpoints and Specification of Concentration-Response Functions from Epidemiologic Studies	C-2
C.1.2	Specification of Demographic and Baseline Incidence Data Inputs.....	C-11
C.1.3	Study Area Selection.....	C-11
C.1.4	Generation of Air Quality Inputs to the Risk Assessment.....	C-17
C.1.5	Risk Modeling Approach.....	C-46
C.2	Supplemental Risk Results.....	C-48
C.2.1	Results from Full Set of 47 Study Areas	C-50
C.2.2	Results from Set of 30 Study Areas controlled by the Annual Standard.....	C-55
C.2.3	Key Observations from the Supplemental Risk Results.....	C-56
C.3	Additional Technical Detail on the At-Risk Analysis.....	C-57
C.3.1	Race/Ethnicity.....	C-58
C.3.2	Concentration-Response Functions	C-59
C.3.3	Age.....	C-61
C.3.4	Baseline Incidence Rates	C-61
C.3.5	Selection of Air Quality Simulation Approach.....	C-63
C.4	Supplemental At-Risk Results	C-63
C.5	Characterizing Variability and Uncertainty in Risk Estimates	C-70
C.5.1	Quantitative Assessment of Uncertainty.....	C-72
C.5.2	Qualitative Uncertainty Analysis.....	C-73
C.5.3	Conclusion	C-81
C.6	PM _{2.5} Design Values for the Air Quality Projections	C-82
	References.....	C-106

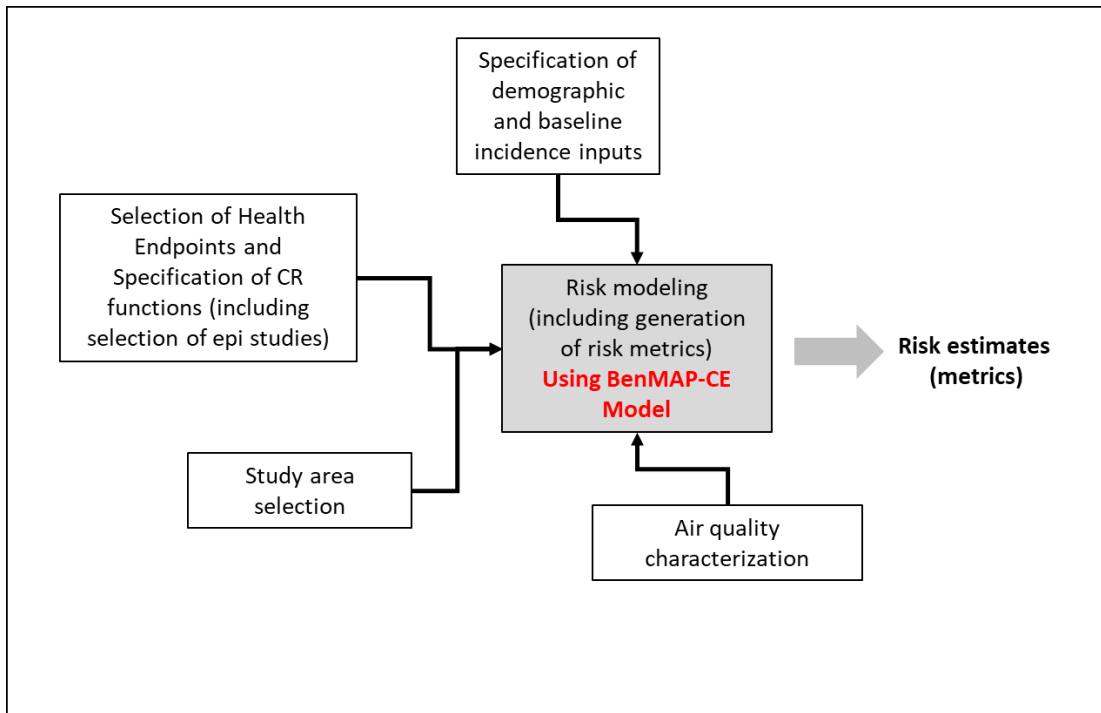
1
2 This appendix provides supplemental information related to the risk assessment described
3 in section 3.4 of this draft PA for the reconsideration of the 2020 final decision on the particulate
4 matter (PM) National Ambient Air Quality Standards (NAAQS), including:

- 5 • Additional technical detail on the risk assessment approach, including sources and
6 derivation of key inputs to the risk modeling process (section C.1).
- 7 • Supplemental risk results (section C.2) intended to provide additional context for the
8 summary risk estimates presented in sections 3.4.2.1-3.4.2.3.
- 9 • Additional technical detail on the at-risk analytic approach, including sources and
10 derivation of key inputs to the risk modeling process (section C.3).
- 11 • Supplemental at-risk analytics (section C.3.4.2) intended to provide additional context for
12 the summary risk estimates presented in section 3.4.2.4.
- 13 • Characterization of variability and uncertainty related to the risk assessment (section C.5)
14 intended to provide additional context for section 3.4.2.5.

16 **C.1 ADDITIONAL TECHNICAL DETAIL ON THE RISK ASSESSMENT** 17 **APPROACH**

18 As discussed in section 3.4, our general approach to estimating PM_{2.5}-associated human
19 health risks in this reconsideration utilizes concentration-response (CR) functions obtained from
20 epidemiologic studies to link ambient PM_{2.5} exposure to risk in the form of mortality incidence
21 (counts). The derivation and use of this type of CR function in modeling PM_{2.5}-attributable risk
22 is well documented both in previous PM NAAQS-related risk assessments (section 3.1.2 of U.S.
23 EPA, 2010) and section C.1.1 of this appendix. Inputs required to model risk using CR functions
24 are identified below (Figure C-1) and include

- 25 (1) the CR functions themselves, obtained from epidemiologic studies (section C.1.1 and
26 C.3.2),
- 27 (2) baseline health incidence data and information on population demographics (section 0
28 and C.3.4),
- 29 (3) study areas (section C.1.3), and
- 30 (4) modeled ambient PM_{2.5} concentrations corresponding to air quality scenarios of interest
31 (section C.1.4).



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

3
4 **C.1.1 Selection of Key Health Endpoints and Specification of Concentration-Response**
5 **Functions from Epidemiologic Studies**

6 In selecting specific CR functions for the risk assessment, we began by considering
7 health outcomes for which the 2019 Integrated Science Assessment (ISA) determined the
8 evidence supports either a “causal” or a “likely to be causal” relationship with short- or long-
9 term PM_{2.5} exposures (U.S. EPA, 2019). As discussed in Chapter 3 (Table 3-1), these outcomes
10 include the following:

- 11 • mortality (resulting from long- and short-term exposure),
12 • cardiovascular effects (resulting from long- and short-term exposure),
13 • respiratory effects (resulting from long- and short-term exposure),
14 • cancer (resulting from long-term exposure), and
15 • nervous system effects (resulting from long-term exposure).

16 We focused the risk assessment on short- and long-term PM exposure-related mortality,
17 reflecting its clear public health importance, the large number of epidemiologic studies available
18 for consideration, and the broad availability of baseline incidence data. The specific set of health
19 effect endpoints included in the risk assessment are:

- 20 • *Long-term PM exposure-related mortality: all-cause*

- 1 • *Short-term PM exposure-related mortality*: all-cause and non-accidental

2 To identify specific epidemiologic studies for potential inclusion in the risk assessment,
3 we focus on U.S. multicity studies assessed in the 2019 ISA. These studies are identified in
4 section 3.4.1.5 of this draft PA. Of these, we used the following criteria to identify the specific
5 set of studies for inclusion in the risk assessment:

- 6 • *National-scale geographic coverage*: We focus on epidemiologic studies reporting
7 national-level CR functions. Epidemiologic studies that focus on individual cities or
8 regions were excluded. Focusing on national-level epidemiologic studies has the benefit
9 of characterizing PM_{2.5}-associated risks broadly across the U.S. and in relatively large
10 populations (compared with single-city or regional studies), which tends to improve
11 precision in the CR functions generated.
- 12 • *Evaluation of relatively lower ambient PM concentrations*: In selecting epidemiology
13 studies, to the extent possible, we focus on those studies which characterized the ambient
14 PM_{2.5}-mortality relationship at levels at or near the current NAAQS, given that the risk
15 assessment would be focusing on evaluating risk associated with the current NAAQS.
- 16 • *Populations with available baseline incidence data*: For some populations (e.g., diesel
17 truck drivers), it can be challenging to model risk at the national-level given uncertainties
18 associated with specifying key inputs for risk modeling (i.e., baseline incidence rates for
19 mortality endpoints and detailed national-level demographics). For that reason, we focus
20 on those epidemiology studies providing CR functions for populations readily
21 generalizable to the broader U.S. population (e.g., specific age groups not differentiated
22 by additional socio-economic, or employment attributes).
- 23 • *Estimates of long-term PM_{2.5} exposures based on hybrid modeling approaches*: For long-
24 term PM_{2.5} exposures, we focus on epidemiologic studies that estimate exposures with
25 hybrid modeling approaches. The rationale for this decision is the agreement between the
26 design of these epidemiology studies (i.e., their use of hybrid-based modeling approaches
27 in characterizing ambient PM) and the hybrid air quality surfaces we are using in this risk
28 assessment. This general agreement between the air modeling surfaces used in long-term
29 mortality epidemiology studies and our air quality modeling reduces uncertainty in the
30 risk assessment.
- 31 • *Estimates of short-term PM_{2.5} exposures based on composite monitor data*: Short-term
32 mortality epidemiology studies utilizing hybrid modeling approaches, which are fewer in
33 number compared with long-term mortality studies, tend to be regional in scope and did
34 not meet the criterion of providing national-scale effect estimates. For that reason, in
35 modeling short-term mortality, epidemiology studies utilizing composite-monitor based
36 exposure surrogates were used as the basis for deriving CR functions. We recognize the
37 uncertainty introduced into the modeling of short-term mortality due to the use of CR
38 functions obtained from studies utilizing composite monitors. However, we felt these use
39 of national-scale epidemiology studies was a more important criterion for selection.
- 40 • *Evaluation of potential confounders and effect modifiers*: To the extent possible,
41 preference was given to studies which more fully address potential confounders and
42 effect modifiers and to those studies which utilize individual (rather than ecological)

1 measures in representing those confounders/effect modifiers. Recognizing that both
2 single- and co-pollutant models have advantages and disadvantages in characterizing the
3 ambient PM-mortality relationship, to the extent possible, we include epidemiology
4 studies (and associated CR functions) based on either single- or co-pollutant models that
5 include ozone. Additional information available in the *Estimating PM_{2.5} and Ozone-
6 Attributable Health Benefits TSD* associated with the 2021 Revised Cross-State Air
7 Pollution Rule Update (RCU) (U.S. EPA, 2021b).

- 8 • *Exploration of multiple approaches for estimating exposures:* For studies that estimate
9 PM_{2.5} exposures using hybrid modeling approaches, preference was given to studies that
10 also explore additional methods for estimating exposures (i.e., multiple hybrid methods
11 or hybrid methods plus monitor-based methods) and compare health effect associations
12 across approaches.

13 Application of the criteria listed above resulted in the selection of the epidemiology
14 studies presented in Table C-1 for inclusion in the risk assessment as sources of effect estimates.
15 Table C-1 includes summary information on study design, details on the selection of effect
16 estimates, the derivation of beta values, and specification of CR functional form based on those
17 effect estimates for use in the risk assessment. The procedure used to derive CR functions
18 (including specification of beta values and mathematical forms for those functions) is described
19 below.

20 The remainder of this section describes the method used in specifying the CR functions
21 used in the PM NAAQS HHRA. Information presented in this section is drawn from the EPA's
22 Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP-CE)
23 Manual, Appendix C.¹ CR functions translate changes in ambient PM_{2.5} into changes in baseline
24 incidence rates for specific disease endpoints utilizing beta (β) values obtained from
25 epidemiology studies studying the association between ambient PM_{2.5} exposure and specific
26 health endpoints. β values (and associated standard errors) are based on effect estimates obtained
27 from the underlying epidemiology studies. In addition, the mathematical forms for the health
28 impact functions specified for use in this risk assessment reflect the models used in the
29 epidemiology studies providing those effect estimates. Consequently, derivation of the β values
30 based on effect estimates from underlying epidemiology studies (and specification of the form of
31 the health impact functions) represents a key step in the design of the HHRA.

32 The majority of the epidemiology studies providing effect estimates for this PM HHRA
33 utilized either Poisson or Cox proportional hazard models which result in exponential (or log-
34 linear) forms for the CR functions, where the natural logarithm of mortality incidence is a linear

¹ <https://www.epa.gov/benmap/benmap-ce-manual-and-appendices>

1 function of PM_{2.5}.² If we let x₀ denote the baseline (starting) PM_{2.5} level, and x₁ denote the
2 control (ending) PM_{2.5} level, y₀ denote the baseline incidences rate of the health effect, and Pop
3 the underlying population count for the applicable demographic group in the spatial unit of
4 analysis³ we can derive the following CR function specifying the relationship between the
5 change in x, Δx= (x₀- x₁) and the corresponding change in y, Δy (mortality incidence):

$$\Delta y = y_0 [1 - e^{-\beta \Delta x}] * \text{Pop}$$

6
7
8
9 Given that the epidemiology studies providing effect estimates for long-term exposure-
10 related mortality and short-term exposure-related mortality in the context of the current PM
11 HHRA (Table C-1) use different categories of models (Cox proportional hazard and
12 Poisson/Logistic, respectively) we describe the process of deriving the betas and specifying CR
13 functional forms separately for each of these endpoint categories. As noted earlier, the logit
14 model utilized in Zanobetti et al., 2014, is discussed at the end of the section covering short-term
15 PM_{2.5}-related mortality.

16 17 Derivation of betas for long-term PM_{2.5} exposure-related mortality

18 Cox proportional hazard models used to evaluate mortality associated with long-term
19 PM_{2.5} exposure are designed to model effects on population survival. This class of epidemiology
20 model is based on a hazard function, defined as the probability that an individual dies at time t,
21 conditional on that individual having survived up to time t. As such, the hazard function
22 represents a time-specific snapshot of the rate of mortality (events per unit time) within a study
23 population. While the risk can vary over time, in the case of the Cox proportional hazard model,
24 it is assumed that the hazard ratio is constant. The proportional hazard model takes the form:

$$h(X, t) = h_0(t)e^{X \cdot \beta}$$

26 Where X is a vector of explanatory variables, β is a vector of associated coefficients and
27 ho(t) is the baseline hazard (the risk when all covariates (X) are set to zero).

28 Epidemiology studies utilizing the Cox proportional hazard model in characterizing
29 ambient PM_{2.5}-health effects typically report hazard ratios (HRs) as the effect estimate. HRs
30 represent the ratio of hazard functions for the baseline and control scenarios reflecting a specific

² One study. Zanobetti et al., 2014, supporting the modeling of short-term PM_{2.5} exposure-related mortality provided a logistic-based model form, which is discussed at the end of this section.

³ Spatial unit of analysis refers to the geographic scale at which the CR function is applied in generating a risk (incidence) estimate (e.g., zip code, county, 12km grid cell). Typically, the spatial unit of analysis used in a REA is based on the spatial scale reflected in the epidemiology study(s) supplying the effect estimates. For this REA, the spatial unit of analysis is the 12km grid cell.

1 difference in ambient PM_{2.5} exposure (often a 10 µg/m³ increment). The HR simplifies as shown
2 (with the baseline hazard ratio dropping out), allowing us to readily derive the β value from this
3 effect estimate:

$$HR = \frac{h(x_0,t)}{h(x_c,t)} = \frac{h_0(t)e^{x_0 \cdot \beta}}{h_0(t)e^{x_c \cdot \beta}} = e^{\Delta PM \cdot \beta}$$

6 It is then possible to calculate the beta as follows:

$$\beta = \frac{\ln(HR)}{\Delta PM}$$

9 As noted in Sutradhar and Austin, 2018, the HR associated with a Cox-proportional
10 hazard model may approximate the RR when the effect estimate (and consequently the β) is
11 relatively small. This is the case with the effect on mortality modeled for long-term exposure to
12 ambient PM_{2.5} (i.e., the size of the effect estimate supports an assumed equivalency between HR
13 and RR). The near equivalency between the HR and RR, allows us to utilize the β derived above
14 in a CR function based on a log-linear functional form of the type presented earlier, to model
15 changes in mortality related to changes in ambient PM.

17 Derivation of betas for short-term PM_{2.5} exposure-related mortality

18 The epidemiology studies selected for use in modeling short-term PM_{2.5} exposure-related
19 mortality utilize both the Poisson (log-linear) model form (Baxter et al., 2017) and the logit
20 model form (Zanobetti et al., 2014).⁴ In both cases, the epidemiology studies provide effects in
21 terms of *percent increase* in mortality.

22 The log-linear (Poisson) model is used to evaluate effects associated with continuous
23 (count) events. With the log-linear (Poisson) model, the relative risk is simply the ratio of the
24 two risks:

$$RR = \frac{y_0}{y_c} = e^{\beta \cdot \Delta PM}$$

26 The derivation of the beta with a Poisson model specified RR is as follows. Taking the
27 natural log of both sides, the beta coefficient in the CR function underlying the relative risk can
28 be derived as:

$$\beta = \frac{\ln(RR)}{\Delta PM}$$

⁴ Note that the Ito et al., 2013 study also utilizes a Poisson model. However, that study provides beta values (including standard errors) and for that reason the results of this study are directly applicable in modeling changes in mortality without any of the derivations presented here for the other studies.

1 The beta derived in this fashion can then be used with a log-linear functional form (as
2 presented earlier) to model changes in mortality related to changes in ambient PM.

3 The logistic model form is used to model dichotomous events. With the logistic model
4 form, when we are provided with a RR value, as is the case here, we can make a similar
5 assumption to that used above with the Cox proportional hazard function (i.e., that the OR and
6 RR approach equivalency under conditions of relatively small effect levels). That observation in
7 turn allows us to assume that

$$RR = \frac{y_0}{y_c} = (1 - y_0) \times e^{-\Delta PM \cdot \beta} + y_0$$

10 Then, assuming (based on the relatively small size of the baseline incidence) that:

$$e^{-\Delta PM \cdot \beta} \cong (1 - y_0) \times e^{-\Delta PM \cdot \beta} + y_0$$

$$\Rightarrow RR \cong e^{-\Delta PM \cdot \beta}$$

14 It is then possible to calculate the underlying beta coefficient as follows:

$$\frac{\ln(RR)}{-\Delta PM} \cong \beta$$

17 Since the derivation of the beta is based on the assumption of a log linear functional
18 form, we can apply the beta in a log-liner CR function of the form described earlier:

$$\Delta y = y_0 [1 - e^{-\beta \Delta x}] * \text{Pop}$$

1 **Table C-1. Details regarding selection of epidemiology studies and specification of concentration-response functions for the**
 2 **risk assessment.**

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemiologic Statistic	Mortality Endpoint	Selected Effect Estimate	Selected Beta	Selected Beta Standard Error (SE)
Long-term exposure-related mortality studies										
Di et al., 2017 Air Pollution and Mortality in the Medicare Population	Exploring relationship between air pollution (ozone, PM _{2.5}) and mortality Key details: - Medicare population (65+) - ecological control for confounders - all-cause mortality only - provides CR function slopes for areas above and below the current PM NAAQS level (but model for areas below current standard only done for low ozone cells)	Exposures estimated at zip code of residence based on a neural network model that incorporates satellite data, chemical transport modeling, land-use terms, meteorology data, monitoring data, and other data	Cox proportional-hazards model with a generalized estimating equation to account for the correlation between ZIP codes	Table 2 Risk of death associated with an increase of 10 µg/m ³ PM _{2.5} or an increase of 10 ppb in ozone concentration. Uses single pollutant model for full analysis.	Using single pollutant, full PM range model (model for <12 µg/m ³ applicable to only low-ozone days) ⁵	Hazard ratio (95 percent CI)	All-cause	1.073 (1.071, 1.075)	7.0E-03	1E-04
Turner et al., 2016 Long-Term Ozone Exposure and Mortality in a Large Prospective Study	Evaluates the relationship between long-term exposure to ambient PM _{2.5} and all-cause and cause-specific mortality. Also, estimated the association between PM _{2.5} , regional PM _{2.5} , and near-source PM _{2.5} and mortality in single-pollutant, copollutant and multipollutant models. - ACS (30+) - Includes lung cancer (otherwise similar results to Pope et al., 2015) - county-level assessment	Exposures estimated at residential locations based on land use data and ground-based monitors	Cox proportional hazard model	Table E4. Adjusted HRs (95 th percentile CI) for all-cause and cause-specific mortality in relation to each 10 unit increase in PM _{2.5} LUR-BME concentrations, follow-up 1982-2004, CPS-II cohort, United States (n = 669,046).	Note that the non-cancer mortality endpoints provided in table E4 appear to mirror those provided in Table 1 of Pope et al., 2015 -so will use long-cancer effect estimate from this study only.	Hazard ratio (95 percent CI)	All-cause	1.06 (1.04-1.08)	5.8E-03	9.6E-04
Short-term exposure-related mortality studies										

⁵ We note that Di et al., 2017 does include a copollutant model-based effect estimate (HR 1.073, 95thCI 1.071-1.075). Had this effect estimate been used in risk modeling (which would translate into a beta value of 7.05E-3), we would anticipate the risk estimates for all-cause mortality to be slightly less (~13% lower based on comparison of calculated betas) than those estimated based on the single-pollutant model used in this risk assessment.

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemiologic Statistic	Mortality Endpoint	Selected Effect Estimate	Selected Beta	Selected Beta Standard Error (SE)
Baxter et al., 2017 Influence of exposure differences in city-to-city heterogeneity in PM _{2.5} -mortality associations in U.S. cities	Uses cluster-based approach to evaluate the impact of residential infiltration factors on inter-city heterogeneity in short-term PM-mortality associations. - Mortality data from NCHS - 77 U.S. CBSAs (all ages) - non-accidental mortality - CBSA-level assessment	Exposure estimates based on data from ground-based monitors	Poisson (log-linear) at city-level then aggregated	Obtained from results section in the text. After pooling the city-specific effect estimates into an overall effect estimate, short-term PM _{2.5} exposure was found to increase 24-hr non-accidental mortality by 0.33% (95% CI: 0.13, 0.53). Based on lag 2 (day 0-1)	NA	Percent increase in 24-hr mortality (95 percent CI)	24-hr non-accidental mortality	0.33 (0.13-0.53)	3.29E-04	1.02E-04
Ito et al., 2013 NPACT study 3. Time-series analysis of mortality, hospitalizations, and ambient PM _{2.5} and its components	Use factor analysis to characterize pollution sources, assess the association between PM _{2.5} and PM _{2.5} components with morbidity and mortality outcomes. Also evaluates pollution levels, land-use, and other variables as modifiers that may explain inter-city variation in PM-mortality effect estimates. - Mortality data from NCHS - 150 and 64 U.S. cities (two analyses) (all ages) - MSA-level assessment	Exposure estimates based on data from ground-based monitors	Poisson GLM	Appendix G, Table G.6 for Figure 4 - use all-year lag 1 Beta: Regression coefficients (beta) and their SE for air pollutants at lag 0 through 3 days used to compute percent excess risks in figures shown in the main text and in Appendices B and G (corresponding figures are noted).	Utilized lag-1 (all year) beta because that had the strongest effect for CVD mortality and wanted our all-cause to reflect that stronger lag-association for the CVD effect (even though focusing on all-cause)	Betas with SE (no conversion required)	24-hr all-cause mortality	Study provided beta and SE	1.45E-04	7.47E-05

Reference and Title	Study Description	Exposure Estimation Approach	CR Function	Location of CR Function(s) in Article	Additional Notes on CR Function(s) Selection	Epidemiologic Statistic	Mortality Endpoint	Selected Effect Estimate	Selected Beta	Selected Beta Standard Error (SE)
Zanobetti et al., 2014 A national case-crossover analysis of the short-term effect of PM _{2.5} on hospitalizations and mortality in subjects with diabetes and neurological disorders	Estimates the effect of short-term exposure to PM _{2.5} on all-cause mortality. Additionally, assesses the potential for pre-existing diseases to modify the association between PM _{2.5} and mortality (neurological disorders and diabetes) - Medicare cohort - 121 U.S. communities (65+) - Community-level assessment (community defined as the county or contiguous counties encompassing a city's population)	Exposure estimates based on data from ground-based monitors	Logistic regression	Table 2. Percent increase for 10 µg/m ³ increase in the two days average PM _{2.5} : Combined across the 121 communities	NA	Percent increase (95 percent CI)	All deaths	0.64 (0.42-0.85)	6.38E-04	1.09E-04

1

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32

C.1.2 Specification of Demographic and Baseline Incidence Data Inputs

This risk analysis requires both demographic and baseline-incidence data for the mortality endpoint categories evaluated. For our analyses, these data are for the year 2015 since the hybrid surfaces included in the analyses are based on a 2015 model year.⁶ The BenMAP-CE model⁷ is used in this risk assessment and the relevant demographic and baseline incidence data for the contiguous U.S., from the sources described below, is readily available within the current version of BenMAP-CE:

- *Demographic data:* BenMAP-CE includes 2010 U.S. Census block-level age, race, ethnicity, and gender-differentiated data which the program can aggregate to various grid-level definitions selected by the user, including the 12 km grid coverage used for risk modeling in this analysis. In addition, BenMAP-CE has the ability to project future demographics using county-level projections provided by Woods & Poole, 2015. See BenMAP-CE manual Appendix J and the *Estimating PM_{2.5} and Ozone-Attributable Health Benefits TSD* associated with the 2021 RCU for additional detail (U.S. EPA, 2021b).
- *Baseline incidence data for mortality endpoints:* County-level mortality and population data from 2012-2014 for seven causes of death in the contiguous U.S. was obtained from the Centers for Disease Control (CDC) WONDER database. To estimate values for 2015, we applied annual adjustment factors, based on a series of Census Bureau projected national mortality rates for all-cause mortality. See BenMAP-CE manual Appendix D for additional detail.

C.1.3 Study Area Selection

In selecting U.S. study areas for inclusion in the risk assessment, we focus on the following characteristics:

- *Available Ambient Monitors:* We have greater confidence in estimating and simulating air quality concentrations over areas with relatively dense ambient monitoring networks, as the modeled air quality surfaces can be compared with monitored concentrations (air quality adjustments are described below in section C.1.4).
- *Geographical Diversity:* Risk assessments including areas that represent a variety of regions across the U.S. and a substantial portion of the U.S. population can be more representative.

⁶ The 2015 model year was the most recent CMAQ modeling platform available at the time of the design of the risk assessment and represents the central year of the 2014-2016 design value (DV) period. A single modeling year was used in the risk assessment, rather than modeling risk for the full three-year design value period, because model inputs for the 2016 period were not available at the time of the study (section C.1.4.3).

⁷ <https://www.epa.gov/benmap>

1 • *Ambient PM_{2.5} Air Quality Concentrations*: Based on 2014-2016 design values, 16 CBSA⁸
2 areas exceeded either or both the current annual and 24-hr PM_{2.5} NAAQS. To include a
3 larger portion of the U.S. in this risk assessment, we also identified CBSA areas with ambient
4 PM_{2.5} concentrations below, but near, the current annual and/or 24-hr PM_{2.5} NAAQS.
5 Inclusion of such areas in the risk assessment necessitates an upward adjustment to PM_{2.5} air
6 quality concentrations in order to simulate just meeting the current standards. Given
7 uncertainty in how such increases could potentially occur, we select areas requiring a
8 relatively modest upward adjustment (i.e., no more than 2.0 µg/m³ for the annual standard
9 and 5 µg/m³ for the 24-hour standard, based on the 2014-2016 design value period). Areas
10 that appeared to be strongly influenced by exceptional events were also excluded (section
11 C.1.4). Using these criteria, 47 urban study areas were identified (PA Figure 3-16 and
12 Appendix section C.1.3), including 30 study areas where just meeting the current standards is
13 controlled by the annual standard,⁹ 11 study areas where just meeting the current standards is
14 controlled by the daily standard,¹⁰ and 6 areas where the controlling standard differed
15 depending on the air quality adjustment approach (PA Figure 3-16).¹¹

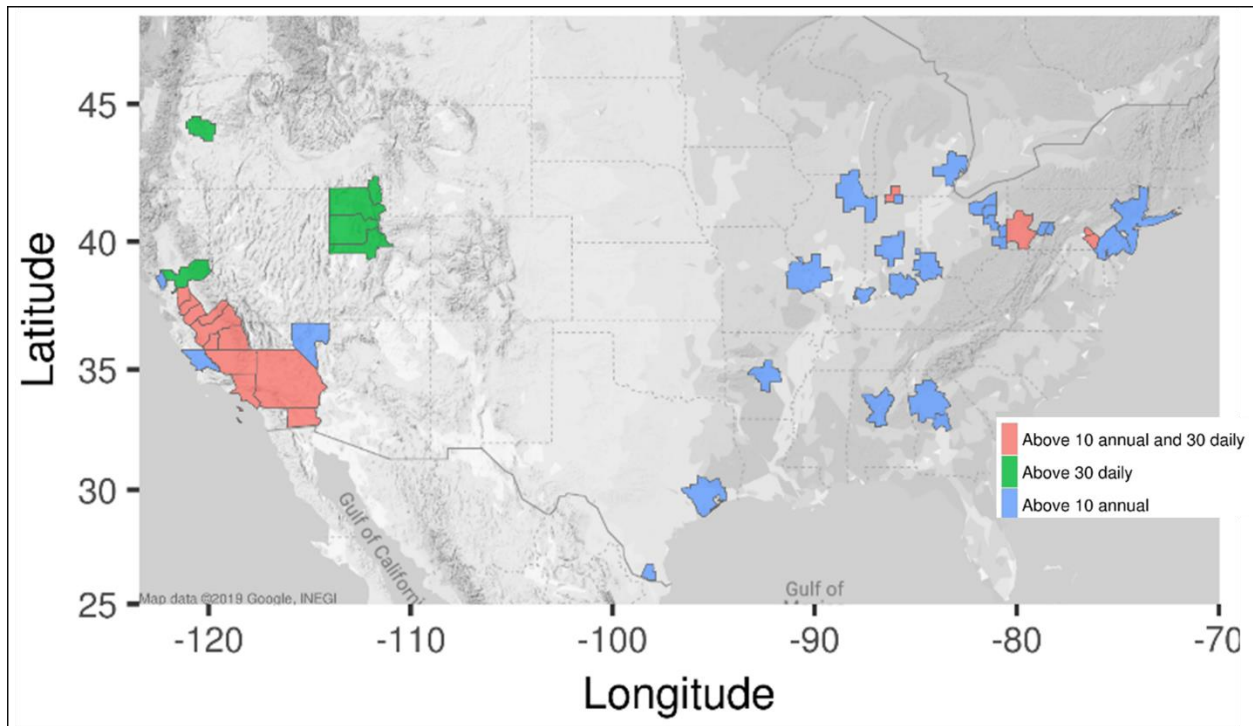
16 Applying these criteria resulted in the inclusion of 47 core-based statistical areas
17 (CBSAs). These 47 study areas are identified in Figure C-2, with colors indicating whether they
18 meet either or both the design value cutoffs. Please note, meeting the criteria for inclusion does
19 not mean the areas exceed the current annual and/or 24-hr PM NAAQS standards. Green
20 indicates areas that only exceed a 24-hr design value of 30 µg/m³, blue indicates areas that only
21 exceed an annual design value of 10 µg/m³, and red indicates areas that exceed both the 24-hr
22 and annual design values.
23

⁸ CBSAs (core-based statistical areas) can include one or more counties. Each CBSA selected included at least one monitor with valid design values and several CBSAs had more than 10 monitors. See Table C-3 in Appendix C.

⁹ For these areas, the annual standard is the “controlling standard” because when air quality is adjusted to simulate just meeting the current or potential alternative annual standards, that air quality also would meet the 24-hour standard being evaluated.

¹⁰ For these areas, the 24-hour standard is the controlling standard because when air quality is adjusted to simulate just meeting the current or potential alternative 24-hour standards, that air quality also would meet the annual standard being evaluated. Some areas classified as being controlled by the 24-hour standard also violate the annual standard.

¹¹ In these 6 areas, the controlling standard depended on the air quality adjustment method used and/or the standard scenarios evaluated.



1

2

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

3

4

5

These 47 urban study areas include many highly populated CBSAs (Figure C-3 and Figure C-4). The population at or above the age of 30 in these areas includes roughly 58.4 million people, or approximately 30% of the total U.S. population above the age of 30.

6

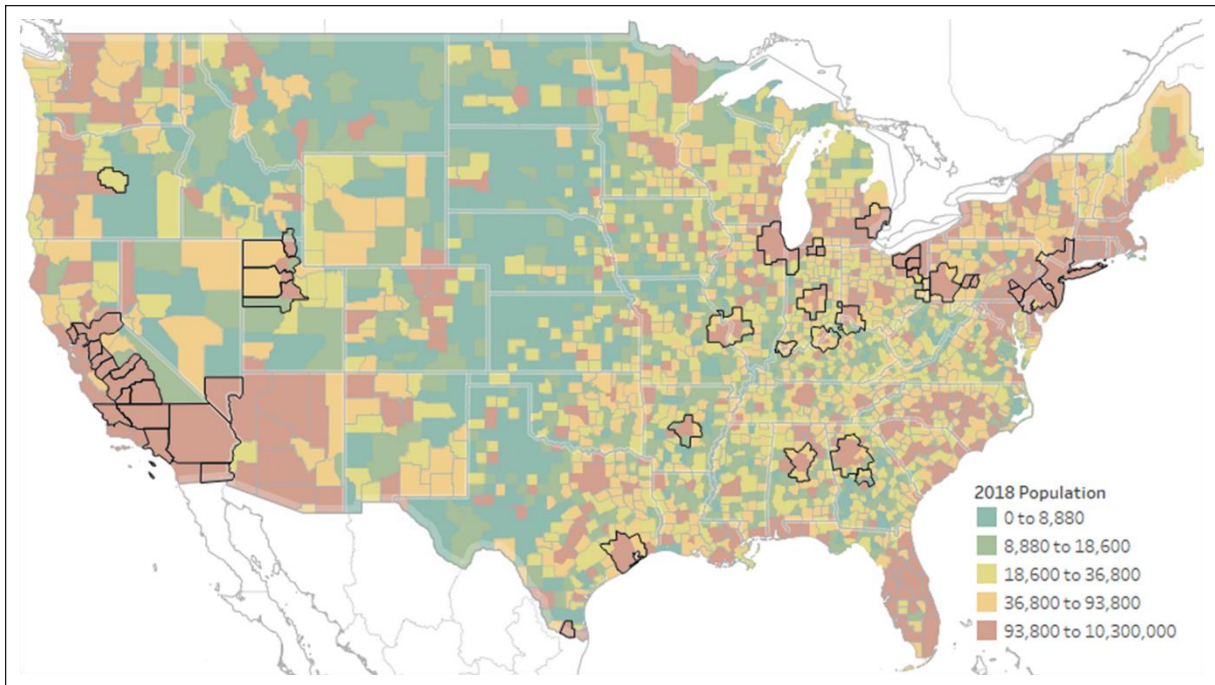
7

8

Additional age-specific population information corresponding to each identified mortality study can be found in Table C-2.

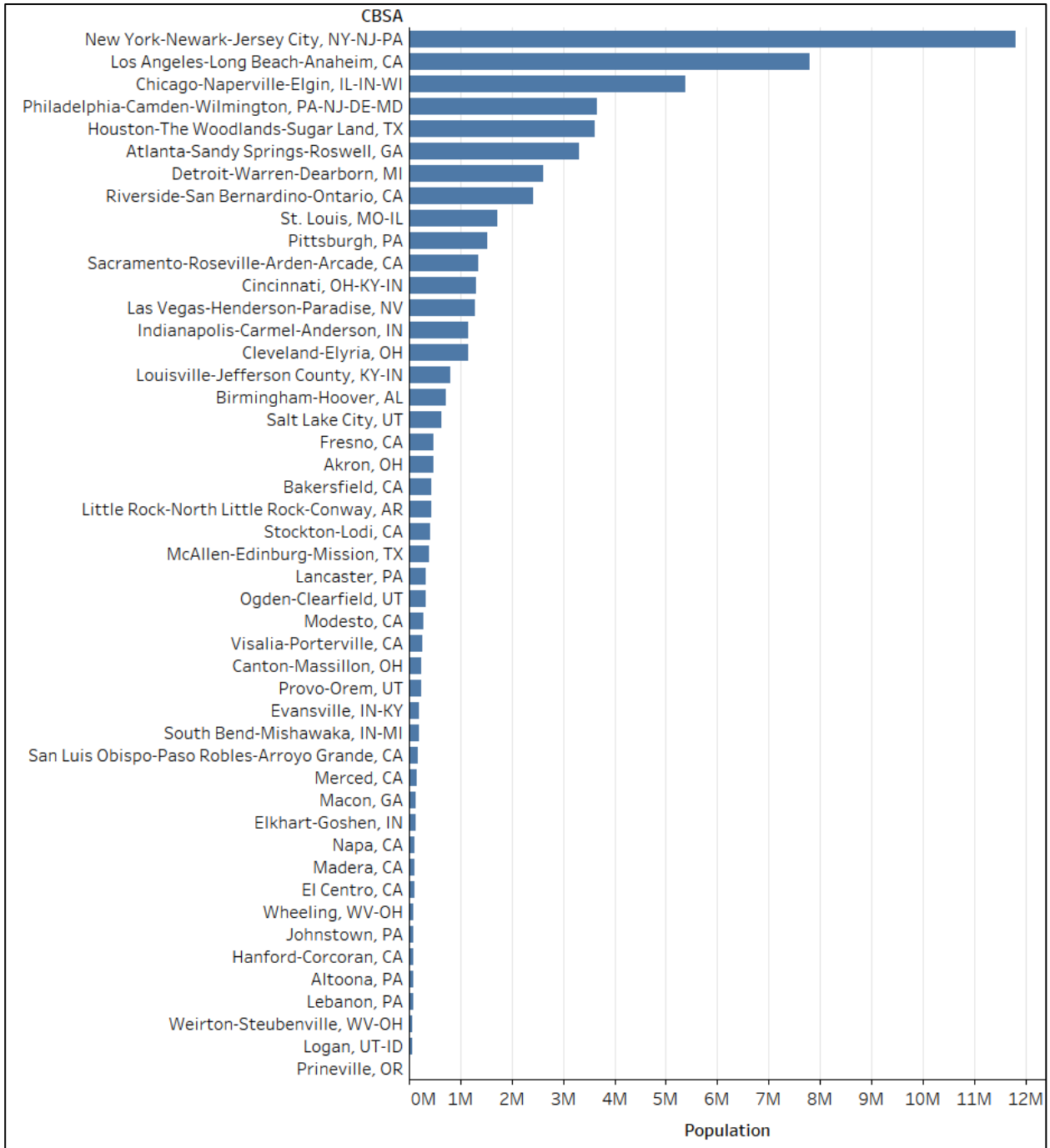
9

10



1
2
3
4

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



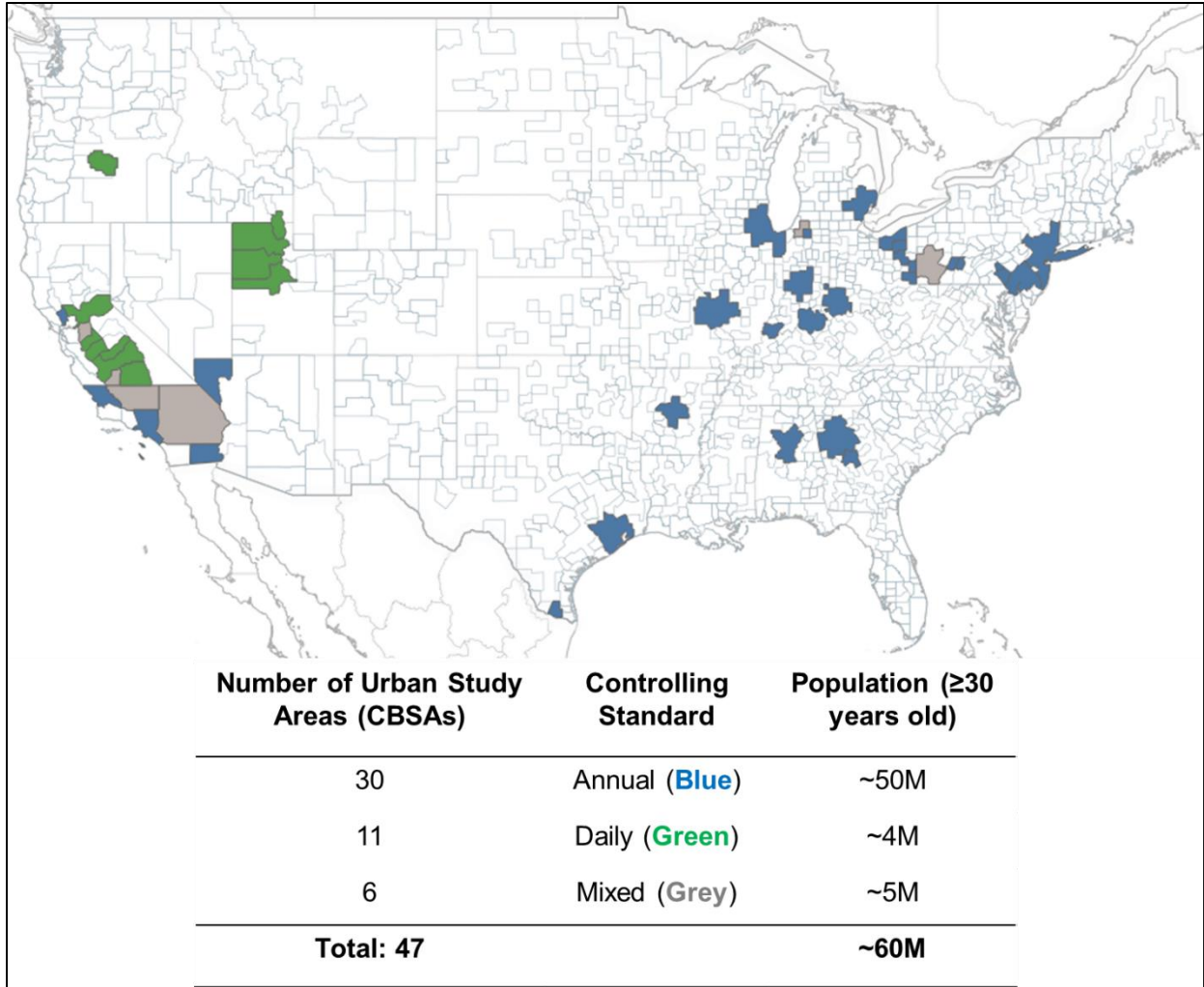
1
2 **Figure C-4. Population counts for ages 30 and above from each of the 47 CBSAs included**
3 **in the risk assessment.**
4

1 **Table C-2. Population of the 47 urban study areas by age range.**

Population Age Range (Years)	Studies Using Age Range	Study Area Groupings (Millions)		
		47	30 (Annual-Controlled)	11 (24-hr-Controlled)
0-99	Baxter et al., 2017 and Ito et al., 2013	98.5	82.5	7.2
30-99	Turner et al., 2016	58.4	49.5	3.9
65-99	Di et al., 2017 and Zanobetti et al., 2014	13.2	11.1	0.8

2
3
4
5
6
7
8
9

As noted in section 3.4 of the draft PA and illustrated in Figure C-5, the 47 urban study areas include 30 study areas where just meeting the simulated standards is controlled by the current annual standard ($12.0 \mu\text{g}/\text{m}^3$), 11 study areas where just meeting the simulated standards is controlled by the current 24-hr standard ($35 \mu\text{g}/\text{m}^3$), and 6 study areas where just meeting the simulated standards is controlled by either the annual or 24-hr standard, depending on the air quality scenario and adjustment strategy (discussed more fully in section C.1.4).



1
 2 **Figure C-5. Map of 47 Urban Study Areas Reflected in Risk Modeling Identifying Subsets**
 3 **Reflected in Risk Modeling (population estimates in millions of people).**

4
 5 **C.1.4 Generation of Air Quality Inputs to the Risk Assessment**

6 As described in detail below, air quality modeling was used to develop gridded PM_{2.5}
 7 concentration fields for the risk assessment. A PM_{2.5} concentration field for 2015 was developed
 8 using a Bayesian statistical model that calibrates chemical transport model (CTM) predictions of
 9 PM_{2.5} to surface measurements (Chapter 2). The 2015 PM_{2.5} concentration field was then
 10 adjusted to correspond to just meeting the existing and potential alternative standards using
 11 response factors developed from CTM modeling with emission changes relative to 2015. The
 12 modeling approach applies realistic spatial response patterns from CTM modeling to a
 13 concentration field, similar to those used in a number of recent epidemiologic studies, to
 14 characterize PM_{2.5} fields at 12 km resolution for study areas.

1 The adjustments to simulate just meeting the current standards and alternative standards
2 are approximations of these air quality scenarios. In reality, changes in PM_{2.5} in an area will
3 depend on what emissions changes occur and the concentration gradients of PM_{2.5} will vary
4 across an area accordingly. For our analyses, two different adjustment approaches were applied
5 to provide two outcomes that could represent potential bounding scenarios of PM_{2.5}
6 concentrations changes across the study area. The two adjustment approaches used to guide the
7 generation of these modeled surfaces were:

- 8 • *Primary PM-based modeling approach (Pri-PM)*: This modeling approach simulates air
9 quality scenarios of interest by preferentially adjusting direct (i.e., primary, directly-
10 emitted) PM emissions. As such, the changes in PM_{2.5} tend to be more localized near the
11 direct emissions sources of PM. In locations for which air quality scenarios cannot be
12 simulated by adjusting modeled primary emissions alone, SO₂ and NO_x precursor
13 emissions are additionally adjusted to simulate changes in secondarily formed PM_{2.5}.
- 14 • *Secondary PM-based modeling approach (Sec-PM)*: This modeling approach simulates
15 air quality scenarios of interest by preferentially adjusting SO₂ and NO_x precursor
16 emissions to simulate changes in secondarily formed PM_{2.5}. In this case, the reductions in
17 PM_{2.5} tend to be more evenly spread across a study area. In locations for which air quality
18 scenarios cannot be simulated by adjusting precursor emissions alone, a proportional
19 adjustment of air quality is subsequently applied.

20 The air quality surfaces generated using these two approaches are not additive. Rather, they
21 should be viewed as reflecting two different broad strategies for adjusting ambient PM_{2.5} levels.

22 In addition, we also employed linear interpolation and extrapolation to simulate air
23 quality under two additional alternative annual standard levels, 11.0, 9.0, and 8.0 µg/m³,
24 respectively (section 3.4.1.3 of the draft PA, Figure 3-15). Interpolation and extrapolation were
25 only performed for grid cells in the subset of 30 urban study areas where the annual standard was
26 controlling in both Pri-PM and Sec-PM simulated air quality scenarios of both 12/35 and 10/30
27 standard combinations. The interpolation and extrapolation were completed at the grid-cell level
28 based on values simulated using hybrid air quality modeling to just meet the current annual
29 standard of 12.0 ug/m³ and alternative annual standard of 10.0 ug/m³ (section 3.4.1.3 of the draft
30 PA, Figure 3-15). A similar linear extrapolation/interpolation was not conducted for additional
31 24-hr standards due to the weaker relationship between the 98th percentile of 24-hr PM_{2.5}
32 concentrations, which are most relevant for simulating air quality that just meets the 24-hour
33 standard, and the concentrations comprising the middle portion of the PM_{2.5} air quality
34 distribution, which are most relevant for estimating risks based on information from
35 epidemiologic studies (i.e., discussed further in sections 3.1.2 and 3.2.3.2 in the draft PA).

36 The sections below provide more detailed information on the air quality modeling
37 approach used to adjust air quality to simulate just meeting the current or alternative primary

1 PM_{2.5} standards. Tables containing PM_{2.5} DVs for the air quality projections can be found in
2 section C.6.

4 **C.1.4.1 Overview of the Air Quality Modeling Approach**

5 To inform risk calculations, recent PM_{2.5} measurements were analyzed to characterize the
6 magnitude and spatial distribution of PM_{2.5} concentrations. These data were then coupled with
7 air quality modeling data to project ambient air quality levels corresponding to just meeting the
8 existing and alternative PM_{2.5} NAAQS¹² in specific areas. An overview of the approach is
9 provided in Figure C-6. The process starts by acquiring PM_{2.5} monitoring data from EPA’s Air
10 Quality System (AQS)¹³ and simulating PM_{2.5} concentrations with the Community Multiscale
11 Air Quality (CMAQ)¹⁴ model for base case and emission-sensitivity scenarios (Figure C-6, Box
12 1). The monitored and modeled data are then fused using the Downscaler model and the
13 Software for Model Attainment Test-Community Edition (SMAT-CE)¹⁵ to develop a baseline
14 spatial field of PM_{2.5} concentrations and relative response factors (RRFs) for projecting PM_{2.5}
15 concentrations, respectively (Figure C-6, Box 2). PM_{2.5} concentrations are projected in two main
16 steps using output from Downscaler and SMAT-CE (Figure C-6, Box 3). First, the PM_{2.5}
17 concentrations measured at monitoring sites in an area are iteratively projected using the RRFs to
18 identify the percent change in anthropogenic emissions required for the highest monitored DV in
19 the area to just meet the controlling standard. Second, gridded spatial fields of PM_{2.5}
20 concentrations are projected using the area-specific percent emission change¹⁶ that corresponds
21 to just meeting the standard at the controlling ambient data site. Additional details on the method
22 are provided in (Kelly et al., 2019a; application of the method to the PM NAAQS risk
23 assessment is described in the remainder of this appendix.

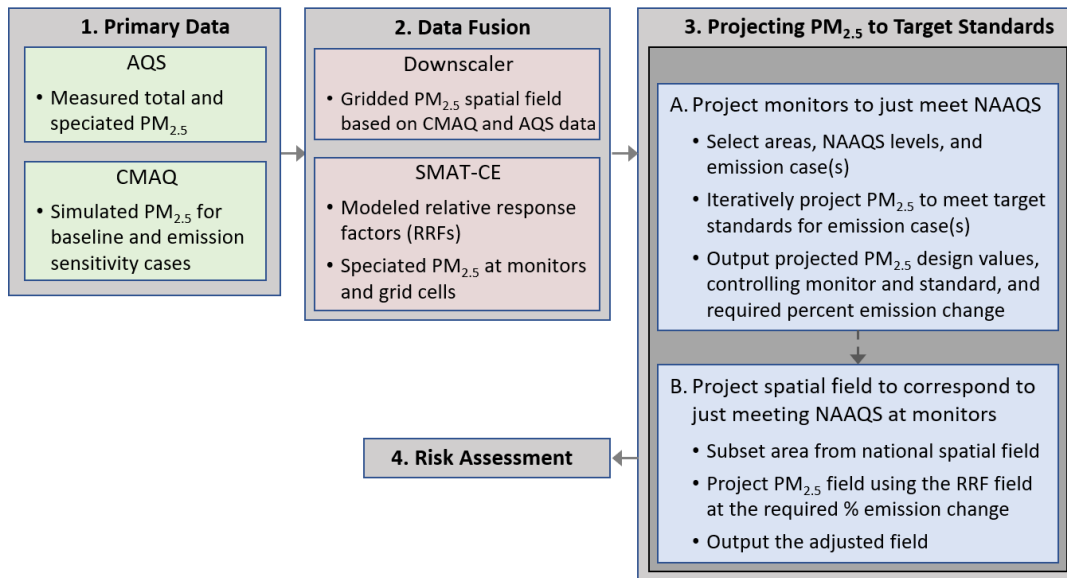
¹² The phrase, “just meeting the PM_{2.5} NAAQS” is defined as the conditions where the highest design value (DV) for the controlling standard in the area equals the existing or alternative NAAQS level under consideration. DVs are statistics used in judging attainment of the NAAQS (www.epa.gov/air-trends/air-quality-design-values).

¹³ www.epa.gov/aqs

¹⁴ www.epa.gov/cmaq

¹⁵ www.epa.gov/scram/photochemical-modeling-tools

¹⁶ Scenarios based on a statistical projection approach were also developed for certain cases as discussed below.



1
2 **Figure C-6. Overview of the system for projecting PM_{2.5} concentrations to correspond to**
3 **just meeting NAAQS.** See section C.1.4.6 and Kelly et al., 2019a for more details.
4

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

6 The 2014-2016 DV period was the most recent period having a complete set of total and
7 speciated PM_{2.5} observations available at the time of the study. PM_{2.5} concentrations from the
8 2014-2016 DV period were used in selecting study areas and as the starting point for air quality
9 projections (Figure C-6, Box 1, “AQS”). Total and speciated PM_{2.5} concentrations for the 2014-
10 2016 DV period were acquired from AQS. For sites in Los Angeles and Chicago, DVs were
11 invalid during the 2014-2016 period. Los Angeles and Chicago have large populations, recent
12 valid DVs for sites in Los Angeles are above existing standards, and Chicago is part of a CBSA
13 that includes sites with valid 2014-2016 DVs in Indiana. For these reasons, invalid data for sites
14 in these areas were replaced with valid data from other recent periods to enable DVs to be
15 approximated for inclusion in the assessment. Specifically, for sites in Los Angeles and Orange
16 Counties in California, observations from April – October 2014 were replaced with observations
17 from the same months in 2013. For sites in Cook, DuPage, Kane, McHenry, and Will Counties in
18 Illinois, observations from January to mid-July 2014 were replaced with observations from the
19 same months in 2015.

20 Of the 56 areas initially identified as above the 10/30 selection threshold¹⁷, DVs for seven
21 areas¹⁸ appeared to meet the threshold due to the influence of wildfires. The influence of

¹⁷ “10/30” indicates an annual standard level of 10 µg/ m³ and a 24-hr standard level of 3 µg m⁻³

¹⁸ Butte-Silver Bow, MT; Helena, MT; Kalispell, MT; Knoxville, TN; Medford, OR; Missoula, MT; and Yakima, WA

1 wildfires on DVs for these areas was estimated in part by recalculating 2014-2016 DVs with
2 days removed that were clearly associated with summertime wildfires in the northwest. Since
3 wildfire influence is often excluded when judging NAAQS attainment, these seven areas were
4 excluded from further consideration. Additionally, the Eugene, OR CBSA was excluded. One
5 monitor in the Eugene CBSA has a 24-hr 2014-2016 DV slightly above the 10/30 selection
6 threshold¹⁹, but the monitor is in a small valley in Oakridge with very local high concentrations
7 of PM_{2.5} in winter that are distinct from conditions in the broader CBSA. Finally, the Phoenix-
8 Mesa-Scottsdale, AZ CBSA was excluded. This CBSA had one monitor slightly above the 10/30
9 DV threshold²⁰, but projecting concentrations for the CBSA was judged to be relatively uncertain
10 because the annual DV is invalid at the only site that exceeded the threshold and the 24-hr DV is
11 just above the threshold.

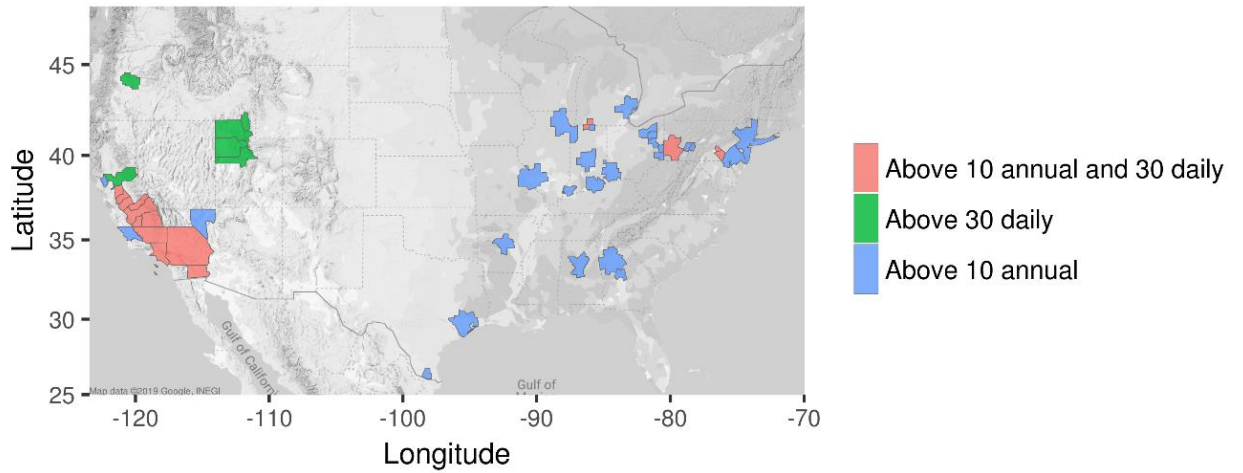
12 The remaining 47 CBSAs were selected for the risk assessment. These areas are shown in
13 Figure C-7. The maximum 2014-2016 DVs and associated sites for each CBSA are provided in
14 Table C-3, and the counties associated with the CBSAs are listed in Table C-4. DVs were
15 calculated to an extra digit of precision for the air quality projections compared with official
16 DVs. This approach is consistent with DV calculations in previous air quality projections (e.g.,
17 USEPA, 2012²¹) and provides a precise target for the iterative projection calculations.

18
19
20
21

¹⁹ The 410392013 monitor in Oakridge has a 24-hr 2014-2016 DV of 31 $\mu\text{g m}^{-3}$

²⁰ The 040213015 monitor in the Phoenix-Mesa-Scottsdale, AZ CBSA has 24-hr 2014-2016 DV of 31 $\mu\text{g m}^{-3}$

²¹ USEPA (2012) Regulatory Impact Analysis for the Final Revisions to the National Ambient Air Quality Standards for Particulate Matter. Office of Air Quality Planning and Standards, Health and Environmental Impacts Division, Research Triangle Park, NC 27711. EPA-452/R-12-005 Available: <https://www3.epa.gov/ttn/ecas/regdata/RIAs/finalria.pdf>



1
2
3
4
5

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

1 **Table C-3. Maximum annual and 24-hr PM_{2.5} DVs for 2014-2016 and associated sites for**
 2 **selected CBSAs.**

CBSA Name	# of Sites	Annual Max Site	Annual Max 14-16 DV	24-hr Max Site	24-hr Max 14-16 DV
Akron, OH	2	391530017	10.99	391530017	23.7
Altoona, PA	1	420130801	10.11	420130801	23.8
Atlanta-Sandy Springs-Roswell, GA	6	131210039	10.38	131210039	19.7
Bakersfield, CA	5	060290016	18.45	060290010	70.0
Birmingham-Hoover, AL	4	010732059	11.25	010730023	22.8
Canton-Massillon, OH	2	391510017	10.81	391510017	23.7
Chicago-Naperville-Elgin, IL-IN-WI ^a	22	170313103	11.10	170310057	26.8
Cincinnati, OH-KY-IN	9	390610014	10.70	390170020	24.2
Cleveland-Elyria, OH	8	390350065	12.17	390350038	25.0
Detroit-Warren-Dearborn, MI	11	261630033	11.30	261630033	26.8
El Centro, CA	3	060250005	12.63	060250005	33.5
Elkhart-Goshen, IN	1	180390008	10.24	180390008	28.6
Evansville, IN-KY	4	181630023	10.11	181630016	22.0
Fresno, CA	4	060195001	14.08	060190011	53.8
Hanford-Corcoran, CA	2	060310004	21.98	060310004	72.0
Houston-The Woodlands-Sugar Land, TX	4	482011035	11.19	482011035	22.4
Indianapolis-Carmel-Anderson, IN	7	180970087	11.44	180970043	26.0
Johnstown, PA	1	420210011	10.68	420210011	25.8
Lancaster, PA	2	420710012	12.83	420710012	32.7
Las Vegas-Henderson-Paradise, NV	4	320030561	10.28	320030561	24.5
Lebanon, PA	1	420750100	11.20	420750100	31.4
Little Rock-North Little Rock-Conway, AR	2	051191008	10.27	051191008	21.7
Logan, UT-ID	1	490050007	6.95	490050007	34.0
Los Angeles-Long Beach-Anaheim, CA ^a	9	060371103	12.38	060371103	32.8
Louisville/Jefferson County, KY-IN	7	180190006	10.64	180190006	23.9
Macon, GA	2	130210007	10.13	130210007	21.2
Madera, CA	1	060392010	13.30	060392010	45.1
McAllen-Edinburg-Mission, TX	1	482150043	10.09	482150043	25.0
Merced, CA	2	060470003	11.81	060472510	39.8
Modesto, CA	2	060990006	13.02	060990006	45.7
Napa, CA	1	060550003	10.36	060550003	25.1
New York-Newark-Jersey City, NY-NJ-PA	17	360610128	10.20	340030003	24.5
Ogden-Clearfield, UT	3	490570002	8.99	490110004	32.6
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	10	420450002	11.46	421010055	27.5
Pittsburgh, PA	10	420030064	12.82	420030064	35.8
Prineville, OR	1	410130100	8.60	410130100	37.6
Provo-Orem, UT	3	490494001	7.74	490494001	30.9
Riverside-San Bernardino-Ontario, CA	2	060658005	14.48	060658005	43.2
Sacramento--Roseville--Arden-Arcade, CA	6	060670006	9.31	060670006	31.4
Salt Lake City, UT	3	490353006	7.62	490353010	41.5
San Luis Obispo-Paso Robles-Arroyo Grande, CA	3	060792007	10.70	060792007	25.9

CBSA Name	# of Sites	Annual Max Site	Annual Max 14-16 DV	24-hr Max Site	24-hr Max 14-16 DV
South Bend-Mishawaka, IN-MI	1	181410015	10.45	181410015	32.5
St. Louis, MO-IL	6	290990019	10.12	295100007	23.7
Stockton-Lodi, CA	2	060771002	12.23	060771002	38.7
Visalia-Porterville, CA	1	061072002	16.23	061072002	54.0
Weirton-Steubenville, WV-OH	4	390810017	11.75	390810017	27.2
Wheeling, WV-OH	2	540511002	10.24	540511002	22.5
^a DVs for Chicago-Naperville-Elgin, IL-IN-WI and Los Angeles-Long Beach-Anaheim, CA were approximated as described in section C.1.4.2.					

1

2 **Table C-4. Counties associated with selected CBSAs**

CBSA Name	Associated Counties
Akron, OH	Portage, Summit
Altoona, PA	Blair
Atlanta-Sandy Springs-Roswell, GA	Barrow, Bartow, Butts, Carroll, Cherokee, Clayton, Cobb, Coweta, Dawson, DeKalb, Douglas, Fayette, Forsyth, Fulton, Gwinnett, Haralson, Heard, Henry, Jasper, Lamar, Meriwether, Morgan, Newton, Paulding, Pickens, Pike, Rockdale, Spalding, and Walton
Bakersfield, CA	Kern
Birmingham-Hoover, AL	Bibb, Blount, Chilton, Jefferson, St. Clair, Shelby, and Walker
Canton-Massillon, OH	Carroll, Stark
Chicago-Naperville-Elgin, IL-IN-WI	Cook, DeKalb, DuPage, Grundy, Kane, Kendall, Lake, McHenry, Will, Jasper, Lake, Newton, Porter, and Kenosha
Cincinnati, OH-KY-IN	Dearborn, Ohio, Union, Boone, Bracken, Campbell, Gallatin, Grant, Kenton, Pendleton, Brown, Butler, Clermont, Hamilton, and Warren
Cleveland-Elyria, OH	Cuyahoga, Geauga, Lake, Lorain, and Medina
Detroit-Warren-Dearborn, MI	Lapeer, Livingston, Macomb, Oakland, St. Clair, and Wayne
El Centro, CA	Imperial
Elkhart-Goshen, IN	Elkhart
Evansville, IN-KY	Posey, Vanderburgh, Warrick, and Henderson
Fresno, CA	Fresno
Hanford-Corcoran, CA	Kings
Houston-The Woodlands-Sugar Land, TX	Austin, Brazoria, Chambers, Fort Bend, Galveston, Harris, Liberty, Montgomery, and Waller
Indianapolis-Carmel-Anderson, IN	Boone, Brown, Hamilton, Hancock, Hendricks, Johnson, Madison, Marion, Morgan, Putnam, and Shelby
Johnstown, PA	Cambria
Lancaster, PA	Lancaster
Las Vegas-Henderson-Paradise, NV	Clark
Lebanon, PA	Lebanon
Little Rock-North Little Rock-Conway, AR	Faulkner, Grant, Lonoke, Perry, Pulaski, and Saline

CBSA Name	Associated Counties
Logan, UT-ID	Franklin, Cache
Los Angeles-Long Beach-Anaheim, CA	Los Angeles and Orange
Louisville/Jefferson County, KY-IN	Clark, Floyd, Harrison, Scott, Washington, Bullitt, Henry, Jefferson, Oldham, Shelby, Spencer, and Trimble
Macon, GA	Bibb, Crawford, Jones, Monroe, and Twiggs
Madera, CA	Madera
McAllen-Edinburg-Mission, TX	Hidalgo
Merced, CA	Merced
Modesto, CA	Stanislaus
Napa, CA	Napa
New York-Newark-Jersey City, NY-NJ-PA	Bergen, Essex, Hudson, Hunterdon, Middlesex, Monmouth, Morris, Ocean, Passaic, Somerset, Sussex, Union, Bronx, Dutchess, Kings, Nassau, New York, Orange, Putnam, Queens, Richmond, Rockland, Suffolk, Westchester, and Pike
Ogden-Clearfield, UT	Box Elder, Davis, Morgan, and Weber
Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	New Castle, Cecil, Burlington, Camden, Gloucester, Salem, Bucks, Chester, Delaware, Montgomery, and Philadelphia
Pittsburgh, PA	Allegheny, Armstrong, Beaver, Butler, Fayette, Washington, and Westmoreland
Prineville, OR	Crook
Provo-Orem, UT	Juab and Utah
Riverside-San Bernardino-Ontario, CA	Riverside and San Bernardino
Sacramento--Roseville--Arden-Arcade, CA	El Dorado, Placer, Sacramento, and Yolo
Salt Lake City, UT	Salt Lake, and Tooele
San Luis Obispo-Paso Robles-Arroyo Grande, CA	San Luis Obispo
South Bend-Mishawaka, IN-MI	St. Joseph and Cass
St. Louis, MO-IL	Bond, Calhoun, Clinton, Jersey, Macoupin, Madison, Monroe, St. Clair, Franklin, Jefferson, Lincoln, St. Charles, St. Louis, Warren, and St. Louis city
Stockton-Lodi, CA	San Joaquin
Visalia-Porterville, CA	Tulare
Weirton-Steubenville, WV-OH	Jefferson, Brooke, and Hancock
Wheeling, WV-OH	Belmont, Marshall, and Ohio

1

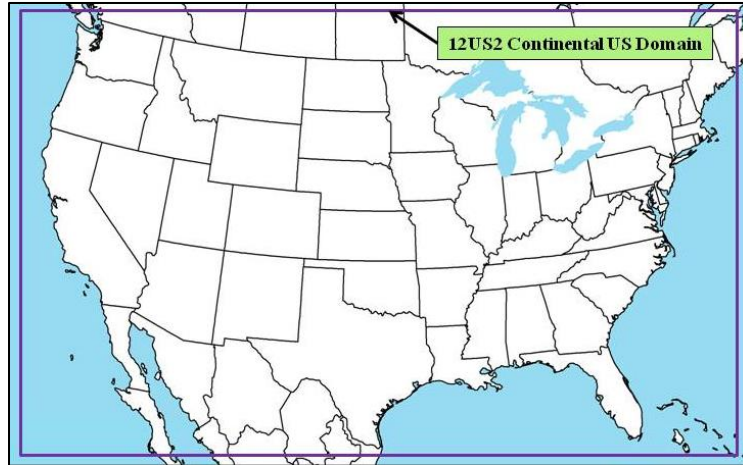
2 **C.1.4.3 Air Quality Modeling**

3 Air quality modeling was conducted using version 5.2.1 of the CMAQ modeling system
4 (Appel, 2018) to develop a continuous national field of PM_{2.5} concentrations and estimates of
5 how concentrations would respond to changes in PM_{2.5} and PM_{2.5} precursor emissions (Figure C-
6 6, “CMAQ”). The CMAQ modeling domain (Figure C-9) covered the contiguous U.S. with 12
7 km horizontal resolution and 35 vertical layers. Since 2015 was the most recent modeling

1 platform available at the time of the study and represents the central year of the 2014-2016 DV
2 period, 2015 was selected as the baseline modeling year for the PM_{2.5} projections. A single
3 modeling year was used due to the time and resources needed to conduct photochemical grid
4 modeling, and because model inputs for the 2016 period were not available at the time of the
5 study.

6 Information on the CMAQ model configuration for the 2015 modeling is provided in
7 Table C-5. The 2015 model simulation and its evaluation against network measurements of
8 speciated and total PM_{2.5} has been described in detail previously (Kelly et al., 2019b). Model
9 performance statistics for PM_{2.5} organic carbon, sulfate, and nitrate were generally similar to or
10 improved compared to the performance for other recent national 12 km model simulations. One
11 exception to the generally good model performance was identified for the Northwest region (OR,
12 WA, and ID). Model performance statistics for this region were generally not as good as in our
13 recent modeling due to issues related to unusually high fire influences in 2015, atmospheric
14 mixing over sites near the Puget Sound, and other factors. However, model performance issues
15 in the Northwest have minimal influence on the risk assessment, because only two of the 47
16 CBSAs are in the Northwest region (i.e., Prineville, OR and part of the Logan, UT-ID, CBSA).
17 Also, the analysis uses ratios of model predictions rather than absolute modeled concentrations,
18 and systematic biases associated with mixing height and fire impact estimates may largely cancel
19 in the ratios. Moreover, fusion of monitor data with model predictions in developing PM_{2.5} RRFs
20 and the baseline concentration field helps mitigate the influence of biases in model predictions
21 (as discussed below). Overall, the model performance evaluation (Kelly et al., 2019b) indicates
22 that the 2015 CMAQ simulation provides concentration estimates that are generally as good or
23 better than in other recent applications and are reliable for use in projecting PM_{2.5} in the risk
24 assessment. Model performance statistics for PM_{2.5} by U.S. climate region and season are
25 provided in Table C-6 and statistic definitions can be found in Table C-7.

26
27



1
2 **Figure C-9. CMAQ modeling domain.**

3
4 **Table C-5. CMAQ model configuration.**

Category	Description
Grid resolution	12 km horizontal; 35 vertical layers
Gas-phase chemistry	Carbon Bond 2006 (CB6r3)
Organic aerosol	Non-volatile treatment for primary organic aerosol; secondary organic aerosol from anthropogenic and biogenic sources
Inorganic aerosol	ISORROPIA II
NH ₃ surface exchange	Bi-directional NH ₃ surface exchange
Windblown dust emissions	Simulated online
Sea-spray emissions	Simulated online
Meteorology	Version 3.8 of Weather Research & Forecasting (WRF) Skamarock et al., 2005 model

5
6 **Table C-6. Model performance statistics^{22,23} for PM_{2.5} at AQS sites for the 2015 base case.**

Region ²³	Season	N	Avg. Obs. (µg m ⁻³)	Avg. Mod. (µg m ⁻³)	MB ²² (µg m ⁻³)	NMB ²² (%)	RMSE ²² (µg m ⁻³)	NME ²² (%)	r ²²
Northeast	Winter	13001	10.04	12.74	2.71	27.0	7.33	48.0	0.68
	Spring	13538	7.97	8.83	0.86	10.8	5.19	44.0	0.59
	Summer	13660	8.38	8.02	-0.36	-4.3	4.06	35.2	0.67
	Fall	13270	7.18	9.08	1.90	26.5	5.40	50.0	0.73
	Annual	53469	8.38	9.64	1.26	15.0	5.60	44.2	0.67
Southeast	Winter	11190	8.07	10.28	2.21	27.4	5.65	47.4	0.58
	Spring	11961	8.06	8.25	0.18	2.3	4.08	33.6	0.55
	Summer	11641	9.78	8.45	-1.33	-13.6	4.86	35.3	0.47
	Fall	11365	6.93	8.13	1.20	17.3	4.32	41.7	0.70

²² See Table C-7 for definition of statistics.

²³ See Figure C-10 for definition of regions.

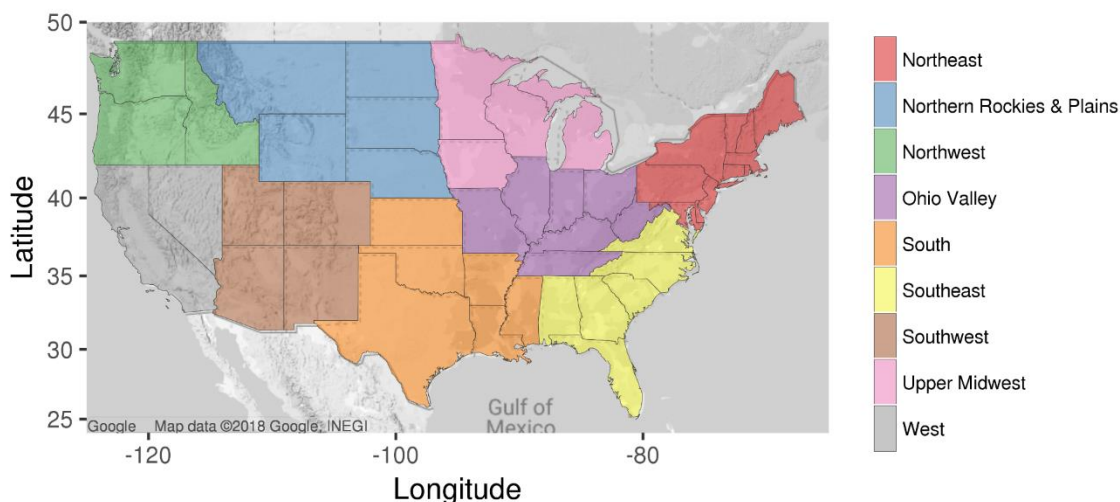
Region ²³	Season	N	Avg. Obs. ($\mu\text{g m}^{-3}$)	Avg. Mod. ($\mu\text{g m}^{-3}$)	MB ²² ($\mu\text{g m}^{-3}$)	NMB ²² (%)	RMSE ²² ($\mu\text{g m}^{-3}$)	NME ²² (%)	r^{22}
	Annual	46157	8.22	8.76	0.54	6.6	4.75	39.1	0.55
Ohio Valley	Winter	10323	9.49	11.60	2.10	22.1	5.75	43.2	0.63
	Spring	10867	8.90	9.85	0.95	10.6	4.60	36.3	0.65
	Summer	10714	10.95	10.56	-0.39	-3.6	5.55	34.3	0.55
	Fall	10568	8.41	10.96	2.54	30.2	6.23	47.1	0.65
	Annual	42472	9.44	10.73	1.29	13.6	5.56	39.8	0.59
Upper Midwest	Winter	6478	8.79	9.72	0.92	10.5	4.75	38.2	0.70
	Spring	6643	7.32	8.27	0.96	13.1	4.30	41.9	0.67
	Summer	6718	7.88	7.85	-0.03	-0.4	5.26	40.8	0.56
	Fall	6664	6.81	9.14	2.33	34.2	4.92	49.3	0.75
	Annual	26503	7.69	8.74	1.04	13.6	4.82	42.2	0.64
South	Winter	8041	7.53	10.13	2.60	34.5	11.81	56.6	0.36
	Spring	8369	8.08	7.12	-0.96	-11.9	4.24	36.3	0.51
	Summer	8440	10.80	8.31	-2.49	-23.0	6.04	40.3	0.34
	Fall	8340	7.55	7.99	0.44	5.9	3.76	35.5	0.63
	Annual	33190	8.50	8.37	-0.13	-1.6	7.15	41.8	0.34
Southwest	Winter	4911	7.46	7.90	0.45	6.0	6.50	55.9	0.52
	Spring	4998	4.88	5.88	1.00	20.6	3.60	48.4	0.44
	Summer	5069	6.12	4.85	-1.27	-20.8	4.15	43.1	0.59
	Fall	5091	5.31	5.90	0.59	11.1	4.35	52.2	0.49
	Annual	20069	5.93	6.12	0.19	3.2	4.77	50.2	0.52
N. Rockies & Plains	Winter	4987	5.57	3.60	-1.98	-35.5	6.80	63.4	0.23
	Spring	5380	4.57	5.00	0.44	9.6	29.58	61.6	0.20
	Summer	5260	9.98	7.68	-2.30	-23.1	17.61	57.4	0.57
	Fall	5010	5.57	5.42	-0.15	-2.7	5.65	56.4	0.44
	Annual	20637	6.43	5.45	-0.99	-15.3	18.06	59.2	0.34
Northwest	Winter	8994	7.90	7.82	-0.08	-1.0	10.20	80.9	0.25
	Spring	9306	5.02	6.84	1.82	36.2	6.65	71.5	0.48
	Summer	9993	9.17	11.12	1.95	21.2	32.40	67.7	0.46
	Fall	9868	7.03	9.39	2.37	33.7	15.33	78.3	0.31
	Annual	38161	7.31	8.85	1.55	21.2	19.26	74.3	0.43
West	Winter	10462	11.67	9.58	-2.08	-17.8	8.09	43.3	0.68
	Spring	10989	7.52	6.95	-0.57	-7.6	4.17	38.3	0.55
	Summer	11065	8.95	8.53	-0.43	-4.8	6.36	43.5	0.51
	Fall	10587	8.61	9.11	0.50	5.8	16.85	46.9	0.37
	Annual	43103	9.16	8.52	-0.64	-7.0	10.02	43.1	0.44

1
2
3
4
5
6
7

1 **Table C-7. Definition of statistics used in the CMAQ model performance evaluation.**

Statistic	Description
$MB (\mu\text{g m}^{-3}) = \frac{1}{n} \sum_{i=1}^n (P_i - O_i)$	Mean bias (MB) is defined as the average difference between predicted (P) and observed (O) concentrations for the total number of samples (n)
$RMSE (\mu\text{g m}^{-3}) = \sqrt{\sum_{i=1}^n (P_i - O_i)^2 / n}$	Root mean-squared error (RMSE)
$NMB (\%) = \frac{\sum_{i=1}^n (P_i - O_i)}{\sum_{i=1}^n O_i} \times 100$	The normalized mean bias (NMB) is defined as the sum of the difference between predictions and observations divided by the sum of observed values
$NME (\%) = \frac{\sum_{i=1}^n P_i - O_i }{\sum_{i=1}^n O_i} \times 100$	Normalized mean error (NME) is defined as the sum of the absolute value of the difference between predictions and observations divided by the sum of observed values
$r = \frac{\sum_{i=1}^n (P_i - \bar{P})(O_i - \bar{O})}{\sqrt{\sum_{i=1}^n (P_i - \bar{P})^2} \sqrt{\sum_{i=1}^n (O_i - \bar{O})^2}}$	Pearson correlation coefficient

2



3

4 **Figure C-10. U.S. climate regions²⁴ used in the CMAQ model performance evaluation.**

5 In addition to the national model performance evaluation just described, CMAQ
6 predictions of PM_{2.5} concentrations were evaluated specifically for the CBSAs considered in the
7 risk assessment. In Table C-8, model performance statistics are provided for predictions at
8 monitors in the 47 CBSAs in 2015. Predictions generally agree well with observations over the
9 full set of areas, with NMBs less than 10% in all seasons except Fall (NMB: 23.6%) and
10 correlation coefficients greater than 0.60 in all seasons except Summer (r: 0.56). Model
11 predictions are compared with observations by CBSA in Figure C-11, and NMBs at individual
12 sites in the CBSAs are shown in Figure C-12. Predictions generally agree well with observations
13 in the individual CBSAs, although underpredictions occurred in the Chicago-Naperville-Elgin

²⁴ <https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php>

1 CBSA when observed $PM_{2.5}$ concentrations were $> 40 \mu g m^{-3}$. The high observed values in
2 Chicago were associated with the 4th of July holiday, and the underpredictions on July 4th and 5th
3 have small influence on the annual $PM_{2.5}$ projections in the risk assessment. The NMB is highest
4 for model predictions in the Birmingham-Hoover CBSA (NMB: 66%). As mentioned above, the
5 effects of model bias are mitigated in part by use of relative response factors (i.e., the ratio model
6 predictions from a base and emission control simulation is used in projecting $PM_{2.5}$
7 concentrations, and some model bias likely cancels in the ratio). For the risk assessment
8 projections, the key aspect of the CMAQ modeling is the spatial of pattern of $PM_{2.5}$ response to
9 changes in emissions. The spatial response pattern was examined in the 47 CBSAs and found to
10 be reasonable even in areas with relatively high bias, such as Birmingham. In Figure C-13, the
11 spatial response pattern associated with the 10/30 projection case for the Birmingham-Hoover
12 CBSA is compared for the proportional projection method and the primary PM projection case
13 based on CMAQ modeling. Relatively high $PM_{2.5}$ responsiveness occurred in the urban part of
14 Birmingham and along arterial roads in the CMAQ-based approach. This spatial pattern is
15 consistent with the location of $PM_{2.5}$ emission sources in Birmingham and provides a realistic
16 spatial response pattern despite the relatively high bias in the concentration predictions. Overall,
17 both the national model performance evaluation and the evaluation for the 47 CBSAs of the risk
18 assessment support use of the CMAQ modeling in this application.

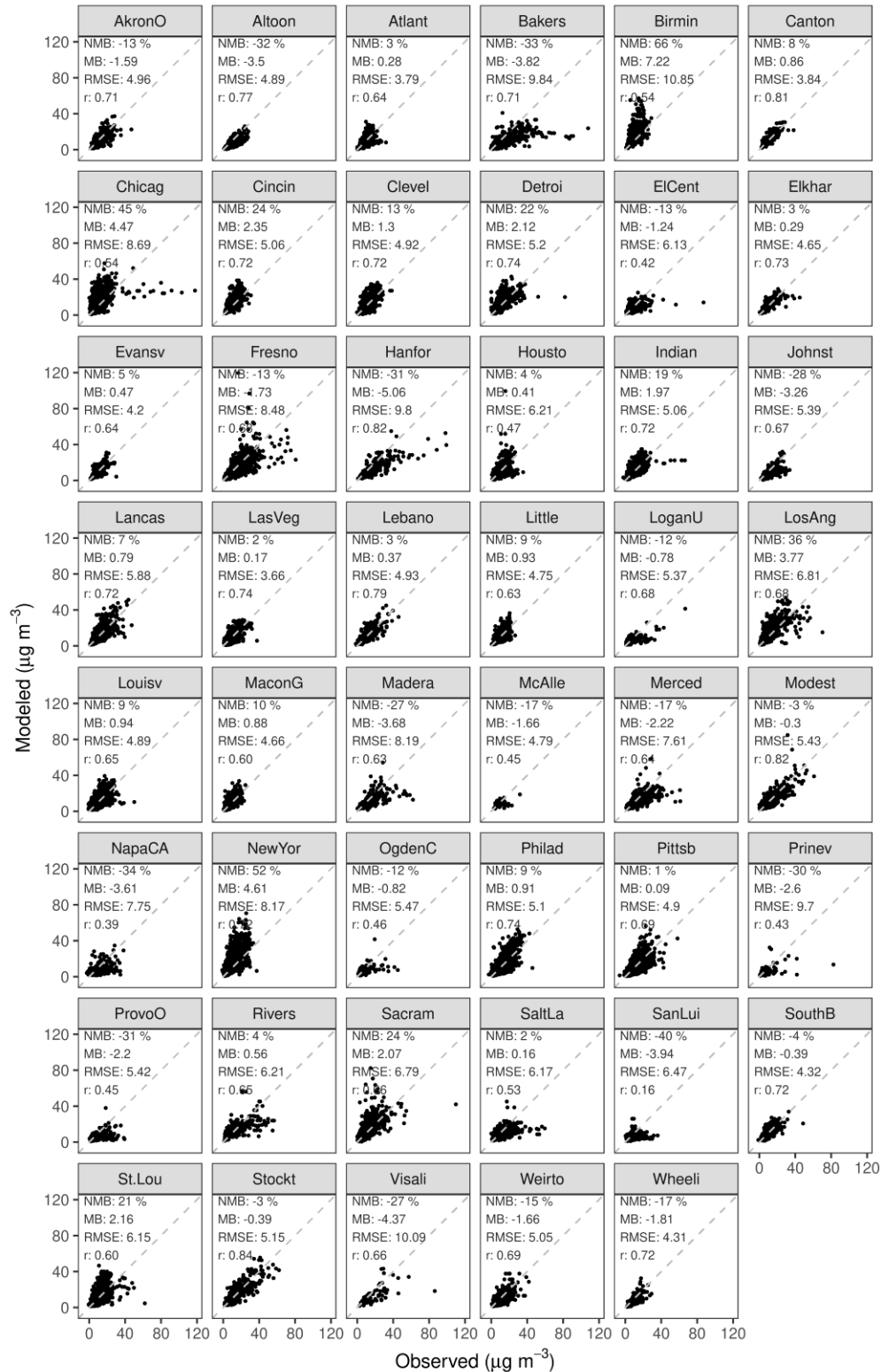
19 To inform $PM_{2.5}$ projections, annual CMAQ modeling was conducted using the same
20 configuration and inputs as the 2015 base case simulation but with anthropogenic emissions of
21 primary $PM_{2.5}$ or NO_x and SO_2 scaled by fixed percentages. Specifically, seven simulations were
22 conducted with changes in anthropogenic NO_x and SO_2 emissions (i.e., combined NO_x and SO_2 ,
23 not separate NO_x and SO_2 simulations) of -100%, -75%, -50%, -25%, +25%, +50%, and +75%.
24 Two simulations were conducted with changes in anthropogenic $PM_{2.5}$ emissions of -50% and
25 +50%. The sensitivity simulations were based on emission changes applied to all anthropogenic
26 sources throughout the year. These “across-the-board” emission changes facilitate projecting the
27 baseline concentrations to just meet a relatively wide range of standards in areas throughout the
28 U.S. using a feasible number of national sensitivity simulations.

29
30
31
32
33

1 **Table C-8. Performance statistics for CMAQ predictions at monitoring sites in the 47**
 2 **CBSAs considered in the risk assessment.**

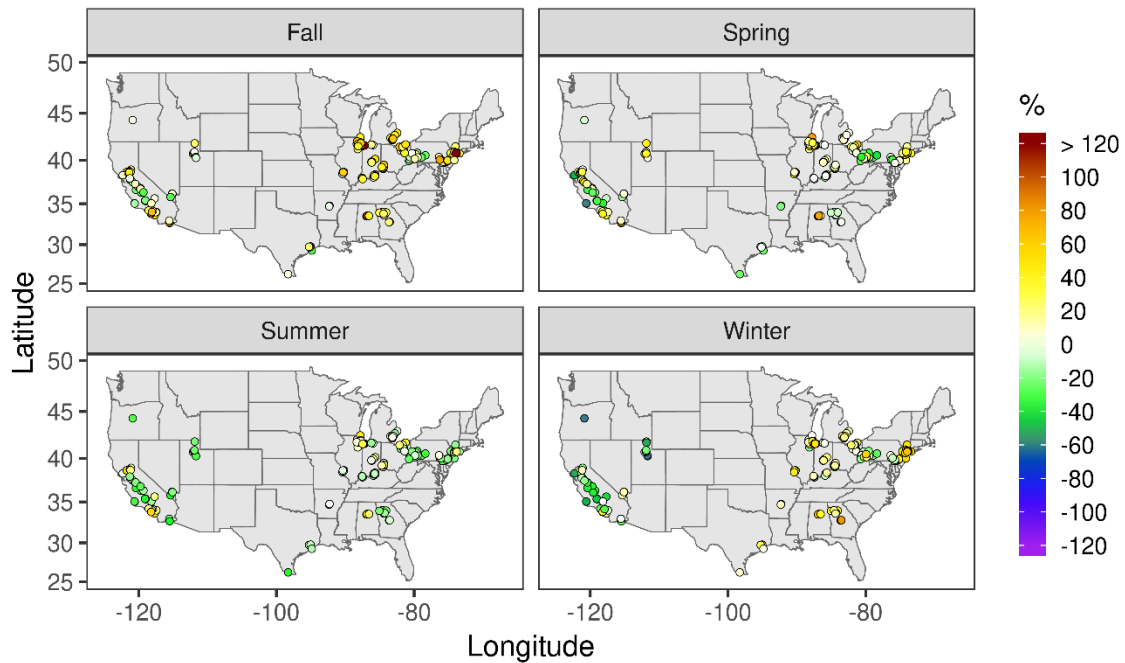
Season	Average Observed ($\mu\text{g m}^{-3}$)	Average Modeled ($\mu\text{g m}^{-3}$)	MB ($\mu\text{g m}^{-3}$)	NMB (%)	RMSE ($\mu\text{g m}^{-3}$)	NME (%)	<i>r</i>
Winter	12.40	13.45	1.05	8.5	8.03	42.4	0.61
Spring	9.17	9.94	0.77	8.4	5.15	38.6	0.62
Summer	10.35	10.08	-0.27	-2.6	5.51	34.6	0.56
Fall	9.00	11.11	2.12	23.6	6.26	45.6	0.67

3



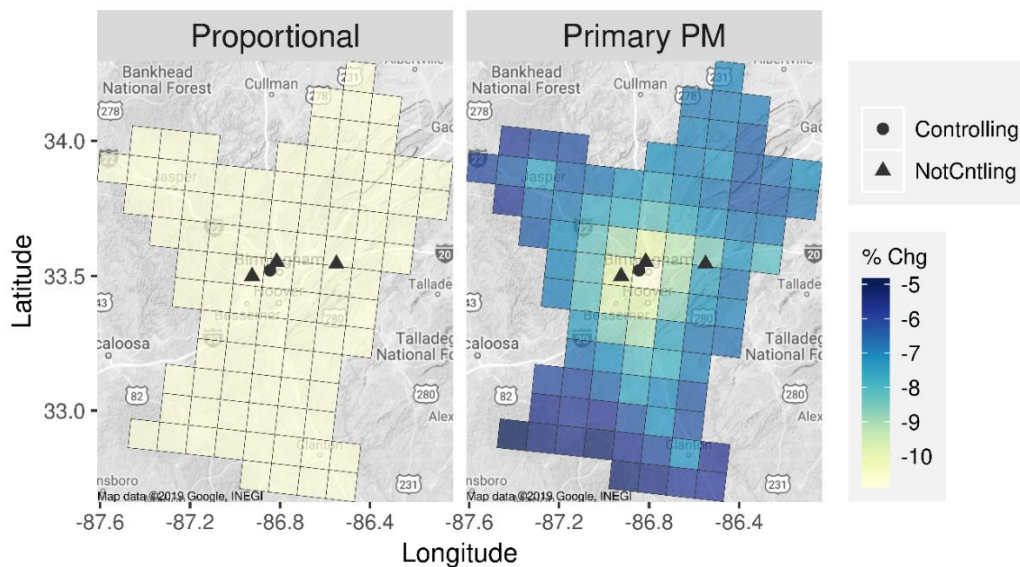
1
 2 **Figure C-11. Comparison of CMAQ predictions and observations at monitoring sites in the**
 3 **47 CBSAs considered in the risk assessment.**
 4

1
2



3
4
5
6

Figure C-12. NMB for CMAQ PM_{2.5} predictions at monitoring sites in the 47 CBSAs by season in 2015.



7
8
9
10
11
12

Figure C-13. Percent change in 2015 annual average PM_{2.5} over the Birmingham CBSA associated with projecting 2014–2016 DVs at monitors to just meet an alternative NAAQS of 10/30 using the proportional projection method and the primary PM_{2.5}, CMAQ-based projection method.

1 The two emission sensitivity scenarios (primary PM_{2.5} and NO_x and SO₂) were selected
 2 to span a wide range of possible PM_{2.5} spatial response patterns. NO_x and SO₂ emission changes
 3 influence concentrations of ammonium nitrate and ammonium sulfate, which are secondary
 4 pollutants that often have broad spatial distributions. Primary PM_{2.5} emission changes have the
 5 greatest influence on PM_{2.5} concentrations close to emission sources. The two distinctly different
 6 PM_{2.5} response patterns for primary PM_{2.5} and NO_x and SO₂ emission changes enable PM_{2.5} to
 7 be projected for a wide range of conditions. Projecting PM_{2.5} for a wide range of conditions is
 8 desirable in this study because many PM_{2.5} spatial response patterns can cause PM_{2.5}
 9 concentrations to just meet NAAQS.

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

11 The 2015 base case and sensitivity modeling results were used to develop RRFs for
 12 projecting PM_{2.5} concentrations to correspond to just meeting NAAQS (Figure C-6, Box 2,
 13 “SMAT-CE”). Baseline PM_{2.5} concentrations are projected by multiplication with RRFs. The
 14 RRF for a PM_{2.5} species is calculated as the ratio of the concentration in the sensitivity
 15 simulation to that in the base case:

$$16 \quad RRF_{species} = \frac{C_{sensitivity,species}}{C_{base,species}} \quad (1)$$

17 where $C_{sensitivity,species}$ is the concentration of the PM_{2.5} species in the sensitivity
 18 simulation, and $C_{base,species}$ is the concentration of the PM_{2.5} species in the base case simulation.
 19 RRFs were calculated for each monitor, grid cell, calendar quarter, standard (annual or 24-hr),
 20 species, and sensitivity simulation using SMAT-CE version 1.2.1. RRFs are used in projecting
 21 air quality to help mitigate the influence of systematic biases in model predictions (National
 22 Resources Council, U.S. EPA, 2018b). More details on the RRF projection method are provided
 23 in EPA’s modeling guidance document (U.S. EPA, 2018b) and the user’s guide for the
 24 predecessor to the SMAT-CE software (Abt Associates, 2014).

25 To apply the RRF approach for the risk assessment projections, RRFs for total PM_{2.5}
 26 were calculated from RRFs for the individual PM_{2.5} species using observation-based estimates of
 27 PM_{2.5} species concentrations in SMAT-CE output. Specifically, total PM_{2.5} RRFs ($RRF_{Tot,PM2.5}$)
 28 were calculated as the weighted average of the speciated RRFs using the observation-based
 29 species concentrations ($C_{species}$) as weights:

$$30 \quad RRF_{Tot,PM2.5} = \frac{\sum RRF_{species} C_{species}}{\sum C_{species}} \quad (2)$$

31 Total PM_{2.5} RRFs were used to project base-case PM_{2.5} concentrations as follows:

$$32 \quad PM_{2.5,projected} = RRF_{Tot,PM2.5} PM_{2.5,base} \quad (3)$$

33 The species concentrations used in calculating the total PM_{2.5} RRFs were generally based
 34 on application of the Sulfate, Adjusted Nitrate, Derived Water, Inferred Carbonaceous material
 35 balance approach (SANDWICH) (Frank, 2006) to measurements of PM_{2.5} species

1 concentrations from the Chemical Speciation Network (CSN)²⁵ and the Interagency Monitoring
2 of Protected Visual Environments (IMPROVE)²⁶ network. The SANDWICH method corrects for
3 different artifacts in the measurements for PM_{2.5} species and total PM_{2.5}. An alternative approach
4 to calculating total PM_{2.5} RRFs was applied for monitors and grid cells in California due to
5 factors including missing data at the Bakersfield speciation monitor²⁷ throughout 2014 and part
6 of 2015. For projections in California, RRFs were calculated directly from the ratio of CMAQ
7 PM_{2.5} concentration predictions in the sensitivity simulation to the base simulation.

8 By default, PM_{2.5} RRFs for the annual standard are calculated using average
9 concentrations over all modeled days in the quarter, and RRFs for the 24-hr standard are
10 calculated using average concentrations over days with the top 10% of modeled PM_{2.5}
11 concentration in the quarter. The default approach was generally followed here, with exceptions
12 for counties in the San Joaquin Valley (SJV) of California and Utah. In these counties²⁸, the
13 average concentration over all days in the quarter was used to calculate RRFs for both the 24-hr
14 and annual standards for sites with valid 24-hr and annual DVs. This approach was used to
15 provide stability in projections of annual fields due the variability in the 24-hr and annual
16 RRFs²⁹. Also, RRFs were set to one³⁰ in the third quarter (July-September) for select counties in
17 the San Joaquin Valley and Utah³¹ to better reflect the seasonal nature of PM_{2.5} in these areas
18 (i.e., PM_{2.5} concentrations are relatively high in winter).

19 RRFs were calculated for each combination of emission sensitivity simulation and the
20 2015 base case. RRFs corresponding to the percent change in emissions for each sensitivity
21 simulation were then interpolated across the range of emission changes from -100 to +100% to
22 facilitate iterative projections of PM_{2.5} concentrations to the nearest percent emission change.
23 PM_{2.5} RRFs are shown in Figure C-14 and Figure C-15 as a function of changes in anthropogenic
24 primary PM_{2.5} and NO_x and SO₂ emissions for monitors in the U.S. during the first and third

²⁵ www.epa.gov/amtic/chemical-speciation-network-csn

²⁶ <http://vista.cira.colostate.edu/Improve/>

²⁷ Site identification number: 060290014

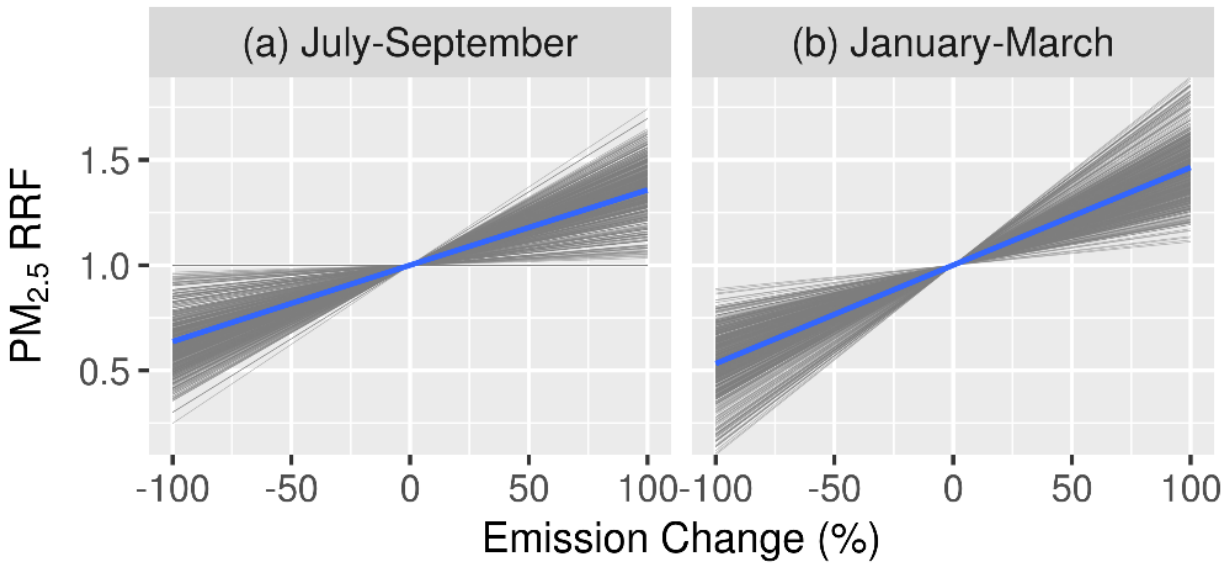
²⁸ SJV counties: Fresno, Stanislaus, Kern, Merced, Madera, Tulare, San Joaquin, and Kings; Utah counties: Cache, Box Elder, Davis, Morgan, Weber, Juab, Utah, Salt Lake, and Tooele.

²⁹ This variability is less of an issue in regional modeling applications where emission changes can be targeted to time periods of elevated PM_{2.5} concentrations in the area.

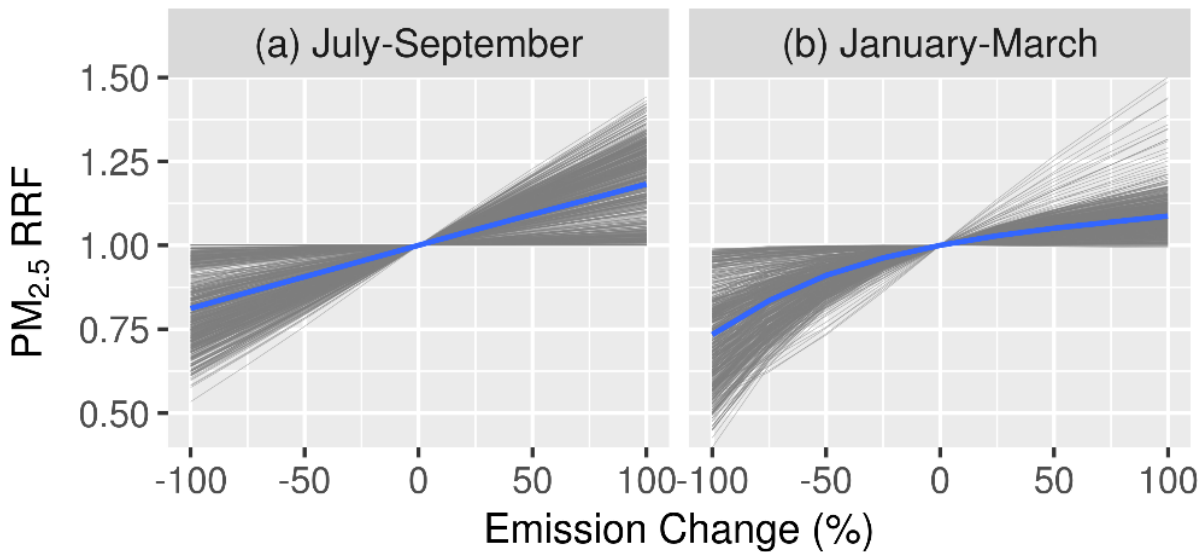
³⁰ When the RRF is 1, the projected concentration equals the base concentration (Equation 3).

³¹ SJV counties: Fresno, Stanislaus, Kern, Merced, and Madera; Utah counties: Cache, Box Elder, Davis, Morgan, Weber, Juab, Utah, Salt Lake, and Tooele. This approach was not applied for Kings, Tulare, and San Joaquin counties in SJV because the percent exceedance of the annual standard was within 10% of the exceedance of the 24-hr standard suggesting that relatively uniform PM_{2.5} concentrations occur throughout the year compared with the other SJV counties.

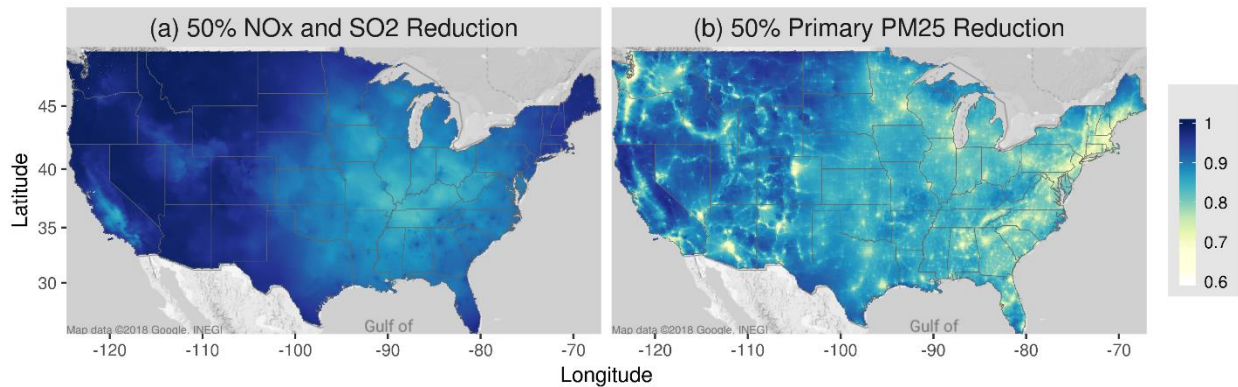
1 calendar quarters. Spatial fields of $PM_{2.5}$ RRFs for 50% reductions in anthropogenic primary
2 $PM_{2.5}$ and NO_x and SO_2 emissions are shown in Figure C-16.
3



4
5 **Figure C-14. Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent**
6 **change in anthropogenic primary $PM_{2.5}$ emissions for monitoring sites in the contiguous**
7 **U.S.**
8



9
10 **Figure C-15. Annual standard $PM_{2.5}$ RRFs for quarters 1 and 3 as a function of the percent**
11 **change in anthropogenic NO_x and SO_2 emissions for monitoring sites in the contiguous**
12 **U.S.**
13



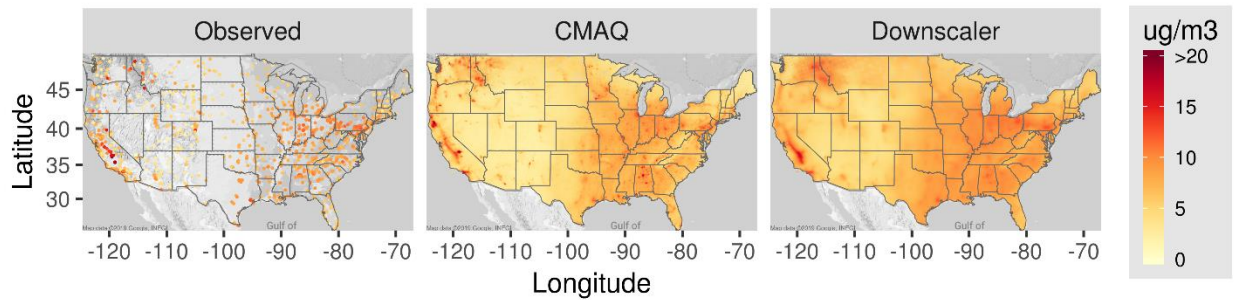
1
2 **Figure C-16. Annual average PM_{2.5} RRFs at CMAQ grid-cell centers for 50% reductions in**
3 **anthropogenic (a) NO_x and SO₂ and (b) primary PM_{2.5} emissions.**

4
5 **C.1.4.5 2015 PM_{2.5} Concentration Fields**

6 To develop a baseline gridded PM_{2.5} concentration field for projection with PM_{2.5} RRFs,
7 a Bayesian statistical model (i.e., Downscaler) was applied (Figure C-6, Box 2, “Downscaler”)
8 (Berrocal et al., 2012). Downscaler makes predictions of PM_{2.5} concentrations to a spatial field
9 of receptor points using PM_{2.5} monitoring data and CMAQ model predictions as inputs.
10 Downscaler takes advantage of the accuracy of the monitoring data and the spatial coverage of
11 the CMAQ predictions to develop new predictions of PM_{2.5} concentration over the U.S.

12 The Downscaler model is routinely applied by U.S. EPA to predict 24-hr average PM_{2.5}
13 concentrations at the centroids of census tracts in the contiguous U.S. (U.S. EPA, 2018a). The
14 model configuration used here is generally consistent with the previous applications, but here
15 predictions were made to the centers of the CMAQ model grid cells rather than to census-tract
16 centroids. Also, PM_{2.5} measurements from the IMPROVE monitoring network were used in
17 addition to measurements included in the AQS database. 24-hr average PM_{2.5} concentrations
18 were predicted for the 2015 period, and the 24-hr PM_{2.5} fields were averaged to the quarterly
19 periods of the PM_{2.5} RRFs for use in projection.

20 Annual average PM_{2.5} concentrations from the monitoring network and CMAQ
21 simulation that were used in model fitting are shown in Figure C-17 along with the resulting
22 Downscaler predictions. Cross-validation statistics are provided in Table C-9 based on
23 comparisons of Downscaler predictions against the 10% of the observations that were randomly
24 withheld from model fitting.



1
2 **Figure C-17. Annual average of the 2015 PM_{2.5} observations and CMAQ predictions used**
3 **in the Downscaler model, and the annual average of the Downscaler PM_{2.5} predictions.**

4
5 **Table C-9. Cross-validation statistics associated with the 2015 Downscaler predictions.**

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

^aThe mean of all biases across the CV cases, where the bias of each prediction is the downscaler prediction minus the observed value.
^bThe bias is squared for each CV prediction, then the square root of the mean of all squared biases across all CV predictions is obtained.
^cA value of 1 is assigned if the measured value lies in the 95th percentile CI of the Downscaler prediction (the Downscaler prediction \pm the Downscaler standard error), and 0 otherwise. This column is the mean of all those 0's and 1's.

6
7 **C.1.4.6 Projecting PM_{2.5} to Just Meet the Standards**

8 PM_{2.5} was projected from baseline concentrations to levels corresponding to just meeting
9 NAAQS using the monitoring data (section C.1.4.2), RRFs (section C.1.4.4), and baseline
10 concentration fields (section C.1.4.5) described above. The projection was done in two steps as
11 shown in Box 3 of Figure C-6. Projections were performed for the existing (12/35)³² and
12 alternative (10/30)³³ standards.

13 First, monitors in the CBSA of interest were identified, and concentrations from these
14 monitors were subset from the national monitoring dataset. The measured concentrations were
15 then projected using the corresponding PM_{2.5} RRF. PM_{2.5} DVs were calculated using the
16 projected concentrations, and the difference between the maximum projected DV and target
17 standard was determined. DV projections over the complete range of percent emission changes (-
18 100 to 100%) were performed using bisection iteration until the difference between the

³² Annual standard level of 12 $\mu\text{g m}^{-3}$ and 24-hr standard level of 35 $\mu\text{g m}^{-3}$

³³ Annual standard level of 10 $\mu\text{g m}^{-3}$ and 24-hr standard level of 30 $\mu\text{g m}^{-3}$

1 maximum projected DV in the CBSA and the standard level was zero or within the difference
2 associated with a 1% emission change. Iterative projections of annual and 24-hr DVs were
3 performed separately, and the controlling standard was determined as the standard requiring the
4 greater percent emission change³⁴. In cases where the emission change needed to just meet the
5 target annual or 24-hr standard was outside of the $\pm 100\%$ range, the standard could not be met
6 using the modeled air quality scenarios. If neither the annual nor 24-hr standard could be just met
7 with emission changes within $\pm 100\%$, then an alternative projection approach was used
8 (discussed below).

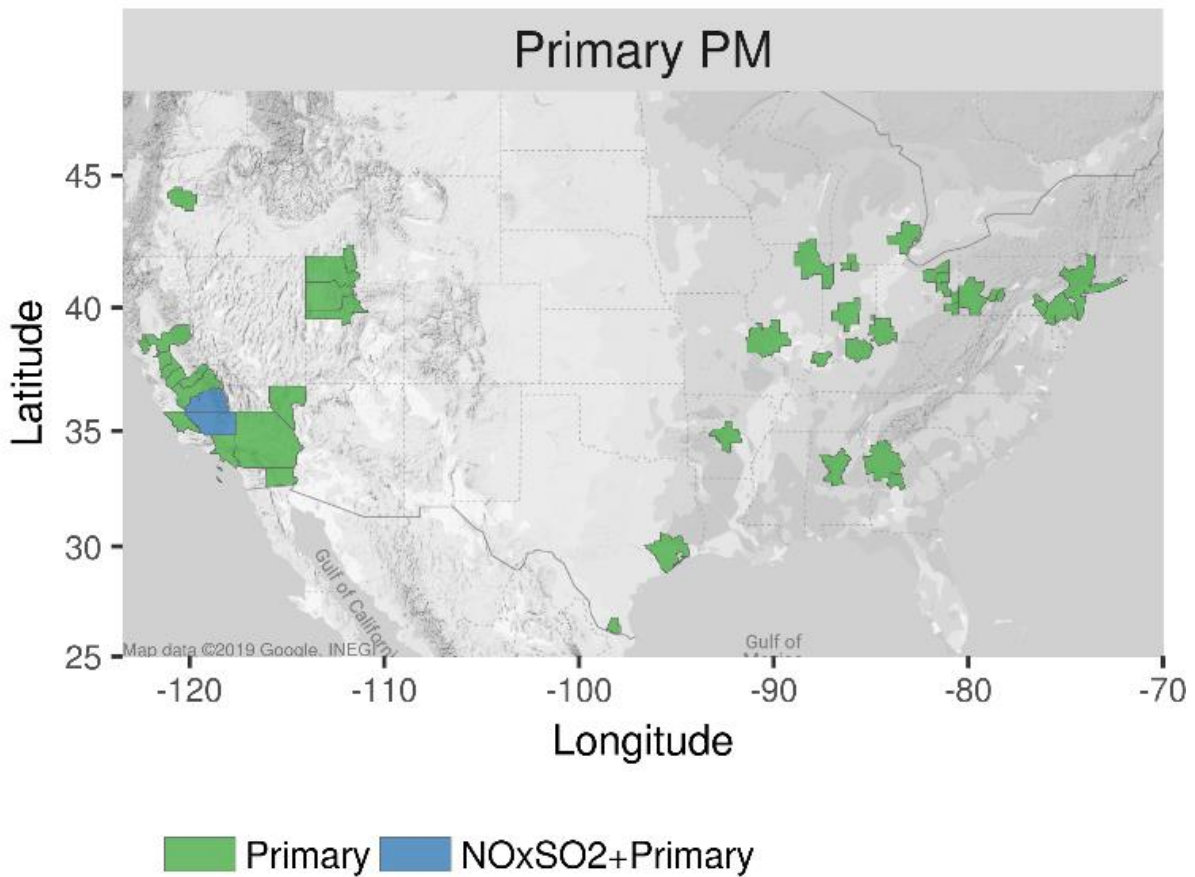
9 Second, 2015 PM_{2.5} concentration fields developed with Downscaler were projected
10 according to the percent emission change required for the maximum projected DV to just meet
11 the controlling standard. The projection was done by multiplying the gridded spatial fields of
12 quarterly average PM_{2.5} concentrations based on Downscaler modeling with the gridded spatial
13 fields of quarterly PM_{2.5} RRFs corresponding to the percent emission change required to just
14 meet the controlling standard. The projected fields of quarterly average PM_{2.5} concentrations
15 were then averaged to produce the annual average projected field.

16 Since PM_{2.5} concentrations can be projected in multiple ways to just meet a standard,
17 projections were done for two scenarios that provide results for a range of PM_{2.5} conditions. The
18 first scenario is referred to as “Primary PM” or Pri-PM because projections were largely based
19 on RRFs developed using CMAQ sensitivity simulations with primary PM_{2.5} emission changes.
20 For three CBSAs³⁵, standards could not be met using primary PM_{2.5} emission reductions alone.
21 PM_{2.5} concentrations were projected for these areas using a combination of primary PM_{2.5} and
22 NO_x and SO₂ emission reductions in the Primary PM scenario³⁶ (Figure C-18).

³⁴ Note that calculations are performed in terms of percent emission reduction. Therefore, in cases where DVs are projected to just meet standards greater than the baseline DVs, the required percent emission reduction is negative (i.e., an emission increase is required), and the smaller absolute percent emission change is selected as the controlling case. For example, the annual standard would be selected as controlling in a case where a 10% emission increase is needed to meet the annual standard and a 50% emission increase is needed to meet the 24-hr standard (because -10 is greater than -50).

³⁵ Bakersfield, Hanford-Corcoran, and Visalia-Porterville (all in California)

³⁶ This approach was applied by using RRFs from the NO_x and SO₂ emission sensitivity simulations to eliminate a fraction of the difference between the maximum base DV and the standard level and then using RRFs from the primary PM_{2.5} emission sensitivity simulations to eliminate the remainder of the difference. The fraction of the difference eliminated with NO_x and SO₂ emission reductions was as follows: 0.4 for Bakersfield, 0.5 for Visalia-Porterville, and 0.6 for Hanford-Corcoran



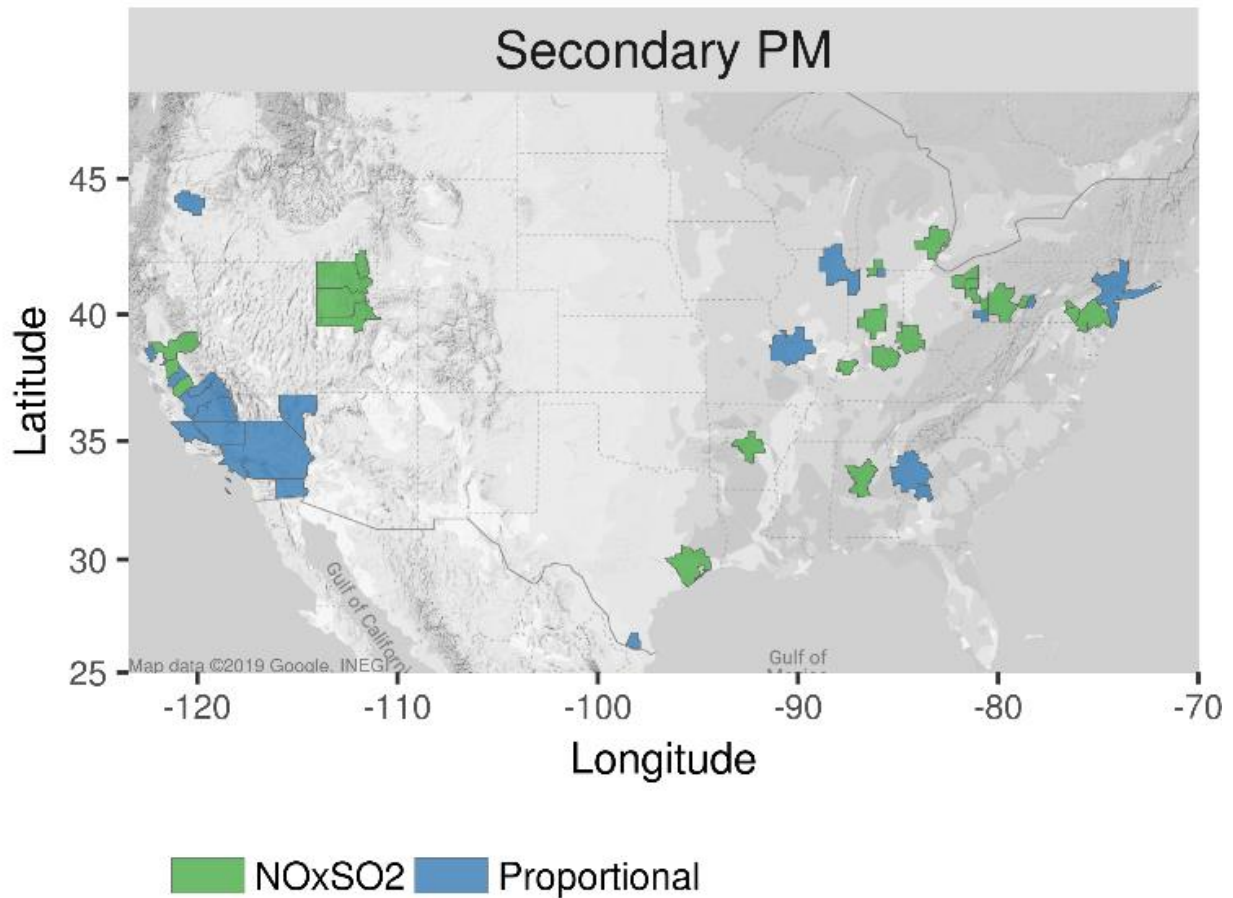
1
 2 **Figure C-18. Projection method used for each CBSA in the “Primary PM” projection case.**
 3 **See text for details.**

4
 5 The second scenario is referred to as “Secondary PM” or Sec-PM because projections
 6 were largely based on RRFs developed using CMAQ modeling with NO_x and SO₂ emission
 7 changes, which affect concentrations of secondary PM components such as ammonium nitrate
 8 and ammonium sulfate. For 22 CBSAs³⁷, standards could not be just met using NO_x and SO₂
 9 emission changes alone. These areas were projected using the proportional scaling method³⁸
 10 (Figure C-19). The proportional method was selected to gap-fill the Secondary PM case because

³⁷ Altoona, PA; Atlanta-Sandy Springs-Roswell, GA; Bakersfield, CA; Chicago-Naperville-Elgin, IL-IN-WI; El Centro, CA; Elkhart-Goshen, IN; Fresno, CA; Hanford-Corcoran, CA; Las Vegas-Henderson-Paradise, NV; Los Angeles-Long Beach-Anaheim, CA; Macon, GA; Madera, CA; McAllen-Edinburg-Mission, TX; Modesto, CA; Napa, CA; New York-Newark-Jersey City, NY-NJ-PA; Prineville, OR; Riverside-San Bernardino-Ontario, CA; St. Louis, MO-IL; San Luis Obispo-Paso Robles-Arroyo Grande, CA; Visalia-Porterville, CA; Wheeling, WV-OH

³⁸ In the proportional method, the spatial field is uniformly scaled by a fixed percentage that corresponds to the percent difference between the controlling standard level and maximum PM_{2.5} DV for the controlling standard. The controlling standard (annual or 24-hr) is identified as the one with the greater percent difference between the maximum DV and the standard level.

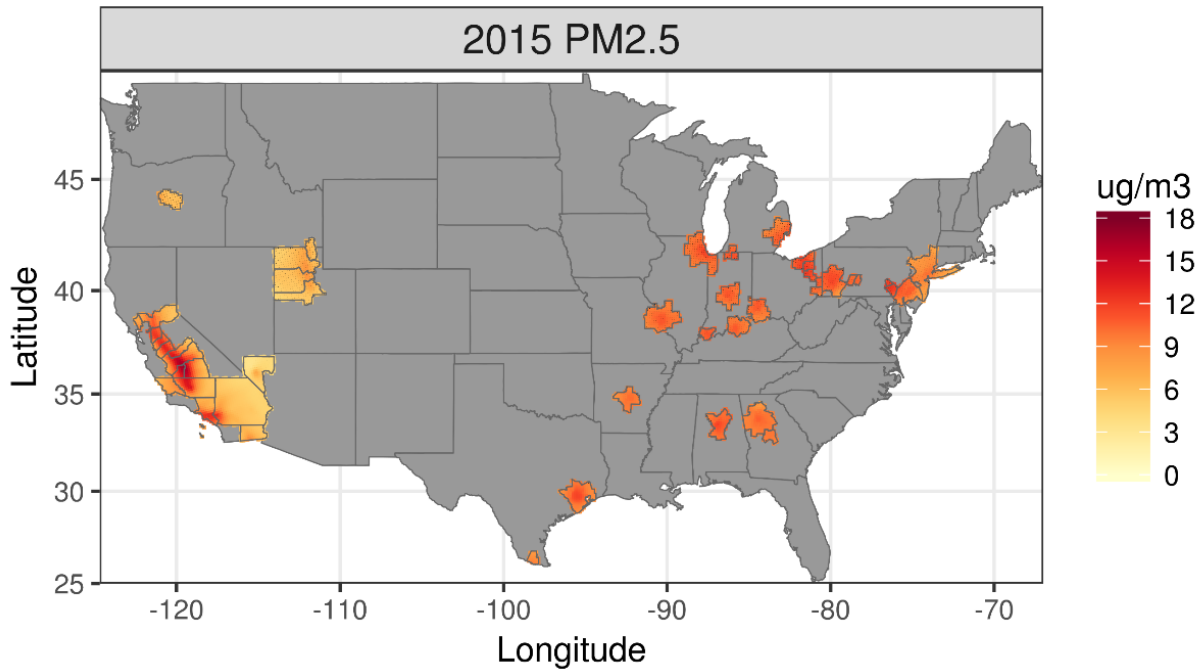
1 it is based on a spatially uniform percent change in $PM_{2.5}$ over the area that is like the
 2 conceptually broad spatial response pattern of $PM_{2.5}$ to changes in secondary $PM_{2.5}$ components.
 3 The proportional method has been used previously in the Risk and Exposure Assessment for the
 4 2012 PM NAAQS review (U.S. EPA, 2010).
 5



6
 7 **Figure C-19. Projection method used for each CBSA in the “Secondary PM” projection**
 8 **case.**

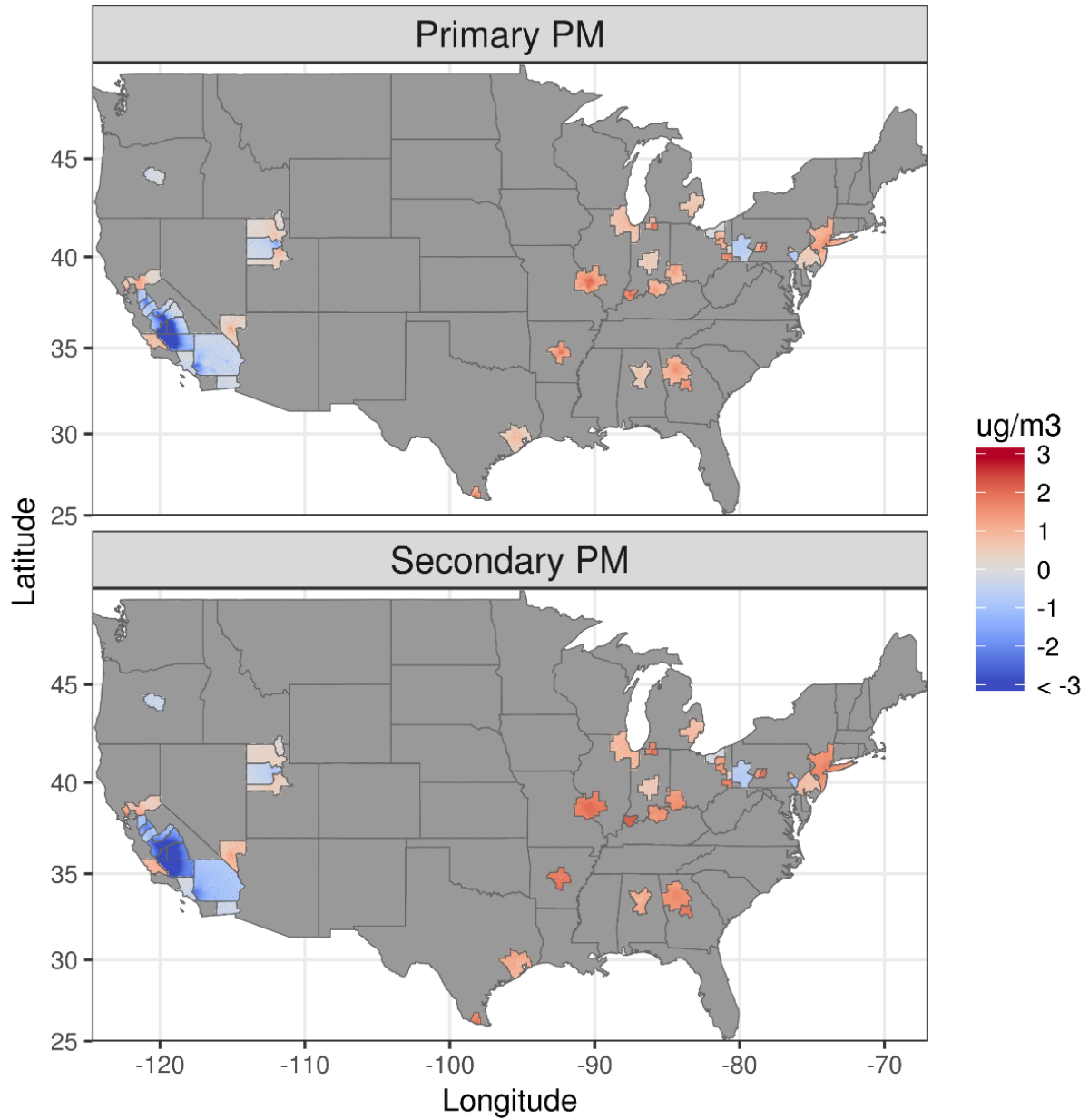
9
 10 The baseline 2015 concentration in the 47 CBSAs is shown in Figure C-20. These
 11 concentrations are the same as those in Figure C-17 but are shown only for the CBSAs included
 12 in the projections. In Figure C-21, the difference in annual concentration projected for the 12/35
 13 case and the 2015 baseline concentration is shown. The positive and negative differences reflect
 14 areas where concentrations were projected to higher and lower levels to just meet the standard,
 15 respectively. In Figure C-22, the difference between the annual concentration projected for the
 16 10/30 case and the and 2015 baseline concentration. Negative values indicate that concentrations

1 were projected to lower levels in all cases for the areas. The difference in projected
2 concentrations for the 10/30 and 12/35 fields is shown in Figure C-23. Baseline and projected
3 PM_{2.5} DVs for monitors in the 47 CBSAs are provided in Table C-19, Table C-20, Table C-21,
4 and Table C-22 in section C.6.³⁹
5



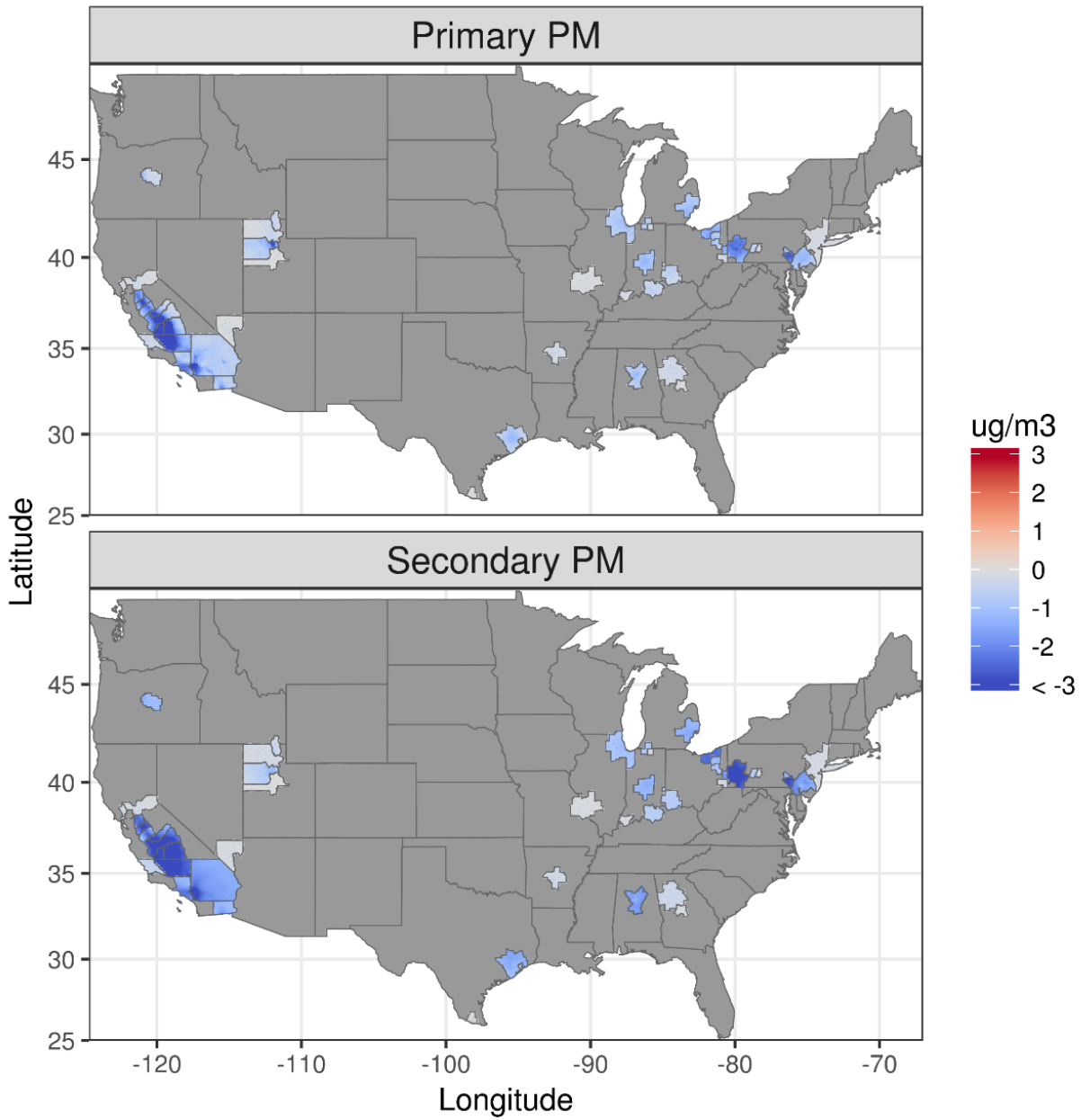
6
7 **Figure C-20. Annual average 2015 PM_{2.5} concentrations in the 47 CBSAs based on**
8 **Downscaler modeling.**
9

³⁹ The tables report the percent emission reduction associated with just meeting standards in the current modeling. These values should not be interpreted as the percent emission reductions that would be required to meet the standards in other application (e.g., attainment demonstrations for state implementation plans). The modeling done here was designed to quickly project PM_{2.5} fields throughout the U.S. with a broad range of model response patterns, rather than to apply model configurations and emission scenarios specific to just meeting standards most efficiently in particular regions.

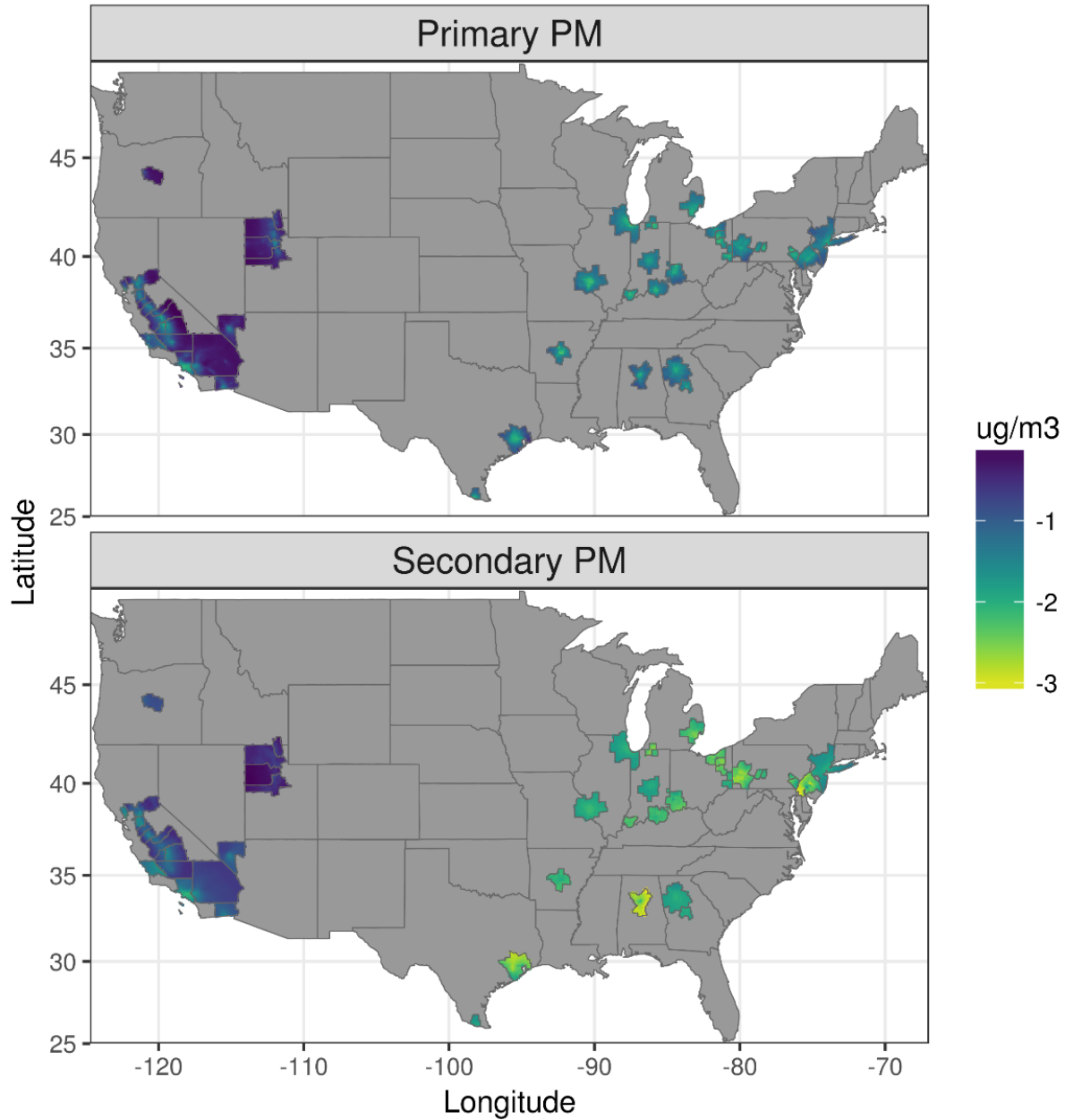


1
2
3
4

Figure C-21. Difference between the annual average projected PM_{2.5} concentrations and the 2015 baseline concentrations for the 12/35 projection cases (i.e., 12/35 – baseline).



1
 2 **Figure C-22. Difference between the annual average projected PM_{2.5} concentrations and**
 3 **the 2015 baseline concentrations for the 10/30 projection cases (i.e., 10/30 – baseline).**
 4



1
 2 **Figure C-23. Difference between the annual average projected PM_{2.5} concentrations in the**
 3 **10/30 and 12/35 cases (i.e., 10/30 – 12/35) for the Primary PM and Secondary PM**
 4 **projection cases.**

5
 6 **C.1.4.7 Limitations**

7 There are several limitations associated with the air quality projections. First, the baseline
 8 and projected concentrations rely on model predictions. Although state-of-the-science modeling
 9 methods were applied, and model performance was generally good, there is uncertainty
 10 associated with the model predictions. Second, due to the national scale of the assessment, the

1 modeling scenarios are based on “across-the-board” emission changes in which emissions of
2 primary PM_{2.5} or NO_x and SO₂ from all anthropogenic sources throughout the U.S. are scaled by
3 fixed percentages. Although this approach tends to target the key sources in each area, it does not
4 tailor emission changes to specific periods or sources. More refined emission scenarios could be
5 beneficial for projections in areas with relatively large seasonal and/or spatial variability in
6 PM_{2.5}. Similarly, fine scale simulations (e.g., 4 km or less), which are not possible due to the
7 national scale of the assessment, would be beneficial in areas with complex terrain and relatively
8 large spatial gradients in PM_{2.5}. A third limitation arises because many emission cases could be
9 applied to project PM_{2.5} concentrations to just meet standards. We applied two projection cases
10 that span a wide range of possible conditions, but these cases are necessarily a subset of the full
11 set of possible projection cases.

12 **C.1.5 Risk Modeling Approach**

13 Risk modeling for this assessment was completed using BenMAP-CE version 1.5.⁴⁰
14 BenMAP-CE was used to estimate risk at the 12 km grid cell level for grid cells intersected by
15 the 47 urban study area CBSAs included in risk modeling. BenMAP-CE is an open-source
16 computer program that calculates the number and economic value of air pollution-related deaths
17 and illnesses. The software incorporates a database that includes many of the CR relationships,
18 population files, and health and economic data needed to quantify these impacts. BenMAP-CE
19 also allows the user to import customized datasets for any of the inputs used in modeling risk.
20 For this analysis, CR functions developed specifically for this assessment were imported into
21 BenMAP-CE (section C.1.1). The BenMAP-CE tool estimates the number of health impacts
22 resulting from changes in air quality. BenMAP-CE can also translate these incidence estimates
23 into monetized benefits, although that functionality was not employed for this risk assessment.
24 Inputs to BenMAP-CE used for this risk assessment are identified above in Figure C-1 and
25 described in detail in sections C.1.1, C.1.2C.1.3, and C.1.4.

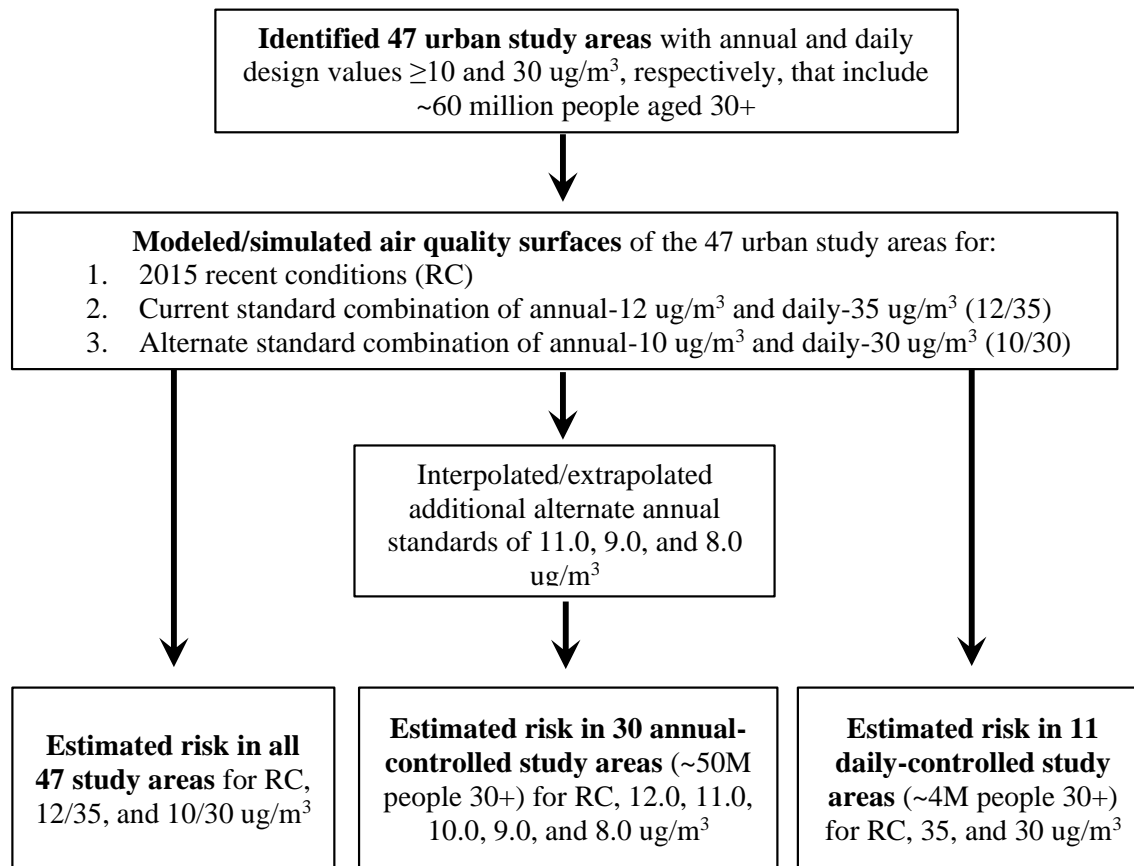
26 An overall flow diagram of the risk assessment approach is provided in Figure C-24.
27 Application of this approach resulted in separate sets of risk estimates being generated for the
28 following three groupings of urban study areas:

- 29 – the full set of 47,
- 30 – the 30 areas controlled by the annual standard, and
- 31 – the 11 areas controlled by the 24-hr standard.

32 Available air quality modeling surfaces for each of the three study area groupings are
33 summarized in Table C-10.

34

⁴⁰ BenMAP-CE is a free program which can be downloaded from: <https://www.epa.gov/benmap>.



1
2 **Figure C-24. Flow diagram of risk assessment technical approach.**

3
4 **Table C-10. Summary of available air quality scenarios for each study area set**

	47 Study Areas (full set)	30 Study Areas (annually controlled)	11 Study Areas (daily controlled)
Recent Conditions (2015)	X	X	X
Just meeting 12/35 $\mu\text{g}/\text{m}^3$	X	X	X
11 $\mu\text{g}/\text{m}^3$ (interpolated)		X	
Just meeting 10/30 $\mu\text{g}/\text{m}^3$	X	X	X
9 $\mu\text{g}/\text{m}^3$ (extrapolated)		X	
8 $\mu\text{g}/\text{m}^3$ (extrapolated)		X	

5
6 Risk estimates are presented and discussed for each of these groupings in draft PA
7 section 3.4.2, with greater emphasis being placed on results generated for the full set of 47 urban
8 study areas and 30 annual-controlled study areas, given interest in national representation and on

1 those study areas where we could also consider the alternative annual standards of 8.0, 9.0 and
2 11.0 $\mu\text{g}/\text{m}^3$.

3 **C.2 SUPPLEMENTAL RISK RESULTS**

4 As noted earlier, this appendix also presents additional granular risk results that supplement the
5 aggregated risk estimates presented and discussed in section 3.4.2 of the draft PA. The
6 supplemental results are intended to provide additional context for the interpretation of summary
7 risk estimates presented in draft PA section 3.4.2 and include additional line plots, maps and
8 scatter plots illustrating the distribution of the grid-level risk estimates across ambient $\text{PM}_{2.5}$
9 concentrations (section C.2). Graphics provide insight into various aspects of the grid-level data
10 underlying the summary tables presented in the draft PA, such as the spatial distribution of risk
11 across the cities included in the risk assessment and how the distribution of grid-cell level risk
12 estimates shifts as lower alternative standards are considered.

13 It can be challenging to understand how patterns of risk are changing under air quality
14 simulated to just meet the current or alternative standards, due to differences in underlying
15 demographics (e.g., size and age of population), health status (e.g., underlying death rates) and
16 exposure (air quality conditions). To better illustrate the distribution of risk under the current
17 standards and how that distribution changes under potential alternative standards, this section
18 presents graphics depicting these changes both in aggregate and at the grid-cell level.

19 As the pattern of risk and risk reduction is similar across mortality endpoints, we focus on
20 a single CR function to illustrate the changes graphically. Consequently, as with the graphics
21 presented in draft PA section 3.4.2, the graphics presented in this section are also based on long-
22 term exposure-related all-cause mortality modeled using a CR function obtained from Turner et
23 al., 2016. The first set of graphics presented in this section (Figure C-25, Figure C-26, Figure C-
24 27, Figure C-28, and Figure C-29) include results for the full set of 47 urban study areas and the
25 second set (Figure C-30 and Figure C-31) include results for the 30 annual-controlled study
26 areas. Graphical plots include:

- 27 • Histograms showing the distribution of 12 km gridded risk estimates across annual-
28 averaged $\text{PM}_{2.5}$ concentrations (Figure C-25 and Figure C-30). These figures allow
29 consideration of how the distribution of risk shifts when simulating air quality that just
30 meets the current standards (12/35 $\mu\text{g}/\text{m}^3$) relative to 2015 recent conditions and
31 subsequently how that distribution of risk shifts downward when simulating air quality
32 that just meets alternative standards of 10/30 $\mu\text{g}/\text{m}^3$.
- 33 • Maps showing the 12 km grid-level risk estimates associated with each of the 47 urban
34 study areas. In these representative maps each grid cell is shown as a square, with the
35 color of the square going from green (lower risk estimates) to red (higher risk estimate)

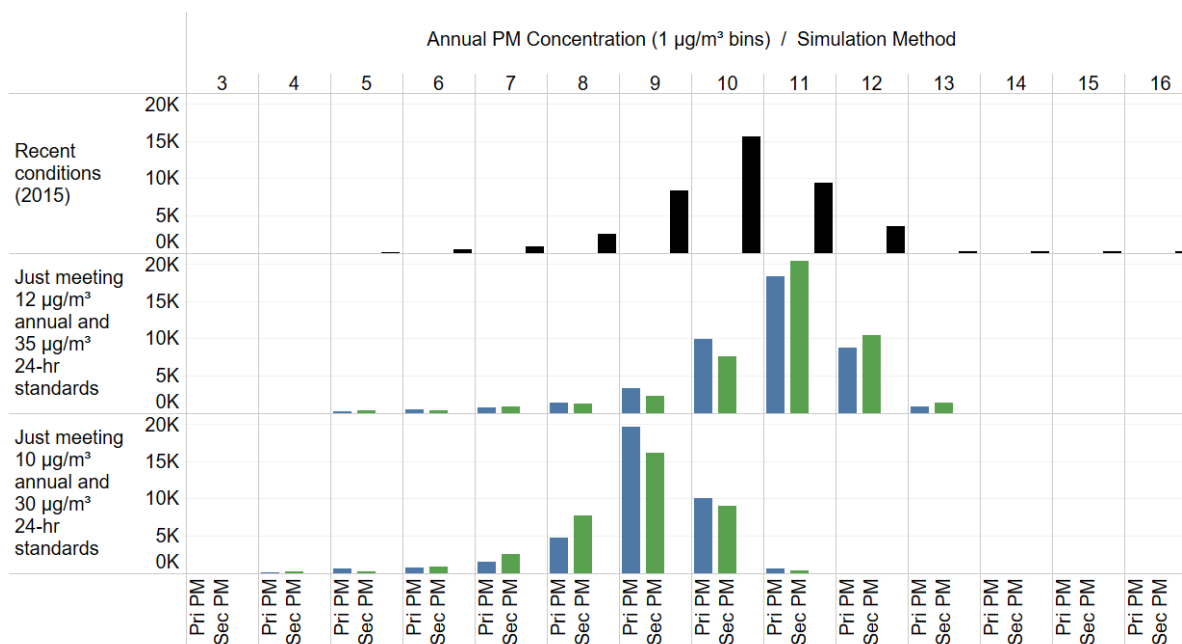
1 colors. The center of the color scales (the beginning of yellow) has been set to a risk
2 estimate of two premature deaths. This means that green squares represent grid cells
3 where 0-1 premature deaths are estimated, yellow squares represent grid cells in which at
4 least two premature deaths are estimated, and as the color graduation approaches red the
5 number of estimated premature deaths increases. Separate maps are presented for
6 (a) the unadjusted 2015 recent conditions simulation (Figure C-26),
7 (b) simulation of the current standards (12/35 $\mu\text{g}/\text{m}^3$) (Figure C-27), and
8 (c) simulation of the change (delta) in risk between the current and alternative
9 standards (10/30 $\mu\text{g}/\text{m}^3$) (Figure C-28).

- 10 • Scatter plots depicting the distribution of modeled risk by annual-average $\text{PM}_{2.5}$
11 concentration (Figure C-29 and Figure C-31). While these scatter plots present similar
12 distributional information as the line graphs, the scatter plots allow for a more detailed
13 consideration of the nature of the risk distribution in relation to ambient $\text{PM}_{2.5}$ levels. In
14 these figures, each grid cell is shown as a dot, with the frequency of dots shown on a
15 color scale from cool (green – lower frequency) to hot (red – higher frequency) colors.⁴¹
16 Consequently, it is possible to consider whether, for example, a shift in risk involves a
17 change in the magnitude of risk across higher-risk cells, or in a change in the density of
18 lower risk cells.

19
20 Key observations resulting from review of these graphics are presented below the figures.
21

⁴¹ For adjusted air quality, a small amount of risk is estimated at concentrations higher than the level of the annual standard (e.g., some risk is estimated at an average concentration of 13 $\mu\text{g}/\text{m}^3$ when air quality is adjusted to just meet the current standard). This can result because risk estimates are for a single year (i.e., 2015) within the 3-year design value period (i.e., 2014 to 2016). While the three-year average design value is 12.0 $\mu\text{g}/\text{m}^3$, a single year can have grid cells with annual average concentrations above or below 12.0 $\mu\text{g}/\text{m}^3$.

1 **C.2.1 Results from Full Set of 47 Study Areas**



2
3
4
5
6
7

Figure C-25. Distribution of estimated PM_{2.5}-associated mortality for recent conditions (2015), current standards (12/35 µg/m³), and alternative standards (10/30 µg/m³) simulated for all 47 urban study areas.⁴²

⁴² Risk is rounded toward zero into whole PM_{2.5} concentration values (e.g., risk estimate at 10 µg/m³ includes risk occurring at 10.0-10.9 µg/m³). Blue lines represent the Pri-PM risk estimates, green lines represent the Sec-PM risk estimates, and black lines represent the 2015 recent conditions risk estimates.

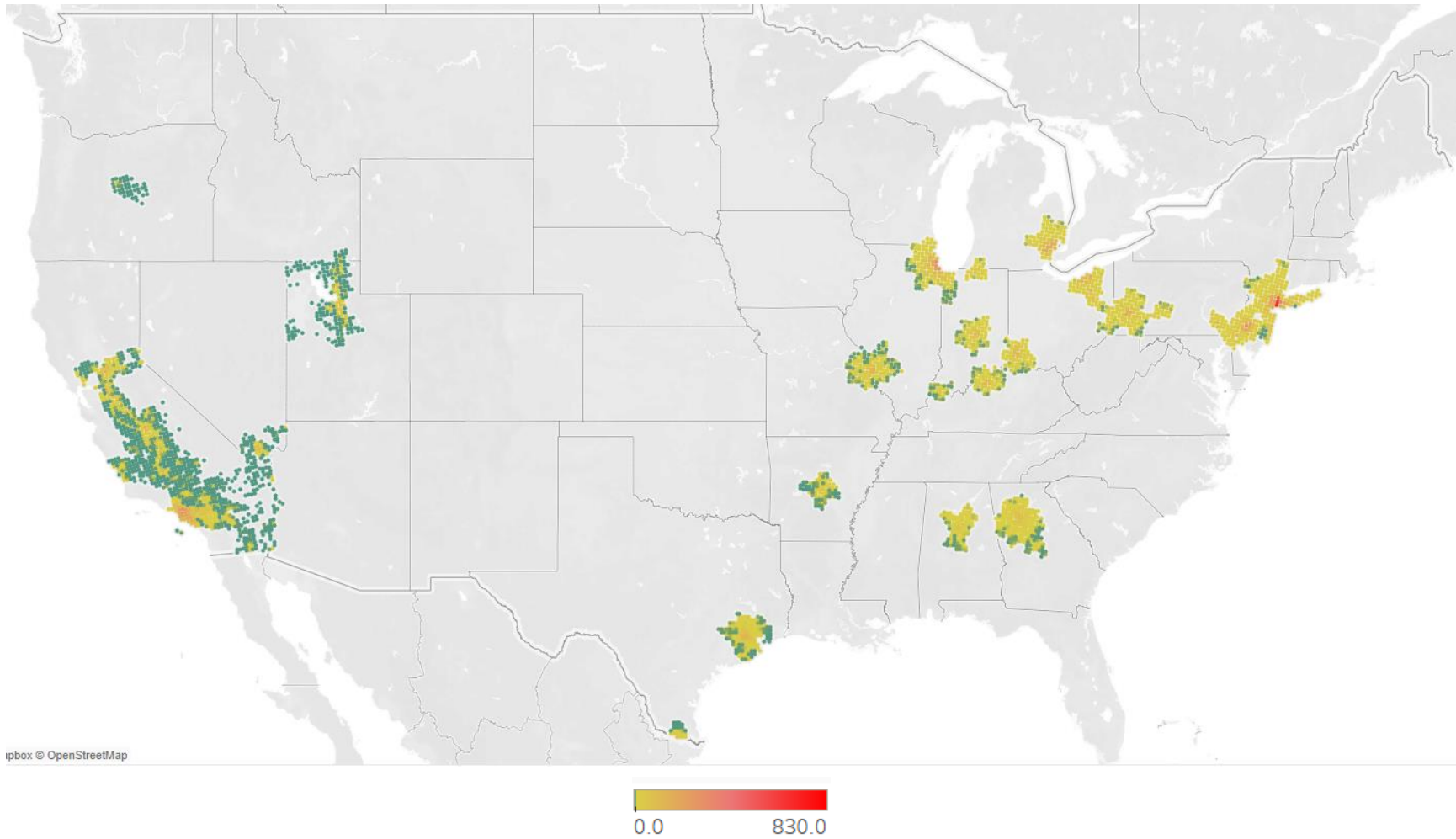


Figure C-26. Estimated number of premature deaths (by 12 km grid cell) under 2015 recent conditions in all 47 study areas.

1

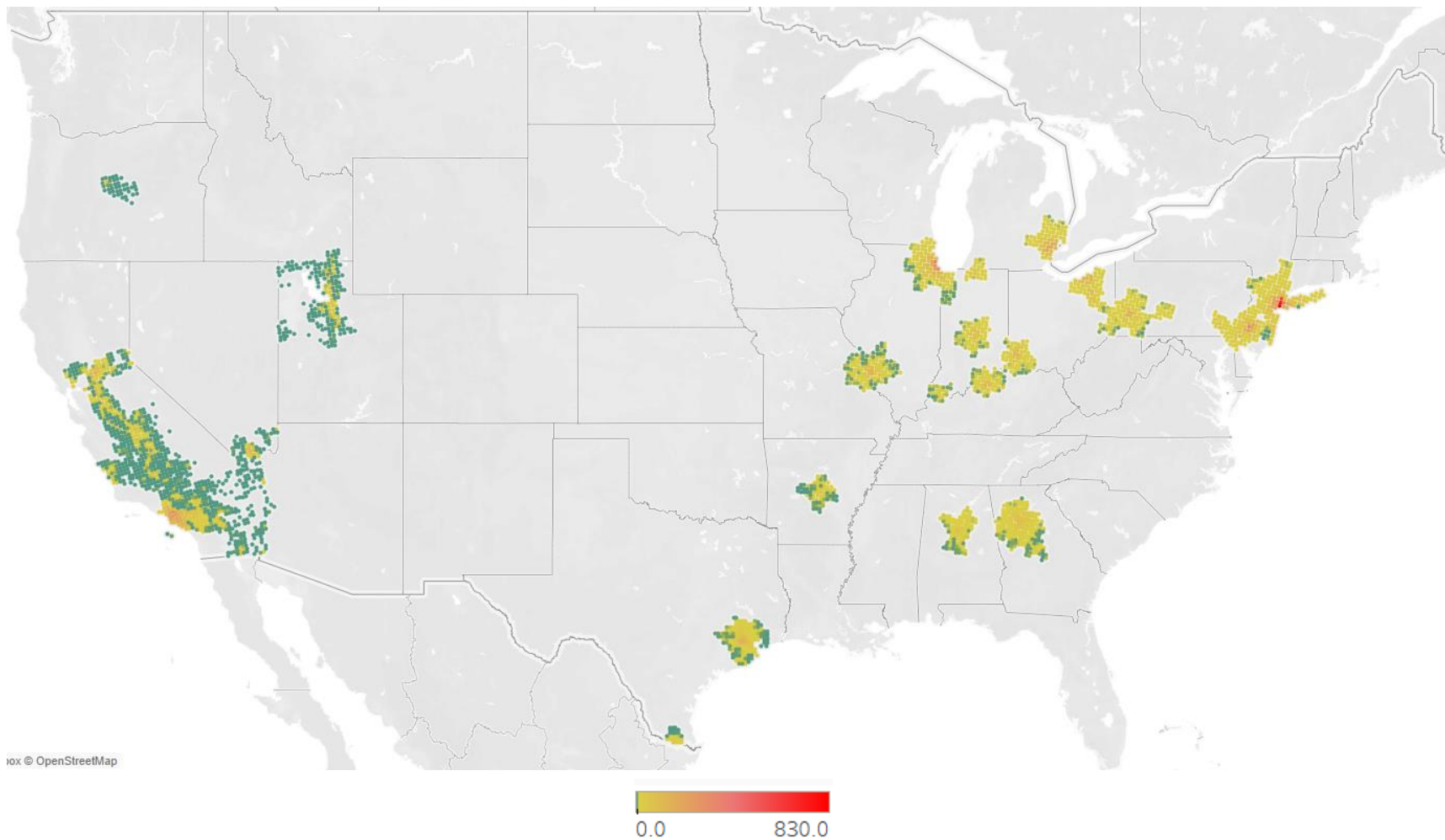


Figure C-27. Estimated number of premature deaths (by 12 km grid cell) when just meeting the current PM standards (12/35) in all 47 study areas (Pri-PM simulation).

1

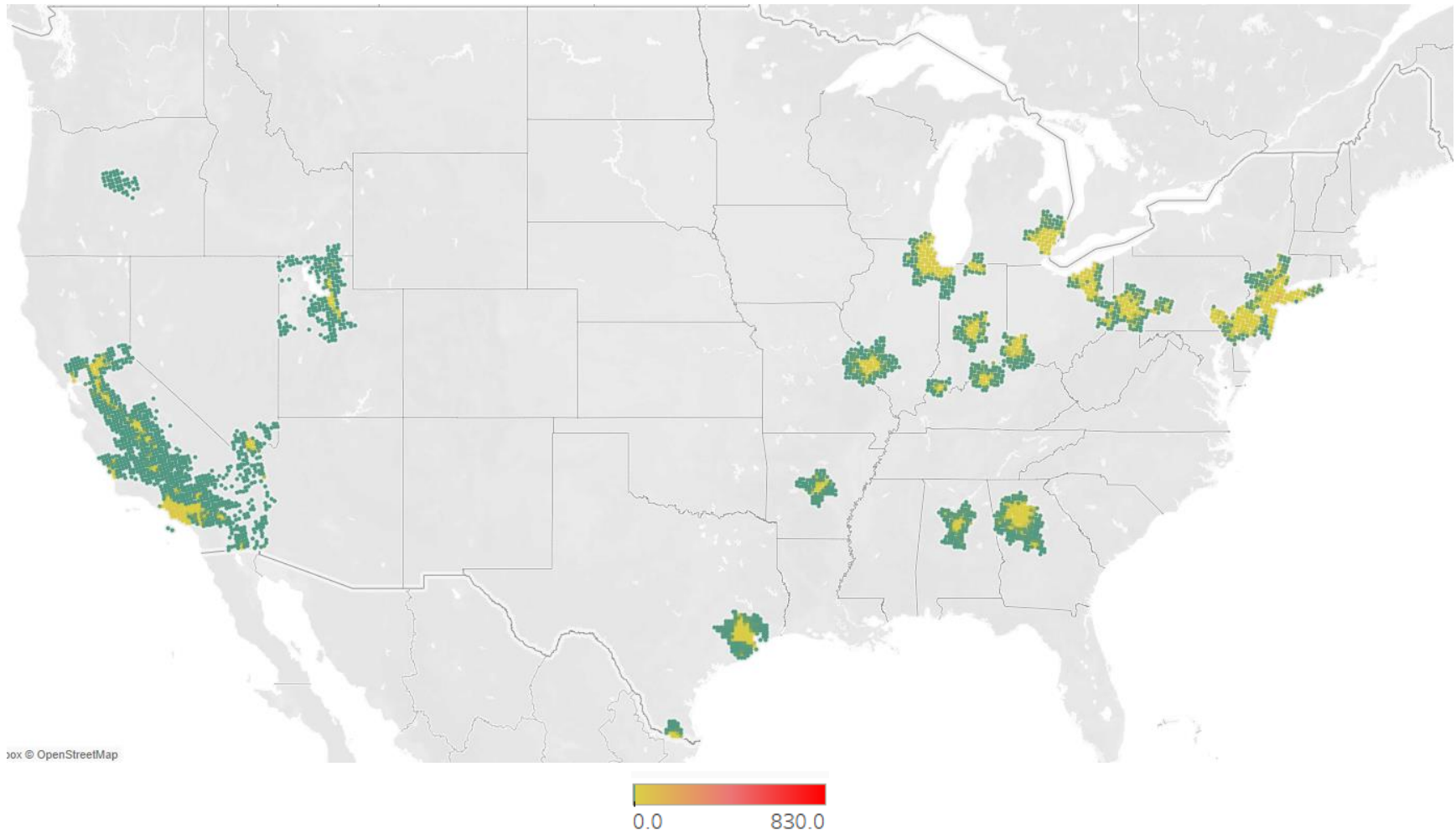
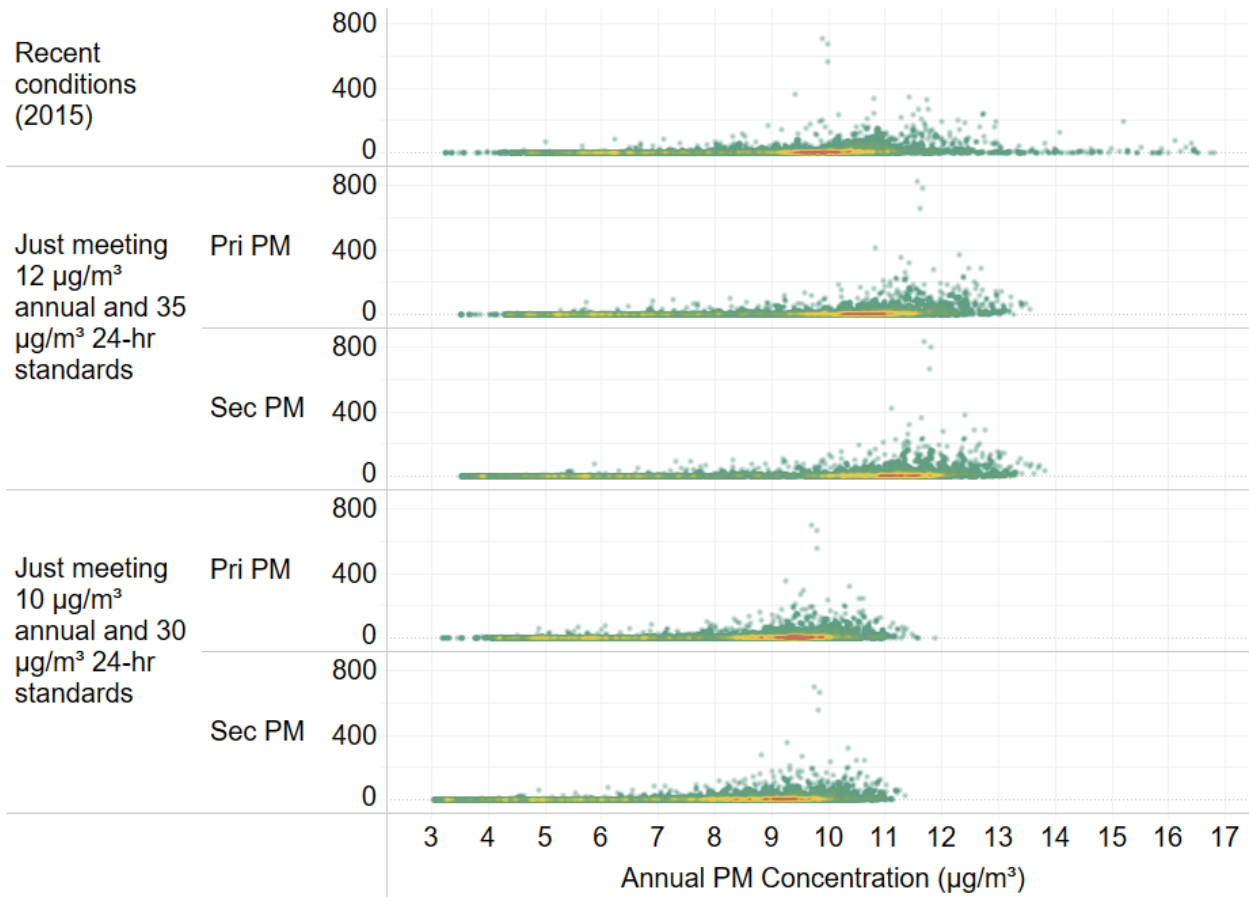


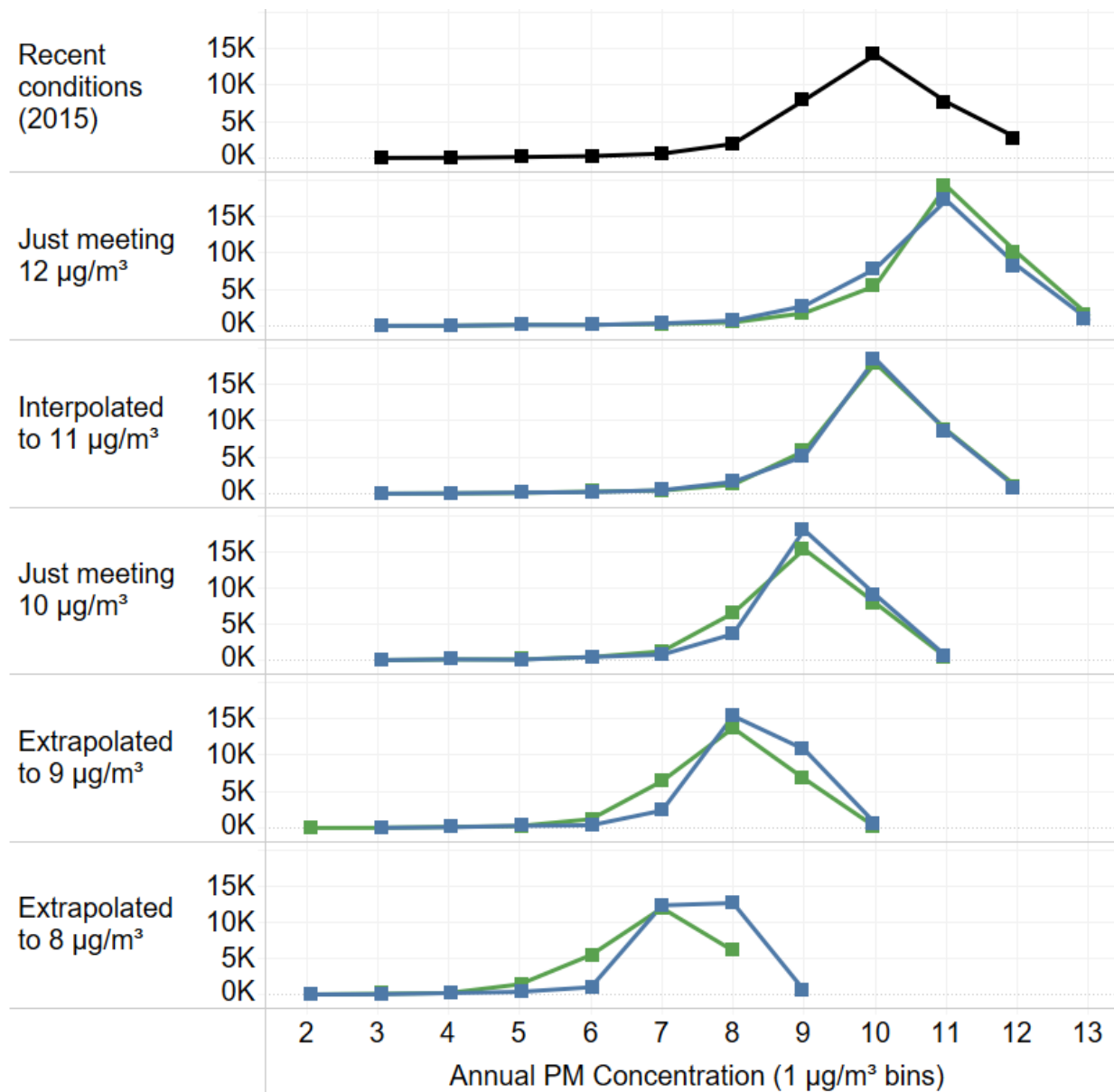
Figure C-28. Estimated reduction in the number of premature deaths (by 12 km grid cell) when going from just meeting the current standards (12/35) to just meeting the alternative standards (10/30) in all 47 study areas (Pri-PM simulation).



1
2
3
4
5

Figure C-29. Distribution of estimated premature death (by 12 km grid cell) for the current standards (12/35 µg/m³), alternative standards (10/30 µg/m³), and recent conditions (2015) for all 47 urban study areas (Pri-PM simulation).

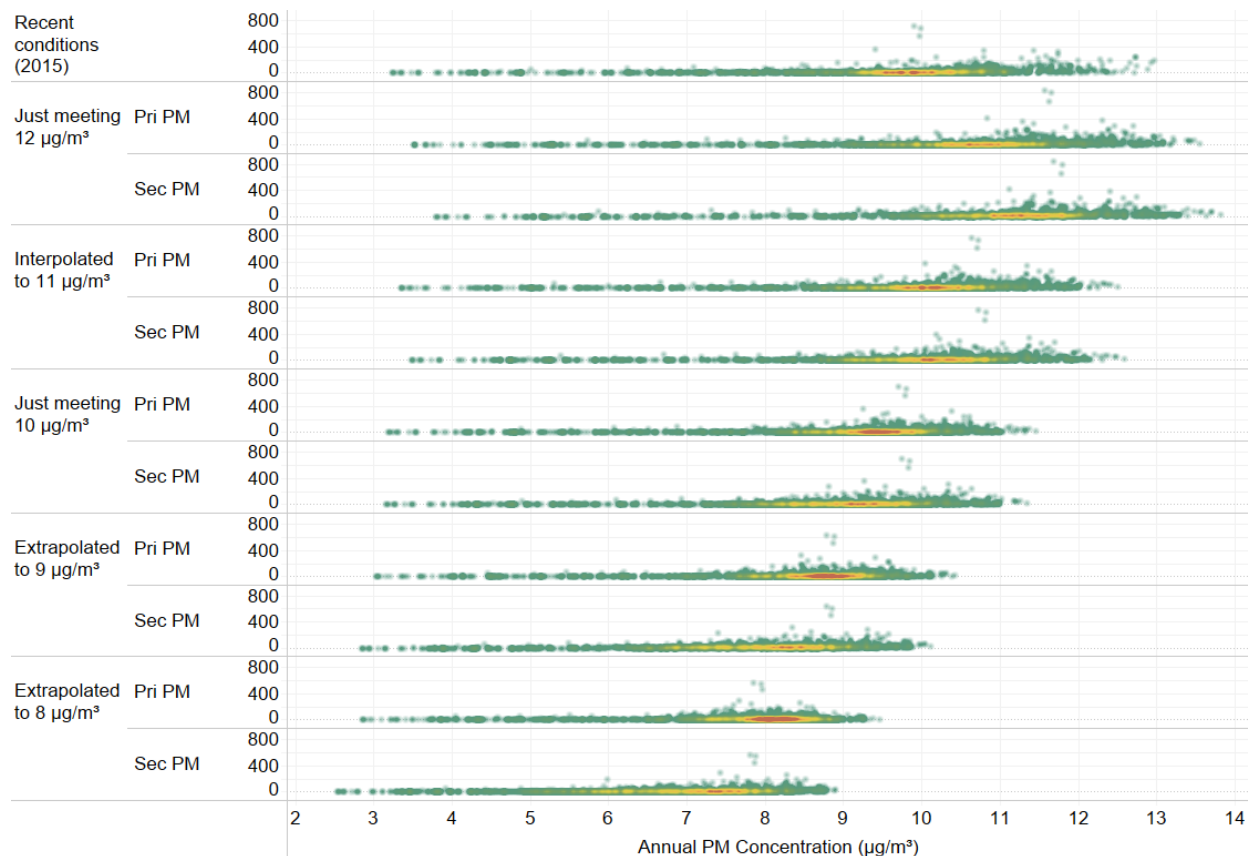
1 **C.2.2 Results from Set of 30 Study Areas controlled by the Annual Standard**



2
3 **Figure C-30. Distribution of estimated PM_{2.5}-associated mortality for recent conditions**
4 **(2015), the current annual standard (12/35 µg/m³), and alternative standards (8.0, 9.0,**
5 **10.0, and 11.0 µg/m³) simulated for the 30 annual-controlled urban study areas (blue**
6 **and green bars represent the Pri-PM_{2.5} and Sec-PM_{2.5} estimates, respectively).⁴³**

7
8

⁴³ Risk is rounded toward zero into whole PM_{2.5} concentration values (e.g., risk estimate at 10 µg/m³ includes risk occurring at 10.0-10.9 µg/m³). Blue lines represent the Pri-PM risk estimates, green lines represent the Sec-PM risk estimates, and black lines represent the 2015 recent conditions risk estimates.



1

2 **Figure C-31. Distribution of estimated premature death (by 12 km grid cell) 47 urban**
 3 **study areas (Pri-PM simulation) for recent conditions (2015), the current annual**
 4 **standard (12.0 µg/m³), alternative annual standards (8.0, 9.0, 10.0, 11.0 µg/m³).**

5

6 **C.2.3 Key Observations from the Supplemental Risk Results**

7 Review of the distributional risk estimates presented in section C.2 further support the
 8 key observations presented in draft PA section 3.4.2. Briefly, these observations include:

- 9
- 10 • Across the full set of alternative annual standards modeled including 11.0, 10.0, 9.0, and
 11 8.0 µg/m³ (each evaluated for the 30 annually-controlled study areas), we see a consistent
 12 reduction in mortality (Figure C-30 and Figure C-31). In addition, we note that these risk
 13 reductions are associated with iteratively lower ambient PM_{2.5} concentrations, such that
 14 with the lowest annual standard considered (8.0 µg/m³) the majority of remaining risk
 15 occurs in grid cells with ambient PM_{2.5} concentrations between 6 and 9 µg/m³. In
 16 contrast, most of the risk occurring under the current standard occurs in grid cells with
 17 ambient concentrations in the range of 10-12 µg/m³ (Figure C-29).
 - 18 • Patterns of risk reduction seen in the summary (aggregated) risk results tables presented
 both in draft PA section 3.4 and this appendix are driven by considerable underlying

1 variability across both CBSAs and across the 12km grid-level risk estimates. Specifically,
2 if we consider the maps and scatter plots presented in section C.2, we see considerable
3 spread (i.e., variability) in the grid-level risk estimates. We note that this underlying
4 variability in risk reflects local patterns of population density, baseline incidence, and
5 modeled ambient PM_{2.5} levels. However, it is important to also note that the underlying
6 variability does not result from differences in CR functions, since for all mortality
7 endpoints modeled in this analysis, national-level effect estimates were utilized.

- 8 • When considering the shift in the distribution of risks for the alternative standards (Figure
9 C-29 and Figure C-31), we note that risk reductions are estimated in grid cells
10 encompassing a wide range of PM_{2.5} concentrations. This includes grid cells with typical
11 (i.e., frequently occurring) concentrations (orange and red dots) as well as cells with
12 concentrations that occur relatively infrequently (green dots). Furthermore, these shifts
13 reflect reductions both in areas with relatively few estimated premature deaths (as
14 represented by points near the bottom of each of the scatter plots) and in areas with much
15 larger numbers of estimated deaths (points higher on the y-axis in these scatter plots).

17 **C.3 ADDITIONAL TECHNICAL DETAIL ON THE AT-RISK ANALYSIS**

18 Our consideration of estimated risks among potentially at-risk populations in the draft PA
19 focuses on addressing the following policy-relevant questions:

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

24
25 Estimating PM_{2.5} exposure and risk within various demographic populations when just
26 meeting the current or alternative annual standard or moving from the current annual standard to
27 an alternative annual standard requires multiple input parameters and several simplifying
28 assumptions. An overall summary of the analytical components is provided in Table C-11 and
29 below we discuss in detail the various data inputs and assumptions associated with the at-risk
30 analysis presented in the draft PA.

1 **Table C-11. Summary of At-Risk Analysis Variables^a**

Race/Ethnicity	Concentration-Response Function	Baseline Incidence Rate
1. White 2. Black 3. Asian 4. Hispanic 5. (Non-Hispanic) 6. (All)	1. (Overall function) 2. Race/Ethnicity-stratified functions	1. (Overall baseline incidence rate) 2. Race/ethnicity-stratified baseline incidence rates

2 ^a Parentheses indicate the variable was used in sensitivity analyses only.

3 **C.3.1 Race/Ethnicity**

4 As the 2019 ISA and the draft ISA Supplement noted strong support for non-White
 5 populations, and particularly Black/African American populations, being at increased risk from
 6 PM_{2.5}-related health effects, in part due to disparities in exposure, we focused on comparing
 7 exposure and risk in Black and White populations. We also included exposure and risk
 8 information from Asians, Native Americans, and Hispanics, although there is less evidence in the
 9 PM ISAs that those demographic groups are at increased risk of PM_{2.5} -related health effects or
 10 experience disparities in PM_{2.5} exposure (U.S. EPA, 2019, U.S. EPA, 2021a).

11 Population information for each demographic group from both the at-risk assessment
 12 population and the original cohort population can be found in Table C-12. In general, the
 13 proportions of White, Black, and Native American people in the Di et al., 2017 study were
 14 comparable to the proportions in the 47 urban study areas, though a slightly higher proportion of
 15 the population in the 47 areas was White. In contrast, the Asian and Hispanic subpopulations
 16 represented a smaller proportion of the Di et al., 2017 cohort than the respective population
 17 proportions in the 47 areas. Importantly, the 0.3% of Native Americans assessed by Di et al.,
 18 2017 equates to approximately 180,000 individuals, which is nearly a third of the ACS cohort
 19 (Turner et al., 2016).

1 **Table C-12. Demographic populations aged 65 and over residing in the full set of 47 study**
 2 **areas, the subset of 30 study areas controlled by the annual standard, and the**
 3 **original cohort.**

Ethnicity & Race	Population in 47 Areas	Percent of Population in 47 Areas	Population in 30 Areas	Percent of Population in 30 Areas	Percent of Population in Di et al., 2017 cohort
White	10,560,891	80.0	8,756,815	78.6	85.4
Black	1,655,695	12.6	1,551,743	13.9	8.7
Asian	927,966	7.0	801,487	7.2	1.8
Native American	51,263	0.4	36,477	0.3	0.3
Non-Hispanic	11,647,164	88.3	9,897,164	88.8	-
Hispanic	1,548,639	11.7	1,249,353	11.2	1.9

4

5 **C.3.2 Concentration-Response Functions**

6 The following eight epidemiologic long-term exposure studies of PM_{2.5} exposure and all-
 7 cause/nonaccidental/total mortality in nonwhite populations were identified in the 2019 ISA and
 8 draft ISA Supplement, met the minimum criteria discussed in the *Estimating PM_{2.5} and Ozone-*
 9 *Attributable Health Benefits TSD* (U.S. EPA, 2019, U.S. EPA, 2021a, U.S. EPA, 2021b), and
 10 were considered for inclusion in the at-risk assessment: Awad et al., 2019, Di et al., 2017,
 11 Kioumourtzoglou et al., 2016, Parker et al., 2018, Lipfert and Wyzga, 2020, Son et al., 2020,
 12 Wang et al., 2017, and Wang et al., 2020. Summary information regarding these eight studies is
 13 available in Table C-13. Consistent with the main risk assessment, we focused on long-term
 14 exposure studies so as to not double-count effects of short-term exposures. No mortality studies
 15 for the at-risk group of children met the initial screening criteria.

1 **Table C-13. Summary information for available epidemiology studies of nonwhite populations considered for the at-risk**
 2 **assessment.**

Study	Cohort	Study Location	Health Outcome	Study Size	Health Years	Air Quality Years	Ages	Exposure Method
Awad et al., 2019	Medicare enrollees	National US	All-cause mortality	12,095,504 movers	2000-2012	2000-2012	>64	Hybrid
Di et al., 2017	Medicare enrollees	National US	All-cause mortality	60,925,443 persons; 22,567,924 deaths	2000-2012	2000-2012	>64	Hybrid or Monitor
Kioumourtzoglou et al., 2016	Medicare enrollees	National US (207 US cities)	All-cause mortality	35,295,005 subjects; 11,411,282 deaths	2000-2010	2000-2010	>64	Monitor
Lipfert and Wyzga, 2020	Veterans	31 VA clinics across 27 states	Mortality risk	Approximately 700,000 males	1976-2001	1999-2001	Average age at entry approximately 52	Hybrid or Monitor
Parker et al., 2018	NHIS	National US	All-cause mortality	657,238 adults	1997-2009	2004	>24	Hybrid
Son et al., 2020	North Carolina residents	North Carolina	Total mortality	775,338 cases (i.e., total deaths) with 3,410,015 control days	2002-2013	2002-2013	All	Hybrid or Monitor
Wang et al., 2017	Medicare enrollees	7 U.S. southeast states: AL, FL, GA, MS, NC, SC, TN	All-cause mortality	13.1 million Medicare beneficiaries; 4.7 million deaths	2000-2013	2000-2013	>64	Hybrid
Wang et al., 2020	Medicare enrollees	National US	Non-accidental mortality	52,954,845 Medicare beneficiaries; 15,324,059 deaths	2000-2008	2000-2008	>64	Hybrid

3

We evaluated the available studies and concentration-response functions to determine if sufficient information exists for use in a quantitative analysis and to determine which study or studies best characterizes at-risk populations across the U.S. Of the available studies from the 2019 ISA, Di et al., 2017 was the largest nationwide study, covered one of the most recent and longest time spans, used a sophisticated exposure estimation technique, and provided sufficient information to apply risk models quantifying increased risks to the following demographic groups: White, Black, Asian, Native American, and Hispanic (Table C-14). Although effect estimates from Di et al., 2017 were derived from a cohort aged 65 and older and the study did not provide a non-Hispanic concentration-response function to directly compare to the Hispanic concentration-response function, it was identified as best characterizing populations potentially at increased risk of long-term PM_{2.5}-attributable all-cause mortality. Health impact functions, including beta parameters and standard errors (SE), were developed for each at-risk population demographic described by Di et al., 2017 and are available in Table C-14.

Table C-14. At-risk hazard ratios, beta coefficients, and standard errors from Di et al., 2017 used in this at-risk assessment.

Demographic Population	Risk of Death Associated with 10 µg/m ³ Increase in PM _{2.5}	Beta Coefficient (SE)
White	1.063 (1.060, 1.065)	0.0061 (0.0001)
All	1.073 (1.071, 1.075)	0.0070 (0.0001)
Hispanic	1.116 (1.100, 1.133)	0.0110 (0.0008)
Black	1.208 (1.199, 1.217)	0.0189 (0.0004)
Asian	1.096 (1.075, 1.117)	0.0092 (0.0010)
Native American	1.100 (1.060, 1.140)	0.0095 (0.0019)

C.3.3 Age

Concentration-response functions stratified by race and ethnicity from Di et al., 2017 were only available for ages 65-99. Therefore, this at-risk analysis only evaluated a single age range group of 65-99 years.

C.3.4 Baseline Incidence Rates

BenMAP-CE includes baseline incidence rates at the most geographically- and age-specific levels available for each health endpoint assessed. For many locations within the U.S., these data are resolved at the county- or state-level, providing a better characterization of the geographic distribution of mortality rates than the national-level rates. Race- and ethnicity-stratified baseline incidence rates from 2007-2016 Census data were recently improved for the all-cause mortality health endpoint, by adding the geographic level option of rural/urban state

1 between county-level and state-level (sections C.3.4.1 and C.3.4.2). Both overall and
2 race/ethnicity-stratified baseline rates are used in this at-risk analysis (section C.3.4.2).

3 **C.3.4.1 Race-Stratified Baseline Incidence Rates**

4 To estimate race-stratified and age-stratified incidence rates at the county level, we
5 downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC
6 WONDER mortality database.⁴⁴ Race-stratified incidence rates were calculated for the following
7 age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-54 years,
8 55-64 years, 65-74 years, 75-84 years, and 85+ years. To address the frequent county-level data
9 suppression for race-specific death counts, we stratified the county-level data into two broad race
10 categories, White and Non-White populations. In a later step, we stratified the non-White
11 incidence rates by race (Black, Asian, Native American) using the relative magnitudes of
12 incidence values by race at the regional level, described in more detail below.

13 We followed methods outlined in Section D.1.1 of the BenMAP User Manual with one
14 notable difference in methodology; we included an intermediate spatial scale between county and
15 state for imputation purposes.⁴⁵ We designated urban and rural counties within each state using
16 CDC WONDER and, where possible, imputed missing data using the state-urban and state-rural
17 classifications before relying on broader statewide data. We followed methods for dealing with
18 suppressed and unreliable data at each spatial scale as described in Section D.1.1.

19 A pooled non-White incidence rate masks important differences in mortality risks by
20 race. To estimate county-level mortality rates by individual race (Black, Asian, Native
21 American), we applied regional race-specific incidence relationships to the county-level pooled
22 non-White incidence rates. We calculated a weighted average of race-specific incidence rates
23 using regional incidence rates for each region/age/race group normalized to one reference
24 population (the Asian race group) and county population proportions based on race-specific
25 county populations from CDC WONDER where available. In cases of population suppression
26 across two or more races per county, we replaced all three race-specific population proportions
27 derived from CDC WONDER with population proportions derived from 2010 Census data in
28 BenMAP-CE (e.g., 50 percent Black, 30 percent Asian, 20 percent Native American).

29 **C.3.4.2 Ethnicity-Stratified Baseline Incidence Rates**

30 To estimate ethnicity-stratified and age-stratified incidence rates at the county level, we
31 downloaded all-cause and respiratory mortality data from 2007 to 2016 from the CDC

⁴⁴ <https://wonder.cdc.gov/>

⁴⁵ https://www.epa.gov/sites/default/files/2015-04/documents/benmap-ce_user_manual_march_2015.pdf

1 WONDER mortality database.⁴⁶ Ethnicity-stratified incidence rates were calculated for the
2 following age groups: < 1 year, 1-4 years, 5-14 years, 15-24 years, 25-34 years, 35-44 years, 45-
3 54 years, 55-64 years, 65-74 years, 75-84 years, and 85+ years. We stratified county-level data
4 by Hispanic origin (Hispanic and non-Hispanic). We followed the methods outlined in Section
5 D.1.1 to deal with suppressed and unreliable data. We also included an intermediate spatial scale
6 between county and state designating urban and rural counties for imputation purposes,
7 described in detail in Section D.1.3 of the BenMAP User Manual.⁴⁷

8 **C.3.5 Selection of Air Quality Simulation Approach**

9 Concentration fields associated with just meeting the current and alternative standards in
10 the 47 urban study areas were based on adjusting 2015 modeled concentrations using CMAQ
11 sensitivity modeling with emission reductions applied throughout the modeling domain. This
12 approach was applied to develop realistic concentration fields that correspond to just meeting
13 standards in the 47 areas. Two distinctly different emission cases were used (Pri-PM and Sec-
14 PM) to examine the sensitivity of results to the air quality adjustment approach. For
15 characterizing risk in at-risk populations, we used air quality fields from the Pri-PM adjustment
16 case alone. In the Pri-PM case, the air quality adjustments for a given area are largely associated
17 with emission reductions within that area due to the local nature of air quality impacts from
18 primary PM sources. For the Sec-PM case, the air quality adjustments may be strongly
19 associated with sources located outside of the area. Since the at-risk calculations are performed
20 for population groups within the 47 urban study areas alone, the Pri-PM adjustment case (in
21 which air quality adjustments are primarily associated with emission sources within the 47 areas)
22 is most appropriate for the at-risk analysis.

23 **C.4 SUPPLEMENTAL AT-RISK RESULTS**

24 Absolute numbers of all-cause premature mortality cases within each racial and ethnic
25 population demographic are available in Table C-15 for total attributable burden under either the
26 current or alternative standards and Table C-16 for the change in risk estimates when moving
27 from the current to a potential alternative annual standard for both the full set of 47 urban study
28 areas and the subset of 30 annually-controlled areas.

29

⁴⁶ <https://wonder.cdc.gov/>

⁴⁷ https://www.epa.gov/sites/default/files/2015-04/documents/benmap-ce_user_manual_march_2015.pdf

1 **Table C-15. Estimates of total PM_{2.5}-associated mortality by demographic population for**
 2 **air quality adjusted to just meet the current or alternative standards.**

Study Areas	Modeling Scenario	Ethnicity & Race				
		White	Black	Hispanic	Asian	Native American
47 areas	Just meeting 12/35 µg/m ³	29,400 (28,200 to 30,400)	13,600 (13,100 to 14,100)	4,850 (4,220 to 5,460)	1,930 (1,530 to 2,300)	125 (77.9 to 169)
	Just meeting 10/30 µg/m ³	25,200 (24,300 to 26,200)	11,700 (11,300 to 12,100)	4,160 (3,610 to 4,680)	1,650 (1,310 to 1,970)	108 (66.9 to 146)
30 areas	Just meeting 12/35 µg/m ³	24,900 (23,900 to 25,800)	12,800 (12,400 to 13,300)	3,970 (3,450 to 4,460)	1,640 (1,300 to 1,960)	87.9 (54.6 to 119)
	Interpolated to 11 µg/m ³	23,100 (22,200 to 24,000)	11,900 (11,500 to 12,400)	3,680 (3,200 to 4,140)	1,520 (1,210 to 1,820)	81.5 (50.6 to 110)
	Just meeting 10/30 µg/m ³	21,300 (20,500 to 22,100)	11,000 (10,600 to 11,400)	3,380 (2,940 to 3,810)	1,400 (1,110 to 1,670)	75.1 (46.5 to 102)
	Extrapolated to 9 µg/m ³	19,600 (18,800 to 20,300)	10,100 (9,740 to 10,500)	3,090 (2,680 to 3,480)	1,280 (1,010 to 1,530)	68.6 (42.4 to 93.0)
	Extrapolated to 8 µg/m ³	17,800 (17,100 to 18,400)	9,180 (8,840 to 9,510)	2,790 (2,420 to 3,140)	1,150 (913 to 1,380)	62.0 (38.3 to 84.3)

3

4 **Table C-16. Change in PM_{2.5}-associated mortality by demographic population for air**
 5 **quality adjusted to just meet the current or alternative standards.**

Study Areas	Modeling Scenario	Ethnicity & Race				
		White	Black	Hispanic	Asian	Native American
47 areas	12/35-10/30 µg/m ³	4,380 (4,200 to 4,540)	2,280 (2,190 to 2,370)	771 (665 to 872)	302 (238 to 364)	18.9 (11.6 to 26.0)
30 areas	12/35-11 (interpolated) µg/m ³	1,890 (1,810 to 1,960)	1,090 (1,050 to 1,130)	327 (282 to 371)	133 (104 to 160)	7.04 (4.29 to 9.68)
	12/35-10/30 µg/m ³	3,760 (3,610 to 3,900)	2,170 (2,080 to 2,250)	652 (563 to 737)	264 (208 to 319)	14.0 (8.57 to 19.3)
	12/35-9 (extrapolated) µg/m ³	5,630 (5,410 to 5,840)	3,220 (3,100 to 3,340)	973 (840 to 1,100)	395 (311 to 476)	21.0 (12.8 to 28.7)
	12/35-8 (extrapolated) µg/m ³	7,490 (7,190 to 7,770)	4,260 (4,090 to 4,420)	1,290 (1,120 to 1,460)	525 (414 to 631)	27.8 (17.0 to 38.1)

6

7

8

9

10

11

12

13

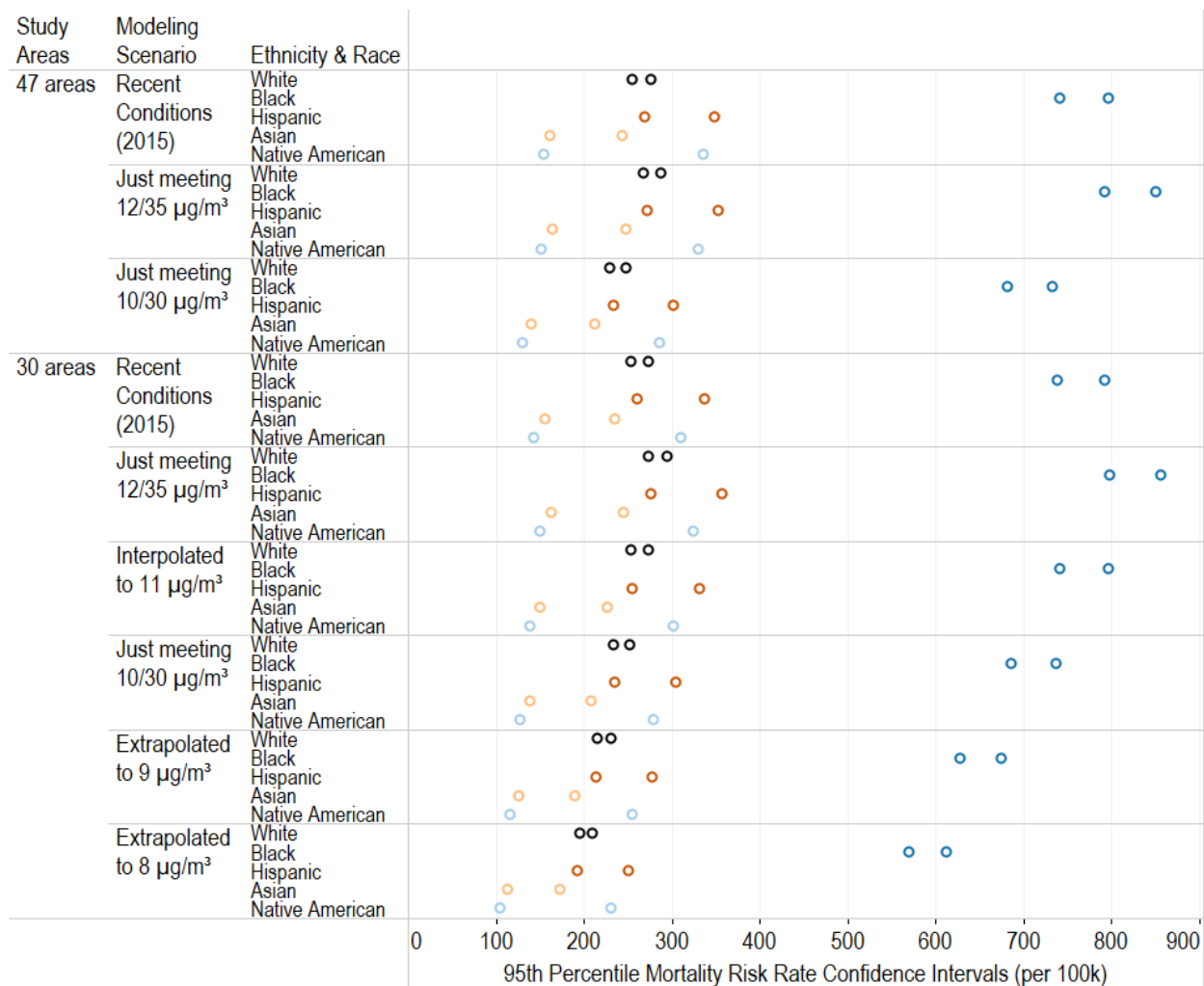
14

15

16

17

For visual purposes only the central risk estimates are included in the at-risk results presented in chapter 3 of the draft PA (section 3.4.2), but an example of the 95th percentile confidence interval (CI) risk estimate spans resulting from the epidemiologic concentration-response functions are provided in Figure C-32. The lower open circle represents the 2.5th percentile and the higher open circle represents the 97.5th percentile CI for each population demographic. CIs are derived from the concentration-response relationships presented in Di et al., 2017 (Figure C-13). While the Hispanic and Native American risk rate CIs often overlap, the Black risk rate estimates are consistently higher than the White risk rates, and the Asian risk rates are consistently lower than the White risk rates (Figure C-32).

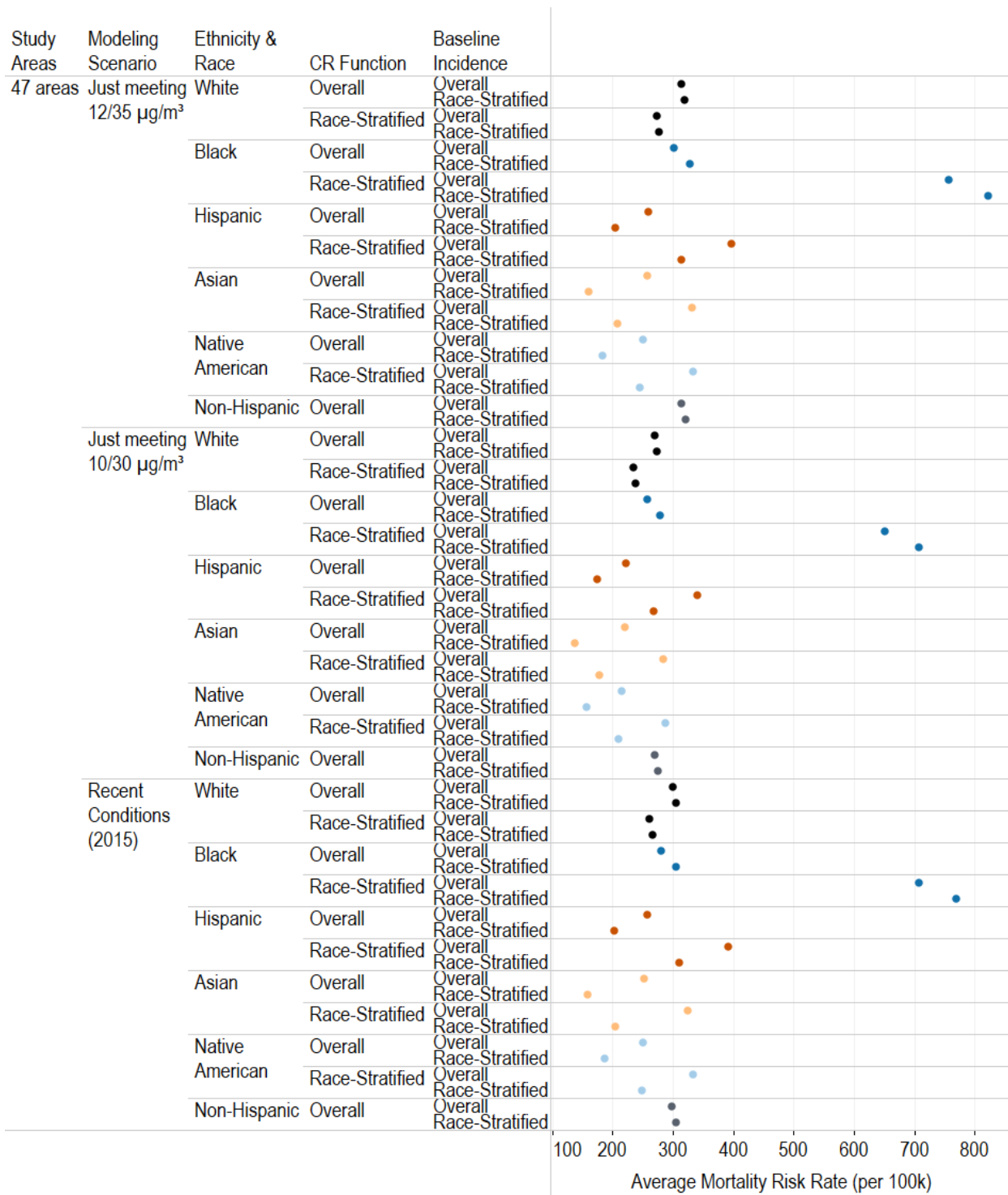


1
2 **Figure C-32. Race- and ethnicity-stratified 95th percentile (2.5th percentile to 97.5th**
3 **percentile) confidence interval risk estimates for recent conditions (2015), the current**
4 **standard, and potential alternative standard air quality surfaces.**

5
6 As the risk rate calculation integrates both population-specific baseline incidence rates
7 and concentration-response relationships with exposure information, we wanted to separate the
8 impacts of each data input. To distinguish the impacts of race-stratified concentration-response
9 functions from baseline incidence rates on the results, we provide the average PM_{2.5}-attributable
10 risk by demographic population in the full set of 47 urban study areas for the current standards,
11 potential alternative standards, and recent condition (2015) air quality surfaces within each
12 demographic group. Figure C-33 and Figure C-34 provide this information when just meeting
13 current and alternative standards or shifting between the current and potential alternative annual
14 standards, respectively.

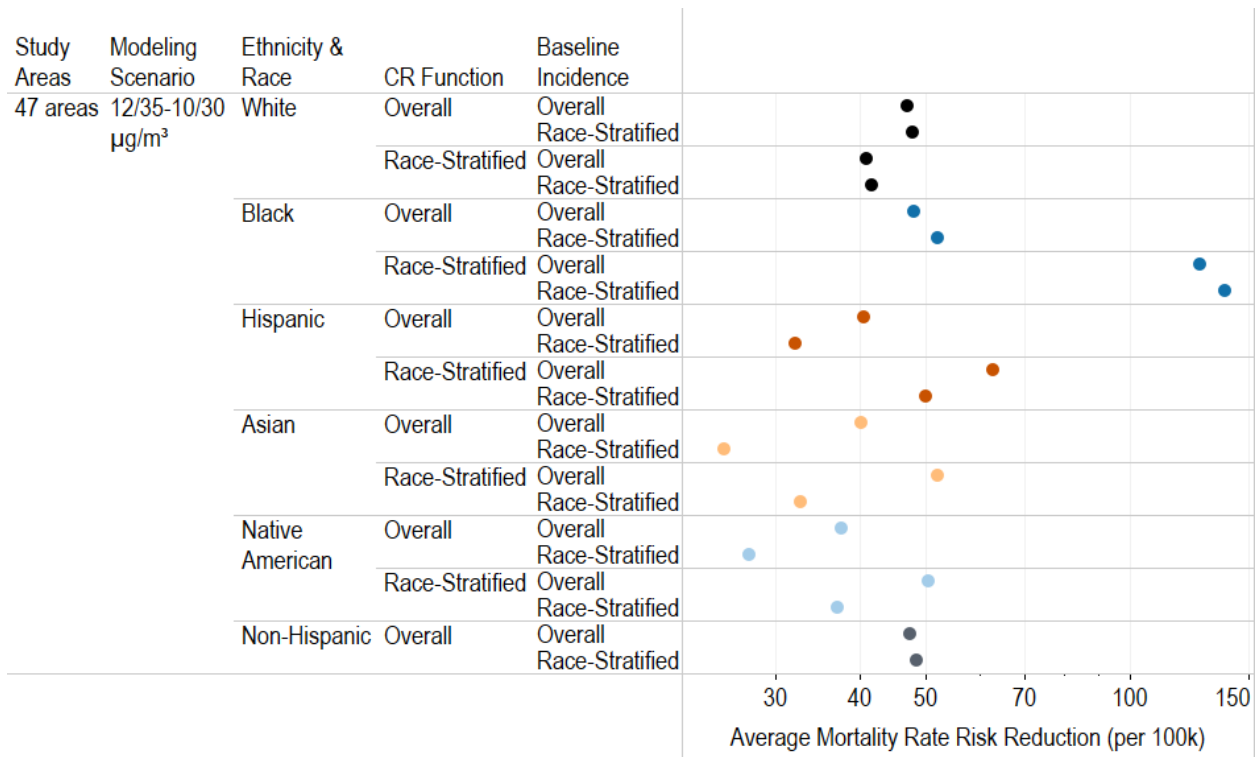
1 Generally, race-stratified concentration-response functions increased the population-
2 normalized risk estimated in nonwhite populations, with the greatest magnitude increase
3 occurring in Black populations, followed by Hispanic populations, and decreased risk estimated
4 in White populations. Di et al., 2017 did not provide a concentration-response function for the
5 non-Hispanic population, so only the overall concentration-response function was applied to
6 non-Hispanics in these supplemental analyses.

7 Many factors effect race/ethnicity-stratified baseline incidence rates, such as access to
8 medical care, socioeconomic status, and underlying health issues. As such, race/ethnicity-
9 stratified baseline incidence rates impacted by each race and ethnicity differently. Race/ethnicity-
10 stratified baseline incidence rates increased risk estimates substantially in Black populations and
11 slightly in White and non-Hispanic populations. In contrast, race/ethnicity-stratified baseline
12 incidence rates decreased risk rates estimated in Hispanic, Asian, and Native American
13 populations.



1
2
3
4
5
6

Figure C-33. Effect of race-stratified concentration-response (CR) functions and baseline incidence rates on the average PM_{2.5}-attributable risk by demographic population in the 47 study areas for the current standard, potential alternative standard, and recent conditions (2015) air quality surfaces within each demographic group.

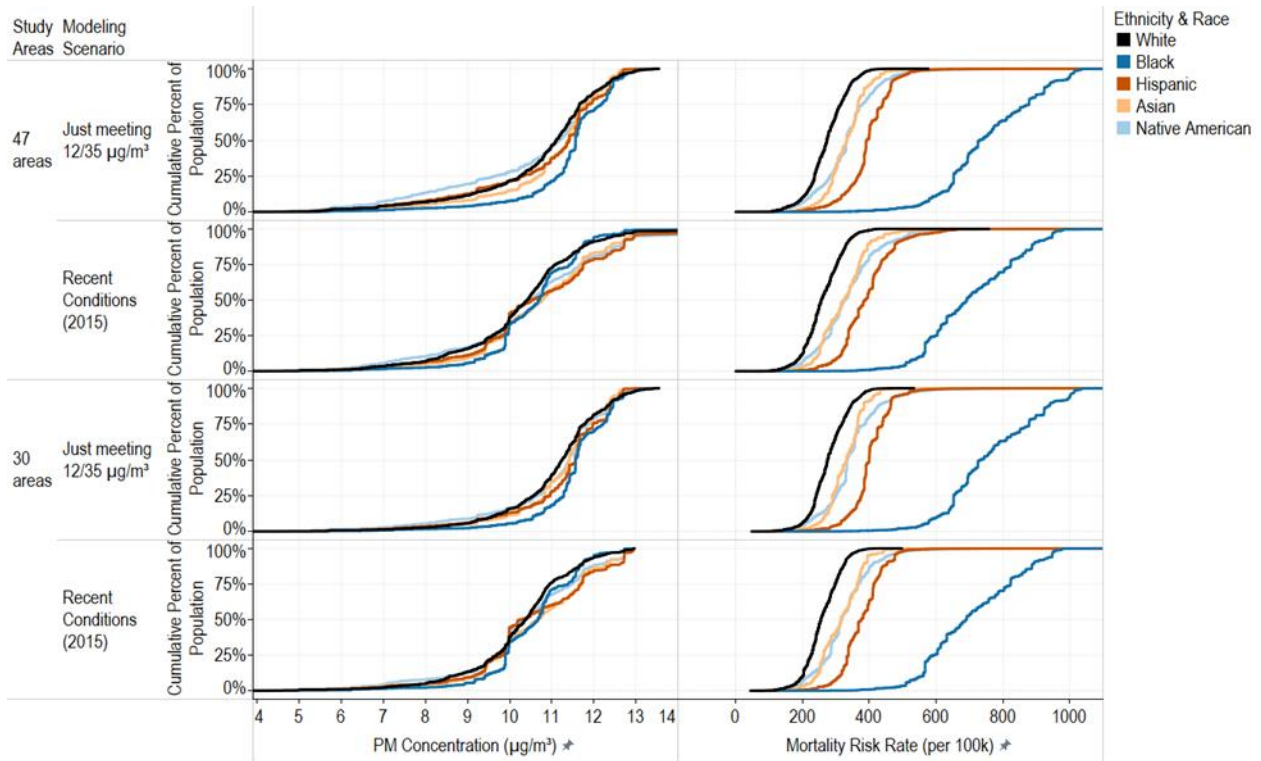


1

2 **Figure C-34. Effect of race-stratified CR functions and baseline incidence rates on the**
 3 **average PM_{2.5}-attributable risk reductions by demographic population in the 47 study**
 4 **areas when shifting from the current to the potential alternative standards within each**
 5 **demographic group.**

6

7 As the annual design values for many study areas required rolling up to just meet the
 8 current standard (section C.1.4.6), for informational purposes we provide cumulative distribution
 9 plots of PM_{2.5} exposure and PM_{2.5}-attributable mortality risk per 100,000 people by demographic
 10 group for the recent condition year 2015, along with the plots for just meeting the current
 11 standards for direct comparison (Figure C-35). Several caveats should be noted when comparing
 12 the recent conditions air quality surface to those adjusted to just meet current or recent air quality
 13 conditions. Importantly, the at-risk analysis focuses on the Pri-PM adjustment approach (section
 14 C.3.4.2), in which emission increases in areas below the current standard occur predominately at
 15 and around the urban cores of the study areas. This could lead to a simulated increase of
 16 disproportionate PM_{2.5} exposures in demographic populations that frequently reside at and
 17 around the urban core. Conversely, disproportionate PM_{2.5} concentrations in demographic
 18 populations residing in areas above the current standards may be obscured when concentrations
 19 are adjusted downward to just meet the current standard.



1
 2 **Figure C-35. PM_{2.5} concentrations and PM_{2.5}-attributable risk by demographic population**
 3 **for recent air quality conditions (2015) and air quality simulated to just meet the**
 4 **current PM standards.**

5
 6 Another aspect of lowering the annual PM_{2.5} standard is the percent of overall risk
 7 attributable to PM_{2.5} exposure. Table C-17 shows that the percent of baseline risk is higher in
 8 racial/ethnic minority demographics in all scenarios analyzed. Additionally, some minority
 9 populations may experience a greater decrease in the percent of baseline PM_{2.5}-attributable risk.

1 **Table C-17. Percent of mortality baseline incidence attributable to PM_{2.5} under the current**
 2 **and potential alternative standards.**

Ethnicity & Race	% of Baseline PM _{2.5} -Attributable Risk Under the Current Standard (12/35)		% of Baseline PM _{2.5} -Attributable Risk Under an Alternative Standard (11)	% of Baseline PM _{2.5} -Attributable Risk Under an Alternative Standard (10/30)		% of Baseline PM _{2.5} -Attributable Risk Under an Alternative Standard (9)	% of Baseline PM _{2.5} -Attributable Risk Under an Alternative Standard (8)
	47 areas	30 areas	30 areas	47 areas	30 areas	30 areas	30 areas
White	6	7	6	5	6	5	5
Black	19	20	18	17	17	15	14
Hispanic	11	12	11	10	10	9	8
Asian	10	10	9	8	8	8	7
Native American	9	10	9	8	9	8	7

3

4 **C.5 CHARACTERIZING VARIABILITY AND UNCERTAINTY IN RISK**
 5 **ESTIMATES**

6 An important component of the risk assessment is the characterization of variability and
 7 uncertainty. Variability refers to the heterogeneity of a variable of interest within a population or
 8 across different populations. Variability is inherent and cannot be reduced through further
 9 research. Hence, the design of a population-level risk assessment is often focused on effectively
 10 characterizing variability in estimated risks across populations. Uncertainty refers to the lack of
 11 knowledge regarding the actual values of inputs to an analysis. In contrast to variability,
 12 uncertainty can be reduced through improved measurement of key variables and ongoing model
 13 refinement. This section discusses our approaches to addressing key sources of variability and
 14 uncertainty in the PM_{2.5} risk assessment.

15 Variability in the risk of PM_{2.5}-associated mortality could result from a number of factors.
 16 These can include variation in PM_{2.5} exposures within and across populations (e.g., due to
 17 differences in behavior patterns, building characteristics, air quality patterns etc.) and in the
 18 health responses to those exposures (e.g., because some groups are at increased risk of PM-
 19 related health effects). There is also variation over space and time in both PM_{2.5} itself (e.g.,
 20 concentrations, air quality patterns) and in the ambient pollutants that co-occur with PM_{2.5}. In the
 21 PM_{2.5} risk assessment discussed in this draft PA, we account for these and other sources of
 22 variability, in part, by estimating risks based on CR functions from a number of epidemiologic
 23 studies. These studies evaluate PM_{2.5} health effect associations for either annual or daily PM_{2.5}
 24 exposures across various time periods; in numerous geographic locations, encompassing much or
 25 all of the U.S.; in various populations, including some with the potential to be at higher risk than

1 the general population (e.g., older adults); and using a variety of methods to estimate PM_{2.5}
2 exposures (e.g., hybrid modeling approaches and monitors) and to control for potential
3 confounders. In selecting areas in which to estimate PM_{2.5}-associated risks, we include areas that
4 cover multiple regions of the U.S., with varying population demographics. Additionally, we use
5 two different strategies for adjusting PM_{2.5} air quality, reflecting the potential for changes in
6 ambient PM_{2.5} concentrations to be influenced by changes in primary PM_{2.5} emissions and by
7 changes in precursor emissions that contribute to secondary particle formation.

8 Beyond the reliance on information from multiple epidemiologic studies to account for
9 the variability in key risk assessment inputs, we use a combination of quantitative and qualitative
10 approaches to characterize the remaining risk estimates uncertainty more explicitly. The
11 characterization of uncertainty associated with risk assessments is often addressed in the
12 regulatory context using a tiered approach in which progressively more sophisticated methods
13 are used to evaluate and characterize sources of uncertainty depending on the overall complexity
14 of the risk assessment (WHO, 2008). Guidance documents developed by the EPA for assessing
15 air toxics-related risk and Superfund Site risks (U.S. EPA, 2004 U.S. EPA, 2001) as well as
16 recent guidance from the World Health Organization (WHO, 2008) specify multitiered
17 approaches for addressing uncertainty. The WHO guidance presents a four-tiered approach,
18 where the decision to proceed to the next tier is based on the outcome of the previous tier's
19 assessment. The four tiers described in the WHO guidance include:

- 20 • Tier 0 – recommended for routine screening assessments, uses default uncertainty factors
21 (rather than developing site-specific uncertainty characterizations);
- 22 • Tier 1 – the lowest level of site-specific uncertainty characterization, involves qualitative
23 characterization of sources of uncertainty (e.g., a qualitative assessment of the general
24 magnitude and direction of the effect on risk results);
- 25 • Tier 2 – site-specific deterministic quantitative analysis involving sensitivity analysis,
26 interval-based assessment, and possibly probability bound (high- and low-end)
27 assessment; and
- 28 • Tier 3 – uses probabilistic methods to characterize the effects on risk estimates of sources
29 of uncertainty, individually and combined.

30 With this four-tiered approach, the WHO framework provides a means for systematically
31 linking the characterization of uncertainty to the sophistication of the underlying risk assessment.
32 Ultimately, the decision as to which tier of uncertainty characterization to include in a risk
33 assessment will depend both on the overall sophistication of the risk assessment and the
34 availability of information for characterizing the various sources of uncertainty. EPA staff used
35 the WHO guidance as a framework for developing the approach used for characterizing
36 uncertainty in this risk assessment. The overall analysis in the PM NAAQS risk assessment is
37 relatively complex, thereby warranting consideration of a full probabilistic (WHO Tier 3)

1 uncertainty analysis. However, limitations in available information prevent this level of analysis
2 from being completed at this time. In particular, the incorporation of uncertainty related to key
3 elements of CR functions (e.g., alternative functional forms, etc.) into a full probabilistic WHO
4 Tier 3 analysis would require that probabilities be assigned to each competing specification of a
5 given model element (with each probability reflecting a subjective assessment of the probability
6 that the given specification is the “correct” description of reality). However, for many model
7 elements there is insufficient information on which to base these probabilities. One approach that
8 has been taken in such cases is expert elicitation; however, this approach is resource- and time-
9 intensive and consequently, it was not feasible to use this technique in the current PM NAAQS
10 reconsideration to support a WHO Tier 3 analysis.

11 For most elements of this risk assessment, rather than conducting a full probabilistic
12 uncertainty analysis, we have included qualitative discussions of the potential impact of
13 uncertainty on risk results (WHO Tier1) and/or completed sensitivity analyses assessing the
14 potential impact of sources of uncertainty on risk results. The remainder of this section is
15 organized as follows. Those sources of uncertainty addressed quantitatively in the risk assessment
16 are discussed in section C.5.1. Those sources of uncertainty addressed qualitatively in the risk
17 assessment are discussed in section C.5.2. Below we summarize key findings from both the
18 qualitative and quantitative assessments of variability and uncertainty in the context of assessing
19 overall confidence in the risk assessment and its estimates.

20 **C.5.1 Quantitative Assessment of Uncertainty**

21 The risk assessment includes three components which allow us to quantitatively evaluate
22 the impact of potentially important sources of uncertainty on the risk estimates generated. Each
23 of these is discussed below including conclusions drawn from each assessment regarding the
24 potential importance of each source of uncertainty:

- 25 • *95 percent CIs around point estimates of mortality risk:* Each of the point estimates
26 presented in the results section includes 95 percent CIs generated by BenMAP-CE,
27 reflecting the standard error associated with the underlying effect estimate (i.e., a
28 measure of the statistical precision of the effect estimate). There is variation in the range
29 of 95 percent CIs associated with the point estimates generated for this analysis, with
30 some CR functions displaying substantially greater variability than others (e.g., Ito et al.,
31 2013, tables in section 3.4.2 of the draft PA). There are a number of factors potentially
32 responsible for the varying degrees of statistical precision in effect estimates, including
33 sample size, exposure measurement error, degree of control for confounders/effect
34 modifiers, and variability in PM_{2.5} concentrations.
- 35 • *Inclusion of multiple mortality estimates reflecting variation in CR functions across*
36 *studies:* For mortality endpoints, we include risk estimates reflecting multiple
37 epidemiology studies and associated study designs (e.g., age ranges, methods for
38 controlling potential confounders). In some instances, we find that the CR function used

1 has only a small impact on risk estimates(e.g., Turner et al., 2016 and Di et al., 2017).
2 The degree to which different CR functions result in different risk estimates could reflect
3 differences in study design and/or study populations evaluated, as well as other factors. In
4 most instances in this risk assessment, the CR function used has only a small impact on
5 risk estimates (e.g., Di et al., 2017). Details regarding the design of epidemiology studies
6 providing effect estimates for this risk assessment are presented in Table C-1.

- 7 • *Evaluation of two different strategies for simulating air quality scenarios:* Two methods
8 are employed to adjust air quality in order to simulate just meeting the current and
9 alternative standards, which could represent potential bounding scenarios of PM_{2.5}
10 concentrations changes across the study area (i.e., the Pri-PM-based method and the Sec-
11 PM based method). Our evaluation of these methods reflects the fact that there is both
12 variability and uncertainty in how emissions in a particular area could change such that
13 the area “just meets” either the current or alternative standards. By modeling risks based
14 on adjusted primary PM_{2.5} emissions and based on adjusted precursor emissions that
15 contribute to secondary PM_{2.5} formation, the risk assessment provides insight into the
16 potential significance of this source of uncertainty. As discussed in section 3.4.2 of this
17 draft PA, the approach to adjusting air quality had relatively modest impacts on overall
18 risk estimates. Specifically, the difference between the absolute risk estimates from two
19 air quality modeling approach methods was generally less than 5% (draft PA section
20 3.4.2).

21 **C.5.2 Qualitative Uncertainty Analysis**

22 While the methods described above address some of the potentially important sources of
23 uncertainty and variability in the risk assessment, there are a range of additional sources that
24 cannot be analyzed quantitatively due to limitations in data, methods and/or resources. We have
25 addressed these additional sources of uncertainty qualitatively (Table C-18).

26 In describing each source of uncertainty, we attempt to characterize both the magnitude
27 and direction of impact on mortality risk estimates, including our rationale for these
28 characterizations. The categories used in describing the potential magnitude of impact (i.e., low,
29 medium, or high) reflect EPA staff judgments on the degree to which a particular source of
30 uncertainty could produce a sufficient impact on risk estimates to influence the interpretation of
31 those estimates in the context of the PM NAAQS reconsideration. Sources classified as having a
32 *low* impact would not be expected to influence conclusions from the risk assessment. Sources
33 classified as having a *medium* impact have the potential to affect such conclusions and sources
34 classified as *high* are likely to influence conclusions. Because this classification of the potential
35 magnitude of impact of sources of uncertainty is qualitative, it is not possible to place a
36 quantitative level of impact on each of the categories.

1 **Table C-18. Qualitative analysis of sources of uncertainty and assessment of potential impact on risk assessment.**

Source of Uncertainty	Description	Direction	Magnitude	Comments
Shape and corresponding statistical uncertainty around the CR function for long-term and short-term exposure-related mortality (especially at lower ambient PM levels)	Interpreting the shapes of concentration-response relationships, particularly at PM _{2.5} concentrations near the lower end of the air quality distribution, can be complicated by relatively low data density in the lower concentration range, the possible influence of exposure measurement error, and variability among individuals with respect to air pollution health effects. These sources of variability and uncertainty tend to smooth and “linearize” population-level concentration-response functions, and thus could obscure the existence of a threshold or nonlinear relationship (U.S. EPA, 2015, section 6.c).	Both	Medium-High	With regard to long-term exposure-related (nonaccidental) mortality, the ISA concludes that the majority of evidence supports a linear, no-threshold concentration-response relationship, though there is initial evidence indicating that the slope of the concentration-response curve may be steeper at lower concentrations for cardiovascular mortality (U.S. EPA, 2019, section 1.5.3.2). For long-term exposure-related mortality, the ISA notes that there is less certainty in the shape of the concentration-response curve at mean annual PM _{2.5} concentrations generally below 8 µg/m ³ because data density is reduced below this concentration (2019 ISA, section 11.2.4). Given that a portion of risk modeling in the risk assessment does involve locations with ambient PM _{2.5} concentrations below 8 µg/m ³ (although most of the population modeled is associated with level above this), we note the potential for significant uncertainty being introduced into the risk assessment (particularly for that portion of risk modeled at or below 8 µg/m ³). With regard to short-term exposure-related mortality, the ISA concludes that, while difficulties remain in assessing the shape of the PM _{2.5} -mortality concentration-response relationship, as identified in the 2009 PM ISA, and studies have not conducted systematic evaluations of alternatives to linearity, recent studies continue to provide evidence of a no-threshold linear relationship, with less confidence at concentrations lower than 5 µg/m ³ .
Representing population-level exposure with 12 km grid	As with long-term exposure-related mortality, short-term exposure-related mortality endpoints were also modeled	Both	Medium-High	Three studies providing effect estimates for short-term exposure-related mortality in the risk assessment all utilized some form of urban-level

Source of Uncertainty	Description	Direction	Magnitude	Comments
cell spatial framework (in context of modeling short-term exposure-related mortality)	using the same 12 km grid cell template. The disconnect between the spatial template used in the underlying short-term epidemiology studies and the 12 km grid template used in the risk assessment introduces uncertainty into risk estimates.			spatial unit in characterizing exposure (e.g., Baxter et al. (2017) utilizes the CBSA, Ito et al. (2013), utilizes the MSA), which are larger (less spatially differentiated) in general than the 12 km grid cells used in modeling risk. This means that we are generally modeling short-term exposure-related mortality at a finer level of spatial resolution in the risk assessment than reflected in the epidemiology studies supplying the effect estimates, which does introduce uncertainty into the analysis.
Representing population-level exposure with 12 km grid cell spatial framework (in context of modeling long-term exposure-related mortality)	The risk assessment utilizes a 12 km grid structure in modeling risk. A source of uncertainty associated with this approach is the mismatch between the 12 km grid cell framework and the exposure estimation approaches used in the epidemiology studies providing effect estimates for the risk assessment. This mismatch can introduce additional exposure error to risk estimates, beyond the error inherent to the underlying epidemiologic study.	Both	Medium	There are a variety of spatial templates used across the epidemiology studies providing CR functions used in the risk assessment and none of them are an exact match with the 12 km grid cell template used in the risk assessment. Jerrett et al. (2013), Pope et al. (2015) Differences between the exposure metric used in the risk assessment and those used in the underlying epidemiologic studies introduce uncertainty into risk estimates.
Simulating just meeting alternative annual standards with levels of 8.0, 9.0, and 11.0 ug/m ³ using linear extrapolation/ interpolation	The use of extrapolation/ interpolation in simulating just meeting annual standards introduces uncertainty into the risk assessment since this approach does not fully capture potential non-linearities associated with the formation of secondary PM _{2.5} .	Both	Medium	Extrapolation to generate the surfaces for 9.0 and 8.0 µg/m ³ are subject to greater uncertainty than interpolation to 11.0 µg/m ³ (i.e., since the former estimates concentrations below those in modeled surfaces, while the latter estimates a surface between two sets of modeled results). In addition, linear extrapolation/interpolation based on the primary-PM modeled surfaces (for current standard and 10.0 µg/m ³) is likely subject to less uncertainty than extrapolation/interpolation based on the secondary-PM modeled surfaces since the latter focus on secondary formation which could involve a higher degree of non-linearity.

Source of Uncertainty	Description	Direction	Magnitude	Comments
Simulating just meeting current and alternative standards using model-based (Downscaler) methods	<p>The baseline and adjusted concentration fields were developed using modeling to fill spatial and temporal gaps in monitoring and to explore air quality scenarios of policy interest. State-of-the-science modeling methods were used, but model-related biases and errors can introduce uncertainty into the PM_{2.5} concentration estimates.</p> <p>b) Due to the national scale of the assessment, the modeling scenarios are based on “across-the-board” emission changes in which emissions of primary PM_{2.5} or NO_x and SO₂ from all anthropogenic sources throughout the U.S. are scaled by fixed percentages. Although this approach tends to target the key sources in each area, it does not tailor emission changes to specific periods or sources.</p> <p>c) Two adjustment cases were applied that span a wide range of emission conditions, but these cases are necessarily a subset of the full set of possible emission cases that could be used to adjust PM_{2.5} concentrations to just meet standards.</p>	This source of uncertainty could bias results in either direction.	Medium	Use of state-of-the-science modeling systems with the relative response factor adjustment approach provides confidence in the broad features of the simulated national PM _{2.5} distributions and how the distributions shift with changing standards levels. Due to challenges in modeling local features in the national annual simulations, quantitative results for individual areas or small subsets of grid cells are relatively uncertain compared with broad features of the national PM _{2.5} distributions.
Potential confounding of the PM _{2.5} -mortality effect	Factors are considered potential confounders if demonstrated in the scientific literature to be related to health effects and correlated with PM. Omitting potential confounders from analyses could either increase or decrease the magnitude of PM _{2.5} effect estimates (e.g., Di et al., 2017, Figure S2 in Supplementary Materials). Thus, not accounting for	Both	Medium	Long-term PM _{2.5} exposure and mortality studies: For studies of long-term exposures, potential confounders are those that vary spatially or temporally. These may include socioeconomic status, race, age, medication use, smoking status, stress, noise, occupational exposures, and copollutant concentrations. Cohort studies used to characterize the PM _{2.5} -mortality relationship used a variety of approaches to account for these and other

Source of Uncertainty	Description	Direction	Magnitude	Comments
	<p>confounders can introduce uncertainty into effect estimates and, consequently, into the risk estimates generated using those effect estimates. Confounders vary according to study design, exposure duration, and health effect. While a range of approaches to control for potential confounders have been adopted across the studies used in the risk assessment, and across the broader body of PM_{2.5} epidemiologic studies assessed in the ISA, no individual study adjusts for all potential confounders.</p>			<p>potential confounders (e.g., see Appendix B). Across studies, a variety of study designs and statistical approaches have been used to account for potential confounding in the PM_{2.5}-mortality relationship. The fact that across this diverse body of evidence epidemiologic studies continue to report consistently positive associations that are often similar in magnitude, adds support the conclusion that the PM_{2.5}-mortality association is robust. Specifically regarding copollutants, the final PM ISA notes that, overall, associations remained relatively unchanged in copollutant models for total (nonaccidental) mortality, cardiovascular, and respiratory adjusted for ozone. Studies focusing on copollutant models with NO₂, PM_{10-2.5}, SO₂ and benzene were examined in individual studies, and across these studies the PM_{2.5}-mortality association was relatively unchanged.</p> <p>Short-term PM_{2.5} exposure and mortality studies: For studies of short-term exposures, potential confounders are those that vary temporally. These may include meteorology (e.g., temperature, humidity), day of week, season, medication use, allergen exposure, copollutant concentrations, and long-term temporal trends. Some recent studies have expanded the examination of potential confounders, including long-term temporal trends, weather, and copollutants. Overall, the ISA concludes that alternative approaches to controlling for long-term temporal trends and for the potential confounding effects of weather may influence the magnitude of the association between PM_{2.5} exposures and mortality, but have not been found to influence the direction of the observed association (U.S. EPA, 2019, section 11.1.5.1). With regard to</p>

Source of Uncertainty	Description	Direction	Magnitude	Comments
				copollutants, recent studies conducted outside the U.S. provide additional evidence that associations between short-term PM _{2.5} exposures and mortality remain positive and relatively unchanged in copollutant models with both gaseous pollutants and PM _{10-2.5} (U.S. EPA, 2019, Section 11.1.4).
Lag structure in short-term exposure-related mortality epidemiology studies	It can be challenging to characterize the timing associated with specific PM _{2.5} -related health effects and consequently specify the lag-structure that should be used in modeling those health effects. This can introduce uncertainty into the modeling of risk for short-term exposure-related endpoints.	Both	Low-Medium	Given the emphasis placed in the risk assessment on mortality, we focus here on lags associated with all-cause mortality.
Compositional and source differences in PM	The composition of PM _{2.5} can differ across study areas reflecting underlying differences in primary and secondary PM _{2.5} sources (both natural and anthropogenic). If these compositional differences lead to differences in public health impacts (per unit concentration in ambient air) for PM _{2.5} , then uncertainty may be introduced into risk estimates that are based on concentration-response relationships for PM _{2.5} mass.	Both	Low	The Integrated Synthesis chapter of the final ISA (Chapter 1, U.S. EPA, 2019) states that, the assessment of PM sources and components confirms and continues to support the conclusion from the 2009 PM ISA: Many PM _{2.5} components and sources are associated with health effects, and the evidence does not indicate that any one source or component is more strongly related with health effects than PM _{2.5} mass.
Temporal mismatch between ambient air quality data characterizing exposure and mortality in long-term exposure-related epidemiology studies	Several of the epidemiology studies for long-term exposure-related mortality have a mismatch between the time period associated with ambient PM _{2.5} concentrations used to characterize population-level exposure and mortality data Jerrett et al. (2016), Pope et al. (2015).	Both	Low	This approach can be reasonable in the context of an epidemiologic study evaluating health effect associations with long-term PM _{2.5} exposures, under the assumption that spatial patterns in PM _{2.5} concentrations are not appreciably different during time periods for which air quality information is not available (e.g., Chen et al. (2016)), Thus, as long as the overall spatial pattern of ambient PM _{2.5} levels in relation to population-level exposure and mortality rates has held relatively stable over time, then a

Source of Uncertainty	Description	Direction	Magnitude	Comments
				temporal disconnect between the time-period associated with mortality and the ambient PM _{2.5} level used in characterizing exposure would not be expected to introduce significant uncertainty into the epidemiology studies and associated effect estimates.
Exposure measurement error in epidemiologic studies assessing the relationship between mortality and exposure to ambient PM _{2.5}	Epidemiologic studies have employed a variety of approaches to estimate population-level PM _{2.5} exposures (e.g., stationary monitors, hybrid modeling approaches). These approaches are based on using measured or predicted ambient PM _{2.5} concentrations as surrogates for population exposures. As such, exposure estimates in epidemiologic studies are subject to exposure error. This error in the underlying epidemiologic studies contributes to uncertainty in the risk estimates that are based on concentration-response relationships in those studies.	Both	Low	Available studies indicate that PM _{2.5} health effect associations are robust across various approaches to estimating PM _{2.5} exposures. This includes recent studies that estimate exposures using ground-based monitors alone and studies that estimate exposures using data from multiple sources (e.g., satellites, land use information, modeling), in addition to monitors. While none of these approaches eliminates the potential for exposure error in epidemiologic studies, such error does not call into question the findings of key PM _{2.5} epidemiologic studies. The ISA notes that, while bias in either direction can occur, exposure error tends to result in underestimation of health effects in epidemiologic studies of PM exposure (U.S. EPA, 2019, section 3.5). Consistent with this, a recent study Hart et al. (2015) reports that correction for PM _{2.5} exposure error using personal exposure information results in a moderately larger effect estimate for long-term PM _{2.5} exposure and mortality (though with wider confidence intervals). While most PM _{2.5} epidemiologic studies have not employed similar corrections for exposure error, several studies report that restricting analyses to populations in close proximity to a monitor (i.e., in order to reduce exposure error) result in larger PM _{2.5} effect estimates (e.g., Willis et al., 2003; Kloog et al., 2013). Thus, to the extent key PM _{2.5} epidemiologic studies are subject to exposure error, correction for that error

Source of Uncertainty	Description	Direction	Magnitude	Comments
				would likely result in larger effect estimates, and thus larger estimates of PM _{2.5} -associated mortality incidence in the risk assessment.
Use of associations reported in epidemiologic studies to estimate how mortality incidence may change with changing PM _{2.5} air quality.	The ISA's determination that the evidence supports a causal relationship between PM _{2.5} exposure and mortality is based on assessing a broad body of evidence from epidemiologic and experimental studies. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate how mortality incidence may change with changing PM _{2.5} air quality is subject to uncertainty.	Both	Low	The ISA assesses a longstanding body of health evidence supporting relationships between PM _{2.5} exposures (short- and long-term) and mortality. Much of this evidence comes from epidemiologic studies conducted in North America, Europe, or Asia that demonstrate generally positive, and often statistically significant, associations between PM _{2.5} exposures and total or cause-specific mortality. In addition, recent experimental evidence, as well as evidence from panel studies, strengthens support for potential biological pathways through which PM _{2.5} exposures could lead to serious health outcomes, including mortality. While this broad body of evidence from across disciplines provides the foundation for the ISA's conclusions, the risk assessment necessarily focuses on a small number of individual studies. Although the studies selected for the risk assessment are part of the evidence base supporting the ISA's causality determinations for mortality, the concentration-response relationship in any given study reflects the particular time period, locations, air quality distribution and populations evaluated in that study. Thus, the use of the concentration-response relationship from any individual epidemiologic study to estimate mortality incidence across the U.S. for populations, locations and PM _{2.5} air quality distributions different from those present during the study period is subject to uncertainty.

1 **C.5.3 Conclusion**

2 To increase overall confidence in the risk assessment, a deliberative process has been
3 used in specifying each of the analytical elements comprising the risk model, including selection
4 of urban study areas as well as specification of other inputs such as CR functions. This
5 deliberative process involved rigorous review of available literature addressing both PM_{2.5}
6 exposure and risk combined with the application of a formal set of criteria to guide development
7 of each of the key analytical elements in the risk assessment. The application of this deliberative
8 process increases overall confidence in the risk estimates by ensuring that the estimates are based
9 on the best available science and data characterizing PM_{2.5} exposure and risk, and that they
10 reflect consideration of input from experts on PM exposure and risk through CASAC and public
11 reviews.

12
13

1 **C.6 PM_{2.5} DESIGN VALUES FOR THE AIR QUALITY PROJECTIONS**

2

3 **Table C-19. PM_{2.5} DVs for the Primary PM projection case and 12/35 standard level.**

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
AkronO	391530017	Annual	Yes	0	-18	10.99	11.99	23.7	25.4
AkronO	391530023	Annual	No	0	-18	9.16	9.90	20.2	21.4
Altoon	420130801	Annual	Yes	0	-41	10.11	12.02	23.8	29.5
Atlant	131210039	Annual	Yes	0	-27	10.38	11.99	19.7	22.6
Atlant	132230003	Annual	No	0	-27	7.82	8.62	16.2	17.5
Atlant	131350002	Annual	No	0	-27	8.84	10.05	17.9	20.2
Atlant	130890002	Annual	No	0	-27	9.34	10.63	19.2	21.7
Atlant	130670003	Annual	No	0	-27	9.51	10.79	18.6	21.0
Atlant	130630091	Annual	No	0	-27	9.86	11.19	19.1	21.6
Bakers	060290010	24-hr	Yes	79	77	16.52	10.23	70.0	35.4
Bakers	060290016	24-hr	No	79	77	18.45	11.45	61.3	31.7
Bakers	060290015	24-hr	No	79	77	5.15	3.97	15.8	13.6
Bakers	060290014	24-hr	No	79	77	16.53	9.81	61.4	31.7
Bakers	060290011	24-hr	No	79	77	6.06	4.84	19.6	16.6
Birmin	010732059	Annual	Yes	0	-10	11.25	12.00	22.3	23.9
Birmin	010732003	Annual	No	0	-10	10.08	10.70	19.0	20.1
Birmin	010731010	Annual	No	0	-10	9.78	10.30	19.2	20.1
Birmin	010730023	Annual	No	0	-10	10.94	11.66	22.8	24.2
Canton	391510017	Annual	Yes	0	-23	10.81	12.04	23.7	26.1
Canton	391510020	Annual	No	0	-23	9.91	10.96	22.0	23.6
Chicag	170313103	Annual	Yes	0	-15	11.10	12.00	22.6	24.2
Chicag	550590019	Annual	No	0	-15	8.04	8.56	20.4	21.5
Chicag	181270024	Annual	No	0	-15	9.51	10.30	22.4	24.1
Chicag	180892004	Annual	No	0	-15	9.84	10.71	24.7	26.7
Chicag	180890031	Annual	No	0	-15	10.12	11.01	23.6	25.6
Chicag	180890026	Annual	No	0	-15	-	-	25.2	27.1
Chicag	180890022	Annual	No	0	-15	-	-	22.7	24.8
Chicag	180890006	Annual	No	0	-15	10.03	10.93	23.1	25.2
Chicag	171971011	Annual	No	0	-15	8.36	8.85	18.4	19.3
Chicag	171971002	Annual	No	0	-15	7.69	8.23	20.0	21.2
Chicag	170890007	Annual	No	0	-15	8.94	9.55	19.2	20.5
Chicag	170890003	Annual	No	0	-15	-	-	19.2	20.0
Chicag	170434002	Annual	No	0	-15	8.87	9.48	19.9	20.7
Chicag	170316005	Annual	No	0	-15	10.79	11.66	24.1	26.1

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Chicag	170314201	Annual	No	0	-15	9.00	9.61	21.4	22.6
Chicag	170314007	Annual	No	0	-15	9.49	10.17	-	-
Chicag	170313301	Annual	No	0	-15	10.37	11.18	23.5	25.2
Chicag	170310076	Annual	No	0	-15	10.18	10.96	22.5	24.0
Chicag	170310057	Annual	No	0	-15	11.03	11.89	26.8	28.4
Chicag	170310052	Annual	No	0	-15	10.00	10.78	23.3	24.9
Chicag	170310022	Annual	No	0	-15	10.38	11.30	22.4	23.9
Chicag	170310001	Annual	No	0	-15	10.13	10.88	21.7	23.4
Cincin	390610014	Annual	Yes	0	-24	10.70	12.02	22.9	24.7
Cincin	390610042	Annual	No	0	-24	10.29	11.47	22.6	24.5
Cincin	390610040	Annual	No	0	-24	9.45	10.53	21.0	22.9
Cincin	390610010	Annual	No	0	-24	9.43	10.41	21.3	22.9
Cincin	390610006	Annual	No	0	-24	9.46	10.56	20.3	21.8
Cincin	390170020	Annual	No	0	-24	-	-	24.2	26.5
Cincin	390170019	Annual	No	0	-24	10.24	11.51	22.0	23.8
Cincin	390170016	Annual	No	0	-24	9.79	10.91	22.1	23.7
Cincin	210373002	Annual	No	0	-24	9.06	10.00	20.9	22.6
Clevel	390350065	Annual	Yes	0	2	12.17	12.03	24.9	24.6
Clevel	391030004	Annual	No	0	2	8.73	8.66	19.6	19.5
Clevel	390933002	Annual	No	0	2	8.10	8.03	20.2	20.1
Clevel	390850007	Annual	No	0	2	7.88	7.82	17.4	17.3
Clevel	390351002	Annual	No	0	2	8.86	8.78	19.5	19.4
Clevel	390350045	Annual	No	0	2	10.61	10.50	22.9	22.7
Clevel	390350038	Annual	No	0	2	11.38	11.25	25.0	24.8
Clevel	390350034	Annual	No	0	2	8.87	8.79	20.4	20.2
Detroi	261630033	Annual	Yes	0	-15	11.30	12.04	26.8	28.4
Detroi	261630039	Annual	No	0	-15	9.11	9.63	22.3	23.7
Detroi	261630036	Annual	No	0	-15	8.68	9.13	21.8	23.2
Detroi	261630025	Annual	No	0	-15	8.98	9.54	24.1	25.2
Detroi	261630019	Annual	No	0	-15	9.18	9.75	22.4	24.1
Detroi	261630016	Annual	No	0	-15	9.62	10.19	24.4	25.4
Detroi	261630015	Annual	No	0	-15	11.19	11.91	25.5	27.0
Detroi	261630001	Annual	No	0	-15	9.50	10.14	23.3	24.9
Detroi	261470005	Annual	No	0	-15	8.89	9.34	24.3	25.4
Detroi	261250001	Annual	No	0	-15	8.86	9.41	24.2	25.7
Detroi	260990009	Annual	No	0	-15	8.80	9.29	26.2	27.6
EICent	060250005	Annual	Yes	0	12	12.63	12.00	33.5	31.3
EICent	060251003	Annual	No	0	12	7.44	7.01	19.8	18.5

CBSA ^a	Site	Controlling Standard	Controlling Site?	NO _x & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
ElCent	060250007	Annual	No	0	12	8.37	7.99	21.5	20.8
Elkhar	180390008	Annual	Yes	0	-47	10.24	12.01	28.6	33.2
Evansv	181630023	Annual	Yes	0	-44	10.11	12.03	21.5	24.0
Evansv	211010014	Annual	No	0	-44	9.64	11.32	20.7	22.3
Evansv	181630021	Annual	No	0	-44	9.84	11.68	21.6	23.3
Evansv	181630016	Annual	No	0	-44	10.02	11.91	22.0	24.0
Fresno	060195001	24-hr	Yes	0	70	14.08	10.87	49.3	35.4
Fresno	060195025	24-hr	No	0	70	13.63	9.98	47.9	31.7
Fresno	060192009	24-hr	No	0	70	8.47	7.26	31.3	25.1
Fresno	060190011	24-hr	No	0	70	14.07	10.01	53.8	34.4
Hanfor	060310004	24-hr	Yes	65	79	21.98	11.79	72.0	35.4
Hanfor	060311004	24-hr	No	65	79	16.49	9.68	58.9	30.7
Housto	482011035	Annual	Yes	0	-14	11.19	12.04	22.4	24.0
Housto	482011039	Annual	No	0	-14	9.22	9.82	21.7	23.1
Housto	482010058	Annual	No	0	-14	9.67	10.37	22.3	23.8
Housto	481671034	Annual	No	0	-14	7.36	7.57	20.3	20.8
Indian	180970087	Annual	Yes	0	-10	11.44	12.01	25.9	26.8
Indian	180970083	Annual	No	0	-10	11.06	11.59	23.9	24.9
Indian	180970081	Annual	No	0	-10	11.07	11.61	25.0	26.0
Indian	180970078	Annual	No	0	-10	10.14	10.60	24.4	24.9
Indian	180970043	Annual	No	0	-10	-	-	26.0	26.4
Indian	180950011	Annual	No	0	-10	9.05	9.40	21.8	22.3
Indian	180570007	Annual	No	0	-10	9.02	9.39	21.4	22.1
Johnst	420210011	Annual	Yes	0	-25	10.68	12.03	25.8	30.3
Lancas	420710012	Annual	Yes	0	12	12.83	12.00	32.7	30.4
Lancas	420710007	Annual	No	0	12	10.57	9.88	29.8	27.4
LasVeg	320030561	Annual	Yes	0	-22	10.28	11.98	24.5	29.4
LasVeg	320032002	Annual	No	0	-22	9.79	11.38	19.8	23.4
LasVeg	320031019	Annual	No	0	-22	5.18	5.70	11.5	12.2
LasVeg	320030540	Annual	No	0	-22	8.80	10.21	21.7	25.9
Lebano	420750100	Annual	Yes	0	-15	11.20	12.02	31.4	33.9
Little	051191008	Annual	Yes	0	-41	10.27	12.03	21.7	24.7
Little	051190007	Annual	No	0	-41	9.78	11.76	20.5	24.0
LoganU	490050007	24-hr	Yes	0	-7	6.95	7.15	34.0	35.4
LosAng	060371103	Annual	Yes	0	5	12.38	12.03	32.8	32.1
LosAng	060592022	Annual	No	0	5	7.48	7.33	15.3	15.0
LosAng	060590007	Annual	No	0	5	9.63	9.37	-	-
LosAng	060374004	Annual	No	0	5	10.25	9.97	27.3	26.7

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
LosAng	060374002	Annual	No	0	5	11.06	10.76	29.2	28.6
LosAng	060371602	Annual	No	0	5	11.86	11.52	32.3	31.5
LosAng	060371302	Annual	No	0	5	11.99	11.64	31.5	30.8
LosAng	060371201	Annual	No	0	5	9.46	9.24	25.6	25.0
LosAng	060370002	Annual	No	0	5	10.52	10.27	29.2	28.6
Louisv	180190006	Annual	Yes	0	-27	10.64	12.04	23.9	26.2
Louisv	211110075	Annual	No	0	-27	10.42	11.84	22.3	24.3
Louisv	211110067	Annual	No	0	-27	9.55	10.78	21.4	23.6
Louisv	211110051	Annual	No	0	-27	10.29	11.48	21.8	23.7
Louisv	211110043	Annual	No	0	-27	10.37	11.72	22.0	24.1
Louisv	180431004	Annual	No	0	-27	9.96	11.20	22.0	24.2
Louisv	180190008	Annual	No	0	-27	8.72	9.69	20.1	21.5
MaconG	130210007	Annual	Yes	0	-39	10.13	12.01	21.2	24.8
MaconG	130210012	Annual	No	0	-39	7.68	8.90	16.6	18.6
Madera	060392010	24-hr	Yes	0	56	13.30	11.03	45.1	35.3
McAlle	482150043	Annual	Yes	0	-67	10.09	12.02	25.0	27.4
Merced	060470003	24-hr	Yes	0	28	11.81	10.97	39.0	35.4
Merced	060472510	24-hr	No	0	28	11.68	10.57	39.8	35.1
Modest	060990006	24-hr	Yes	0	51	13.02	10.70	45.7	35.3
Modest	060990005	24-hr	No	0	51	-	-	38.8	32.5
NapaCA	060550003	Annual	Yes	0	-47	10.36	12.03	25.1	29.1
NewYor	360610128	Annual	Yes	0	-26	10.20	12.00	23.9	27.8
NewYor	361030002	Annual	No	0	-26	7.18	8.10	18.8	21.0
NewYor	360810124	Annual	No	0	-26	7.52	8.65	19.5	22.4
NewYor	360710002	Annual	No	0	-26	6.95	7.81	17.5	19.6
NewYor	360610134	Annual	No	0	-26	9.70	11.38	21.6	25.0
NewYor	360610079	Annual	No	0	-26	8.42	9.82	22.8	25.6
NewYor	360470122	Annual	No	0	-26	8.66	10.10	20.5	23.7
NewYor	360050133	Annual	No	0	-26	9.05	10.53	24.0	28.0
NewYor	360050110	Annual	No	0	-26	7.39	8.56	19.4	22.8
NewYor	340392003	Annual	No	0	-26	8.59	9.87	23.6	26.3
NewYor	340390004	Annual	No	0	-26	9.87	11.40	24.2	27.3
NewYor	340310005	Annual	No	0	-26	8.42	9.63	22.2	24.7
NewYor	340292002	Annual	No	0	-26	7.23	8.04	18.1	19.8
NewYor	340273001	Annual	No	0	-26	6.78	7.56	17.1	18.8
NewYor	340171003	Annual	No	0	-26	8.79	10.15	23.4	26.9
NewYor	340130003	Annual	No	0	-26	8.89	10.21	23.8	27.3
NewYor	340030003	Annual	No	0	-26	8.90	10.22	24.5	27.4

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
OgdenC	490110004	24-hr	Yes	0	-18	7.28	7.77	32.6	35.4
OgdenC	490570002	24-hr	No	0	-18	8.99	9.73	-	-
OgdenC	490030003	24-hr	No	0	-18	6.35	6.76	-	-
Philad	420450002	Annual	Yes	0	-8	11.46	12.04	26.0	27.2
Philad	421010057	Annual	No	0	-8	10.86	11.37	27.0	28.4
Philad	421010055	Annual	No	0	-8	11.43	12.03	27.5	29.0
Philad	421010048	Annual	No	0	-8	10.27	10.77	25.6	27.0
Philad	420290100	Annual	No	0	-8	9.64	10.03	23.9	25.1
Philad	340150004	Annual	No	0	-8	8.33	8.69	20.6	21.5
Philad	340071007	Annual	No	0	-8	8.84	9.23	21.0	22.0
Philad	340070002	Annual	No	0	-8	10.19	10.61	23.5	24.6
Philad	240150003	Annual	No	0	-8	8.70	9.02	22.6	23.4
Philad	100031012	Annual	No	0	-8	9.04	9.40	23.0	23.8
Pittsb	420030064	Annual	Yes	0	13	12.82	12.00	35.8	32.8
Pittsb	421290008	Annual	No	0	13	8.65	8.15	19.6	18.9
Pittsb	421255001	Annual	No	0	13	8.35	7.89	17.8	17.2
Pittsb	421250200	Annual	No	0	13	8.95	8.44	19.3	18.2
Pittsb	421250005	Annual	No	0	13	11.02	10.38	22.7	21.2
Pittsb	420070014	Annual	No	0	13	10.11	9.48	21.9	20.5
Pittsb	420050001	Annual	No	0	13	11.03	10.30	21.9	20.5
Pittsb	420031301	Annual	No	0	13	11.00	10.30	24.8	23.0
Pittsb	420031008	Annual	No	0	13	9.78	9.16	20.5	19.3
Pittsb	420030008	Annual	No	0	13	9.50	8.85	20.5	19.0
Prinev	410130100	24-hr	Yes	0	10	8.60	8.17	37.6	35.3
ProvoO	490494001	24-hr	Yes	0	-30	7.74	8.57	30.9	35.3
ProvoO	490495010	24-hr	No	0	-30	6.73	7.52	-	-
ProvoO	490490002	24-hr	No	0	-30	7.41	8.31	28.9	33.2
Rivers	060658005	24-hr	Yes	0	36	14.48	11.51	43.2	35.3
Rivers	060658001	24-hr	No	0	36	-	-	36.5	29.6
Sacram	060670006	24-hr	Yes	0	-23	9.31	10.40	31.4	35.4
Sacram	061131003	24-hr	No	0	-23	6.62	7.19	15.8	17.3
Sacram	060670012	24-hr	No	0	-23	7.30	8.01	19.8	21.2
Sacram	060670010	24-hr	No	0	-23	8.67	9.65	26.5	29.9
Sacram	060610006	24-hr	No	0	-23	7.58	8.47	20.3	22.3
Sacram	060610003	24-hr	No	0	-23	6.71	7.26	19.3	20.2
SaltLa	490353010	24-hr	Yes	0	44	-	-	41.5	35.3
SaltLa	490353006	24-hr	No	0	44	7.62	6.19	36.8	30.2
SaltLa	490351001	24-hr	No	0	44	7.07	5.85	32.1	25.8

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
SanLui	060792007	Annual	Yes	0	-46	10.70	12.04	25.9	29.4
SanLui	060798002	Annual	No	0	-46	5.71	6.33	-	-
SanLui	060792004	Annual	No	0	-46	8.25	9.26	19.8	21.4
SouthB	181410015	24-hr	Yes	0	-23	10.45	11.37	32.5	35.4
St.Lou	290990019	Annual	Yes	0	-39	10.12	12.02	22.8	24.9
St.Lou	295100094	Annual	No	0	-39	9.57	11.38	23.3	25.9
St.Lou	295100093	Annual	No	0	-39	-	-	23.7	26.6
St.Lou	295100085	Annual	No	0	-39	10.10	12.01	23.6	26.2
St.Lou	295100007	Annual	No	0	-39	9.78	11.52	23.7	26.4
St.Lou	291893001	Annual	No	0	-39	9.85	11.72	22.4	25.2
Stockt	060771002	24-hr	Yes	0	17	12.23	11.30	38.7	35.4
Stockt	060772010	24-hr	No	0	17	10.74	9.96	37.3	34.3
Visali	061072002	24-hr	Yes	48	56	16.23	10.93	54.0	35.4
Weirto	390810017	Annual	Yes	0	-5	11.75	12.02	27.2	27.8
Weirto	540090011	Annual	No	0	-5	9.75	9.95	22.8	23.5
Weirto	540090005	Annual	No	0	-5	10.52	10.74	22.4	22.9
Weirto	390810021	Annual	No	0	-5	9.29	9.47	22.2	22.6
Wheeli	540511002	Annual	Yes	0	-44	10.24	12.02	22.5	25.4
Wheeli	540690010	Annual	No	0	-44	9.61	11.32	19.7	22.6

^a CBSA names are the first six characters of the full CBSAs names in Table C-3.
^b Percent reduction in NOx and SO₂ emissions associated with just meeting the standard in this case.
^c Percent reduction in Primary PM_{2.5} emissions associated with just meeting the standard in this case.

1
2

1 Table C-20. PM_{2.5} DVs for the Secondary PM projection case and 12/35 standard level.

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
AkronO	391530017	Annual	Yes	-67	0	10.99	12.04	23.7	26.8
AkronO	391530023	Annual	No	-67	0	9.16	10.20	20.2	21.8
Altoon	420130801	Annual	Yes	N/A	N/A	10.11	12.04	23.8	28.3
Atlant	131210039	Annual	Yes	N/A	N/A	10.38	12.04	19.7	22.9
Atlant	132230003	Annual	No	N/A	N/A	7.82	9.07	16.2	18.8
Atlant	131350002	Annual	No	N/A	N/A	8.84	10.25	17.9	20.8
Atlant	130890002	Annual	No	N/A	N/A	9.34	10.83	19.2	22.3
Atlant	130670003	Annual	No	N/A	N/A	9.51	11.03	18.6	21.6
Atlant	130630091	Annual	No	N/A	N/A	9.86	11.44	19.1	22.2
Bakers	060290010	24-hr	Yes	N/A	N/A	16.52	10.40	70.0	35.4
Bakers	060290016	24-hr	No	N/A	N/A	18.45	11.61	61.3	31.0
Bakers	060290015	24-hr	No	N/A	N/A	5.15	3.24	15.8	8.0
Bakers	060290014	24-hr	No	N/A	N/A	16.53	10.40	61.4	31.1
Bakers	060290011	24-hr	No	N/A	N/A	6.06	3.81	19.6	9.9
Birmin	010732059	Annual	Yes	-56	0	11.25	12.03	22.3	24.2
Birmin	010732003	Annual	No	-56	0	10.08	10.86	19.0	21.5
Birmin	010731010	Annual	No	-56	0	9.78	10.68	19.2	21.4
Birmin	010730023	Annual	No	-56	0	10.94	11.73	22.8	25.3
Canton	391510017	Annual	Yes	-78	0	10.81	12.04	23.7	26.1
Canton	391510020	Annual	No	-78	0	9.91	11.14	22.0	24.8
Chicag	170313103	Annual	Yes	N/A	N/A	11.10	12.04	22.6	24.5
Chicag	550590019	Annual	No	N/A	N/A	8.04	8.72	20.4	22.1
Chicag	181270024	Annual	No	N/A	N/A	9.51	10.32	22.4	24.3
Chicag	180892004	Annual	No	N/A	N/A	9.84	10.67	24.7	26.8
Chicag	180890031	Annual	No	N/A	N/A	10.12	10.98	23.6	25.6
Chicag	180890026	Annual	No	N/A	N/A	-	-	25.2	27.3
Chicag	180890022	Annual	No	N/A	N/A	-	-	22.7	24.6
Chicag	180890006	Annual	No	N/A	N/A	10.03	10.88	23.1	25.1
Chicag	171971011	Annual	No	N/A	N/A	8.36	9.07	18.4	20.0
Chicag	171971002	Annual	No	N/A	N/A	7.69	8.34	20.0	21.7
Chicag	170890007	Annual	No	N/A	N/A	8.94	9.70	19.2	20.8
Chicag	170890003	Annual	No	N/A	N/A	-	-	19.2	20.8
Chicag	170434002	Annual	No	N/A	N/A	8.87	9.62	19.9	21.6
Chicag	170316005	Annual	No	N/A	N/A	10.79	11.70	24.1	26.1
Chicag	170314201	Annual	No	N/A	N/A	9.00	9.76	21.4	23.2
Chicag	170314007	Annual	No	N/A	N/A	9.49	10.29	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	11.25	23.5	25.5

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Chicag	170310076	Annual	No	N/A	N/A	10.18	11.04	22.5	24.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	11.96	26.8	29.1
Chicag	170310052	Annual	No	N/A	N/A	10.00	10.85	23.3	25.3
Chicag	170310022	Annual	No	N/A	N/A	10.38	11.26	22.4	24.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	10.99	21.7	23.5
Cincin	390610014	Annual	Yes	-72	0	10.70	12.04	22.9	26.1
Cincin	390610042	Annual	No	-72	0	10.29	11.66	22.6	26.2
Cincin	390610040	Annual	No	-72	0	9.45	10.79	21.0	25.4
Cincin	390610010	Annual	No	-72	0	9.43	10.75	21.3	24.4
Cincin	390610006	Annual	No	-72	0	9.46	10.75	20.3	24.3
Cincin	390170020	Annual	No	-72	0	-	-	24.2	27.8
Cincin	390170019	Annual	No	-72	0	10.24	11.40	22.0	24.5
Cincin	390170016	Annual	No	-72	0	9.79	11.06	22.1	25.1
Cincin	210373002	Annual	No	-72	0	9.06	10.42	20.9	25.1
Clevel	390350065	Annual	Yes	6	0	12.17	12.04	24.9	24.7
Clevel	391030004	Annual	No	6	0	8.73	8.61	19.6	19.2
Clevel	390933002	Annual	No	6	0	8.10	7.99	20.2	19.9
Clevel	390850007	Annual	No	6	0	7.88	7.78	17.4	17.1
Clevel	390351002	Annual	No	6	0	8.86	8.74	19.5	19.2
Clevel	390350045	Annual	No	6	0	10.61	10.49	22.9	22.6
Clevel	390350038	Annual	No	6	0	11.38	11.26	25.0	24.7
Clevel	390350034	Annual	No	6	0	8.87	8.75	20.4	20.1
Detroi	261630033	Annual	Yes	-56	0	11.30	12.04	26.8	30.2
Detroi	261630039	Annual	No	-56	0	9.11	9.88	22.3	24.8
Detroi	261630036	Annual	No	-56	0	8.68	9.39	21.8	23.4
Detroi	261630025	Annual	No	-56	0	8.98	9.75	24.1	26.5
Detroi	261630019	Annual	No	-56	0	9.18	9.97	22.4	24.1
Detroi	261630016	Annual	No	-56	0	9.62	10.38	24.4	27.4
Detroi	261630015	Annual	No	-56	0	11.19	11.97	25.5	28.2
Detroi	261630001	Annual	No	-56	0	9.50	10.20	23.3	25.0
Detroi	261470005	Annual	No	-56	0	8.89	9.50	24.3	26.1
Detroi	261250001	Annual	No	-56	0	8.86	9.65	24.2	26.7
Detroi	260990009	Annual	No	-56	0	8.80	9.48	26.2	28.4
EICent	060250005	Annual	Yes	N/A	N/A	12.63	12.04	33.5	31.9
EICent	060251003	Annual	No	N/A	N/A	7.44	7.09	19.8	18.9
EICent	060250007	Annual	No	N/A	N/A	8.37	7.98	21.5	20.5
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	12.04	28.6	33.6
Evansv	181630023	Annual	Yes	-89	0	10.11	12.03	21.5	32.5

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Evansv	211010014	Annual	No	-89	0	9.64	11.58	20.7	30.2
Evansv	181630021	Annual	No	-89	0	9.84	11.79	21.6	32.4
Evansv	181630016	Annual	No	-89	0	10.02	11.95	22.0	32.8
Fresno	060190011	24-hr	Yes	N/A	N/A	14.07	10.46	53.8	35.4
Fresno	060195025	24-hr	No	N/A	N/A	13.63	10.13	47.9	31.5
Fresno	060195001	24-hr	No	N/A	N/A	14.08	10.47	49.3	32.4
Fresno	060192009	24-hr	No	N/A	N/A	8.47	6.30	31.3	20.6
Hanford	060310004	24-hr	Yes	N/A	N/A	21.98	10.81	72.0	35.4
Hanford	060311004	24-hr	No	N/A	N/A	16.49	8.11	58.9	29.0
Housto	482011035	Annual	Yes	-91	0	11.19	12.04	22.4	25.2
Housto	482011039	Annual	No	-91	0	9.22	10.16	21.7	24.9
Housto	482010058	Annual	No	-91	0	9.67	10.52	22.3	24.8
Housto	481671034	Annual	No	-91	0	7.36	8.27	20.3	23.3
Indian	180970087	Annual	Yes	-24	0	11.44	12.02	25.9	27.5
Indian	180970083	Annual	No	-24	0	11.06	11.64	23.9	25.2
Indian	180970081	Annual	No	-24	0	11.07	11.65	25.0	26.7
Indian	180970078	Annual	No	-24	0	10.14	10.72	24.4	26.2
Indian	180970043	Annual	No	-24	0	-	-	26.0	27.6
Indian	180950011	Annual	No	-24	0	9.05	9.51	21.8	23.1
Indian	180570007	Annual	No	-24	0	9.02	9.52	21.4	22.8
Johnst	420210011	Annual	Yes	-86	0	10.68	12.04	25.8	27.9
Lancas	420710012	Annual	Yes	40	0	12.83	12.03	32.7	31.6
Lancas	420710007	Annual	No	40	0	10.57	9.78	29.8	28.5
LasVeg	320030561	Annual	Yes	N/A	N/A	10.28	12.04	24.5	28.7
LasVeg	320032002	Annual	No	N/A	N/A	9.79	11.47	19.8	23.2
LasVeg	320031019	Annual	No	N/A	N/A	5.18	6.07	11.5	13.5
LasVeg	320030540	Annual	No	N/A	N/A	8.80	10.31	21.7	25.4
Lebano	420750100	Annual	Yes	-61	0	11.20	12.04	31.4	32.4
Little	051191008	Annual	Yes	-98	0	10.27	12.04	21.7	26.7
Little	051190007	Annual	No	-98	0	9.78	11.40	20.5	25.5
LoganU	490050007	24-hr	Yes	-28	0	6.95	7.12	34.0	35.4
LosAng	060371103	Annual	Yes	N/A	N/A	12.38	12.04	32.8	31.9
LosAng	060592022	Annual	No	N/A	N/A	7.48	7.27	15.3	14.9
LosAng	060590007	Annual	No	N/A	N/A	9.63	9.37	-	-
LosAng	060374004	Annual	No	N/A	N/A	10.25	9.97	27.3	26.6
LosAng	060374002	Annual	No	N/A	N/A	11.06	10.76	29.2	28.4
LosAng	060371602	Annual	No	N/A	N/A	11.86	11.53	32.3	31.4
LosAng	060371302	Annual	No	N/A	N/A	11.99	11.66	31.5	30.6

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
LosAng	060371201	Annual	No	N/A	N/A	9.46	9.20	25.6	24.9
LosAng	060370002	Annual	No	N/A	N/A	10.52	10.23	29.2	28.4
Louisv	180190006	Annual	Yes	-65	0	10.64	12.04	23.9	28.4
Louisv	211110075	Annual	No	-65	0	10.42	11.76	22.3	26.4
Louisv	211110067	Annual	No	-65	0	9.55	10.84	21.4	25.4
Louisv	211110051	Annual	No	-65	0	10.29	11.67	21.8	25.9
Louisv	211110043	Annual	No	-65	0	10.37	11.71	22.0	26.1
Louisv	180431004	Annual	No	-65	0	9.96	11.32	22.0	25.8
Louisv	180190008	Annual	No	-65	0	8.72	10.07	20.1	24.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	12.04	21.2	25.2
MaconG	130210012	Annual	No	N/A	N/A	7.68	9.13	16.6	19.7
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	11.15	45.1	35.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	12.04	25.0	29.8
Merced	060472510	24-hr	Yes	32	0	11.68	10.79	39.8	35.4
Merced	060470003	24-hr	No	32	0	11.81	10.89	39.0	34.1
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	10.82	45.7	35.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	30.1
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	12.04	25.1	29.2
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	12.04	23.9	28.2
NewYor	361030002	Annual	No	N/A	N/A	7.18	8.48	18.8	22.2
NewYor	360810124	Annual	No	N/A	N/A	7.52	8.88	19.5	23.0
NewYor	360710002	Annual	No	N/A	N/A	6.95	8.20	17.5	20.7
NewYor	360610134	Annual	No	N/A	N/A	9.70	11.45	21.6	25.5
NewYor	360610079	Annual	No	N/A	N/A	8.42	9.94	22.8	26.9
NewYor	360470122	Annual	No	N/A	N/A	8.66	10.22	20.5	24.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	10.68	24.0	28.3
NewYor	360050110	Annual	No	N/A	N/A	7.39	8.72	19.4	22.9
NewYor	340392003	Annual	No	N/A	N/A	8.59	10.14	23.6	27.9
NewYor	340390004	Annual	No	N/A	N/A	9.87	11.65	24.2	28.6
NewYor	340310005	Annual	No	N/A	N/A	8.42	9.94	22.2	26.2
NewYor	340292002	Annual	No	N/A	N/A	7.23	8.53	18.1	21.4
NewYor	340273001	Annual	No	N/A	N/A	6.78	8.00	17.1	20.2
NewYor	340171003	Annual	No	N/A	N/A	8.79	10.38	23.4	27.6
NewYor	340130003	Annual	No	N/A	N/A	8.89	10.49	23.8	28.1
NewYor	340030003	Annual	No	N/A	N/A	8.90	10.51	24.5	28.9
OgdenC	490110004	24-hr	Yes	-53	0	7.28	7.65	32.6	35.4
OgdenC	490570002	24-hr	No	-53	0	8.99	9.37	-	-
OgdenC	490030003	24-hr	No	-53	0	6.35	6.70	-	-

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Philad	420450002	Annual	Yes	-75	0	11.46	12.04	26.0	27.4
Philad	421010057	Annual	No	-75	0	10.86	11.54	27.0	28.1
Philad	421010055	Annual	No	-75	0	11.43	12.03	27.5	28.8
Philad	421010048	Annual	No	-75	0	10.27	10.91	25.6	27.4
Philad	420290100	Annual	No	-75	0	9.64	10.38	23.9	25.2
Philad	340150004	Annual	No	-75	0	8.33	8.94	20.6	23.2
Philad	340071007	Annual	No	-75	0	8.84	9.51	21.0	21.9
Philad	340070002	Annual	No	-75	0	10.19	10.95	23.5	24.6
Philad	240150003	Annual	No	-75	0	8.70	9.47	22.6	23.7
Philad	100031012	Annual	No	-75	0	9.04	9.81	23.0	23.6
Pittsb	420030064	Annual	Yes	30	0	12.82	12.02	35.8	34.8
Pittsb	421290008	Annual	No	30	0	8.65	8.06	19.6	18.0
Pittsb	421255001	Annual	No	30	0	8.35	7.78	17.8	16.4
Pittsb	421250200	Annual	No	30	0	8.95	8.32	19.3	18.2
Pittsb	421250005	Annual	No	30	0	11.02	10.30	22.7	21.7
Pittsb	420070014	Annual	No	30	0	10.11	9.52	21.9	20.6
Pittsb	420050001	Annual	No	30	0	11.03	10.45	21.9	20.4
Pittsb	420031301	Annual	No	30	0	11.00	10.28	24.8	23.6
Pittsb	420031008	Annual	No	30	0	9.78	9.20	20.5	19.0
Pittsb	420030008	Annual	No	30	0	9.50	8.89	20.5	19.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	8.10	37.6	35.4
ProvoO	490494001	24-hr	Yes	-76	0	7.74	8.29	30.9	35.4
ProvoO	490495010	24-hr	No	-76	0	6.73	7.21	-	-
ProvoO	490490002	24-hr	No	-76	0	7.41	7.95	28.9	33.2
Rivers	060658005	24-hr	Yes	N/A	N/A	14.48	11.87	43.2	35.4
Rivers	060658001	24-hr	No	N/A	N/A	-	-	36.5	29.9
Sacram	060670006	24-hr	Yes	-99	0	9.31	10.04	31.4	35.3
Sacram	061131003	24-hr	No	-99	0	6.62	7.08	15.8	19.0
Sacram	060670012	24-hr	No	-99	0	7.30	7.85	19.8	21.3
Sacram	060670010	24-hr	No	-99	0	8.67	9.30	26.5	30.2
Sacram	060610006	24-hr	No	-99	0	7.58	8.08	20.3	22.2
Sacram	060610003	24-hr	No	-99	0	6.71	7.04	19.3	20.7
SaltLa	490353010	24-hr	Yes	58	0	-	-	41.5	35.4
SaltLa	490353006	24-hr	No	58	0	7.62	6.91	36.8	31.5
SaltLa	490351001	24-hr	No	58	0	7.07	6.30	32.1	25.8
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	12.04	25.9	29.1
SanLui	060798002	Annual	No	N/A	N/A	5.71	6.43	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	9.28	19.8	22.3

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
SouthB	181410015	Annual	Yes	-92	0	10.45	12.04	32.5	34.8
St.Lou	290990019	Annual	Yes	N/A	N/A	10.12	12.04	22.8	27.1
St.Lou	295100094	Annual	No	N/A	N/A	9.57	11.39	23.3	27.7
St.Lou	295100093	Annual	No	N/A	N/A	-	-	23.7	28.2
St.Lou	295100085	Annual	No	N/A	N/A	10.10	12.02	23.6	28.1
St.Lou	295100007	Annual	No	N/A	N/A	9.78	11.64	23.7	28.2
St.Lou	291893001	Annual	No	N/A	N/A	9.85	11.72	22.4	26.6
Stockt	060771002	24-hr	Yes	42	0	12.23	11.41	38.7	35.4
Stockt	060772010	24-hr	No	42	0	10.74	9.96	37.3	34.3
Visali	061072002	24-hr	Yes	N/A	N/A	16.23	10.64	54.0	35.4
Weirto	390810017	Annual	Yes	-14	0	11.75	12.03	27.2	27.5
Weirto	540090011	Annual	No	-14	0	9.75	10.02	22.8	23.6
Weirto	540090005	Annual	No	-14	0	10.52	10.80	22.4	23.1
Weirto	390810021	Annual	No	-14	0	9.29	9.55	22.2	22.8
Wheeli	540511002	Annual	Yes	N/A	N/A	10.24	12.04	22.5	26.5
Wheeli	540690010	Annual	No	N/A	N/A	9.61	11.30	19.7	23.2

^a CBSA names are the first six characters of the full CBSAs names in Table C-3.

^b Percent reduction in NOx and SO₂ emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

^c Percent reduction in Primary PM_{2.5} emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

1
2

1 Table C-21. PM_{2.5} DVs for the Primary PM projection case and 10/30 standard level.

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
AkronO	391530017	Annual	Yes	0	17	10.99	10.03	23.7	22.6
AkronO	391530023	Annual	No	0	17	9.16	8.46	20.2	19.1
Altoon	420130801	Annual	Yes	0	2	10.11	10.02	23.8	23.5
Atlant	131210039	Annual	Yes	0	6	10.38	10.01	19.7	19.0
Atlant	132230003	Annual	No	0	6	7.82	7.64	16.2	15.9
Atlant	131350002	Annual	No	0	6	8.84	8.57	17.9	17.3
Atlant	130890002	Annual	No	0	6	9.34	9.04	19.2	18.7
Atlant	130670003	Annual	No	0	6	9.51	9.22	18.6	18.2
Atlant	130630091	Annual	No	0	6	9.86	9.56	19.1	18.5
Bakers	060290016	Annual	Yes	91	100	18.45	10.01	61.3	29.1
Bakers	060290015	Annual	No	91	100	5.15	3.66	15.8	13.6
Bakers	060290014	Annual	No	91	100	16.53	8.37	61.4	26.0
Bakers	060290011	Annual	No	91	100	6.06	4.58	19.6	15.9
Bakers	060290010	Annual	No	91	100	16.52	8.87	70.0	27.9
Birmin	010732059	Annual	Yes	0	16	11.25	10.03	22.3	19.8
Birmin	010732003	Annual	No	0	16	10.08	9.06	19.0	17.2
Birmin	010731010	Annual	No	0	16	9.78	8.94	19.2	17.7
Birmin	010730023	Annual	No	0	16	10.94	9.77	22.8	20.6
Canton	391510017	Annual	Yes	0	15	10.81	10.01	23.7	22.6
Canton	391510020	Annual	No	0	15	9.91	9.21	22.0	21.0
Chicag	170313103	Annual	Yes	0	18	11.10	10.01	22.6	21.0
Chicag	550590019	Annual	No	0	18	8.04	7.42	20.4	18.8
Chicag	181270024	Annual	No	0	18	9.51	8.55	22.4	20.4
Chicag	180892004	Annual	No	0	18	9.84	8.78	24.7	22.8
Chicag	180890031	Annual	No	0	18	10.12	9.05	23.6	21.1
Chicag	180890026	Annual	No	0	18	-	-	25.2	22.8
Chicag	180890022	Annual	No	0	18	-	-	22.7	20.4
Chicag	180890006	Annual	No	0	18	10.03	8.93	23.1	20.5
Chicag	171971011	Annual	No	0	18	8.36	7.78	18.4	17.4
Chicag	171971002	Annual	No	0	18	7.69	7.04	20.0	18.7
Chicag	170890007	Annual	No	0	18	8.94	8.21	19.2	17.8
Chicag	170890003	Annual	No	0	18	-	-	19.2	18.1
Chicag	170434002	Annual	No	0	18	8.87	8.13	19.9	18.9
Chicag	170316005	Annual	No	0	18	10.79	9.73	24.1	21.7
Chicag	170314201	Annual	No	0	18	9.00	8.25	21.4	19.9
Chicag	170314007	Annual	No	0	18	9.49	8.66	-	-
Chicag	170313301	Annual	No	0	18	10.37	9.38	23.5	21.3

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Chicag	170310076	Annual	No	0	18	10.18	9.24	22.5	20.7
Chicag	170310057	Annual	No	0	18	11.03	9.99	26.8	25.1
Chicag	170310052	Annual	No	0	18	10.00	9.06	23.3	21.4
Chicag	170310022	Annual	No	0	18	10.38	9.28	22.4	20.9
Chicag	170310001	Annual	No	0	18	10.13	9.22	21.7	19.7
Cincin	390610014	Annual	Yes	0	12	10.70	10.04	22.9	21.8
Cincin	390610042	Annual	No	0	12	10.29	9.69	22.6	21.6
Cincin	390610040	Annual	No	0	12	9.45	8.91	21.0	20.0
Cincin	390610010	Annual	No	0	12	9.43	8.93	21.3	20.5
Cincin	390610006	Annual	No	0	12	9.46	8.91	20.3	19.5
Cincin	390170020	Annual	No	0	12	-	-	24.2	23.3
Cincin	390170019	Annual	No	0	12	10.24	9.60	22.0	21.1
Cincin	390170016	Annual	No	0	12	9.79	9.22	22.1	21.2
Cincin	210373002	Annual	No	0	12	9.06	8.58	20.9	20.0
Clevel	390350065	Annual	Yes	0	33	12.17	10.00	24.9	21.3
Clevel	391030004	Annual	No	0	33	8.73	7.57	19.6	17.8
Clevel	390933002	Annual	No	0	33	8.10	6.95	20.2	18.7
Clevel	390850007	Annual	No	0	33	7.88	6.84	17.4	15.4
Clevel	390351002	Annual	No	0	33	8.86	7.64	19.5	17.5
Clevel	390350045	Annual	No	0	33	10.61	8.84	22.9	20.1
Clevel	390350038	Annual	No	0	33	11.38	9.37	25.0	22.0
Clevel	390350034	Annual	No	0	33	8.87	7.58	20.4	18.2
Detroi	261630033	Annual	Yes	0	26	11.30	10.00	26.8	24.9
Detroi	261630039	Annual	No	0	26	9.11	8.21	22.3	20.3
Detroi	261630036	Annual	No	0	26	8.68	7.88	21.8	19.8
Detroi	261630025	Annual	No	0	26	8.98	7.99	24.1	21.7
Detroi	261630019	Annual	No	0	26	9.18	8.18	22.4	19.7
Detroi	261630016	Annual	No	0	26	9.62	8.63	24.4	22.6
Detroi	261630015	Annual	No	0	26	11.19	9.94	25.5	22.8
Detroi	261630001	Annual	No	0	26	9.50	8.39	23.3	20.4
Detroi	261470005	Annual	No	0	26	8.89	8.11	24.3	22.4
Detroi	261250001	Annual	No	0	26	8.86	7.90	24.2	22.2
Detroi	260990009	Annual	No	0	26	8.80	7.94	26.2	23.8
EICent	060250005	Annual	Yes	0	50	12.63	10.01	33.5	25.0
EICent	060251003	Annual	No	0	50	7.44	5.67	19.8	14.6
EICent	060250007	Annual	No	0	50	8.37	6.80	21.5	18.5
Elkhar	180390008	Annual	Yes	0	6	10.24	10.01	28.6	27.8
Evansv	181630023	Annual	Yes	0	2	10.11	10.02	21.5	21.5

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Evansv	211010014	Annual	No	0	2	9.64	9.56	20.7	20.7
Evansv	181630021	Annual	No	0	2	9.84	9.76	21.6	21.5
Evansv	181630016	Annual	No	0	2	10.02	9.94	22.0	21.9
Fresno	060195001	24-hr	Yes	0	100	14.08	9.49	49.3	30.3
Fresno	060195025	24-hr	No	0	100	13.63	8.41	47.9	26.4
Fresno	060192009	24-hr	No	0	100	8.47	6.74	31.3	22.2
Fresno	060190011	24-hr	No	0	100	14.07	8.27	53.8	27.1
Hanfor	060310004	Annual	Yes	82	98	21.98	10.00	72.0	29.5
Hanfor	060311004	Annual	No	82	98	16.49	8.36	58.9	25.2
Housto	482011035	Annual	Yes	0	19	11.19	10.01	22.4	20.2
Housto	482011039	Annual	No	0	19	9.22	8.40	21.7	19.6
Housto	482010058	Annual	No	0	19	9.67	8.70	22.3	20.3
Housto	481671034	Annual	No	0	19	7.36	7.07	20.3	19.6
Indian	180970087	Annual	Yes	0	25	11.44	10.01	25.9	24.2
Indian	180970083	Annual	No	0	25	11.06	9.72	23.9	22.5
Indian	180970081	Annual	No	0	25	11.07	9.71	25.0	23.4
Indian	180970078	Annual	No	0	25	10.14	8.97	24.4	22.8
Indian	180970043	Annual	No	0	25	-	-	26.0	24.6
Indian	180950011	Annual	No	0	25	9.05	8.17	21.8	20.7
Indian	180570007	Annual	No	0	25	9.02	8.07	21.4	20.0
Johnst	420210011	Annual	Yes	0	12	10.68	10.02	25.8	23.5
Lancas	420710012	Annual	Yes	0	41	12.83	9.98	32.7	25.5
Lancas	420710007	Annual	No	0	41	10.57	8.20	29.8	22.0
LasVeg	320030561	Annual	Yes	0	4	10.28	9.97	24.5	23.6
LasVeg	320032002	Annual	No	0	4	9.79	9.50	19.8	19.2
LasVeg	320031019	Annual	No	0	4	5.18	5.08	11.5	11.3
LasVeg	320030540	Annual	No	0	4	8.80	8.55	21.7	20.9
Lebano	420750100	Annual	Yes	0	21	11.20	10.04	31.4	28.0
Little	051191008	Annual	Yes	0	6	10.27	10.00	21.7	21.3
Little	051190007	Annual	No	0	6	9.78	9.48	20.5	20.1
LoganU	490050007	24-hr	Yes	0	19	6.95	6.40	34.0	30.3
LosAng	060371103	Annual	Yes	0	34	12.38	9.99	32.8	27.8
LosAng	060592022	Annual	No	0	34	7.48	6.43	15.3	13.3
LosAng	060590007	Annual	No	0	34	9.63	7.84	-	-
LosAng	060374004	Annual	No	0	34	10.25	8.36	27.3	23.7
LosAng	060374002	Annual	No	0	34	11.06	9.02	29.2	24.9
LosAng	060371602	Annual	No	0	34	11.86	9.55	32.3	26.5
LosAng	060371302	Annual	No	0	34	11.99	9.64	31.5	27.0

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
LosAng	060371201	Annual	No	0	34	9.46	7.93	25.6	21.6
LosAng	060370002	Annual	No	0	34	10.52	8.81	29.2	25.0
Louisv	180190006	Annual	Yes	0	12	10.64	10.01	23.9	22.8
Louisv	211110075	Annual	No	0	12	10.42	9.79	22.3	21.4
Louisv	211110067	Annual	No	0	12	9.55	8.99	21.4	20.5
Louisv	211110051	Annual	No	0	12	10.29	9.76	21.8	21.2
Louisv	211110043	Annual	No	0	12	10.37	9.77	22.0	21.2
Louisv	180431004	Annual	No	0	12	9.96	9.41	22.0	21.0
Louisv	180190008	Annual	No	0	12	8.72	8.29	20.1	19.5
MaconG	130210007	Annual	Yes	0	2	10.13	10.03	21.2	21.0
MaconG	130210012	Annual	No	0	2	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	0	84	13.30	9.89	45.1	30.4
McAlle	482150043	Annual	Yes	0	2	10.09	10.03	25.0	24.9
Merced	060470003	24-hr	Yes	0	65	11.81	9.87	39.0	30.4
Merced	060472510	24-hr	No	0	65	11.68	9.11	39.8	28.8
Modest	060990006	24-hr	Yes	0	77	13.02	9.52	45.7	30.3
Modest	060990005	24-hr	No	0	77	-	-	38.8	29.2
NapaCA	060550003	Annual	Yes	0	9	10.36	10.04	25.1	24.6
NewYor	360610128	Annual	Yes	0	3	10.20	9.99	23.9	23.5
NewYor	361030002	Annual	No	0	3	7.18	7.07	18.8	18.6
NewYor	360810124	Annual	No	0	3	7.52	7.39	19.5	19.1
NewYor	360710002	Annual	No	0	3	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	0	3	9.70	9.51	21.6	21.2
NewYor	360610079	Annual	No	0	3	8.42	8.26	22.8	22.5
NewYor	360470122	Annual	No	0	3	8.66	8.49	20.5	20.2
NewYor	360050133	Annual	No	0	3	9.05	8.87	24.0	23.6
NewYor	360050110	Annual	No	0	3	7.39	7.25	19.4	19.1
NewYor	340392003	Annual	No	0	3	8.59	8.44	23.6	23.2
NewYor	340390004	Annual	No	0	3	9.87	9.69	24.2	23.8
NewYor	340310005	Annual	No	0	3	8.42	8.28	22.2	21.9
NewYor	340292002	Annual	No	0	3	7.23	7.13	18.1	17.9
NewYor	340273001	Annual	No	0	3	6.78	6.69	17.1	16.9
NewYor	340171003	Annual	No	0	3	8.79	8.64	23.4	22.9
NewYor	340130003	Annual	No	0	3	8.89	8.73	23.8	23.4
NewYor	340030003	Annual	No	0	3	8.90	8.75	24.5	24.1
OgdenC	490110004	24-hr	Yes	0	15	7.28	6.89	32.6	30.3
OgdenC	490570002	24-hr	No	0	15	8.99	8.39	-	-
OgdenC	490030003	24-hr	No	0	15	6.35	6.02	-	-

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Philad	420450002	Annual	Yes	0	20	11.46	9.99	26.0	22.9
Philad	421010057	Annual	No	0	20	10.86	9.56	27.0	23.4
Philad	421010055	Annual	No	0	20	11.43	9.94	27.5	24.2
Philad	421010048	Annual	No	0	20	10.27	9.00	25.6	22.7
Philad	420290100	Annual	No	0	20	9.64	8.66	23.9	21.2
Philad	340150004	Annual	No	0	20	8.33	7.43	20.6	18.2
Philad	340071007	Annual	No	0	20	8.84	7.86	21.0	18.8
Philad	340070002	Annual	No	0	20	10.19	9.11	23.5	20.6
Philad	240150003	Annual	No	0	20	8.70	7.90	22.6	20.5
Philad	100031012	Annual	No	0	20	9.04	8.15	23.0	21.1
Pittsb	420030064	Annual	Yes	0	44	12.82	10.04	35.8	26.2
Pittsb	421290008	Annual	No	0	44	8.65	6.96	19.6	16.9
Pittsb	421255001	Annual	No	0	44	8.35	6.78	17.8	15.7
Pittsb	421250200	Annual	No	0	44	8.95	7.22	19.3	15.7
Pittsb	421250005	Annual	No	0	44	11.02	8.85	22.7	18.0
Pittsb	420070014	Annual	No	0	44	10.11	7.98	21.9	17.5
Pittsb	420050001	Annual	No	0	44	11.03	8.58	21.9	17.8
Pittsb	420031301	Annual	No	0	44	11.00	8.64	24.8	18.7
Pittsb	420031008	Annual	No	0	44	9.78	7.68	20.5	16.1
Pittsb	420030008	Annual	No	0	44	9.50	7.30	20.5	16.3
Prinev	410130100	24-hr	Yes	0	33	8.60	7.19	37.6	30.4
ProvoO	490494001	24-hr	Yes	0	3	7.74	7.65	30.9	30.4
ProvoO	490495010	24-hr	No	0	3	6.73	6.65	-	-
ProvoO	490490002	24-hr	No	0	3	7.41	7.32	28.9	28.4
Rivers	060658005	24-hr	Yes	0	58	14.48	9.69	43.2	30.4
Rivers	060658001	24-hr	No	0	58	-	-	36.5	25.4
Sacram	060670006	24-hr	Yes	0	6	9.31	9.02	31.4	30.4
Sacram	061131003	24-hr	No	0	6	6.62	6.47	15.8	15.4
Sacram	060670012	24-hr	No	0	6	7.30	7.11	19.8	19.4
Sacram	060670010	24-hr	No	0	6	8.67	8.41	26.5	25.7
Sacram	060610006	24-hr	No	0	6	7.58	7.34	20.3	19.9
Sacram	060610003	24-hr	No	0	6	6.71	6.56	19.3	19.0
SaltLa	490353010	24-hr	Yes	0	85	-	-	41.5	30.4
SaltLa	490353006	24-hr	No	0	85	7.62	4.85	36.8	23.8
SaltLa	490351001	24-hr	No	0	85	7.07	4.72	32.1	21.0
SanLui	060792007	Annual	Yes	0	22	10.70	10.04	25.9	24.9
SanLui	060798002	Annual	No	0	22	5.71	5.42	-	-
SanLui	060792004	Annual	No	0	22	8.25	7.76	19.8	19.2

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
SouthB	181410015	24-hr	Yes	0	18	10.45	9.72	32.5	30.3
St.Lou	290990019	Annual	Yes	0	2	10.12	10.02	22.8	22.7
St.Lou	295100094	Annual	No	0	2	9.57	9.48	23.3	23.2
St.Lou	295100093	Annual	No	0	2	-	-	23.7	23.5
St.Lou	295100085	Annual	No	0	2	10.10	10.00	23.6	23.4
St.Lou	295100007	Annual	No	0	2	9.78	9.69	23.7	23.6
St.Lou	291893001	Annual	No	0	2	9.85	9.76	22.4	22.3
Stockt	060771002	24-hr	Yes	0	43	12.23	9.86	38.7	30.3
Stockt	060772010	24-hr	No	0	43	10.74	8.75	37.3	29.6
Visali	061072002	24-hr	Yes	58	74	16.23	9.67	54.0	30.4
Weirto	390810017	Annual	Yes	0	33	11.75	10.00	27.2	22.6
Weirto	540090011	Annual	No	0	33	9.75	8.42	22.8	19.8
Weirto	540090005	Annual	No	0	33	10.52	9.07	22.4	19.8
Weirto	390810021	Annual	No	0	33	9.29	8.06	22.2	19.3
Wheeli	540511002	Annual	Yes	0	5	10.24	10.03	22.5	22.1
Wheeli	540690010	Annual	No	0	5	9.61	9.42	19.7	19.4

^a CBSA names are the first six characters of the full CBSAs names in Table C-3.

^b Percent reduction in NOx and SO₂ emissions associated with just meeting the standard in this case.

^c Percent reduction in Primary PM_{2.5} emissions associated with just meeting the standard in this case.

1 Table C-22. PM_{2.5} DVs for the Secondary PM projection case and 10/30 standard level.

CBSA ^a	Site	Controlling Standard	Controlling Site?	NO _x & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
AkronO	391530017	Annual	Yes	45	0	10.99	10.04	23.7	20.8
AkronO	391530023	Annual	No	45	0	9.16	8.24	20.2	17.7
Altoon	420130801	Annual	Yes	N/A	N/A	10.11	10.04	23.8	23.6
Atlant	131210039	Annual	Yes	N/A	N/A	10.38	10.04	19.7	19.1
Atlant	132230003	Annual	No	N/A	N/A	7.82	7.56	16.2	15.7
Atlant	131350002	Annual	No	N/A	N/A	8.84	8.55	17.9	17.3
Atlant	130890002	Annual	No	N/A	N/A	9.34	9.03	19.2	18.6
Atlant	130670003	Annual	No	N/A	N/A	9.51	9.20	18.6	18.0
Atlant	130630091	Annual	No	N/A	N/A	9.86	9.54	19.1	18.5
Bakers	060290010	24-hr	Yes	N/A	N/A	16.52	8.99	70.0	30.4
Bakers	060290016	24-hr	No	N/A	N/A	18.45	10.04	61.3	26.6
Bakers	060290015	24-hr	No	N/A	N/A	5.15	2.80	15.8	6.9
Bakers	060290014	24-hr	No	N/A	N/A	16.53	9.00	61.4	26.7
Bakers	060290011	24-hr	No	N/A	N/A	6.06	3.30	19.6	8.5
Birmin	010732059	Annual	Yes	71	0	11.25	10.04	22.3	20.2
Birmin	010732003	Annual	No	71	0	10.08	8.86	19.0	16.1
Birmin	010731010	Annual	No	71	0	9.78	8.39	19.2	16.6
Birmin	010730023	Annual	No	71	0	10.94	9.72	22.8	20.3
Canton	391510017	Annual	Yes	36	0	10.81	10.04	23.7	21.7
Canton	391510020	Annual	No	36	0	9.91	9.13	22.0	19.4
Chicag	170313103	Annual	Yes	N/A	N/A	11.10	10.04	22.6	20.4
Chicag	550590019	Annual	No	N/A	N/A	8.04	7.27	20.4	18.5
Chicag	181270024	Annual	No	N/A	N/A	9.51	8.60	22.4	20.3
Chicag	180892004	Annual	No	N/A	N/A	9.84	8.90	24.7	22.3
Chicag	180890031	Annual	No	N/A	N/A	10.12	9.15	23.6	21.3
Chicag	180890026	Annual	No	N/A	N/A	-	-	25.2	22.8
Chicag	180890022	Annual	No	N/A	N/A	-	-	22.7	20.5
Chicag	180890006	Annual	No	N/A	N/A	10.03	9.07	23.1	20.9
Chicag	171971011	Annual	No	N/A	N/A	8.36	7.56	18.4	16.6
Chicag	171971002	Annual	No	N/A	N/A	7.69	6.96	20.0	18.1
Chicag	170890007	Annual	No	N/A	N/A	8.94	8.09	19.2	17.4
Chicag	170890003	Annual	No	N/A	N/A	-	-	19.2	17.4
Chicag	170434002	Annual	No	N/A	N/A	8.87	8.02	19.9	18.0
Chicag	170316005	Annual	No	N/A	N/A	10.79	9.76	24.1	21.8
Chicag	170314201	Annual	No	N/A	N/A	9.00	8.14	21.4	19.4
Chicag	170314007	Annual	No	N/A	N/A	9.49	8.58	-	-
Chicag	170313301	Annual	No	N/A	N/A	10.37	9.38	23.5	21.3

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Chicag	170310076	Annual	No	N/A	N/A	10.18	9.21	22.5	20.4
Chicag	170310057	Annual	No	N/A	N/A	11.03	9.98	26.8	24.2
Chicag	170310052	Annual	No	N/A	N/A	10.00	9.05	23.3	21.1
Chicag	170310022	Annual	No	N/A	N/A	10.38	9.39	22.4	20.3
Chicag	170310001	Annual	No	N/A	N/A	10.13	9.16	21.7	19.6
Cincin	390610014	Annual	Yes	28	0	10.70	10.03	22.9	21.2
Cincin	390610042	Annual	No	28	0	10.29	9.61	22.6	20.8
Cincin	390610040	Annual	No	28	0	9.45	8.78	21.0	19.0
Cincin	390610010	Annual	No	28	0	9.43	8.78	21.3	19.6
Cincin	390610006	Annual	No	28	0	9.46	8.82	20.3	18.4
Cincin	390170020	Annual	No	28	0	-	-	24.2	22.5
Cincin	390170019	Annual	No	28	0	10.24	9.66	22.0	20.6
Cincin	390170016	Annual	No	28	0	9.79	9.16	22.1	20.1
Cincin	210373002	Annual	No	28	0	9.06	8.38	20.9	18.9
Clevel	390350065	Annual	Yes	79	0	12.17	10.04	24.9	20.5
Clevel	391030004	Annual	No	79	0	8.73	6.75	19.6	13.9
Clevel	390933002	Annual	No	79	0	8.10	6.28	20.2	13.8
Clevel	390850007	Annual	No	79	0	7.88	6.10	17.4	12.9
Clevel	390351002	Annual	No	79	0	8.86	6.81	19.5	14.4
Clevel	390350045	Annual	No	79	0	10.61	8.50	22.9	17.0
Clevel	390350038	Annual	No	79	0	11.38	9.33	25.0	19.7
Clevel	390350034	Annual	No	79	0	8.87	6.90	20.4	15.4
Detroi	261630033	Annual	Yes	60	0	11.30	10.03	26.8	24.3
Detroi	261630039	Annual	No	60	0	9.11	7.82	22.3	18.8
Detroi	261630036	Annual	No	60	0	8.68	7.43	21.8	19.1
Detroi	261630025	Annual	No	60	0	8.98	7.63	24.1	19.1
Detroi	261630019	Annual	No	60	0	9.18	7.83	22.4	20.3
Detroi	261630016	Annual	No	60	0	9.62	8.33	24.4	21.3
Detroi	261630015	Annual	No	60	0	11.19	9.88	25.5	22.0
Detroi	261630001	Annual	No	60	0	9.50	8.26	23.3	20.1
Detroi	261470005	Annual	No	60	0	8.89	7.81	24.3	20.6
Detroi	261250001	Annual	No	60	0	8.86	7.49	24.2	20.5
Detroi	260990009	Annual	No	60	0	8.80	7.57	26.2	21.8
EICent	060250005	Annual	Yes	N/A	N/A	12.63	10.04	33.5	26.6
EICent	060251003	Annual	No	N/A	N/A	7.44	5.91	19.8	15.7
EICent	060250007	Annual	No	N/A	N/A	8.37	6.65	21.5	17.1
Elkhar	180390008	Annual	Yes	N/A	N/A	10.24	10.04	28.6	28.0
Evansv	181630023	Annual	Yes	3	0	10.11	10.03	21.5	21.2

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Evansv	211010014	Annual	No	3	0	9.64	9.56	20.7	20.3
Evansv	181630021	Annual	No	3	0	9.84	9.76	21.6	21.2
Evansv	181630016	Annual	No	3	0	10.02	9.95	22.0	21.7
Fresno	060190011	24-hr	Yes	N/A	N/A	14.07	9.48	53.8	30.4
Fresno	060195025	24-hr	No	N/A	N/A	13.63	9.18	47.9	27.1
Fresno	060195001	24-hr	No	N/A	N/A	14.08	9.49	49.3	27.9
Fresno	060192009	24-hr	No	N/A	N/A	8.47	5.71	31.3	17.7
Hanford	060310004	24-hr	Yes	N/A	N/A	21.98	9.28	72.0	30.4
Hanford	060311004	24-hr	No	N/A	N/A	16.49	6.96	58.9	24.9
Housto	482011035	Annual	Yes	84	0	11.19	10.04	22.4	19.6
Housto	482011039	Annual	No	84	0	9.22	8.09	21.7	18.7
Housto	482010058	Annual	No	84	0	9.67	8.57	22.3	19.1
Housto	481671034	Annual	No	84	0	7.36	6.29	20.3	17.8
Indian	180970087	Annual	Yes	48	0	11.44	10.03	25.9	21.8
Indian	180970083	Annual	No	48	0	11.06	9.64	23.9	21.4
Indian	180970081	Annual	No	48	0	11.07	9.66	25.0	20.8
Indian	180970078	Annual	No	48	0	10.14	8.73	24.4	19.9
Indian	180970043	Annual	No	48	0	-	-	26.0	20.9
Indian	180950011	Annual	No	48	0	9.05	7.86	21.8	18.3
Indian	180570007	Annual	No	48	0	9.02	7.75	21.4	17.8
Johnst	420210011	Annual	Yes	31	0	10.68	10.04	25.8	25.1
Lancas	420710012	Annual	Yes	98	0	12.83	10.01	32.7	26.2
Lancas	420710007	Annual	No	98	0	10.57	7.81	29.8	23.4
LasVeg	320030561	Annual	Yes	N/A	N/A	10.28	10.04	24.5	23.9
LasVeg	320032002	Annual	No	N/A	N/A	9.79	9.56	19.8	19.3
LasVeg	320031019	Annual	No	N/A	N/A	5.18	5.06	11.5	11.2
LasVeg	320030540	Annual	No	N/A	N/A	8.80	8.59	21.7	21.2
Lebano	420750100	Annual	Yes	53	0	11.20	10.03	31.4	28.6
Little	051191008	Annual	Yes	11	0	10.27	10.04	21.7	21.1
Little	051190007	Annual	No	11	0	9.78	9.57	20.5	19.9
LoganU	490050007	24-hr	Yes	56	0	6.95	6.51	34.0	30.4
LosAng	060371103	Annual	Yes	N/A	N/A	12.38	10.04	32.8	26.6
LosAng	060592022	Annual	No	N/A	N/A	7.48	6.07	15.3	12.4
LosAng	060590007	Annual	No	N/A	N/A	9.63	7.81	-	-
LosAng	060374004	Annual	No	N/A	N/A	10.25	8.31	27.3	22.1
LosAng	060374002	Annual	No	N/A	N/A	11.06	8.97	29.2	23.7
LosAng	060371602	Annual	No	N/A	N/A	11.86	9.62	32.3	26.2
LosAng	060371302	Annual	No	N/A	N/A	11.99	9.72	31.5	25.5

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
LosAng	060371201	Annual	No	N/A	N/A	9.46	7.67	25.6	20.8
LosAng	060370002	Annual	No	N/A	N/A	10.52	8.53	29.2	23.7
Louisv	180190006	Annual	Yes	24	0	10.64	10.02	23.9	22.0
Louisv	211110075	Annual	No	24	0	10.42	9.83	22.3	20.3
Louisv	211110067	Annual	No	24	0	9.55	8.96	21.4	19.9
Louisv	211110051	Annual	No	24	0	10.29	9.68	21.8	20.2
Louisv	211110043	Annual	No	24	0	10.37	9.77	22.0	20.2
Louisv	180431004	Annual	No	24	0	9.96	9.37	22.0	20.4
Louisv	180190008	Annual	No	24	0	8.72	8.13	20.1	18.3
MaconG	130210007	Annual	Yes	N/A	N/A	10.13	10.04	21.2	21.0
MaconG	130210012	Annual	No	N/A	N/A	7.68	7.61	16.6	16.5
Madera	060392010	24-hr	Yes	N/A	N/A	13.30	10.04	45.1	30.4
McAlle	482150043	Annual	Yes	N/A	N/A	10.09	10.04	25.0	24.9
Merced	060472510	24-hr	Yes	68	0	11.68	9.74	39.8	30.4
Merced	060470003	24-hr	No	68	0	11.81	9.82	39.0	29.8
Modest	060990006	24-hr	Yes	N/A	N/A	13.02	9.75	45.7	30.4
Modest	060990005	24-hr	No	N/A	N/A	-	-	38.8	25.8
NapaCA	060550003	Annual	Yes	N/A	N/A	10.36	10.04	25.1	24.3
NewYor	360610128	Annual	Yes	N/A	N/A	10.20	10.04	23.9	23.5
NewYor	361030002	Annual	No	N/A	N/A	7.18	7.07	18.8	18.5
NewYor	360810124	Annual	No	N/A	N/A	7.52	7.40	19.5	19.2
NewYor	360710002	Annual	No	N/A	N/A	6.95	6.84	17.5	17.2
NewYor	360610134	Annual	No	N/A	N/A	9.70	9.55	21.6	21.3
NewYor	360610079	Annual	No	N/A	N/A	8.42	8.29	22.8	22.4
NewYor	360470122	Annual	No	N/A	N/A	8.66	8.52	20.5	20.2
NewYor	360050133	Annual	No	N/A	N/A	9.05	8.91	24.0	23.6
NewYor	360050110	Annual	No	N/A	N/A	7.39	7.27	19.4	19.1
NewYor	340392003	Annual	No	N/A	N/A	8.59	8.46	23.6	23.2
NewYor	340390004	Annual	No	N/A	N/A	9.87	9.72	24.2	23.8
NewYor	340310005	Annual	No	N/A	N/A	8.42	8.29	22.2	21.9
NewYor	340292002	Annual	No	N/A	N/A	7.23	7.12	18.1	17.8
NewYor	340273001	Annual	No	N/A	N/A	6.78	6.67	17.1	16.8
NewYor	340171003	Annual	No	N/A	N/A	8.79	8.65	23.4	23.0
NewYor	340130003	Annual	No	N/A	N/A	8.89	8.75	23.8	23.4
NewYor	340030003	Annual	No	N/A	N/A	8.90	8.76	24.5	24.1
OgdenC	490110004	24-hr	Yes	29	0	7.28	7.01	32.6	30.4
OgdenC	490570002	24-hr	No	29	0	8.99	8.71	-	-
OgdenC	490030003	24-hr	No	29	0	6.35	6.10	-	-

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
Philad	420450002	Annual	Yes	86	0	11.46	10.04	26.0	22.3
Philad	421010057	Annual	No	86	0	10.86	9.12	27.0	22.5
Philad	421010055	Annual	No	86	0	11.43	9.95	27.5	23.9
Philad	421010048	Annual	No	86	0	10.27	8.70	25.6	21.1
Philad	420290100	Annual	No	86	0	9.64	7.87	23.9	19.5
Philad	340150004	Annual	No	86	0	8.33	6.99	20.6	16.9
Philad	340071007	Annual	No	86	0	8.84	7.23	21.0	17.1
Philad	340070002	Annual	No	86	0	10.19	8.40	23.5	20.2
Philad	240150003	Annual	No	86	0	8.70	6.90	22.6	17.5
Philad	100031012	Annual	No	86	0	9.04	7.21	23.0	17.7
Pittsb	420030064	24-hr	Yes	100	0	12.82	9.22	35.8	30.4
Pittsb	421290008	24-hr	No	100	0	8.65	6.04	19.6	12.9
Pittsb	421255001	24-hr	No	100	0	8.35	5.90	17.8	11.1
Pittsb	421250200	24-hr	No	100	0	8.95	6.10	19.3	13.7
Pittsb	421250005	24-hr	No	100	0	11.02	7.78	22.7	18.1
Pittsb	420070014	24-hr	No	100	0	10.11	7.38	21.9	15.2
Pittsb	420050001	24-hr	No	100	0	11.03	8.39	21.9	15.5
Pittsb	420031301	24-hr	No	100	0	11.00	7.79	24.8	19.7
Pittsb	420031008	24-hr	No	100	0	9.78	7.11	20.5	14.7
Pittsb	420030008	24-hr	No	100	0	9.50	6.81	20.5	14.2
Prinev	410130100	24-hr	Yes	N/A	N/A	8.60	6.95	37.6	30.4
ProvoO	490494001	24-hr	Yes	6	0	7.74	7.68	30.9	30.4
ProvoO	490495010	24-hr	No	6	0	6.73	6.68	-	-
ProvoO	490490002	24-hr	No	6	0	7.41	7.36	28.9	28.4
Rivers	060658005	Annual	Yes	N/A	N/A	14.48	10.04	43.2	30.0
Rivers	060658001	Annual	No	N/A	N/A	-	-	36.5	25.3
Sacram	060670006	24-hr	Yes	18	0	9.31	9.11	31.4	30.4
Sacram	061131003	24-hr	No	18	0	6.62	6.50	15.8	15.1
Sacram	060670012	24-hr	No	18	0	7.30	7.17	19.8	19.3
Sacram	060670010	24-hr	No	18	0	8.67	8.50	26.5	25.5
Sacram	060610006	24-hr	No	18	0	7.58	7.45	20.3	19.9
Sacram	060610003	24-hr	No	18	0	6.71	6.63	19.3	18.9
SaltLa	490353010	24-hr	Yes	79	0	-	-	41.5	30.3
SaltLa	490353006	24-hr	No	79	0	7.62	6.46	36.8	29.3
SaltLa	490351001	24-hr	No	79	0	7.07	5.88	32.1	23.2
SanLui	060792007	Annual	Yes	N/A	N/A	10.70	10.04	25.9	24.3
SanLui	060798002	Annual	No	N/A	N/A	5.71	5.36	-	-
SanLui	060792004	Annual	No	N/A	N/A	8.25	7.74	19.8	18.6

CBSA ^a	Site	Controlling Standard	Controlling Site?	NOx & SO ₂ Reduction (%) ^b	Primary PM _{2.5} Reduction (%) ^c	Base Annual DV (µg m ⁻³)	Projected Annual DV (µg m ⁻³)	Base 24-hr DV (µg m ⁻³)	Projected 24-hr DV (µg m ⁻³)
SouthB	181410015	24-hr	Yes	30	0	10.45	9.68	32.5	30.4
St.Lou	290990019	Annual	Yes	N/A	N/A	10.12	10.04	22.8	22.6
St.Lou	295100094	Annual	No	N/A	N/A	9.57	9.49	23.3	23.1
St.Lou	295100093	Annual	No	N/A	N/A	-	-	23.7	23.5
St.Lou	295100085	Annual	No	N/A	N/A	10.10	10.02	23.6	23.4
St.Lou	295100007	Annual	No	N/A	N/A	9.78	9.70	23.7	23.5
St.Lou	291893001	Annual	No	N/A	N/A	9.85	9.77	22.4	22.2
Stockt	060771002	Annual	Yes	97	0	12.23	10.04	38.7	29.7
Stockt	060772010	Annual	No	97	0	10.74	8.69	37.3	28.4
Visali	061072002	24-hr	Yes	N/A	N/A	16.23	9.14	54.0	30.4
Weirto	390810017	Annual	Yes	62	0	11.75	10.02	27.2	23.8
Weirto	540090011	Annual	No	62	0	9.75	8.14	22.8	19.9
Weirto	540090005	Annual	No	62	0	10.52	8.82	22.4	18.8
Weirto	390810021	Annual	No	62	0	9.29	7.68	22.2	18.5
Wheeli	540511002	Annual	Yes	N/A	N/A	10.24	10.04	22.5	22.1
Wheeli	540690010	Annual	No	N/A	N/A	9.61	9.42	19.7	19.3

^a CBSA names are the first six characters of the full CBSAs names in Table C-3.

^b Percent reduction in NOx and SO₂ emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

^c Percent reduction in Primary PM_{2.5} emissions associated with just meeting the standard in this case; N/A indicates 'not applicable' where proportional projection was used.

1 REFERENCES

- 2 Abt Associates, Inc. (2014). User's guide: modeled attainment test software. U.S. Environmental
3 Protection Agency. Research Triangle Park, NC. Available:
4 https://www3.epa.gov/ttn/scram/guidance/guide/MATS_2-6-1_manual.pdf.
- 5 Appel, KW, Napelenok, S., Hogrefe, C., Pouliot, G., Foley, K.M., Roselle, S.J., Pleim, J.E.,
6 Bash, J., Pye, H.O.T., Heath, N., Murphy, B., Mathur, R. (2018). Overview and
7 evaluation of the Community Multiscale Air Quality (CMAQ) Modeling System Version
8 5.2. *Air Pollution Modeling and Its Application XXV*. C. Mensink, Kallos, G. Springer
9 International Publishing, Cham. Switzerland: 69-73.
- 10 Awad, YA, Di, Q, Wang, Y, Choirat, C, Coull, BA, Zanobetti, A and Schwartz, J (2019).
11 Change in PM_{2.5} exposure and mortality among Medicare recipients: Combining a
12 semi-randomized approach and inverse probability weights in a low exposure population.
13 *Environmental Epidemiology (Philadelphia, Pa.)* 3(4).
- 14 Baxter, LK, Crooks, JL and Sacks, JD (2017). Influence of exposure differences on city-to-city
15 heterogeneity in PM_{2.5}-mortality associations in US cities. *Environmental Health: A*
16 *Global Access Science Source* 16(1): 1.
- 17 Berrocal, VJ, Gelfand, AE and Holland, DM (2012). Space-time data fusion under error in
18 computer model output: an application to modeling air quality. *Biometrics* 68(3): 837-
19 848.
- 20 Chen, H, Burnett, RT, Copes, R, Kwong, JC, Villeneuve, PJ, Goldberg, MS, Brook, RD, van
21 Donkelaar, A, Jerrett, M, Martin, RV, Brook, JR, Kopp, A and Tu, JV (2016). Ambient
22 fine particulate matter and mortality among survivors of myocardial infarction:
23 population-based cohort study. *Environmental Health Perspectives* 124(9): 1421-1428.
- 24 Di, Q, Wang, Y, Zanobetti, A, Wang, Y, Koutrakis, P, Choirat, C, Dominici, F and Schwartz, JD
25 (2017). Air pollution and mortality in the Medicare population. *New England Journal of*
26 *Medicine* 376(26): 2513-2522.
- 27 Frank, NH (2006). Retained nitrate, hydrated sulfates, and carbonaceous mass in federal
28 reference method fine particulate matter for six eastern US cities. *Journal of the Air*
29 *Waste Management Association* 56(4): 500-511.
- 30 Hart, JE, Liao, X, Hong, B, Puett, RC, Yanosky, JD, Suh, H, Kioumourtzoglou, MA,
31 Spiegelman, D and Laden, F (2015). The association of long-term exposure to PM_{2.5} on
32 all-cause mortality in the Nurses' Health Study and the impact of measurement-error
33 correction. *Environmental Health: A Global Access Science Source* 14: 38.
- 34 Ito, K, Ross, Z, Zhou, J, Nádas, A, Lippmann, M and Thurston, GD (2013). National Particle
35 Component Toxicity (NPACT) initiative: Study 3. Time-series analysis of mortality,
36 hospitalizations, and ambient PM_{2.5} and its components. 177. Health Effects Institute.
37 Boston, MA. [https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-](https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-AppendixG_0.pdf)
38 [AppendixG_0.pdf](https://www.healtheffects.org/system/files/RR177-Lippmann-Study3-AppendixG_0.pdf).

- 1 Jerrett, M, Burnett, RT, Beckerman, BS, Turner, MC, Krewski, D, Thurston, G, Martin, RV, van
2 Donkelaar, A, Hughes, E, Shi, Y, Gapstur, SM, Thun, MJ and Pope, CA, 3rd (2013).
3 Spatial analysis of air pollution and mortality in California. *Am J Respir Crit Care Med*
4 188(5): 593-599.
- 5 Jerrett, M, Turner, MC, Beckerman, BS, Pope, CA, van Donkelaar, A, Martin, RV, Serre, M,
6 Crouse, D, Gapstur, SM, Krewski, D, Diver, WR, Coogan, PF, Thurston, GD and
7 Burnett, RT (2016). Comparing the health effects of ambient particulate matter estimated
8 using ground-based versus remote sensing exposure estimates. *Environmental Health*
9 *Perspectives* 125(4): 552-559.
- 10 Kelly, JT, Jang, CJ, Timin, B, Gantt, B, Reff, A, Zhu, Y, Long, S and Hanna, A (2019a). A
11 system for developing and projecting PM_{2.5}. 5 spatial fields to correspond to just meeting
12 national ambient air quality standards. *Atmospheric Environment* 2: 100019.
- 13 Kelly, JT, Koplitz, SN, Baker, KR, Holder, AL, Pye, HOT, Murphy, BN, Bash, JO, Henderson,
14 BH, Possiel, NC, Simon, H, Eyth, AM, Jang, CJ, Phillips, S and Timin, B (2019b).
15 Assessing PM_{2.5} model performance for the conterminous U.S. with comparison to model
16 performance statistics from 2007-2015. *Atmospheric Environment* 214: 116872.
- 17 Kioumourtzoglou, MA, Schwartz, J, James, P, Dominici, F and Zanobetti, A (2016). PM_{2.5} and
18 mortality in 207 us cities: Modification by temperature and city characteristics.
19 *Epidemiology* 27(2): 221-227.
- 20 Kloog, I, Ridgway, B, Koutrakis, P, Coull, BA and Schwartz, JD (2013). Long- and short-term
21 exposure to PM_{2.5} and mortality: using novel exposure models. *Epidemiology* 24(4):
22 555-561.
- 23 Lipfert, FW and Wyzga, RE (2020). Environmental predictors of survival in a cohort of US
24 military veterans: A multi-level spatio-temporal analysis stratified by race.
25 *Environmental research* 183: 108842.
- 26 National Resources Council (2004). *Air Quality Management in the United States*. The National
27 Academies Press. Washington, DC. NRC.
- 28 Parker, JD, Kravets, N and Vaidyanathan, A (2018). Particulate matter air pollution exposure and
29 heart disease mortality risks by race and ethnicity in the United States: 1997 to 2009
30 National Health Interview Survey with mortality follow-up through 2011. *Circulation*
31 137(16): 1688-1697.
- 32 Pope, CA, Turner, MC, Burnett, R, Jerrett, M, Gapstur, SM, Diver, WR, Krewski, D and Brook,
33 RD (2015). Relationships between fine particulate air pollution, cardiometabolic
34 disorders and cardiovascular mortality. *Circulation Research* 116(1): 108-U258.
- 35 Skamarock, WC, Klemp, JB, Dudhia, J, Gill, DO, Barker, DM, Wang, W and Powers, JG
36 (2005). A description of the advanced research WRF version 2. National Center For
37 Atmospheric Research Boulder Co Mesoscale and Microscale

- 1 Son, J-Y, Lane, KJ, Miranda, ML and Bell, ML (2020). Health disparities attributable to air
2 pollutant exposure in North Carolina: Influence of residential environmental and social
3 factors. *Health & place* 62: 102287.
- 4 Sutradhar, R and Austin, PC (2018). Relative rates not relative risks: addressing a widespread
5 misinterpretation of hazard ratios. *Annals of epidemiology* 28(1): 54-57.
- 6 Turner, MC, Jerrett, M, Pope, A, III, Krewski, D, Gapstur, SM, Diver, WR, Beckerman, BS,
7 Marshall, JD, Su, J, Crouse, DL and Burnett, RT (2016). Long-term ozone exposure and
8 mortality in a large prospective study. *American Journal of Respiratory and Critical Care*
9 *Medicine* 193(10): 1134-1142.
- 10 U.S. EPA (2001). Risk Assessment Guidance for Superfund: Volume III - Part A, Process for
11 Conducting Probabilistic Risk Assessment. Office of Emergency and Remedial
12 Response. Washington, D.C. U.S. EPA. EPA 540-R-02-002. Available at :
13 https://www.epa.gov/sites/production/files/2015-09/documents/rags3adt_complete.pdf.
- 14 U.S. EPA (2004). An Examination of EPA Risk Assessment Principles and Practices. Office of
15 Research and Development. Washington, D.C. U.S. EPA. EPA/100/B-04/001. Available
16 at: <https://semspub.epa.gov/work/10/500006305.pdf>.
- 17 U.S. EPA (2010). Quantitative Health Risk Assessment for Particulate Matter (Final Report).
18 Office of Air Quality Planning and Standards, Health and Environmental Impacts
19 Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-10-005. June 2010.
20 Available at: <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P1007RFC.txt>.
- 21 U.S. EPA (2015). Preamble to the Integrated Science Assessments. U.S. Environmental
22 Protection Agency, Office of Research and Development, National Center for
23 Environmental Assessment, RTP Division. Research Triangle Park, NC. U.S. EPA.
24 EPA/600/R-15/067. November 2015. Available at:
25 <https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=310244>.
- 26 U.S. EPA (2018a). Bayesian space-time downscaling fusion model (downscaler) -Derived
27 Estimates of Air Quality for 2014. Office of Air Quality Planning and Standards.
28 Research Triangle Park, NC. U.S. EPA. EPA-454/R-18-008. Available at:
29 <https://www.epa.gov/hesc/rsig-related-downloadable-data-files>.
- 30 U.S. EPA (2018b). Modeling Guidance for Demonstrating Attainment of Air Quality Goals for
31 Ozone, PM_{2.5}, and Regional Haze. Office of Air Quality Planning and Standards.
32 Research Triangle Park, NC. U.S. EPA. EPA 454/R-18-009. Available at:
33 [https://www3.epa.gov/ttn/scram/guidance/guide/O3-PM-RH-Modeling_Guidance-](https://www3.epa.gov/ttn/scram/guidance/guide/O3-PM-RH-Modeling_Guidance-2018.pdf)
34 [2018.pdf](https://www3.epa.gov/ttn/scram/guidance/guide/O3-PM-RH-Modeling_Guidance-2018.pdf).
- 35 U.S. EPA (2019). Integrated Science Assessment (ISA) for Particulate Matter (Final Report).
36 U.S. Environmental Protection Agency, Office of Research and Development, Center for
37 Public Health and Environmental Assessment. Research Triangle Park, NC. U.S. EPA.
38 EPA/600/R-19/188. December 2019. Available at:

- 1 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
2 [assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 3 U.S. EPA (2021a). Integrated Science Assessment (ISA) for Particulate Matter (Draft
4 Supplement). U.S. Environmental Protection Agency, Office of Research and
5 Development, Center for Public Health and Environmental Assessment. Research
6 Triangle Park, NC. U.S. EPA. EPA/600/R-19/. September 2021. Available at:
7 [https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review)
8 [assessments-current-review](https://www.epa.gov/naaqs/particulate-matter-pm-standards-integrated-science-assessments-current-review).
- 9 U.S. EPA (2021b). Technical Support Document (TSD)for the Final Revised Cross-State Air
10 Pollution Rule Update for the 2008 Ozone Season NAAQS. U.S. Environmental
11 Protection Agency. Durham, NC. Office of Air Quality Planning and Standrads. EPA-
12 HQ-OAR-2020-0272. Available at: [https://www.epa.gov/sites/default/files/2021-](https://www.epa.gov/sites/default/files/2021-03/documents/estimating_pm2.5-_and_ozone-attributable_health_benefits_tsd.pdf)
13 [03/documents/estimating_pm2.5-_and_ozone-attributable_health_benefits_tsd.pdf](https://www.epa.gov/sites/default/files/2021-03/documents/estimating_pm2.5-_and_ozone-attributable_health_benefits_tsd.pdf).
- 14 Wang, B, Eum, K-D, Kazemiparkouhi, F, Li, C, Manjourides, J, Pavlu, V and Suh, H (2020).
15 The impact of long-term PM 2.5 exposure on specific causes of death: Exposure-response
16 curves and effect modification among 53 million US Medicare beneficiaries.
17 *Environmental Health* 19(1): 1-12.
- 18 Wang, Y, Shi, L, Lee, M, Liu, P, Di, Q, Zanobetti, A and Schwartz, JD (2017). Long-term
19 exposure to PM_{2.5} and mortality among older adults in the Southeastern US.
20 *Epidemiology* 28(2): 207-214.
- 21 WHO (2008). Uncertainty and Data Quality in Exposure Assessment. The Internaltional
22 Programme on Chemical Safety. Geneva. WHO.
23 https://www.who.int/ipcs/publications/methods/harmonization/exposure_assessment.pdf.
- 24 Willis, A, Jerrett, M, Burnett, R and Krewski, D (2003). The association between sulfate air
25 pollution and mortality at the county scale: an exploration of the impact of scale on a
26 long-term exposure study. *Journal of Toxicology and Environmental Health Part A*
27 66(16-19): 1605-1624.
- 28 Woods & Poole (2015). Complete Demographic Database.
- 29 Zanobetti, A, Dominici, F, Wang, Y and Schwartz, JD (2014). A national case-crossover analysis
30 of the short-term effect of PM_{2.5} on hospitalizations and mortality in subjects with
31 diabetes and neurological disorders. *Environmental Health: A Global Access Science*
32 *Source* 13(1): 38.

33

APPENDIX D. QUANTITATIVE ANALYSES FOR VISIBILITY IMPAIRMENT

D.1 BACKGROUND

To inform the EPA's decision in the 2012 review on the adequacy of protection provided by the secondary PM standards the EPA conducted a technical analysis of the relationships between a 3-year average daily visibility metric and the 24-hour PM_{2.5} mass-based standard (Kelly et al., 2012). The 3-year visibility metric was calculated as the 3-year average of the 90th percentile of daily visibility index values.¹ Light extinction coefficient (b_{ext}) values for the visibility index were calculated using the original IMPROVE equation (Equation D-1 in section D.2.2 below), which at the time of the 2012 review, the EPA considered to be better suited to urban sites that were the focus of the analysis than other versions of the IMPROVE equation, with a few modifications to the equation: excluding the coarse mass² and sea salt³ terms in the equation and using a multiplier of 1.6 for converting OC to OM.⁴

¹ The visibility index is a logarithmic transformation of the light extinction coefficient, b_{ext} , the use of which ensures that increases or decreases in light extinction coefficient always produce, respectively, increases or decreases in visibility index (Kelly et al., 2012).

² PM_{2.5} is the size fraction of PM responsible for most of the visibility impairment in urban areas (U.S. EPA, 2009, section 9.2.2.2). Data available at the time of the 2012 review suggested that, generally, PM_{10-2.5} was a minor contributor to visibility impairment most of the time (U.S. EPA, 2010) although the coarse fraction may be a major contributor in some areas in the desert southwestern region of the country. Moreover, at the time of the 2012 review, there were few data available from continuous PM_{10-2.5} monitors to quantify the contribution of coarse PM to calculated light extinction.

³ In estimating light extinction in the 2012 review, the EPA did not consider it appropriate to include the term for hygroscopic sea salt in evaluating urban light extinction, given that sea salt is not a major contributor to light extinction in urban areas compared with more remote coastal locations. In particular, Pitchford (2010) estimated that the contribution of sea salt to PM_{2.5} light extinction was generally well below 5% for PM_{2.5} light extinction greater than 24 dv (U.S. EPA, 2010, p. 3-22; U.S. EPA, 2012, p. IV-5).

⁴ At the time of the 2012 review, the EPA considered the multiplier of 1.8 recommended by Pitchford et al. (2007) to convert OC to OM for use in the revised IMPROVE equation (Equation D-2 below) to be too high for urban environments. The composition of, and the mix of emission sources contributing to, PM_{2.5} differ between urban and remote areas, and consequently, the light extinction may differ between urban and remote areas. Organic mass in urban areas is often from local and regional sources and would have a greater percentage of fresh emissions compared with aged emissions, which tend to be more prominent in rural areas, and a different PM mass to OC ratio than in urban areas. The EPA also considered the multiplier of 1.4 used with the original IMPROVE equation to be too low to adequately account for the contribution of OM to visibility impairment, particularly in urban areas where OM concentrations tend to be higher. Based on these considerations, along with an evaluation of the OC to OM relationship at CSN sites (2011 PA, Appendix F, section F.6), the EPA chose to use a multiplier of 1.6 to convert OC to OM in the light extinction calculations used in the 2012 review (U.S. EPA, 2012, pages IV-5-IV-8).

1 Using 2008-2010 air quality data for 102 CSN network sites,⁵ the 2012 analysis explored
2 the relationship between the 3-year design values for the existing 24-hour PM_{2.5} standard and
3 values of the 3-year visibility metric.⁶ The analysis indicated that increases in 24-hour PM_{2.5}
4 design values generally correspond to increases in the 3-year visibility metric values, and vice-
5 versa (78 FR 3201, January 15, 2013). The analysis also found linear correlations between the
6 24-hour PM_{2.5} design values and the 3-year visibility metric with an average r² value of 0.75
7 across all of the sites (Kelly et al., 2012). A key implication of this analysis was that for the level
8 proposed by the EPA for a visibility index-based standard, the 24-hour PM_{2.5} standard of 35
9 µg/m³ would be controlling in almost all or all instances (78 FR 3202, January 15, 2013).

10 **D.2 ANALYSIS: METHODS AND INPUTS**

11 Consistent with the analyses conducted in the 2012 review described above and the 2020
12 review described in the 2020 PA ({U.S. EPA, 2020 #285}, section 5.2.1.2), we have conducted
13 analyses examining the relationship between PM mass concentrations and estimated light
14 extinction in terms of a PM visibility metric. These analyses are intended to inform our
15 understanding of visibility impairment in the U.S. under recent air quality conditions,
16 particularly those conditions that meet the current standards, and our understanding of the
17 relative influence of various factors on light extinction. These analyses were conducted using
18 three versions of the IMPROVE equation (Equations D-1 through D-3 below) to estimate light
19 extinction to better understand the influence of variability in inputs across the three equations.
20 This analysis included 60 monitoring sites that are geographically distributed across the U.S. in
21 both urban and rural areas (see Figure D-1). The data set is comprised of sites with data for the
22 2017-2019 period that supported a valid 24-hour PM_{2.5} design value⁷ and met strict criteria for
23 PM species. Light extinction calculations at these 60 monitoring sites also included the coarse
24 fraction in the IMPROVE equations.⁸ Results for these analyses are presented in Figures 5-3 and
25 5-4 and discussed in section 5.2.1.2 of Chapter 5 and presented in Table D-7 and Figure D-2 in
26 section D.3 below.

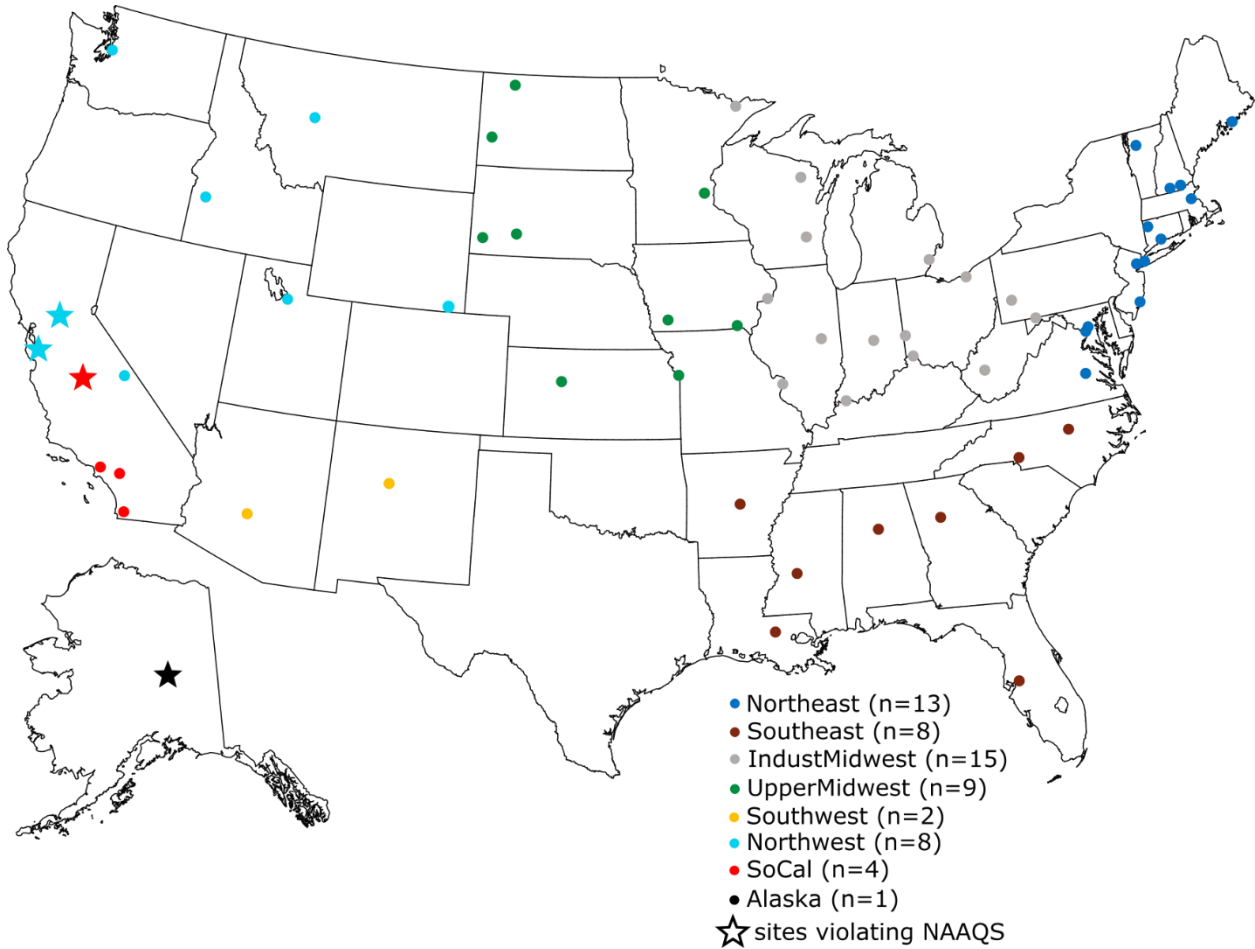
⁵ The 102 sites included in the Kelly et al. (2012) analysis were those sites that met the data completeness criteria used for that analysis (Kelly et al., 2012, p. 15).

⁶ The EPA used monthly average relative humidity values rather than shorter-term (e.g., hourly) values to estimate light extinction in the 2012 review in order to capture seasonal variability of relative humidity and its effects on visibility impairment. This was intended to focus more on the underlying aerosol contributions to visibility impairment and less on the day-to-day variations in humidity (U.S. EPA, 2012, p. IV-10).

⁷ The design value (DV) for the standard is the metric used to determine whether areas meet or exceed the NAAQS. A design value is a statistic that describes the air quality status of a given area relative to the NAAQS.

⁸ In the 2020 analyses, PM₁₀ data were available for only a subset of 20 of the 67 monitoring sites included in the analysis ({U.S. EPA, 2020 #285}, section 5.2.1.2).

1



2

3 **Figure D-1. Locations of monitoring sites with data for 2017-2019 with a valid PM_{2.5} design**
4 **value and meeting completeness criteria for PM species.**

5

6 **D.2.1 Data Sources for Inputs to Estimate Light Extinction**

7 **D.2.1.1 Relative Humidity**

8 Relative humidity data were downloaded from the North American Regional Reanalysis
9 (NARR). NARR is the National Centers for Environmental Prediction’s (NCEP) high resolution
10 combined model and assimilated meteorological dataset. NARR is an extension of the NCEP
11 Global Reanalysis which is run over North American using the Eta Model (32 km) together with
12 the Regional Data Assimilation System. Files for 3-hour average 10 m relative humidity data for
13 2017-2019 are available at <https://esrl.noaa.gov/psd/data/gridded/data.narr.html>.

14 Using NARR latitudes, relative humidity data were reassigned to each grid cell from
15 coordinated universal time (UTC) to their closest time zone and the 3-hour relative humidity data
16 were then averaged to 24-hour local time averages in order to approximate the 24-hour averaging

1 time (midnight-midnight) of the daily PM_{2.5} measurements. The PM_{2.5} and PM_{2.5} component
2 daily mass data (described in subsequent sections) were temporally and spatially matched with
3 the closest 24-hour average relative humidity grid cell.

4 **D.2.1.2 PM_{2.5} Concentrations**

5 The raw data for PM_{2.5} site-level daily mass concentrations came from an Air Quality
6 System (AQS)⁹ query of the daily site-level concentrations. Data files used were for 24-hour
7 average values from regulatory monitors for all sites in the U.S. for all available days (including
8 potential exceptional events) for 2017-2019. When a single site had multiple monitors, the
9 previously-determined primary monitor concentration was used. If the primary monitor value
10 was missing, the average of the collocated monitors was used. These data were screened so that
11 all days either had a valid filter-based 24-hour concentration measurement¹⁰ or at least 18 valid
12 hourly concentrations measurements.

13 **D.2.1.3 Coarse PM Concentrations**

14 The raw data for PM_{10-2.5} monitor-level daily mass concentrations came from an AQS
15 query of the daily monitor-level concentrations. Data files used were for 24-hour average
16 concentrations from monitors mainly in the Interagency Monitoring of Protected Visual
17 Environments (IMPROVE) network and NCore Multipollutant Monitoring Network. Data were
18 included for sites with ≥ 11 valid days for each quarter of 2017-2019.

19 **D.2.1.4 PM_{2.5} Component Concentrations**

20 The raw data for PM_{2.5} component concentrations for the components listed in Table D-1
21 came from an AQS query of the daily monitor-level concentrations. Data files used were for
22 filter-based, 24-hour average concentrations from monitors in the Interagency Monitoring of
23 Protected Visual Environments (IMPROVE) network, Chemical Speciation Network (CSN), and
24 NCore Multipollutant Monitoring Network. Data were included for days with valid data for all
25 chemical components listed in Table D-1 below and for sites with ≥ 11 valid days for each
26 quarter of 2017-2019.

27

⁹ The Air Quality System is an EPA database of ambient air quality monitoring data (<https://www.epa.gov/aqs>).

¹⁰ A valid filter-based 24-hour concentration measurement is one collected via FRM, and that has undergone laboratory equilibration (at least 24 hours at standardized conditions of 20-23°C and 30-40% relative humidity) prior to analysis (see Appendix L of 40 CFR Part 50 for the 2012 NAAQS for PM).

1 **Table D-1. PM_{2.5} components from AQS used in IMPROVE equations.**

PM _{2.5} Component Drawn from AQS	AQS Parameter Code
Sulfate	88403
Nitrate	88306
OC (TOR ^a)	88320, 88370
EC (TOR ^a)	88321, 88380
Aluminum (Al), Silica (Si), Calcium (Ca), Iron (Fe), Titanium (Ti)	88104 (Al), 88165 (Si), 88111 (Ca), 88126 (Fe), 88161 (Ti)
Chloride, Chlorine	88115 (Chlorine), 88203 (Chloride)
^a OC and EC values are based on the thermal optical reflectance (TOR) analytical method, which replaced the NIOSH 5040-like thermal optical transmittance (TOT) method in the CSN network after 2009 (Spada and Hyslop, 2018).	

2

3 **D.2.1.5 24-Hour PM_{2.5} Design Values**

4 Files for 24-hour PM_{2.5} design values for 2017-2019 are located at
 5 <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2017-2019 PM_{2.5}
 6 design values is described in Appendix N of 40 CFR Part 50 for the 2012 National Ambient Air
 7 Quality Standards (NAAQS) for Particulate Matter (PM).

8

9 **D.2.1.6 24-Hour PM₁₀ Design Values**

10 Files for 24-hour PM₁₀ design values for 2017-2019 are located at
 11 <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2017-2019 PM₁₀
 12 design values is described in Appendix K of 40 CFR Part 50.

13

14 **D.2.1.7 Annual PM_{2.5} Design Values**

15 Files for annual PM_{2.5} design values for 2017-2019 are located at
 16 <https://www.epa.gov/air-trends/air-quality-design-values>. Data handling of the 2017-2019 PM_{2.5}
 17 design values is described in Appendix N of 40 CFR Part 50 for the 2012 National Ambient Air
 18 Quality Standards (NAAQS) for Particulate Matter (PM).

19

20 **D.2.2 Calculating Light Extinction for Visibility Impairment Analyses**

21 For all days with a valid relative humidity value, PM_{2.5} mass concentration, and all
 22 chemical components listed in Table D-1, daily light extinction was calculated using three
 23 versions of the IMPROVE equation, as shown below. Formulas for derivation of the equation
 24 variables from the AQS parameters are presented in Table D-6.

25

Original IMPROVE Equation (Malm et al., 1994):

$$b_{ext} \cong 3f(RH)([AS] + [AN]) + 4[OM] + 10[EC] + 1[FS] + 0.6[CM] + 10$$

Equation D-1

where:

[AS] is concentration in $\mu\text{g}/\text{m}^3$ of ammonium sulfate,

[AN] is concentration in $\mu\text{g}/\text{m}^3$ of ammonium nitrate,

[OM] is concentration in $\mu\text{g}/\text{m}^3$ of organic matter,

[EC] is concentration in $\mu\text{g}/\text{m}^3$ of elemental carbon,

[FS] is concentration in $\mu\text{g}/\text{m}^3$ of fine soil,

[CM] is concentrations in $\mu\text{g}/\text{m}^3$ of coarse mass, and

f(RH) is the relative-humidity-dependent water growth function, assigned values as shown in Table D-2:

Table D-2. Relatively-humidity-dependent water growth function for use in the original IMPROVE equation.

RH (%)	1-36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56
f(RH)	1	1.02	1.04	1.06	1.08	1.1	1.13	1.15	1.18	1.2	1.23	1.26	1.28	1.31	1.34	1.37	1.41	1.44	1.47	1.51	1.54
RH (%)	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77
f(RH)	1.58	1.62	1.66	1.7	1.74	1.79	1.83	1.88	1.93	1.98	2.03	2.08	2.14	2.19	2.25	2.31	2.37	2.43	2.5	2.56	2.63
RH (%)	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98 ^a
f(RH)	2.7	2.78	2.86	2.94	3.03	3.12	3.22	3.33	3.45	3.58	3.74	3.93	4.16	4.45	4.84	5.37	6.16	7.4	9.59	14.1	26.4

Note: See fRHOriginalIMPROVE.csv file from <http://vista.cira.colostate.edu/Improve/the-improve-algorithm/> (Malm et al., 1994).

^a For our application, any relative humidity values greater than 98% were assigned the f(RH) value associated with 98%, the highest value available for the relative humidity function.

1 The various coefficients are the empirically derived extinction efficiency (mass scattering and
 2 absorption) coefficients, as originally specified by Malm et al. (1994).

3

4 **Revised IMPROVE Equation (Pitchford et al., 2007):**

$$\begin{aligned}
 5 \quad b_{ext} \cong & 2.2f_S(RH)[small\ sulfate] + 4.8f_L(RH)[large\ sulfate] + 2.4f_S(RH)[small\ nitrate] \\
 6 \quad & + 5.1f_L(RH)[large\ nitrate] + 2.8[small\ OM] + 6.1[large\ OM] + 10[EC] \\
 7 \quad & + 1[FS] + 1.7f_{SS}(RH)[SS] + 0.6[CM] + 10
 \end{aligned}$$

8

Equation D-2

9 where:

10 [small sulfate], [large sulfate], [small nitrate], [large nitrate], [small OM] and [large OM]
 11 are defined as follows in Table D-3:

12 **Table D-3. Values for use in the revised IMPROVE equation for small and large sulfate,**
 13 **nitrate, and organic matter concentrations.**

	If [] ≥ 20	If [] <20
Large sulfate	[AS]	[AS]÷20
Small sulfate	0	[AS] - ([AS]÷20)
Large nitrate	[AN]	[AN]÷20
Small nitrate	0	[AN] - ([AN]÷20)
Large OM	[OM]	[OM]÷20
Small OM	0	[OM] - ([OM]÷20)
Note: [AS], [AN] and [OM] are defined as for Equation D-1.		

14

15 [SS] is sea salt; and,

16 $f_{SS}(RH)$, $f_S(RH)$, and $f_L(RH)$ are defined as shown in Table D-4:

17

1 **Table D-4. Relatively-humidity-dependent water growth function for sea salt, small**
 2 **particles, and large particles for use in the revised IMPROVE equation.**

RH (%)	1-36	37	38	39	40	41	42	43	44	45	46	47	48	49	50
f_{ss}(RH)	1	1	1	1	1	1	1	1	1	1	1	2.3584	2.3799	2.4204	2.4488
f_s(RH)	1	1.38	1.4	1.42	1.44	1.46	1.48	1.49	1.51	1.53	1.55	1.57	1.59	1.62	1.64
f_L(RH)	1	1.31	1.32	1.34	1.35	1.36	1.38	1.39	1.41	1.42	1.44	1.45	1.47	1.49	1.5
RH (%)	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65
f_{ss}(RH)	2.4848	2.5006	2.5052	2.5279	2.5614	2.5848	2.5888	2.616	2.6581	2.6866	2.7341	2.7834	2.8272	2.8287	2.8594
f_s(RH)	1.66	1.68	1.71	1.73	1.76	1.78	1.81	1.83	1.86	1.89	1.92	1.95	1.99	2.02	2.06
f_L(RH)	1.52	1.54	1.55	1.57	1.59	1.61	1.63	1.65	1.67	1.69	1.71	1.73	1.75	1.78	1.8
RH (%)	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80
f_{ss}(RH)	2.8943	2.9105	2.9451	3.0105	3.0485	3.1269	3.1729	3.2055	3.2459	3.2673	3.3478	3.4174	3.5202	3.5744	3.6329
f_s(RH)	2.09	2.13	2.17	2.22	2.26	2.31	2.36	2.41	2.47	2.54	2.6	2.67	2.75	2.84	2.93
f_L(RH)	1.83	1.86	1.89	1.92	1.95	1.98	2.01	2.05	2.09	2.13	2.18	2.22	2.27	2.33	2.39
RH (%)	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95 ^a
f_{ss}(RH)	3.6905	3.808	3.9505	4.0398	4.1127	4.2824	4.494	4.6078	4.8573	5.1165	5.3844	5.7457	6.1704	6.7178	7.3492
f_s(RH)	3.03	3.15	3.27	3.42	3.58	3.76	3.98	4.23	4.53	4.9	5.35	5.93	6.71	7.78	9.34
f_L(RH)	2.45	2.52	2.6	2.69	2.79	2.9	3.02	3.16	3.33	3.53	3.77	4.06	4.43	4.92	5.57
Note: See fRHRevisedIMPROVE.csv file from http://vista.cira.colostate.edu/Improve/the-improve-algorithm/ (Pitchford et al., 2007).															
^a For our application, any relative humidity values greater than 95% were assigned the f(RH) value associated with 95%, the highest value available for the relative humidity function.															

3
4 and

5 [EC], [FS] and [CM] are defined as for Equation D-1.

6 This equation is generally dividing PM components into small and large particle sizes¹¹ with
 7 separate mass scattering efficiencies and hygroscopic growth functions for each size (included in
 8 the equation as f_s(RH) for small particles, f_L(RH) for large particles, and f_{ss}(RH) for sea salt).
 9

¹¹ The large mode for sulfate, nitrate, and OM represents aged and/or cloud processed particles, whereas the small mode represents freshly formed particles. These size modes are described by log-normal mass size distributions with geometric mean diameters and geometric standard deviations of 0.2 μm and 2.2 for small mode and 0.5 μm and 1.5 for the large mode, respectively.

Lowenthal and Kumar (2016) Equation:

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

Equation D-3

where:

$f_s(RH)_{OM}$ and $f_L(RH)_{OM}$ are the relative-humidity-dependent water growth function for small and large organic matter, respectively, as defined in Table D-5 below.

Table D-5. Relatively-humidity-dependent water growth function for small organic matter and large organic matter for use in the original IMPROVE equation.

RH (%)	0-29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45
$f_s(RH)_{OM}$	1.000	1.321	1.325	1.329	1.333	1.337	1.340	1.343	1.346	1.349	1.352	1.354	1.356	1.358	1.360	1.362	1.364
$f_L(RH)_{OM}$	1.000	1.267	1.271	1.274	1.278	1.280	1.283	1.286	1.288	1.290	1.292	1.294	1.296	1.297	1.299	1.300	1.302
RH (%)	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62
$f_s(RH)_{OM}$	1.366	1.368	1.369	1.371	1.373	1.75	1.377	1.379	1.382	1.384	1.387	1.390	1.393	1.397	1.400	1.404	1.409
$f_s(RH)_{OM}$	1.303	1.305	1.306	1.308	1.309	1.311	1.306	1.308	1.309	1.311	1.313	1.314	1.316	1.318	1.320	1.323	1.325
RH (%)	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79
$f_s(RH)_{OM}$	1.413	1.419	1.424	1.430	1.437	1.444	1.452	1.460	1.469	1.478	1.489	1.500	1.511	1.524	1.537	1.51	1.566
$f_s(RH)_{OM}$	1.328	1.331	1.334	1.338	1.342	1.346	1.350	1.355	1.385	1.393	1.401	1.409	1.418	1.428	1.438	1.449	1.461
RH (%)	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95 ^a	
$f_s(RH)_{OM}$	1.582	1.599	1.617	1.637	1.657	1.679	1.703	1.727	1.754	1.782	1.812	1.843	1.877	1.912	1.950	1.989	
$f_s(RH)_{OM}$	1.473	1.486	1.500	1.515	1.531	1.548	1.566	1.585	1.605	1.626	1.648	1.672	1.696	1.722	1.750	1.779	

Note: See Table 1 in Lowenthal and Kumar (2016).

^a For our application, any relative humidity values greater than 95% were assigned the $f(RH)$ value associated with 95%, the highest value available for the relative humidity function.

and

[small sulfate], [large sulfate], [small nitrate], [large nitrate], [small OM], [large OM], [EC], [FS], [SS], [CM], $f_s(RH)$, $f_L(RH)$ and $f_{SS}(RH)$ are defined as above for Equation D-2.

This equation updates the multiplier for estimating the concentration organic matter, [OM], from the concentration of organic carbon to 2.1 and incorporates $f_s(RH)_{OM}$ and $f_L(RH)_{OM}$ representing water absorption by soluble organic matter as a function of relative humidity for small and large organic matter, respectively.

Based on each equation, site-specific visibility metrics were derived for each site as follows. Daily light extinction values were derived for 2017, 2018, and 2019, the 90th percentile of daily values for each year was calculated, and the three years of values were averaged. The 3-year averages of the 90th percentiles of daily light extinction values were paired with the 2017-2019 PM_{2.5} 24-hour design values for each site having valid data for both statistics.

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

Equation Variable	How Calculated from AQS Parameter Values
Ammonium Sulfate	All three equations: $1.375 \times [\text{Sulfate}]^A$
Ammonium Nitrate	All three equations: $1.29 \times [\text{Nitrate}]^B$
Organic Matter	Original IMPROVE equation: $1.6 \times [\text{OC}]^C$ Revised IMPROVE equation: $1.6 \times [\text{OC}]^C$ Lowenthal and Kumar (2016) equation: $2.1 \times [\text{OC}]$
Elemental Carbon	$[\text{EC}]$
Fine Soil	All three equations: ^D $2.2 \times [\text{Al}] + 2.49 \times [\text{Si}] + 1.63 \times [\text{Ca}] + 2.42 \times [\text{Fe}] + 1.94 \times [\text{Ti}]$
Sea Salt	Revised IMPROVE and Lowenthal and Kumar, 2016 equations: ^D $1.8 \times [\text{Chloride}]$ $1.8 \times [\text{Chlorine}]$ (if chloride is missing)
^A This formula is based on molar molecular weights of ammonium sulfate and sulfate (Malm et al., 1994). ^B This formula is based on molar molecular weights of ammonium nitrate and nitrate (Malm et al., 1994). ^C See footnote 4 earlier in this appendix. ^D This formula is documented in Malm et al. (1994).	

D.3 SUMMARY OF RESULTS

Results for the visibility impairment analyses are discussed in section 5.2.1.2 of Chapter 5. Table D-7 presents the 24-hour PM_{2.5} design values, 24-hour PM₁₀ design values, annual PM_{2.5} design values, and 3-year visibility metrics based on light extinction calculations using the three versions of the IMPROVE equation with the coarse mass fraction included in the analyses. Figure 5-3 and 5-4 in Chapter 5 show a comparison of the 3-year visibility metric and the 24-hour PM_{2.5} design values for the 60 monitoring sites in the analyses where light extinction was calculated using the original IMPROVE equation¹² and the Lowenthal and Kumar IMPROVE equation. Figure D-2 below presents the 3-year visibility metric and the 24-hour PM_{2.5} design values for the 60 monitoring sites with light extinction calculated using the revised IMPROVE equation.¹³

¹² For this analysis, the original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM from the light extinction calculation, consistent with the modifications in the 2012 and 2020t review.

¹³ For this analysis, the revised IMPROVE equation in Equation D-2 was modified to use a 1.6 multiplier to convert OC to OM, consistent with the modifications in the 2012 and 2020 reviews.

1 **Table D-7. Summary of 24-hour PM_{2.5}, 24-hour PM₁₀, and annual PM_{2.5} design values, and 3-year visibility metrics at 60**
 2 **monitoring sites (2017-2019).**

Monitor ID	State	Region	24-hour PM _{2.5} Design Value (µg/m ³) ^A	24-hour PM ₁₀ Design Value (number of exceedances) ^{B,C}	Annual PM _{2.5} Design Value (µg/m ³) ^D	3-year Visibility Metric (deciviews) ^E		
						Original IMPROVE Equation ^F	Revised IMPROVE Equation ^G	Lowenthal & Kumar IMPROVE Equation
010730023	Alabama	Southeast	21	0	10.0	23	23	26
020900034	Alaska	Alaska	40	1.4	8.9	24	25	27
040139997	Arizona	Southwest	21	0.7	7.4	21	21	24
051190007	Arkansas	Southeast	19	0	9.3	21	21	24
060270002	California	Northwest	23	3	5.6	13	14	15
060190011	California	SoCal	56	1	14.1	29	27	32
060371103	California	SoCal	31		11.9	26	25	28
060658001	California	SoCal	31	0	12.1	26	25	28
060670006	California	Northwest	37	4.1	10.2	25	25	30
060731022	California	SoCal	19	0	9.3	21	21	24
060850005	California	Northwest	43	0	10.5	22	22	26
090050005	Connecticut	Northeast	12		4.1	15	16	18
090090027	Connecticut	Northeast	18	0	6.9	23	23	26
110010043	District Of Columbia	Northeast	20	0	8.9	23	23	25
120573002	Florida	Southeast	18		7.9	18	19	21
130890002	Georgia	Southeast	19	0	8.4	20	20	24
160010010	Idaho	Northwest	29		7.4	23	22	26
170191001	Illinois	IndustrialMidwest	18		7.8	22	22	23
180970078	Indiana	IndustrialMidwest	20	0	9.0	24	24	26
181630021	Indiana	IndustrialMidwest	17	0	8.2	22	22	24
191370002	Iowa	UpperMidwest	16		6.6	21	22	22

191630015	Iowa	IndustrialMidwest	20	0	8.0	23	23	25
191770006	Iowa	UpperMidwest	16	0	7.0	21	22	23
201950001	Kansas	UpperMidwest	14		5.0	16	17	18
202090021	Kansas	UpperMidwest	26		9.4	23	23	26
220330009	Louisiana	Southeast	21	0	8.8	22	23	25
230090103	Maine	Northeast	11	0	3.2	16	18	18
240230002	Maryland	IndustrialMidwest	13		5.7	16	17	18
240330030	Maryland	Northeast	15	0	6.7	19	20	23
250250042	Massachusetts	Northeast	18		7.4	19	20	21
261630001	Michigan	IndustrialMidwest	22		8.8	23	23	25
270031002	Minnesota	UpperMidwest	20	0	7.3	23	23	25
270750005	Minnesota	IndustrialMidwest	13		3.8	16	16	17
280490020	Mississippi	Southeast	17		9.1	20	20	24
295100085	Missouri	IndustrialMidwest	21		8.7	24	24	26
300490004	Montana	Northwest	23		3.9	19	18	22
330115001	New Hampshire	Northeast	10		3.0	13	14	15
330150018	New Hampshire	Northeast	12		4.9	17	17	19
340010006	New Jersey	Northeast	15		6.6	18	19	20
340130003	New Jersey	Northeast	20	0	8.4	23	23	25
350010023	New Mexico	Southwest	15	0	5.6	16	17	19
360810124	New York	Northeast	18	0	7.0	21	22	24
371190041	North Carolina	Southeast	16		8.1	19	20	23
371830014	North Carolina	Southeast	13	0	7.7	19	19	23
380070002	North Dakota	UpperMidwest	15		3.9	18	18	20
380130004	North Dakota	UpperMidwest	16	0	3.6	20	20	21
390350060	Ohio	IndustrialMidwest	24	0	9.9	25	25	27
390610040	Ohio	IndustrialMidwest	20	0	9.4	22	23	24
391351001	Ohio	IndustrialMidwest	18		8.1	22	22	23
420030008	Pennsylvania	IndustrialMidwest	20		9.1	23	23	25
460330132	South Dakota	UpperMidwest	14	0	3.8	13	14	15
460710001	South Dakota	UpperMidwest	14	0	4.1	14	15	17

490353006	Utah	Northwest	30		7.5	26	26	28
500070007	Vermont	Northeast	12	0	4.3	15	16	17
510870014	Virginia	Northeast	15	0	7.1	19	20	23
530330080	Washington	Northwest	26		6.3	21	22	24
540390020	West Virginia	IndustrialMidwest	15		7.9	21	21	24
550270001	Wisconsin	IndustrialMidwest	21	0	7.0	24	24	26
550410007	Wisconsin	IndustrialMidwest	15		4.7	19	19	21
560210100	Wyoming	Northwest	11	0	3.2	13	14	15

^A The 24-hour PM_{2.5} design value is the 3-year average of the 98th percentile of daily PM_{2.5} mass concentrations. The current 24-hour PM_{2.5} NAAQS is set at a level of 35 µg/m³.

^B The 24-hour PM₁₀ design value is not to be exceeded more than once per year on average over three years. The current 24-hour PM₁₀ NAAQS is set at a level of 150 µg/m³.

^C For some monitoring locations, PM₁₀ design values are not available because of a lack of collocated PM₁₀ monitoring at the site or insufficient data after applying completeness criteria for calculating PM₁₀ design values.

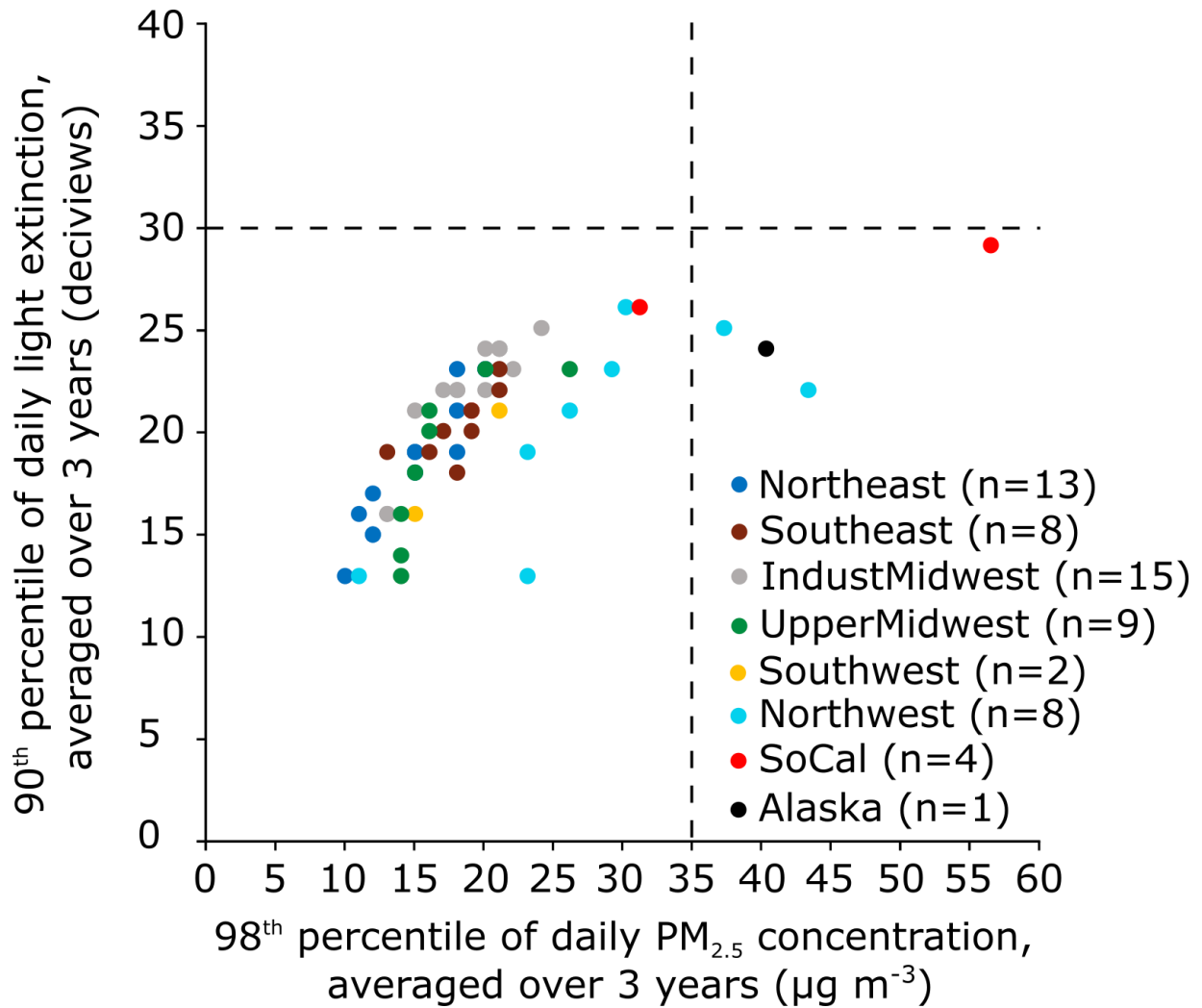
^D The annual PM_{2.5} design value is the annual mean, averaged over three years. The current secondary annual PM_{2.5} NAAQS is set at a level of 15.0 µg/m³.

^E The 3-year visibility metric is the 3-year average of the 90th percentile of daily light extinction. In the 2012 and 2020 reviews, the target level of protection identified for the 3-year visibility metric was 30 deciviews.

^F The original IMPROVE equation in Equation D-1 was modified to use a 1.6 multiplier to convert OC to OM from the light extinction calculation, consistent with the modifications in the 2012 and 2020 reviews.

^G The revised IMPROVE equation in Equation D-2 was modified to use a 1.6 multiplier to convert OC to OM, consistent with the modifications in the 2012 and 2020 reviews.

1
2



1
2
3
4
5
6
7

Figure D-2. Comparison of 90th percentile of daily light extinction, averaged over three years, and 98th percentile of daily PM_{2.5} concentrations, averaged over three years, for 2017-2019 using the revised IMPROVE equation. (Note: Dashed lines indicate the level of current 24-hour PM_{2.5} standard (35 µg/m³) and the target level of protection identified for the 3-year visibility metric (30 dv).)

1 REFERENCES

- 2 Abt Associates, Inc. (2001). Assessing public opinions on visibility impairment due to air
3 pollution: Summary report. U.S. Environmental Protection Agency. Research Triangle
4 Park, NC.
- 5 Kelly, J, Schmidt, M, Frank, N, Timin, B, Solomon, D and Venkatesh, R. (2012). Memorandum
6 to PM NAAQS Review Docket (EPA-HQ-OAR-2007-0492). Technical Analyses to
7 Support Surrogacy Policy for Proposed Secondary PM_{2.5} NAAQS under NSR/PSD
8 Programs. June 14, 2012. Docket ID No. EPA-HQ-OAR-2007-0492. Office of Air
9 Quality Planning and Standards Research Triangle Park, NC. Available at:
10 <https://www3.epa.gov/ttn/naaqs/standards/pm/data/20120614Kelly.pdf>.
- 11 Lowenthal, DH and Kumar, N (2016). Evaluation of the IMPROVE Equation for estimating
12 aerosol light extinction. Journal of the Air and Waste Management Association 66(7):
13 726-737.
- 14 Malm, WC, Sisler, JF, Huffman, D, Eldred, RA and Cahill, TA (1994). Spatial and seasonal
15 trends in particle concentration and optical extinction in the United States. Journal of
16 Geophysical Research 99(D1): 1347-1370.
- 17 Pitchford, M. (2010). Memorandum to PM NAAQS Review Docket (EPA-HQ-OAR-2007-
18 0492). Assessment of the Use of Speciated PM_{2.5} Mass-Calculated Light Extinction as a
19 Secondary PM NAAQS Indicator of Visibility. . November 17, 2010. Docket ID No.
20 EPA-HQ-OAR-2007-0492. Office of Air Quality Planning and Standards Research
21 Triangle Park, NC. Available at:
22 <https://www3.epa.gov/ttn/naaqs/standards/pm/data/Pitchford11172010.pdf>.
- 23 Pitchford, M, Maim, W, Schichtel, B, Kumar, N, Lowenthal, D and Hand, J (2007). Revised
24 algorithm for estimating light extinction from IMPROVE particle speciation data. Journal
25 of the Air and Waste Management Association 57(11): 1326-1336.
- 26 Spada, NJ and Hyslop, NP (2018). Comparison of elemental and organic carbon measurements
27 between IMPROVE and CSN before and after method transitions. Atmos Environ 178:
28 173-180.
- 29 U.S. EPA (2009). Integrated Science Assessment for Particulate Matter (Final Report). Office of
30 Research and Development, National Center for Environmental Assessment. Research
31 Triangle Park, NC. U.S. EPA. EPA-600/R-08-139F. December 2009. Available at:
32 <https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=216546>.
- 33 U.S. EPA (2010). Particulate Matter Urban-Focused Visibility Assessment (Final Document).
34 Office of Air Quality Planning and Standards, Health and Environmental Impacts
35 Division. Research Triangle Park, NC. U.S. EPA. EPA-452/R-10-004. July 2010.
36 Available at: <https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100FO5D.txt>.
- 37 U.S. EPA (2012). Responses to Significant Comments on the 2012 Proposed Rule on the
38 National Ambient Air Quality Standards for Particulate Matter (June 29, 2012; 77 FR

1 38890). Research Triangle Park, NC. U.S. EPA. Docket ID No. EPA-HQ-OAR-2007-
2 0492. Available at: <https://www3.epa.gov/ttn/naaqs/standards/pm/data/20121214rtc.pdf>.

1 **ATTACHMENT: SUMMARY OF VISIBILITY PREFERENCE**
2 **STUDIES**

3
4 The preference studies available at the time of the 2012 and 2020 reviews were
5 conducted in four urban areas. Three western preference studies were available, including one in
6 Denver, Colorado (Ely et al., 1991), one in the lower Fraser River valley near Vancouver, British
7 Columbia, Canada (Pryor, 1996), and one in Phoenix, Arizona (BBC Research & Consulting,
8 2003). A pilot focus group study was also conducted for Washington, DC (Abt Associates,
9 2001), and a replicate study with 26 participants was also conducted for Washington, DC (Smith
10 and Howell, 2009).¹⁴ Study specific details for these preference studies are shown in Table D-8.

¹⁴ The replicate study with 26 participants was one test group of three included in Smith and Howell (2009). This study also included two additional test groups to assess varying light extinction conditions using the same scene as was used in the first test group. Study details in Table D-8 reflect all three test groups included in the study. However, for reasons described in section 2.5.2 of U.S. EPA (2010), results from the other two test groups were not included in the EPA’s evaluation of levels of acceptable visibility impairment from the preference studies.

1 **Table D-8. Summary of visibility preference studies.** (Adapted from Table 9-2 in U.S. EPA,
 2 2009).

	Denver, CO	Phoenix, AZ	Vancouver, British Columbia	Washington, DC	Washington, DC
Report Date	1991	2003	1996	2001	2009
Duration of session		45 minutes	50 minutes	2 hours	
Compensation	None	\$50	None	\$50	None
# focus group sessions	16 ^a	27 ^b	4	1	3 tests
# participants	214	385	180	9	64
Age range	Adults	18-65+	University students	27-58	Adults
Annual or seasonal	Wintertime	Annual	Summertime	Annual	Annual
# and type of scene presented	Single scene of downtown Denver with the mountains in the south in the background	Single scene of downtown Phoenix with the Estrella Mountains in the background, 42 km max. distance	Single scene from each of two suburbs in the lower Fraser River valley – Chilliwack and Abbotsford ^c	Single scene of Potomac River, Washington Mall and downtown Washington, DC, 8 km max. sight	Single scene of DC Mall and downtown, 8 km maximum sight
# total visibility conditions presented	20 conditions (+ 5 duplicates)	21 conditions (+ 4 duplicates)	20 conditions (10 from each city)	20 conditions (+ 5 duplicates)	22 conditions
Source of slides	Actual photos taken between 9am and 3pm	WinHaze	Actual photos taken at 1pm or 4pm	WinHaze	WinHaze
Medium of presentation	Slide projection	Slide projection	Slide projection	Slide projection	Slide projection
Ranking scale used	7 point scale	7 point scale	7 point scale	7 point scale	7 point scale
Visibility range presented (dv)	11-40	15-35	Chilliwack: 13-25 Abbotsford: 13.5-31.5	9-38	9-45
Health issue directions	Ignore potential health impacts; visibility only	Judge solely on visibility, do not consider health	Judge solely on visibility, do not consider health	Health never mentioned, "Focus only on visibility"	Health never mentioned, "Focus only on visibility"
Key questions asked	<ul style="list-style-type: none"> •Rank VAQ (1-7 scale) •Is each slide "acceptable" •"How much haze is too much?" 	<ul style="list-style-type: none"> •Rank VAQ (1-7 scale) •Is each slide "acceptable" •How many days a year would this picture be "acceptable" 	<ul style="list-style-type: none"> •Rank VAQ (1-7 scale) •Is each slide "acceptable" 	<ul style="list-style-type: none"> •Rank VAQ (1-7 scale) •Is each slide "acceptable" •If this hazy, how many hours would it be acceptable (3 slides only) •Valuation question 	<ul style="list-style-type: none"> •Rank VAQ (1-7 scale) •Is each slide "acceptable"
Mean dv found "acceptable"	20.3	23-25	Chilliwack: ~23 Abbotsford: ~19	~20 (range 20-25)	~30

^a No preference data were collected at a 17th focus group session due to a slide projector malfunction.
^b The 27 focus groups were conducted in 6 neighborhood locations in Phoenix, with 3 focus groups held in Spanish.
^c Chilliwack scene includes downtown buildings in the foreground with mountains in the background up to 65 km away. Abbotsford scene has fewer manmade objects in the foreground and is primarily a more rural scene with mountains in the background up to 55 km away.

1 **REFERENCES**

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

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

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

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

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

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

United States
Environmental Protection
Agency

Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

Publication No. EPA-452/P-21-001
October 2021
